"CLINICAL EFFICACY OF TWO ANTIMICROBIALS (CEFTRIAXONE AND METRONIDAZOLE) VERSUS THREE ANTIMICROBIALS (CEFTRIAXONE , METRONIDAZOLE AND AMIKACIN)IN PERFORATIVE PERITONITIS"

By

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DISSERTATION SUBMITTED TO SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH, KOLAR, KARNATAKA IN PARTIAL FULFILLMENT OF THE REQUIREMENTS FOR THE DEGREE OF

MASTER OF SURGERY

IN

GENERAL SURGERY

Under the guidance of

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2018

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LIST OF ABBREVATIONS

1. B.C- before Christ
2. e.g.- example
3. A.D- anno Domini
4. D&C- Dilatation and Curretage
5. E. coli- Escherichia coli
6. No.- Number
7. Sl. No-Serial number
8. CNS- Central Nervous System
9. AST- Aspartate Transaminase
10. IL- Interleukin
11. MOFS- Multi-Organ Failure Syndrome
12. AIDS- Acquired Immuno Deficiency Syndrome
13. mm- milli metre
14. CAPD- Continuous Ambulatory Peritoneal Dialysis
15. PBP- Primary Bacterial Peritonitis
16. PMN- Polymorpho Nuclear Leukocytes
17. NSAIDS- Non Steroidal Anti Inflammatory Drugs
18. HSV- Highly Selective Vagotomy
19. TV+ D- Truncal Vagotomy + Drainage
20. ERCP- Endoscopic Retrograde Cholangio-Pancreaticography
21. CT- Computed Tomography
22. ARDS- Acute Respiratory Distress Syndrome
23. PE- Pulmonary Embolism

LIST OF ABBREVATIONS

24. DVT- Deep Vein thrombosis
25. Hb- Hemoglobin
26. WBC- White Blood Cell
27. ABG- Arterial Blood Gas
28. CRP- C- Reactive Protein
29. US- Ultrasound
30. PPFA-Peri-Portal Free Air
31. GIT- Gatro Intestinal Tract
32. DU- Duodenal
33. PP- Prepyloric
34. APP- Appendicular
35. IL- Interleukin
36. JE- Jejunum
37. SSI- Surgical site infection
38. EB- Enterobacter
39. ECOC- Enterococcus
40. ACI- Acinetobacter
41. KLE- Klebsiella
42. NG- No Growth

ABSTRACT

BACKGROUND :

Peritonitis is classified as primary, secondary and tertiary. In primary peritonitis (spontaneous bacterial peritonitis) and continuous ambulatory peritoneal dialysis-associated peritonitis, the source of the infection is not due a breach in the gastrointestinal tract and usually caused by a single organism. Secondary peritonitis ensues, which may be localized and contained or diffuse carrying a high mortality in the absence of surgical intervention and appropriate antimicrobial therapy. Another sequelae of perforated viscus is intra-abdominal abscesses, located in the intra or retroperitoneal space, which occur in partially treated diffuse peritonitis, postoperatively or in localized disease where the omentum has sealed off the perforation and formed an inflammatory barrier. In contrast, secondary peritonitis following perforation of the gastrointestinal tract or an infection originating in an intra-abdominal structure, e.g. gall bladder, pancreas etc. Tertiary peritonitis is an ill-defined entity, which occurs despite adequate treatment of primary or secondary peritonitis.

Combination antibiotic therapy has been used to provide the patient with broad-spectrum coverage against the many potential pathogens encountered in abdominal sepsis. Several potential benefits of the clinical use of antibiotic combinations have been advanced. So this study will be conducted to focus on the efficacy of combination of two versus three antimicrobial drug in the management of patients with perforated peritonitis.

OBJECTIVES OF THE STUDY:

1.To assess the efficacy of two antimicrobials(Ceftriaxone And Metronidazole) in perforative peritonitis.

2.To assess the efficacy of three antimicrobials(Ceftriaxone, Metronidazole

And Amikacin)in perforative peritonitis.

3.To compare the clinical outcome of perforative peritonitis with two and three antimicrobials in the terms of reduction in postoperative infections and hospital stay.

MATERIALS AND METHODS

SOURCE OF DATA:

This is a prospective clinical study conducted on 140 consecutive patients who presented to the surgical department of R. L. Jalappa Hospital and Research Centre, Tamaka, Kolar with peritonitis secondary to hollow viscus perforation. Study period was from December 2015 to June 2017. This is a randomized study and all the patients were divided in two groups.

GROUP A: Patients with all odd serial numbers were included in this group and treated with two antimicrobials (Inj Ceftriaxone 1gm IV BD and Inj Metronidazole 500mg IV TID).

GROUP B: Patients with all even serial numbers were included in this group and treated with three antimicrobials(Inj Ceftriaxone 1gm IV BD , Inj Metronidazole 500mg IV TID and Inj Amikacin 500mg IV BD).

INCLUSION CRITERIA:

1.Patients with peritonitis secondary to hollow viscus perforation.

2.Patients with age >18years and <70years.

EXCLUSION CRITERIA:

1.Peritonitis secondary to trauma to the abdomen.

2.Peritonitis secondary to gynaecological interventions like D&C.

3.Peritonitis secondary to malignancies and immuno-compromised state

4.Patients allergic to Ceftriaxone, Metronidazole and Amikacin.

5.Tertiary peritonitis.

RESULTS:

The clinical outcome in the form reduction in postoperative complications and hospital stay were assessed in 140 patients, in Group A(with the usage of two antimicrobials , Ceftriaxone and Metronidazole) and Group B(with usage of three antimicrobials, Ceftriaxone, Metronidazole and Amikacin). There was decrease in postoperative complications and hospital stay in Group B.

The p-value was significant in Group B patients <0.05(0.007).

There were 6 deaths, all of them had severe form of peritonitis with massive contamination and delayed presentation to the hospital. This study also revealed that men are commonly affected and duodenal ulcer perforation is the commonest site of perforation. Escherichia coli is the most common organism isolated.

CONCLUSION:

In our study peritonitis is more common in men compared to women. The most common age group is in between 21 – 40 years in cases of peritonitis with the mean age of 37 years. Duodenal ulcer perforation is the commonest site of perforation. Escherichia coli is the most common organism isolated in the peritoneal fluid. Use of with usage of three antimicrobials, Ceftriaxone, Metronidazole and Amikacin($p<0.05$) is beneficial in reduction in postoperative complications and hospital stay when compared to usage of two antimicrobials, Ceftriaxone and Metronidazole which is statistically significant.

KEYWORDS: Perforative peritonitis, Ceftriaxone, Metronidazole, Amikacin, Postoperative complications and Hospital stay.

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INTRODUCTION

INTRODUCTION

Peritonitis presents most commonly due to the localized or generalized infection caused from various factors. Secondary peritonitis is the most common form that follows an intraperitoneal source usually from perforation of hollow viscera. Acute generalized peritonitis due to underlying hollow viscus perforation is a critical and life-threatening condition. It is a common surgical emergency in most of the general surgical units across the world[1].

The rate of secondary infection is higher as majority of patients being from rural areas, present late to the hospital due to low awareness, local beliefs and faith in native medicine. It is often associated with significant morbidity and mortality[2]. The study aims to compare the efficacy of two antimicrobials (Ceftriaxone And Metronidazole) and three antimicrobials (Ceftriaxone, Metronidazole And Amikacin) in perforative peritonitis and to ensure adequate control of infection and to decrease the chances of post operative wound infection thereby preventing prolonged hospital stay.

The multifaceted nature of abdominal surgical infections makes it difficult to precisely define the disease and to assess its severity and therapeutic progress. Both the anatomic source of infection and to a greater degree, the physiologic compromise it inflicts affects the outcome[1].

Perforation of an intra-abdominal hollow organ with spillage of contents into the peritoneal cavity always leads to severe pain, shock, sepsis and a high risk of death. However in posterior perforation, the features are less dramatic[3].

According the various studies on the pathology of peritonitis, the pathogenesis of perforative peritonitis is mainly based on the local and systemic release of pro and anti-inflammatory mediators triggered by the presence of bacteria and their products in the abdominal cavity.

Therefore, treatment consists of preoperative initiation of broad spectrum antibiotic therapy, focal restoration, intraoperative debridement and lavage[4].

Closure of perforation with a thorough peritoneal wash under systemic antibiotic coverage has been the important step in managing peritonitis, which is practiced now-a-days. In cases of small intestinal perforation, resection anastomosis can be performed[5].

High-risk patients require timely and aggressive treatment especially in severe peritonitis. Early prognostic evaluation is desirable so as to be able to select high-risk patients for more aggressive treatment especially in severe peritonitis[1].

The prognosis and outcome of peritonitis depend upon the interaction of several factors, which includespatient-related factors, disease-specific factors, diagnostic and therapeutic interventions[6].

According to Surgical Infections Society and the Infection Diseases Society of American guidelines, "an Antimicrobial therapy for an established infections should be continued until resolution of clinical signs of infection occurs, including normalization of temperature and white blood cell count and return to gastrointestinal function". In most trials involving antibiotic therapy, an arbitrarily fixed period ranging from 5 to 14 days is used for all patients with intraabdominal infections, irrespective of the severity of peritonitis[7].

AIMS AND OBJECTIVES

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REVIEW OF LITERATURE

REVIEW OF LITERATURE

Nearly all surgical interventions into the abdominal cavity had ended up with peritonitis and death till the 19th century. The operations are all defective in one important aspect in management of sepsis and fatal peritonitis is the inevitable consequence. Discoveries by Pasteur and introduction of antiseptic surgery by Lister led to dramatic decline in the mortality following surgical exploration of the peritoneal cavity[8].

An experimental study in rats conducted revealed that survival in established peritonitis depends on adequate antibiotic therapy as well as topical antibiotic peritoneal lavage[1].

Another study recommended that antibiotic therapy and peritoneal lavage to be an effective , safe and simple alternative to the surgical treatment of generalized peritonitis wherein peritoneal lavage was combined intraoperatively with debridement of any peritoneal or any viscus exudates and there was postoperative intraperitoneal sepsis in 4 patients out of 722 patients who eventually underwent reoperation[9].

Recent advances in the field of research have shown that the peritoneal mesothelial cells have a remarkable capacity to respond to peritoneal insult. They generate an intense biological response and play an important role in formation of adhesions. Yao V and associates studied mesothelial cascading process that has evolved to protect life in the absence of surgery. They suggested modification in the activity of the mesothelial cells by molecular strategies, could help in more advancement in managing peritonitis[10]. Researchers have also shown that the mesothelial cell is a critical component of the peritoneal membrane.

The preoperative antibiotics and intraperitoneal use of several antibiotics, which is been done now-a-days, can lead to apoptosis of the peritoneal mesothelial cells[11].

Triple antibiotic therapy providing broad-spectrum coverage of gram-positive, gram-negative, and anaerobic bacteria has been the standard treatment of perforative peritonitis.

Treatment with multiple antibiotics in perforative peritonitis seemed to be therapeutically effective in this study which was similar to the study conducted by Nathens and Colleagues [12] where in treatment with multiple antibiotic combination in peritonitis was one of the treatment options in peritonitis which proved beneficial when compared to single antibiotic. There was reduction in postoperative complication rate in this study. The study done by Hunt and Colleagues[13] whether to use multiple antibiotics or not in perforative peritonitis also revealed reduction in complications in the postoperative period and patient survival rate.

Another experimental study in rats done by Hau and Colleagues[14] who investigated on a group of rats with fecal peritonitis revealed that peritoneal irrigation along with use of systemic antibiotics were effective.

HISTORICAL PERSPECTIVE

One of the earliest citations to peritoneum can be traced back to around 1700 years ago in *Edwin Smith Surgical Papyrus*, a manuscript written around the time of Imhotep (the Egyptian patron god of medicine). Breasted who translated these works, described, "*I felt as if I had been peering through a newly revealed window, opening upon the once impenetrable gloom enveloping man's earliest endeavors to understand the world he lived in it was as if I had watched a hand slowly raising the curtain that covered this window and then suddenly the hand had refused to lift, the curtain further*". The curtain may have meant peritoneum.

Since the establishment of medical antiquity, humans have been challenged with the varied spectra of peritonitis. Chronicles from the past civilizations illustrate the recognized value of therapeutic drainage in peritonitis. The first description of a patient with peritonitis was described by Hippocrates and the Koic School of Medicine as:

"*The patient looks sick and wasted. The nose is pointed, the temples sunken, the eyes lay deep, rimmed and dull. The face expresses fear, the tongue is furrowed, the skin shiny. The patient avoids all movements and breathes shallow. The abdominal wall is rigid with muscular guarding, no bowel sounds can be heard. The pulse is quick and small. A hard, tender mass in the hypochondrium is a bad prognostic sign if it involves the whole area. The presence of such a mass at the beginning of the fever indicates that death is imminent".*

The above depiction is presently known as the **Hippocrates facies** [15].

He also described septic shock as "*A protrusive nose, hollow eyes, sunken temples, cold ears that are drawn in with the lobes turned outwards, the forehead's skin rough and tense like parchment and the whole face greenish or black or leadened".*

In the second century A.D., Galen, apparently performed several surgeries including suturing of the lacerated bowel. He described about the appearance of suppuration in post-operative period and proposed that such suppuration (laudable pus) was critical for proper wound healing.

Sushruta in 6th century B.C. wrote the oldest known descriptions of bowel surgery and described using a cautery over the swelling of strangulated hernias and used the mandibles of black ants to clamp the edges of bowel wounds together.

Fabriciusd'Aquapendente in 12th century described a procedure of intestinal repair involving end-to-end anastomosis. Lanfranc in 13th century used animal tracheas to connect divided segments of bowel.

The first description of gastric ulcer is attributed to the Italian physician Marcello Donati in 1586 and the first case of perforated gastric ulcer was documented by Christopher Rawlinson in England in 1727.

Douglas Best in 1730 gave a detailed description of the peritoneum.

Winslow in 1732 described greater and lesser omentum, lesser sac and foramen.

Duodenal ulcer was first described by Georg Hamberger in Germany in 1746 and in 1793 Jacopo Penada from Italy recorded a duodenal perforation[16].

In 1938, Graham popularized simple closure technique with omental patch for perforated peptic ulcer[17].

The three milestones that fostered an understanding of the peritonitis disease process comprised the foundation of experimental physiology by Francois Magendie and Claude Bernard, an

understanding of cellular pathology by Rudolph Virchow and the advent of germ theory by Pasteur and Koch.

Froriep in 1812, described the anatomy of peritoneum and omentum.

George Wegener first reported in 1879, a series of experiments attempting to elucidate the normal physiology of the peritoneum. Reginald Fitz published his paper in 1886 on inflammation of the vermiform appendix highlighting the importance of early diagnosis and treatment. Thus the modern surgical treatment of appendix was initiated[18].

Kriege in 1892 described the successful surgical management of a perforated peptic ulcer followed by Carlisle in 1894. The first major trial of conservative treatment was undertaken by Herman Taylor at the King George hospital, Illford in 1957. He reported a 10 year experience of managing 256 patients with perforated ulcer, 208 of who had been treated by conservative method.

Later on Smith, Travers and Elliston in the early 19th century gave a clear description of peritonitis independently[19]. With the advent of general anaesthesia and the advent of asepsis and antisepsis techniques, laparotomy as a part of management of peritonitis was gradually established. In 19th century, treatment of peritonitis consisted of absolute rest, purgation, abstinence from food intake, application of cold to the abdomen, opium administration, etc.

Lee *et al.*[20],in 1977 documented that pneumo-peritoneum in perforated duodenal ulcer disease is seen in 50-60% of patients within 8 hours of perforation and after 8 hours in over 80% of patients. Hodnett *et al.,* in 1989 showed that most gastric ulcer perforated anteriorly with only 9% perforated posteriorly. Laparoscopic repair of perforated duodenal ulcer was first reported in 1990 by Mouret and Colleagues[20].

William WT *et al.,* in 1988 showed that suturing of perforated gastric ulcer primarily without omental patch had significantly higher mortality and early postoperative complications rates than when patches were applied[21].

Surgery for the management of typhoid perforation began in the late 1800 with the appreciated works of Finney and Cushing[22].

The understanding of peritoneum was further enriched by John B. Murphy in his writings[23]- *"There are no stomata or stigmata in the peritoneum. The endothelial lining is everywhere, continuous".*

Herbert E. Durham analyzed the peritoneal fluid and proposed a chain of cellular events which are divided in 5 stages- stage before leucopenia, the leukopenic stage, the microxyphil stage, the macrophage stage and the recovery stage[24].

Meleney *et al.*[26], demonstrated the existence of the bacterial synergism and that combinations of aerobic and anaerobic bacteria produced severe sepsis than from individual strains[25].

In 1891, the first published report on the surgery for intestinal tuberculosis was by Hartman and Piteit[26].

Singhai *et al.,* in 1964 showed that ileocaecal region was the commonest site of involvement in abdominal TB[27].In 1968, Bhansali *et al.,* testified that closure of tubercular perforation with or without bypass attributes to poor results[28]. Resection and anastomosis was recommended by Aston and Decosta in 1985.

Various surgical procedures have been used for distal ileal perforations with variable results. The postoperative mortality and morbidity remains high. Resection anastomosis carries a high morbidity and mortality. Wani RA *et al.*[29], proposed end to side ileotransverse anastomosis with closure of distal stump as a better procedure for the distal ileal perforation over other techniques with regards to the morbidity[29].

The complication rates in various perforations vary. Nair SK *et al.*[30],in 1981 reported maximal morbidity in the form of wound infection in 52% of patients, followed by fecal fistula in 16% of patients, septicemia in 8% of patients and respiratory infection in 4% of patients[30].

The current therapy of peritonitis was summarized by Martin Kirschner[31] in 1926. His therapeutic principles are valid to this day and his article represents a hallmark in the therapy of intraperitoneal infections.

Its conclusions were:

1)Every patient with acute diffuse peritonitis should be operated immediately unless there is an absolute contraindication to surgery. Exceptions are gonococcal and pneumococcal peritonitis.

2)The operative procedure and the anesthesia should be conducted as gently as possible.

3) The incision should be made over the focus of infection. If there is any doubt, a midline laparotomy should be performed. The incision should be long enough to allow easy access to the infectious focus.

4) The most important aim of surgery is the elimination of the source of infection. This should be done by the simplest possible procedure. Eventration of the bowel should be avoided.

5) Exudate and debris found in the peritoneal cavity are removed by irrigation with normal saline solution. Medications should not be instilled into the peritoneal cavity.

6) Mechanical emptying of the bowel or primary construction of stomata should be avoided.

7) The free peritoneal cavity cannot be drained and drains should not be used. Only if secure elimination of the infectious focus is not possible, drainage is indicated.

Khosrovani in 1994 identified 3 factors of immediate mortality for perforation of duodenopyloric ulcers- age over 70 years, admission delayed by more than 24hours and preoperative hemodynamic shock[32].

The peritoneal cavity is a potential space lined by a mesothelial layer and sub serosal layer. The peritoneal cavity is divided into general peritoneal cavity and lesser omental bursa. They communicate through the foramen of Winslow. For the purpose of description anterior abdominal wall is divided into 9 regions, by 2 horizontal and 2 vertical lines. The use of quadrants help in topographic location of pain[33].

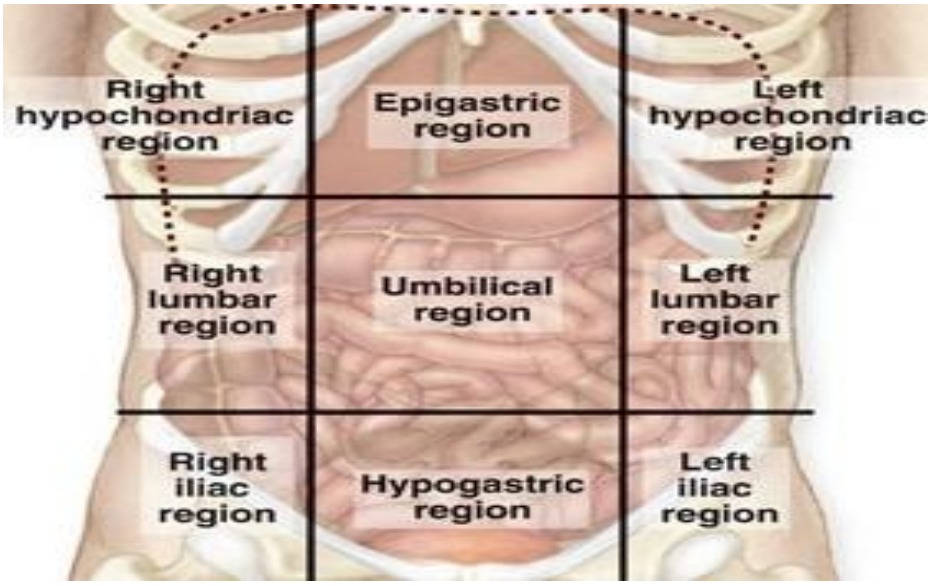


Figure 1: Nine quadrants of the abdomen

ANATOMY

Embryology

At the end of the third week, intra embryonic mesoderm differentiates into paraxial mesoderm, intermediate mesoderm and lateral plate mesoderm that is involved in forming the body cavity. Clefts appear in the lateral plate mesoderm that coalesces to split the solid layer into:

(a) The parietal (somatic) layer adjacent to the surface ectoderm and continuous with the extra embryonic parietal mesoderm layer over the amnion.

(b) The visceral (splanchnic) layer adjacent to endoderm forming the gut tube and continuous with the visceral layer of extra embryonic mesoderm covering the yolk sac.

Embryo at 19 days: Intercellular clefts are visible in the lateral plate mesoderm.

Embryo at 20 days: The lateral plate is divided into somatic and visceral mesoderm layers that line the intraembryonic cavity. Tissue bordering the intraembryonic cavity differentiates into serous membranes.

The space created between the two layers of lateral plate mesoderm comprises the primitive body cavity. Cells of the parietal layer of lateral plate mesoderm lining the intra embryonic cavity become mesothelial and form the parietal layer of the serous membranes lining the outside of the peritoneal, pleural and pericardial cavities. In a same way, cells of the visceral layer of lateral

plate mesoderm form the visceral layer of the serous membranes covering the abdominal organs, lungs and heart[34].

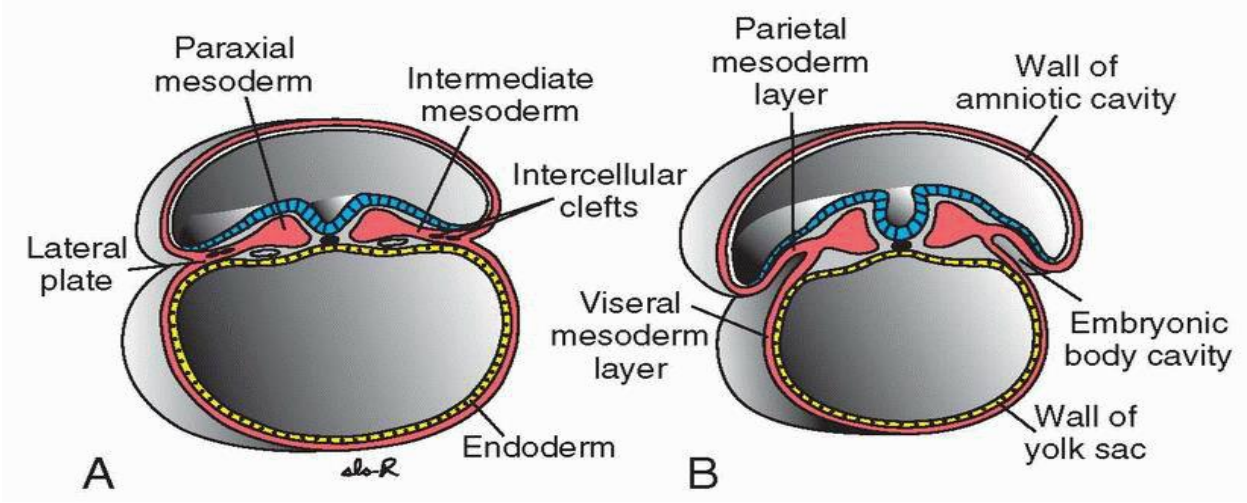


Figure 2: Embryology of peritoneum. A. Transverse section through an embryo of approximately 19 days B. Section through an embryo of approximately 20 days[34].

Formation of the Peritoneal Ligaments and Mesenteries[35]

The peritoneal ligaments are developed from the ventral and dorsal mesenteries. The ventral mesentery is formed from the mesoderm of the septum transversum (derived from the cervical somites which migrate downward). The ventral mesentery forms the falciform ligament, the lesser omentum and the coronary and triangular ligaments of the liver.

The dorsal mesentery is formed from the fusion of the splanchnopleuric mesoderm on the two sides of the embryo. It extends from the posterior abdominal wall to the posterior border of the abdominal part of the gut. The dorsal mesentery forms the gastrophrenic ligament, the gastrosplenic omentum, the splenorenal ligament, the greater omentum and the mesenteries of the small and large intestines.

Formation of the Lesser and Greater Peritoneal Sacs[35]

The extensive growth of the right lobe of the liver pulls the ventral mesentery to the right and causes rotation of the stomach and duodenum. By this means, the upper right part of the peritoneal cavity becomes incorporated into the lesser sac. The right free border of the ventral mesentery becomes the right border of the lesser omentum and the anterior boundary of the entrance into the lesser sac.

The remaining part of the peritoneal cavity, which is not included in the lesser sac is called the greater sac and the two sacs are in communication through the epiploic foramen.

Formation of the Greater Omentum[35]

The spleen develops from the upper part of the dorsal mesentery and the greater omentum is formed as a result of the rapid and extensive growth of the dorsal mesentery caudal to the spleen. To begin with, the greater omentum extends from the greater curvature of the stomach to the posterior abdominal wall superior to the transverse mesocolon. With continued growth, it reaches inferiorly as an apronlike double layer of peritoneum anterior to the transverse colon.

Later, the posterior layer of the omentum fuses with the transverse mesocolon; as a result, the greater omentum becomes attached to the anterior surface of the transverse colon. As development proceeds, the omentum becomes laden with fat. The inferior recess of the lesser sac extends inferiorly between the anterior and the posterior layers of the fold of the greater omentum.

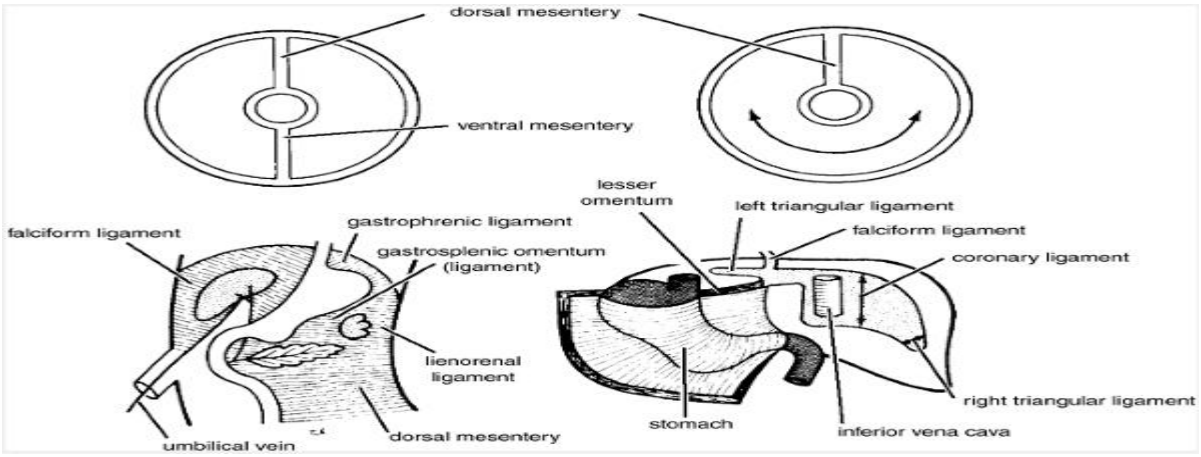


Figure 3: Ventral and dorsal mesenteries and the organs that develop within them

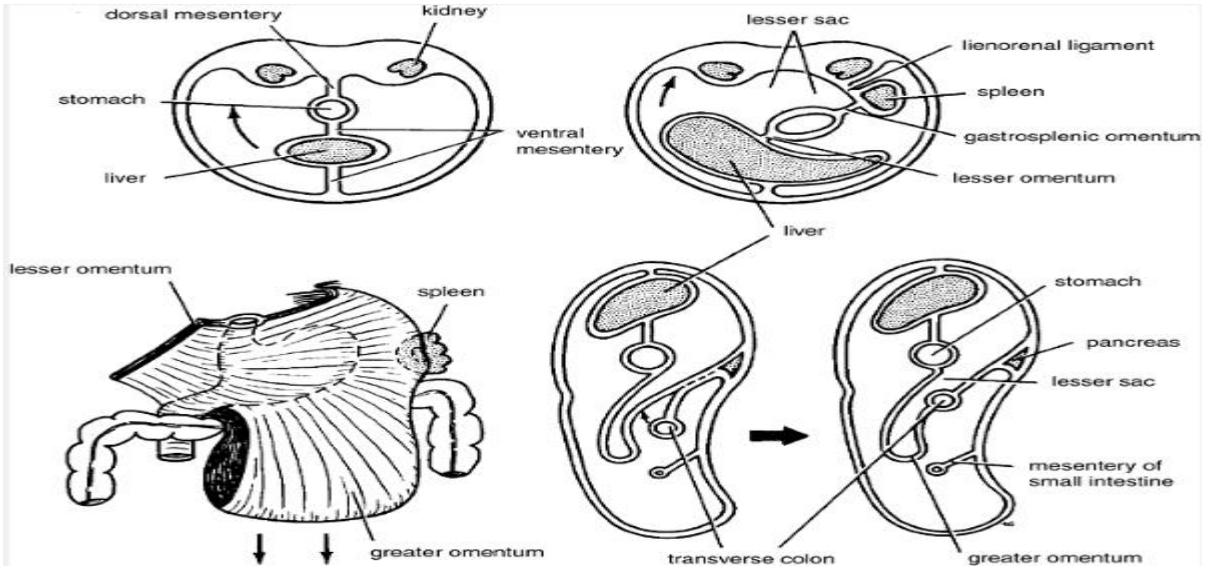


Figure 4: Rotation of the stomach and the formation of the greater omentum and lesser sac.

Surgical anatomy

A. Abdominal cavity:

The abdominal cavity forms the superior and major part of the abdominopelvic cavity, the continuous cavity that extends between the thoracic diaphragm and the pelvic diaphragm. The abdominal cavity has no floor of its own because it is continuous with the pelvic cavity. The plane of the pelvic inlet (superior pelvic aperture) arbitrarily, but not physically, separates the abdominal and the pelvic cavities. The abdominal cavity extends superiorly into the osseocartilaginous thoracic cage to the 4th intercostal space. The more superiorly placed abdominal organs like spleen, liver and stomach are protected by the thoracic cage. The greater pelvis i.e., expanded portion of the pelvis superiorly to the pelvic inlet supports and protects the lower abdominal organs like lower portion of ileum, cecum and sigmoid colon[36].

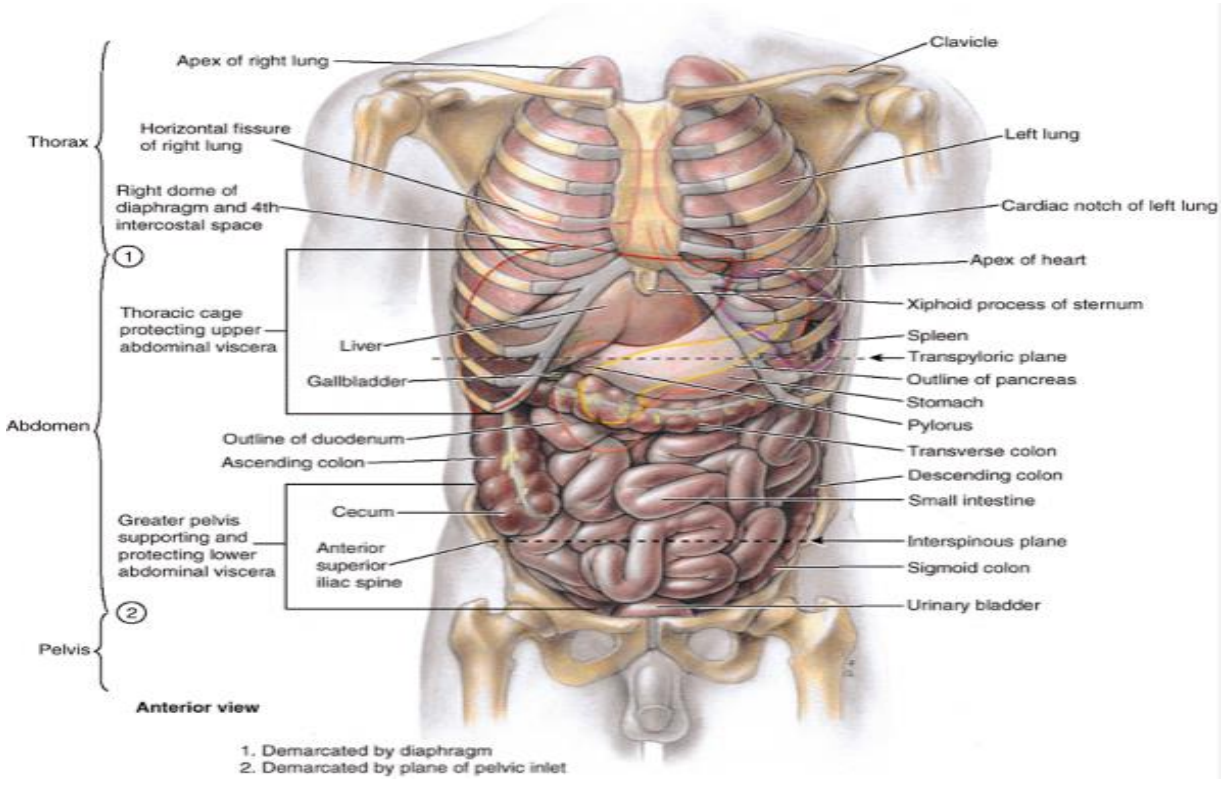


Figure 5: Overview of viscera of thorax and abdomen in situ[36].

In summary, the abdominal cavity is[36]

- The major part of the abdominopelvic cavity.
- Located between the diaphragm and the pelvic inlet.
- Separated from the thoracic cavity by the thoracic diaphragm.
- Continuous inferiorly with the pelvic cavity.
- Under cover of the thoracic cage superiorly.
- Supported and partially protected inferiorly by the greater pelvis.
- Enclosed antero-laterally by multi-layered, musculo-aponeurotic abdominal walls.
- The location of most digestive organs, parts of the urogenital system (kidneys and the ureters) and the spleen.

B. Peritoneum:

The peritoneal cavity is the largest cavity in the body with the surface area of its lining membrane being 2 m^2 in an adult which is nearly equal to that of the skin. It is divided into parietal and visceral portions. The parietal layer lines the abdominal and pelvic cavities and the abdominal surface of the diaphragm. The visceral layer covers the abdominal and pelvic organs and includes the mesenteries.

The peritoneum consists of a fibrous layer (the tunica subserosa) and a surface layer of mesothelium (the tunica serosa).

The parietal peritoneum is only loosely connected with the body wall, separated from it by an adipose layer, the telasubserosa; whereas the visceral peritoneum is usually tightly attached to the organs it covers[37].

Table 1: Parts of the Peritoneum[37]

Omenta	Greaterомentum
	Lesser omentum
Mesenteries	Mesentery of the small bowel
	Mesoappendix
	Transverse mesocolon
	Pelvic mesocolon
Ligaments	Of liver
	Of urinary bladder
	Of uterus
Fossae	Duodenal
	Cecal
	Intersigmoid

Vascular Supply of the Peritoneum[37]

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

Lymphatics of the Peritoneum[37]

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

In 1863, Von Recklinghausen was the first to describe the modified lymphatics which are able to remove particles from the peritoneal fluid during the process of respiration. The relaxed diaphragm permits opening of the stomata of these lymphatic vessels and the fluid enters the lymphatic circulation. Higgins et al. reported that contractions of the diaphragm pump the lymph and its contents (particulate matter and molecular substances) upward, aided by one-way valves which are located within the lymphatics of the retrosternal area.

Innervations of the Peritoneum[37]

The parietal peritoneum contains somatic afferent nerves for the sensation of pain; the anterior portion of the parietal peritoneum is especially sensitive.

In contrast, the visceral peritoneum is relatively insensitive to pain. Sensations are poorly perceived and not clearly localized by the brain and is characteristic of visceral afferent fibers carried by autonomic nerves to viscera in general. The principal stimulus which can evoke pain from visceral peritoneum is tension upon or stretching of the tissue or ischemia. A perforated viscus may, perhaps, produce anterior abdominal wall rigidity and an intraperitoneal fluid collection may produce pain like sensations of traction or tension on the mesentery in the retroperitoneal space, but not localized pain[37].

Spaces in the peritoneum:

The peritoneal cavity is subdivided into interconnected compartments or spaces by 11 ligaments and mesenteries[38].

The peritoneal ligaments or mesenteries include the[38]

1. Coronary,
2. Gastrohepatic,
3. Hepatoduodenal,
4. Falciform,
5. Gastrocolic,
6. Duodenocolic,
7. Gastrosplenic,
8. Splenorenal,
9. Phrenicocolic ligaments,
10. The transverse mesocolon,
11. Small bowel mesentery.

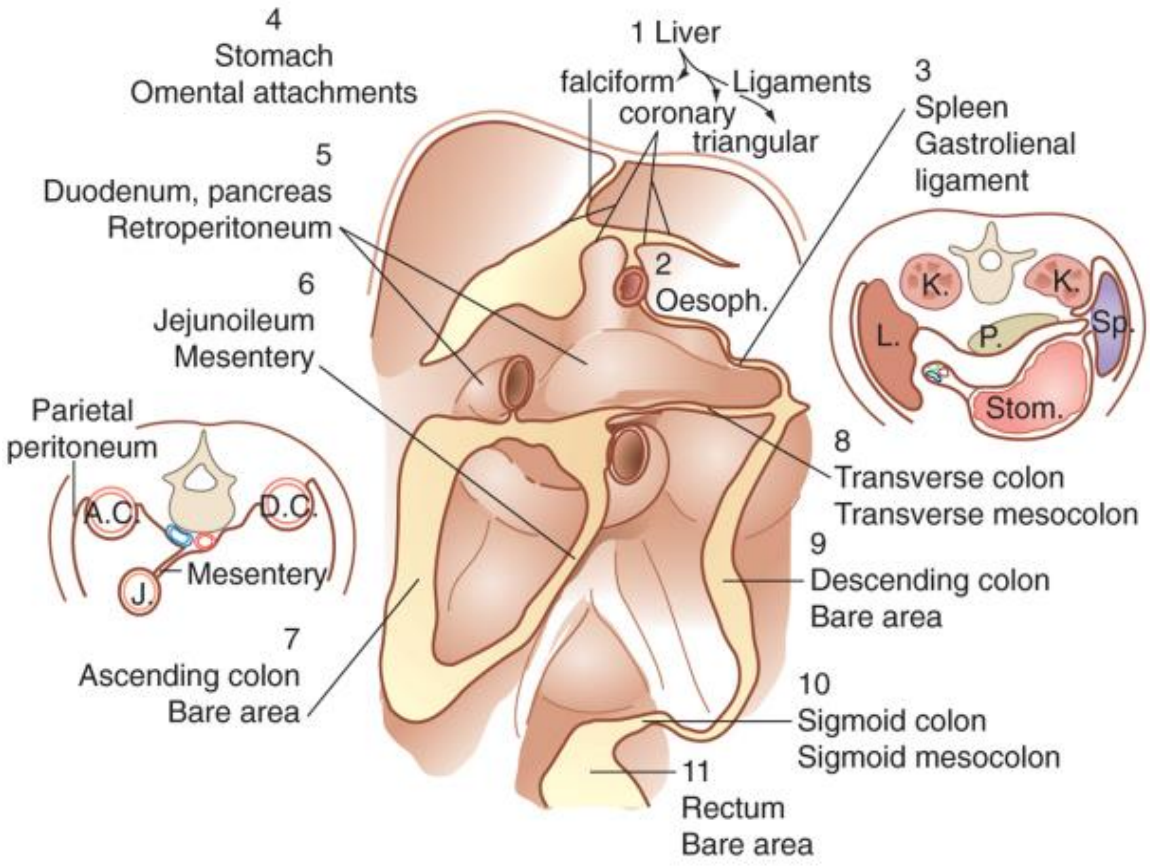


Figure 6**:**Peritoneal ligaments and mesenteric reflections in the adult.[38]

Peritoneal recesses, Spaces, and Gutters

These ligaments partition the abdomen into nine potential spaces:[38]

1. Right and left subphrenic,
2. Subhepatic,
3. Supramesenteric
4. Inframesenteric,
5. Right and left paracolic gutters,
6. Pelvis and
7. Lesser space.

These ligaments, mesenteries and peritoneal spaces direct the circulation of fluid in the peritoneal cavity and thus may be useful in predicting the route of spread of infectious and malignant diseases. For example, perforation of the duodenum from peptic ulcer disease may result in the movement of fluid and the development of abscesses in the subhepatic space, the right paracolic gutter and the pelvis[38]. These attachments partition the abdomen into nine potential spaces and are represented in figure no 7.

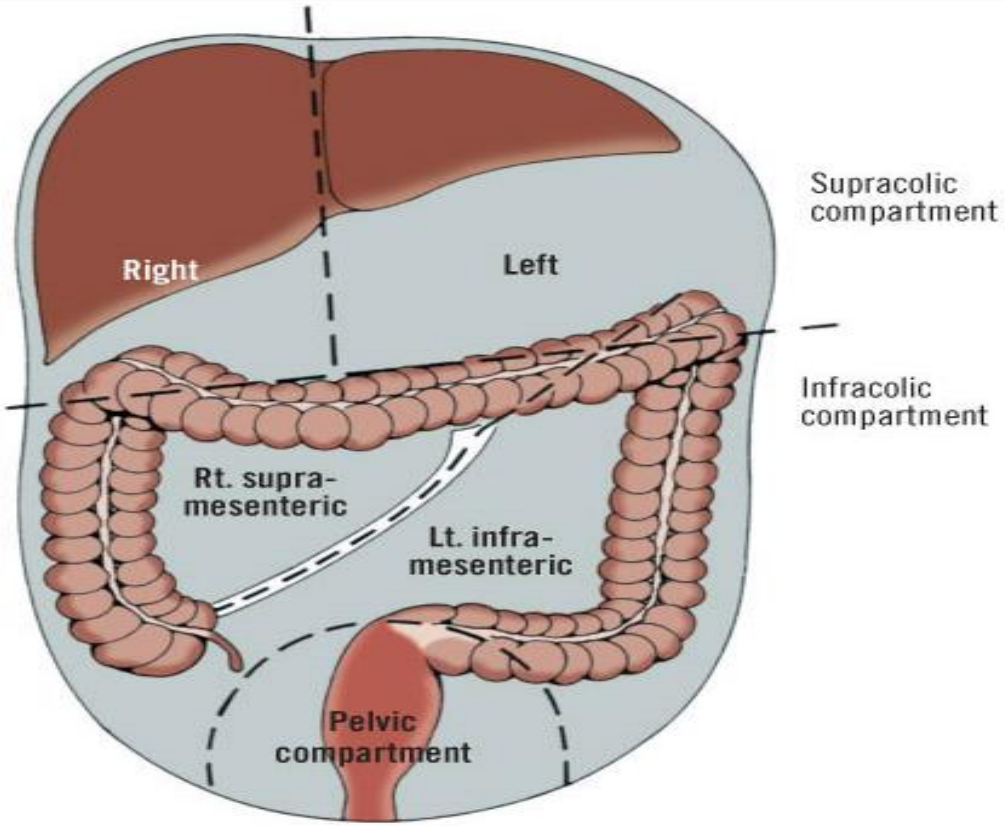


Figure 7: Spaces in the peritoneum:

Greater sac:

The peritoneal cavity is the largest cavity in the body and is divided into two parts: the greater sac and the lesser sac (fig: 8 and 9). The greater sac is the main compartment and extends from the diaphragm down into the pelvis.

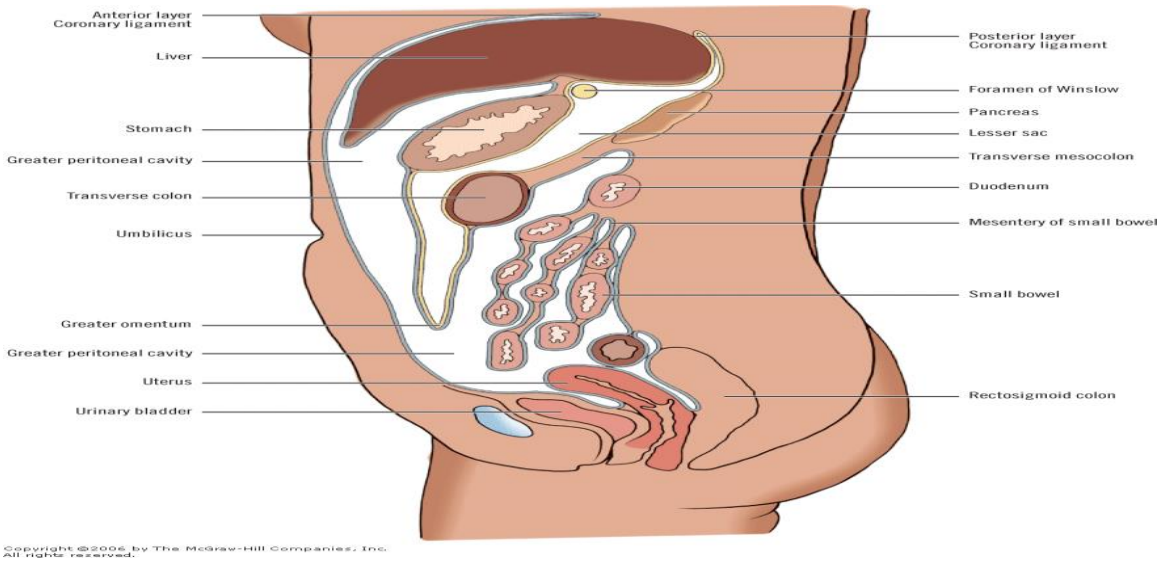


Figure 8: Vertical disposition of the peritoneum (abdominopelvic cavity).[37]

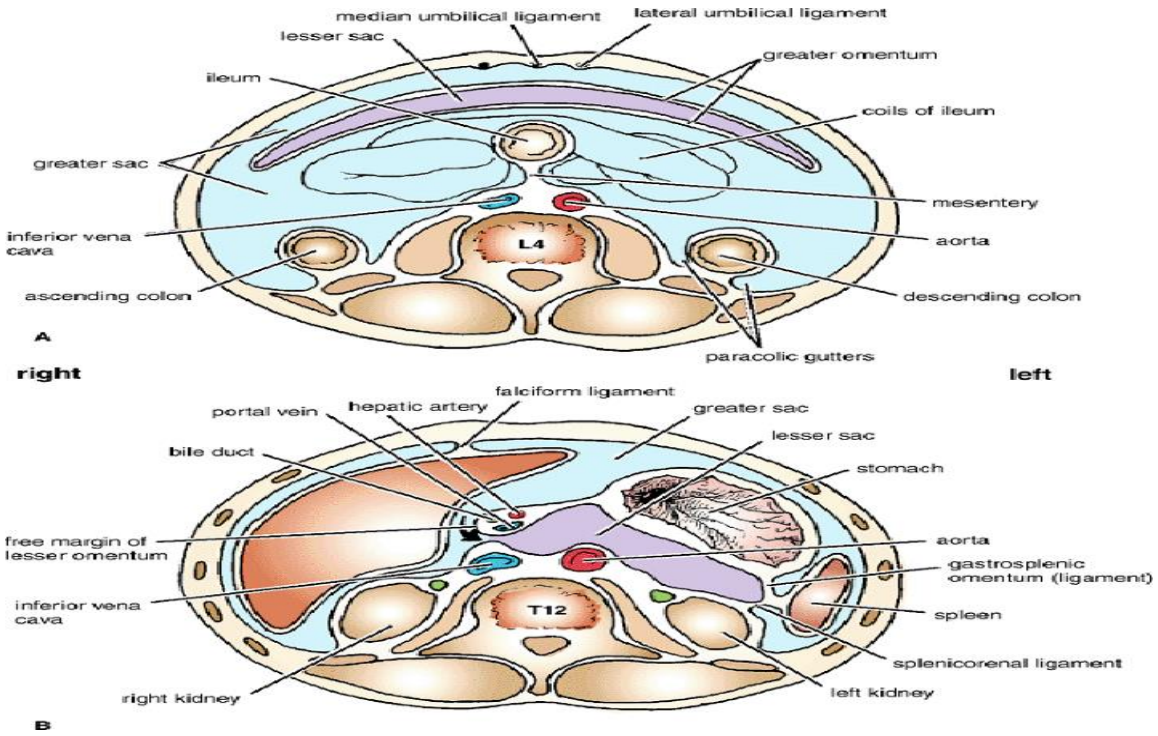


Figure 9: Transverse sections of the abdomen showing the arrangement of the peritoneum[35]

Lesser Sac

The lesser sac lies behind the stomach and the lesser omentum. It extends upward as far as the diaphragm and downward between the layers of the greater omentum. The left margin of the sac is formed by the spleen and the gastrosplenicomentum and splenicorenal ligament. The right margin opens into the greater sac which is the main part of the peritoneal cavity through the opening of the lesser sac i.e., epiploic foramen (Foramen of Winslow).

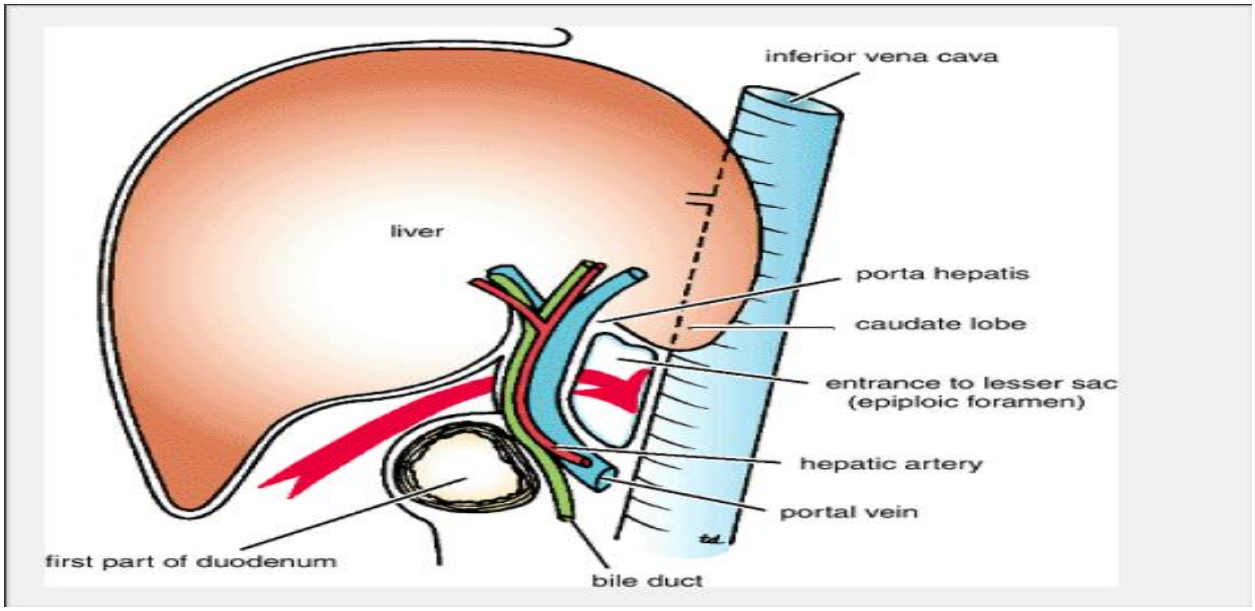


Figure 10: lesser sac

Duodenal Recesses

Close to the duodeno-jejunal junction, there are4 small pocketlike pouches of peritoneum called the superior duodenal, inferior duodenal, paraduodenal and retroduodenal recesses as depicted in figure 11.

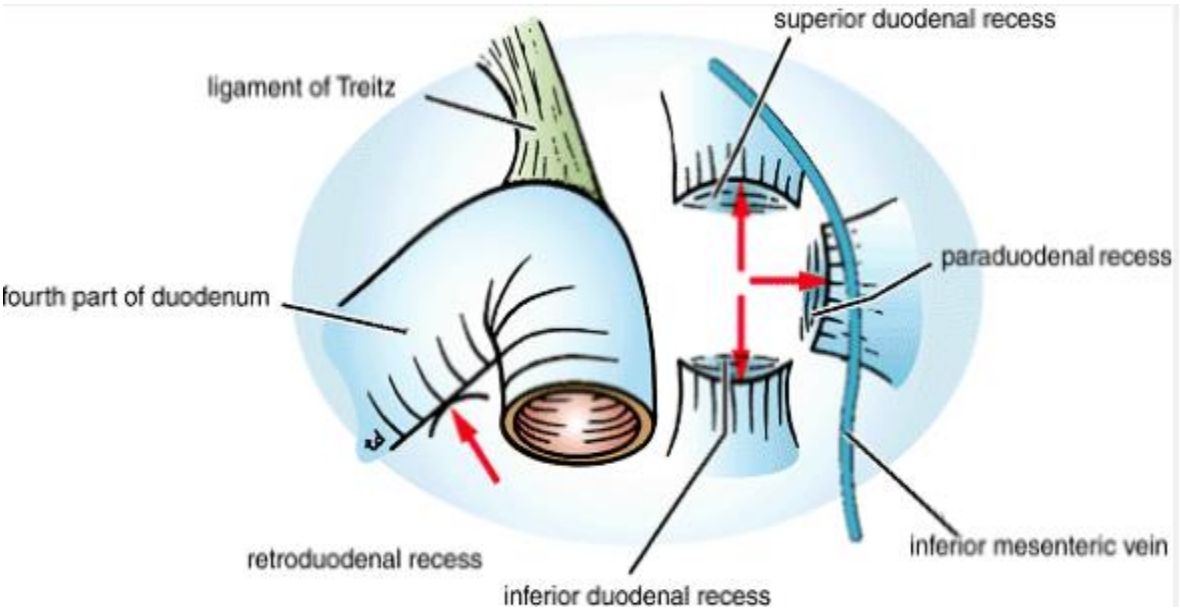


Figure 11: Peritoneal recesses forming the paraduodenal recess.

Cecal Recesses

Folds of peritoneum close to the cecum produce three peritoneal recesses called the superior ileocecal, the inferior ileocecal and the retrocecal recesses (Fig. 12).

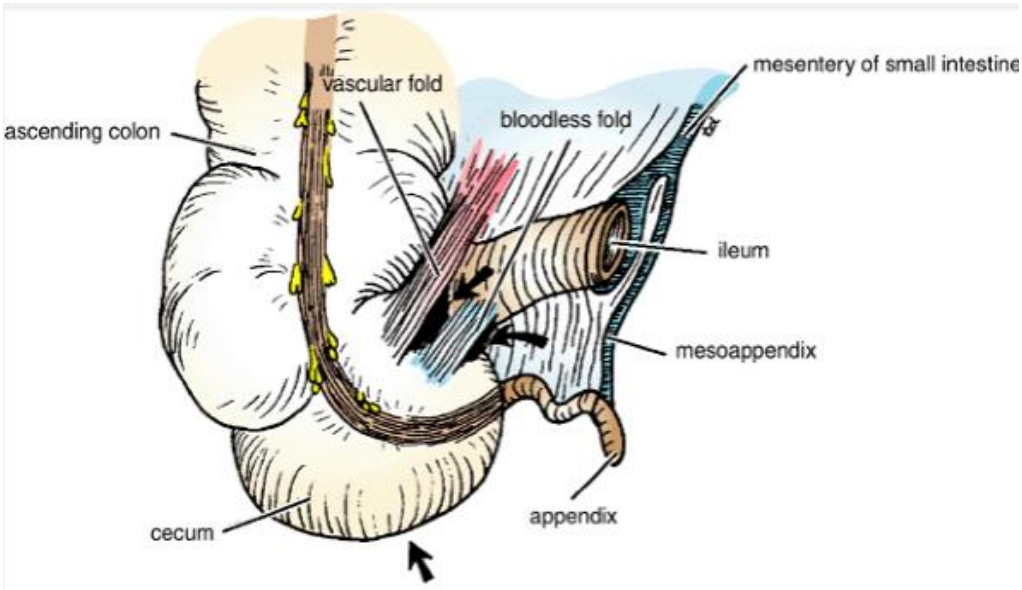


Figure 12: cecal recess

Inter-sigmoid Recess

The inter-sigmoid recess is situated at the apex of the inverted, V-shaped root of the sigmoid mesocolon (Fig. 13); its mouth opens downward.

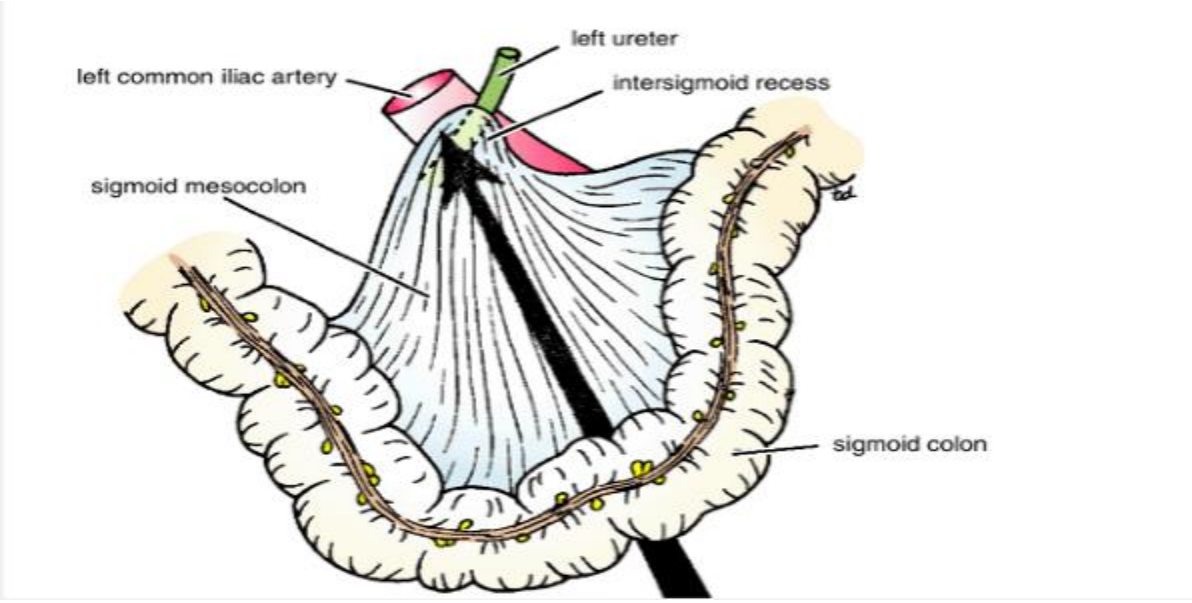


Figure 13: Inter sigmoid recess

Paracolic Gutters

The paracolic gutters lie on the lateral and medial sides of the ascending and descending colons respectively. The subphrenic spaces and the paracolic gutters are clinically important because they are the areas for the fluid or pus collection and movement of infected peritoneal fluid.

Peritoneal fluid

A small amount of serous fluid is normally present in the peritoneal space, potential space containing approximately 50 ml of isotonic fluid which lubricates the surfaces, allowing frictionless movements of the gastrointestinal tract and contains:

- protein content (consisting mainly of albumin) of <30 g/L
- White blood cells<300 per microliter (WBCs, generally mononuclear cells).[39]

Peritoneal spread of disease[37]

The spread of fluid in the peritoneal cavity depends on all of the following:

- Location of the source and the rate of fluid production
- Pressure differences in the abdomen
- Mesenteric partitions and peritoneal fossae
- Position of the body in relation to gravity

The large surface area of the peritoneal cavity allows infection and malignant disease to spread easily throughout the abdomen. If malignant cells enter the peritoneal cavity by direct invasion (e.g. from colon or ovarian cancer) spread may be rapid.

The peritoneal cavity can also act as a barrier to and container of disease. Intra-abdominal infection therefore tends to remain below the diaphragm rather than spread into other body cavities.[40]

The circulation of fluid and potential areas for abscess formation is shown in figure 14 and 15. Some compartments collect fluid or pus more often than others. These compartments include the pelvis (the lowest portion), the subphrenic spaces on the right and left sides and Morrison's pouch, which is a postero-superior extension of the subhepatic spaces and is the lowest part of the paravertebral groove when a patient is recumbent. The falciform ligament separating the right and left subphrenic spaces appears to act as a barrier to the spread of infection; consequently, it is unusual to find bilateral subphrenic collections.[39]

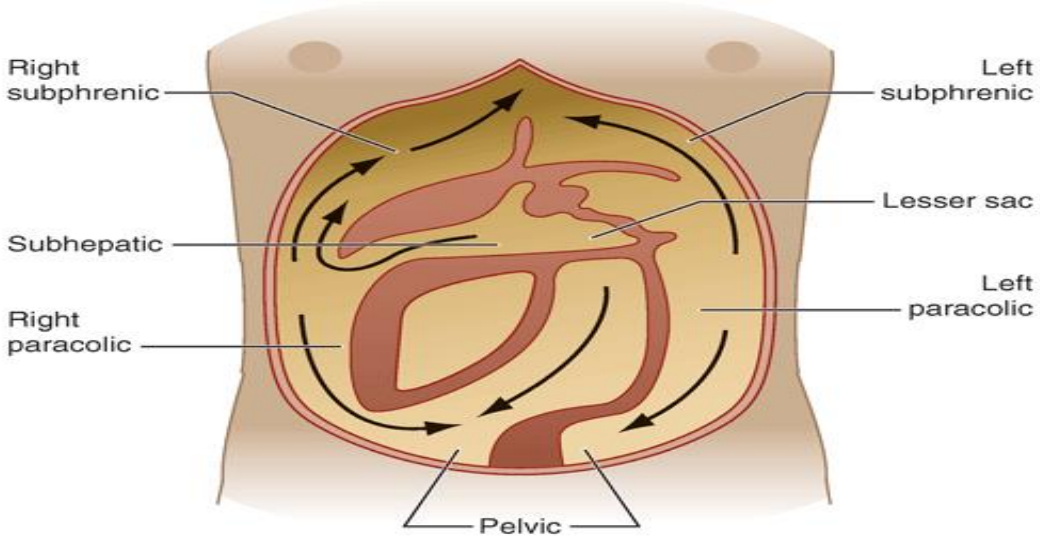


Figure 14: peritoneal spread of disease[39]

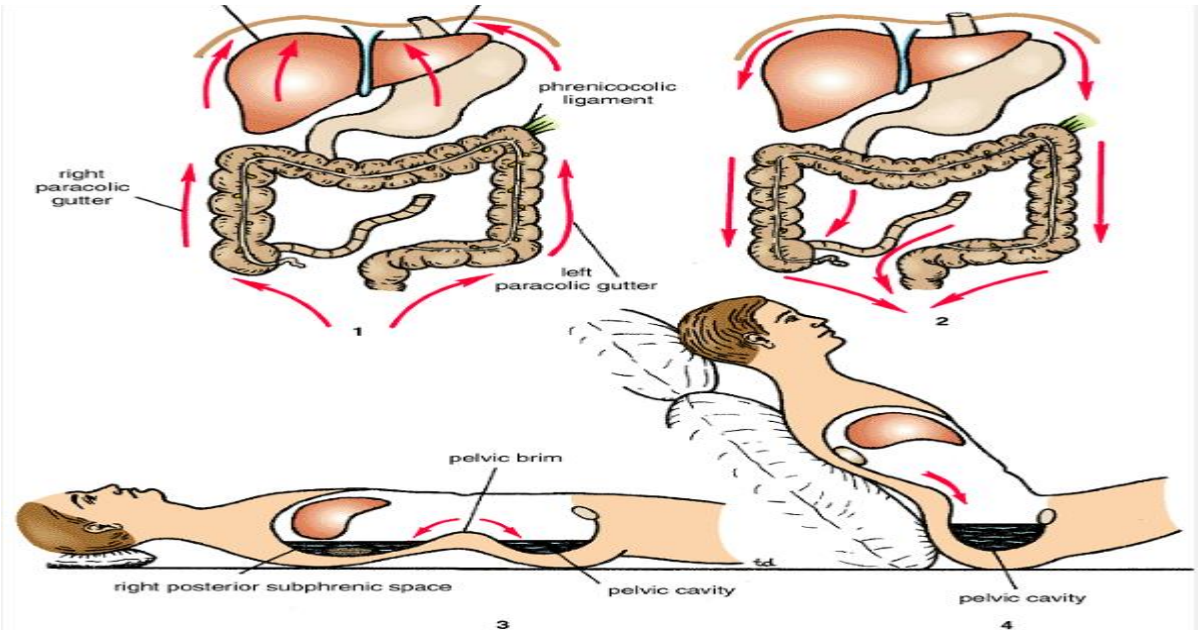


Figure 15: Direction of flow of the peritoneal fluid. 1. Normal flow upward to the subphrenic spaces. 2. Flow of inflammatory exudate in peritonitis. 3. The two sites where inflammatory exudate tends to collect when the patient is nursed in the supine position. 4. Accumulation of inflammatory exudate in the pelvis when the patient is nursed in the inclined position.

Intraperitoneal and Retroperitoneal Relationships[35]

The descriptive terms like intraperitoneal and retroperitoneal are used to explain the relationship of various organs to their peritoneal covering. An organ is said to be intraperitoneal when it is almost totally covered with visceral peritoneum. The stomach, jejunum, ileum and spleen are the best examples of intraperitoneal organs. Retroperitoneal organs lie behind the peritoneal layer and are covered with the visceral peritoneum partially. The pancreas and the ascending and descending parts of the colon are examples of retroperitoneal organs. No organ, however, is actually within the peritoneal cavity. An intraperitoneal organ, such as the stomach, appears to be surrounded by the peritoneal cavity, but it is covered with visceral peritoneum and is attached to other organs by omenta.

The relationship of the viscera to the peritoneum is as follows: [36]

- Intra peritoneal organs are totally covered with visceral layer of the peritoneum like the stomach, spleen etc. Intra peritoneal organs have conceptually, if not literally, invaginated into the closed sac, like pressing your fist into an inflated balloon.
- Extra peritoneal, retro and sub peritoneal vital organs are outside the peritoneal cavity and are covered with peritoneum partially on one surface.

The peritoneal cavity is within the abdominal cavity and continues inferiorly into the pelvic cavity. The peritoneal cavity is totally closed in men; in contrast there is a communication in females to the exterior of the body through the fallopian tubes, uterine cavity and vagina. This communication constitutes a potential pathway of infection from the exterior.[36]

Histology of peritoneum:

Both parietal and visceral parts of the peritoneum have the same histologic formation:

Basement membrane covered by a single layer of mesothelial cells. Loss of these cells produces non physiologic adhesions between the two parts.

PATHOPHYSIOLOGY

Peritonitis is simply defined as inflammation of the peritoneum and may be localized or generalised.[41]

Factors influencing diaphragmatic uptake of fluid and particles[42].

1. Mesothelial cells contain the contractile filaments, actin, which when paralyzed, markedly enlarges in size.

2. Most important is the state of diaphragmatic contraction. With exhalation, the diaphragm relaxes, the stomata open and because of the negative pressure induced by the diaphragm moving upward, fluid and particulate material are sucked up to the open stomata and then to the substernal lymph nodes and from there to the thoracic duct.

3. Presence of inflammation, which increases stomata patency by inducing mesothelial cell retraction.

4. The diaphragmatic lymphatics play a major role in the absorption of fluid and particulate matter from the peritoneal cavity, both under normal circumstances and during peritonitis.

Response of the peritoneum and peritoneal cavity to infection[38]:

1. Bacteria are rapidly removed from the peritoneal cavity through the diaphragmatic stomata and lymphatics, as described above.
2. Peritoneal macrophages release pro-inflammatory mediators that promote the migration of leukocytes into the peritoneal cavity from the surrounding microvasculature.
3. Degranulation of peritoneal mast cells releases histamine and other vasoactive products, causing local vasodilatation and the extravasation of protein rich fluid containing complement and immunoglobulins into the peritoneal space.
4. Protein within the peritoneal fluid opsonizes bacteria, which along with activation of the complement cascade, promotes neutrophil and macrophage-mediated bacterial phagocytosis and destruction.
5. Bacteria become sequestered within fibrin matrices, thereby promoting abscess formation and limiting the generalized spread of the infection.[38]

Paths to peritoneal infection[41]**:**

- Gastrointestinal perforation e.g.: perforated ulcer, appendix, diverticulum.
- Transmural translocation[no perforation] e.g.: pancreatitis, ischemic bowel.
- Exogenous contamination e.g.: drains, open surgery, trauma.
- Female genital tract infection, e.g.: pelvic inflammatory disease.
- Haematogenous spread [rare] e.g.: septicaemia.

Phases of Peritonitis[43]

Phase I:

This involves the rapid removal of contaminants from the peritoneal cavity into the systemic circulation. It occurs because contaminated peritoneal fluid moves cephalad in response to pressure gradients generated by the diaphragm. The fluid passes through natural opening in the diaphragmatic peritoneum and is absorbed into lymphatics. The lymph flows into the main lymphatic ducts via the substernal nodes. The resultant septicemia predominantly involves gram-negative facultative anaerobes and is associated with high morbidity.

Phase II:

This involves synergistic interactions between aerobes and anaerobes as they encounter host complement and phagocytes. The activation of complement is a first-line event in peritonitis and involves innate and acquired immunity; activation occurs mainly by the classical pathway, with the alternative and lectin pathways in support. Phospholipids produced by the peritoneal mesothelial cellular structures work synergistically with complement to increase opsonization and phagocytosis. Peritoneal mesothelial cells are also potent secretors of pro-inflammatory mediators, including interleukin-6, IL-8, monocyte chemoattractant protein-1, macrophage inflammatory protein-1α and tumor necrosis factor-α.Therefore, peritoneal mesothelial cells play a central role in the cell signaling pathways leading to the recruitment of phagocytes to the peritoneal cavity and the up regulation of mast cells and fibroblasts in the sub-mesothelium.

Phase III:

It is an attempt by host defenses to localize infection, mainly via production of fibrinous exudates that traps microbes within its matrix and promotes local phagocytic effectors mechanisms. It also serves to promote the development of abscesses. Regulation of the formation and degradation of fibrinous exudates is vital to this process. The plasminogen-activating activity generated by peritoneal mesothelial cells determines whether the fibrin that forms after peritoneal injury is lysed or organized into fibrous adhesions. In particular, tumor necrosis factor-α stimulates the production of plasminogen activator-inhibitor-1 by peritoneal mesothelial cells, which inhibits degradation of fibrin.

Microbiology of peritonitis

The commonest organisms causing peritonitis are Escherichia coli, aerobic and anaerobic streptococci and bacteroides. Less frequently encountered organisms are Clostridium welchi, staphylococci or Klebsiella pneumoniae(Friedländer's bacillus).[41]

Source of peritonitis:

Stomach and duodenum are the major source of peritonitis.[37]

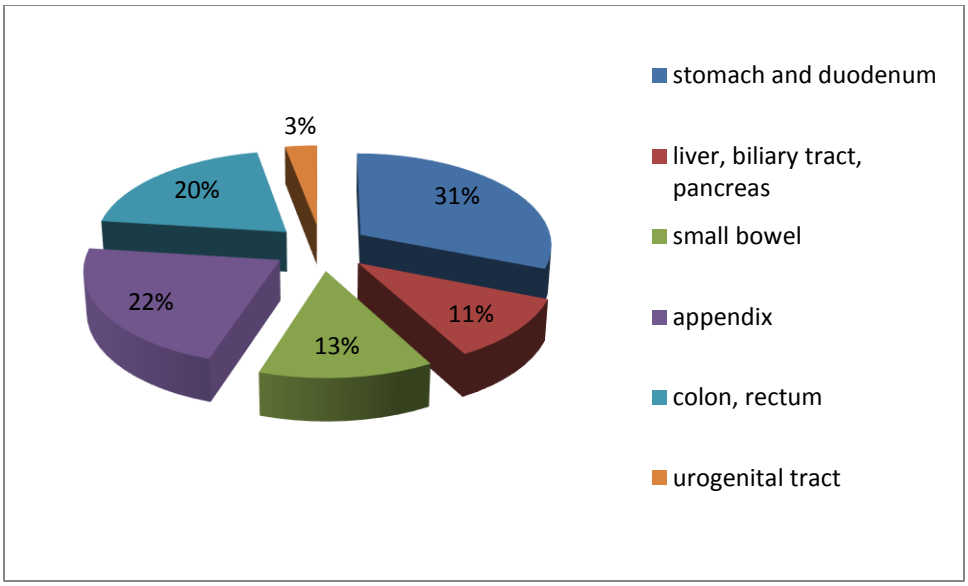


Figure 16: Source of peritonitis [44]

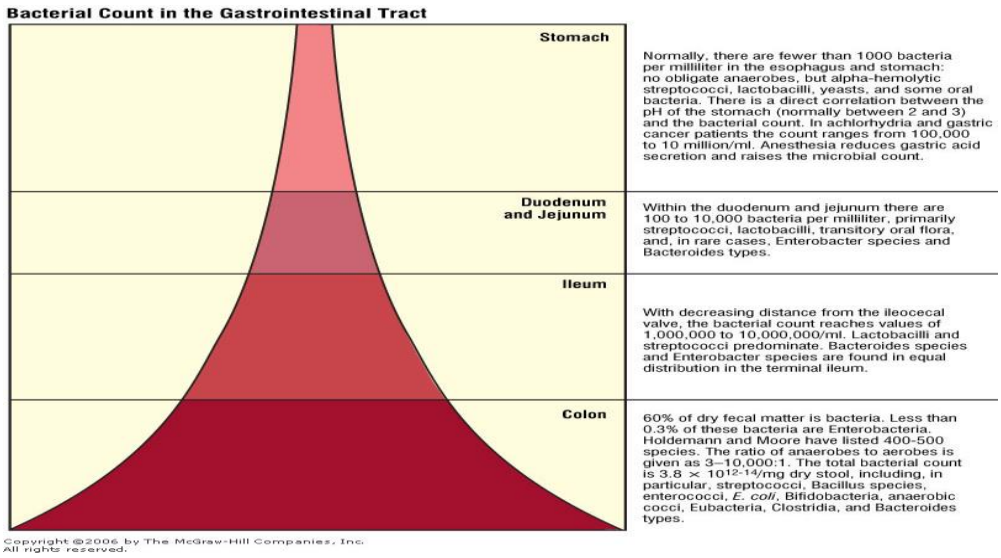


Figure 17: Bacterial count in gastrointestinal tract

Microorganisms in peritonitis[41]

Gastrointestinal source:

- Escherichia coli
- Streptococci
- Bacteroides
- Clostridium
- Klebsiella pneumoniae

Other sources

- Chlamydia trachomatis
- Neisseria gonorrhoeae
- Haemolytic streptococci
- Staphylococcus
- Streptococcus pneumoniae
- Mycobacterium tuberculosis and other species
- Fungal infections

Primary Bacterial Peritonitis

In Primary Bacterial Peritonitis, a single organism is typically isolated; enteric gram-negative bacilli such as Escherichia coli are most commonly encountered, gram-positive organisms such as streptococci, enterococci or even pneumococci are sometimes found.[39]

Secondary peritonitis

Secondary peritonitis develops when bacteria contaminate the peritoneum as a result of spillage from an intraabdominal affected structure. The most common causes are perforation and inflammation. The organisms isolated almost constitutegram-negative bacteria and anaerobes predominantly, especially when the contaminating source is colonic.

The organisms isolated from the peritoneum also vary with the source of the initial process and the normal flora at that site. The normal flora of the stomach comprises the same organisms found in the oropharynx but in lower numbers. Thus, the bacterial burden in a ruptured ulcer is negligible compared with that in a ruptured appendix. The normal flora of the colon below the ligament of Treitz contains 10^{11} anaerobic organisms per gram of feces but only10^{8} aerobes per gram; therefore, anaerobic species account for 99.9% of the bacteria. Leakage of colonic contents (pH 7–8) does not cause significant chemical peritonitis, but infection is intense because of the heavy bacterial load.[39]

Factors favouring localization of peritonitis[41]

1.Anatomical:

Transverse colon and Transverse mesocolon deters the spread of infection from supracolic to infracolic compartment of peritoneal cavity. When supracolic region overflows, as is often the case when a peptic ulcer perforates, it does over the colon into the infracolic compartment or by the right paracolic gutter to the right iliac fossa and hence to the pelvis.

2.Pathological :

The clinical course is determined in part by the manner in which adhesions form around the affected organ. Inflamed peritoneum loses its glistening appearance and becomes red and velvety. Flakes of fibrin appear and cause loops of intestine to become adherent to one another and to the parities. There is an outpouring of inflammatory exudates rich in leukocytes and plasma proteins that will becomes turbid, if localization occurs, the turbid fluid becomes frank pus. Peristalsis is retarded in affected bowel and this helps to prevent distribution of the infection. The greater omentum by enveloping and becoming adherent to inflamed structures often forms a substantial barrier to the spread of infection.

Factors favoring diffuse generalized peritonitis[41]

- Speed of peritoneal contamination is prime factor. If an inflamed appendix or hollow viscous perforates before localization has taken place, there will be an efflux of contents into the peritoneal cavity.
- Stimulation of peristalsis by the ingestion of food or even water, hinders localization. Violent peristalsis occasioned by the administration of a purgative or an enema may cause the widespread distribution of an infection that would otherwise have remained localized.
- The virulence of the infecting organism may be so great as to render the localization of infection difficult or impossible.

- Smaller size of omentum in young children makes them vulnerable for infection.
- Disruption of localized collections may occur with injudicious and rough handling, e.g. appendicular mass or pericolic abscess.
- Deficient natural resistance ('immunodeficiency') may result from drugs (e.g. steroids), disease (e.g. AIDS) or old age.

Sequelae leading to multiorgan failure[45]

Sepsis is the major risk factor in the development of multiorgan failure syndrome (MOFS). MOFS increases with severity and duration of shock. Injury to micro vascular system especially microvascular endothelium, is common to ischaemia reperfusion injury and multiorgan failure syndrome. Toxic neutrophil products like proteases, elastase, collagenase, cathepsin G are bactericidal and during endothelial cell injury, produce free oxygen radicals which causes endothelial activation and injury directly through both membrane peroxidation and increased neutrophil adherence in chemotaxis. Miles and Burke suggested decisive period for bacterial infection. This period refers to the time required for bacterial numbers in fluid or tissue to exceed 10^5 / mm3 or (per gm. of tissue) and to establish an infection. Infection must be dealt with before bacterial numbers reach these levels.

ETIOLOGY

Causes of peritoneal inflammation.[46]

TABLE 2: Causes of peritoneal inflammation

Bacterial	gastrointestinal and non gastrointestinal
Chemical	bile, barium
Allergic	starch peritonitis
Traumatic	operative handling
Ischemic	strangulated bowel, vascular occlusion
Miscellaneous	Familial Mediterranean fever

Table 3: CLASSIFICATION OF INTRAABDOMINAL INFECTIONS[46]

1	Primary peritonitis Diffuse bacterial peritonitis in the absence of disruption of intraabdominal organs	A. Spontaneous peritonitis in children
		B. Spontaneous peritonitis in adults
		C. Peritonitis in CAPD
		D. Tuberculous/granulomatous peritonitis
2	Secondary peritonitis Localized (abscess) or diffuse peritonitis	A. Acute perforative peritonitis 1. Gastrointestinal perforation

	originating from a defect in abdominal viscus	2. Intestinal ischemia 3. Pelvic peritonitis and other forms
		B. Postoperative peritonitis 1. Anastomotic leak 2. Accidental perforation and Devascularization
		C. Post-traumatic peritonitis 1. After blunt abdominal trauma 2. After penetrating abdominal Trauma
3	Tertiary peritonitis Peritonitis like syndrome occurring late due to disturbance in the host's immune response	A. Peritonitis without evidence for Pathogens
		B. Peritonitis with fungi
		C. Peritonitis with low-grade virus

Table 4: Aetiology of peritonitis[46]

Acute peritonitis	Chronic (sclerosing) peritonitis
• **Primary (spontaneous)** • **Secondary** • **Acute suppurative** • **Granulomatous** • **Chemical (aseptic)** • **Interventional** • **Traumatic** • **Drug-induced**	• Infectious • Drug-induced • Chemical • Foreign-body • Carcinomatous

Peritonitis:

Peritonitis is simply defined as inflammation of the peritoneum which may be localized or generalised.[41]

Stages of peritonitis:

Stage 1: Stage of peritonism: This stage involves irritation of the peritoneum due to leakage of gastric juice into the peritoneal cavity(chemical peritonitis) which usually lasts for about six hours. On examination there might be a slight variation in the pulse, respiration and temperature. Tenderness and muscle guarding are constantly present over the site of perforation. Great importance should be given to diagnose this condition at this stage as chances of survival of the patient gradually declines with passage of time.

Stage 2:Stage of reaction

The irritant fluid becomes diluted with the peritoneal exudates. Symptoms are relieved but signs of peritoneal reaction should be looked for. Muscular rigidity continues to be present. The other two features are obliteration of liver dullness and shifting dullness. Rectal examination may elicit tenderness in the recto-vesical or rectouterine pouch. Erect x-ray of the abdomen will show air under the diaphragm in 70% of the cases.

Stage 3: Stage of diffuse peritonitis

The pinched and anxious face, sunken eyes and hollow cheeks- the so called Hippocratic facies, with raising pulse rate which is low in volume and tension, persistent vomiting, board like rigidity of the abdomen, increasing distention of the abdomen all give hint to the diagnosis of this condition and imminent death.

PRIMARY (SPONTANEOUS) BACTERIAL PERITONITIS:[39](PBP)

In adults, primary bacterial peritonitis (PBP) is most commonly seen in hepatic failure patients secondary to cirrhosis of liver. However, the disease has been reported in adults with metastatic malignant disease, post-necrotic cirrhosis, chronic active hepatitis, acute viral hepatitis, congestive heart failure, systemic lupus erythematosus and lymphedema as well as in patients with no underlying disease. Although PBP virtually always develops in patients with preexisting ascites,in general it is an uncommon event, occurring in 10% of cirrhotic patients. The cause of PBP has not been established definitively but is believed to involve haematogenous spread of organisms in a patient in whom a diseased liver and altered portal circulation result in a defect in the usual filtration function. Organisms multiply in ascites, a good medium for growth.

The proteins of the complement cascade have been found in peritoneal fluid, with lower levels in cirrhotic patients than in patients with ascites of other etiologies. The opsonic and phagocytic properties of PMNs are diminished in patients with advanced liver disease.[39]

SECONDARY PERITONITIS:

Secondary peritonitis develops when bacteria contaminate the peritoneum as a result of spillage from an intraabdominal viscus. Secondary peritonitis can result primarily from chemical irritation and/or bacterial contamination. For example, as long as the patient is not achlorhydric, a ruptured gastric ulcer will release low-pH gastric contents that will serve as a chemical irritant.[39]

Secondary peritonitis due to hollow viscus perforation

Perforative peritonitis is the most common surgical emergency in India.

Despite advances in surgical techniques, antimicrobial therapy and intensive care support, the management of peritonitis continues to be highly demanding and complex[47].

Peritonitis is inflammation of the peritoneum and peritoneal cavity and is most commonly due to a localized or generalized infection. Primary peritonitis results from bacterial, chlamydial, fungal or mycobacterial infection in the absence of perforation of the gastrointestinal tract, whereas secondary peritonitis occurs in the setting of gastrointestinal perforation. Frequent causes of secondary bacterial peritonitis include peptic ulcer disease, acute appendicitis, colonic diverticulitis and pelvic inflammatory disease[48].

Perforations due to peptic ulcer disease were a common entity and a major cause of morbidity and mortality until the latter half of the 20th century. The incidence has fallen in parallel with the general decline in the prevalence of peptic ulcer disease.

Duodenal ulcer perforations are 2-3 times more common than gastric perforations and about one third of gastric perforations are due to gastric carcinomas. The overall mortality rate is relatively high (20-40%), largely because of complications such as septicaemia and multi organ failure syndrome.[49]

Peptic ulcer perforation

The peptic ulcer perforation is one of the most common surgical emergencies after acute appendicitis and acute intestinal obstruction. There is a decline in the incidence of peptic ulcers and the elective surgeries for the same, which is attributed to the era of H2 blockers and proton pump inhibitors. But the incidence of emergency surgeries, hospitalization and mortality for the perforated peptic ulcer in general has remained stable through the last two decades, probably due to increased inadvertent use of NSAIDS, corticosteroids and irregular use of H2 antagonists.

Approximately 98-99% of peptic ulcers occur in the first portion of duodenum or in the stomach.[48]

Perforation of peptic ulcer may be classified as acute perforation, sub-acute perforation, chronic perforation, perforation associated with haemorrhage, perforation of intra thoracic gastric ulceration and pseudo perforation.

Perforation is the second most common complication of peptic ulcer. Surgery is almost always indicated, although occasionally nonsurgical treatment can be used in a stable patient without peritonitis in whom radiologic studies document a sealed perforation. Patients with acute perforation and GI blood loss (either chronic or acute) should be suspected of having a second ulcer.[50] The options for surgical treatment of perforated duodenal ulcer are simple patch closure, patch closure and highly selective vagotomy(HSV) or patch closure and truncal vagotomy and drainage(TV+D).

Simple patch closure alone should be done in patients with hemodynamic instability and/or exudative peritonitis signifying a perforation >24 hours old. In all other patients, the addition of HSV may be considered because studies have reported a negligible mortality with this approach. Perforated gastric ulcer results in a higher mortality rate than perforated duodenal ulcer (10 to 40%) due to the advanced age of the patients, increased medical comorbidities, delay in seeking medical attention and the larger size of gastric ulcers. In the stable patient without multiple operative risk factors, perforated gastric ulcers are best treated by distal gastric resection. Vagotomy is usually added for type II and III gastric ulcers. Patch closure with biopsy or local excision and closure or biopsy, closure, truncal vagotomy, and drainage are alternative operations in the unstable or high-risk patient or in the patient with a perforation in an inopportune location (e.g., juxta-pyloric). All perforated gastric ulcers, even those in the pre-pyloric position, should be biopsied if they are not removed at surgery.

Perforated Appendicitis[51]

Appendicular inflammation may progress to necrosis and ultimately to perforation. Perforation can develop more rapidly. When acute appendicitis has progressed to appendicular perforation, other symptoms may be present. Patients will often complain of two or more days of severe abdominal pain, usually localizing to the right lower quadrant if the perforation has been walled off by surrounding intra-abdominal structures. It may be diffuse if generalized peritonitis ensues often with rigors and high fever to up to 102°F (38.9°C) or above. A history of poor oral intake and dehydration may also be present.

Most patients with perforated appendicitis present with symptoms related to the inflamed appendix itself or to a localized intraperitoneal abscess from perforation.

Abscess can be seen in the retroperitoneal region due to perforation of a retrocecal appendix or in the liver from hematogenous spread of infection through the portal vein. An intraperitoneal abscess could fistulize to the skin, resulting in anenterocutaneous fistula. Pylephlebitis (septic portal vein thrombosis) presents with high fevers and jaundice and can be confused with cholangitis; it is a dreaded complication of acute appendicitis and carries a high mortality.

Small Bowel Perforation[50]

Today, iatrogenic injury incurred during GI endoscopy is the most common cause of small bowel perforation. Other etiologies of small bowel perforation include infections (tuberculosis, typhoid), Crohn's disease, ischemia, drugs (e.g., potassium and NSAID-induced ulcers), radiation-induced injury, Meckel's and acquired diverticula, neoplasms (lymphoma, adenocarcinoma and melanoma)etc.

Among iatrogenic injuries, duodenal perforation during ERCP with endoscopic sphincterotomy is the most common. This complication occurs in 0.3 to 2% of cases. Patients who have undergone Billroth II gastrectomy are at increased risk of duodenal perforations as well as free jejunal perforations during ERCP.[50]

CT scan is the most sensitive test for diagnosing duodenal perforations. Positive findings include pneumoperitoneum for free perforations, retroperitoneal air, contrast extravasation and paraduodenal fluid collections. Intraperitoneal duodenal perforations require surgical repair with pyloric exclusion and gastrojejunostomy or a tube duodenostomy. Perforation of the jejunum and ileum require surgical repair or segmental resection.

Typhoid Enteritis[48]

Typhoid fever remains a significant problem in developing countries, most commonly in areas with contaminated water supplies and inadequate waste disposal. Children and young adults are most often affected.

Typhoid enteritis is an acute systemic infection caused primarily by Salmonella typhi. The pathological events are initiated in the intestinal tract after oral ingestion of the typhoid bacillus. These organisms penetrate the small bowel mucosa, making their way rapidly to the lymphatics and then systemically. Hyperplasia of the reticuloendothelial system, including lymph nodes, liver and spleen occurs. Peyer's patches in the small bowel become hyperplastic and may subsequently ulcerate with complications of hemorrhage or perforation.

The diagnosis of typhoid fever is by isolating the organism from blood (positive in 90% of the patients during the first week of the illness), bone marrow and stool cultures. High titres of agglutinins against the O and H antigens are strongly suggestive of typhoid fever.

Complications requiring potential surgical intervention include hemorrhage and perforation. The incidence of hemorrhage was reported to be as high as 20%. Intestinal perforation through an ulcerated peyer's patch occurs in about 2% of cases. Typically, it is a single perforation in the terminal ileum and simple closure of the perforation is the treatment of choice. With multiple perforations, which occur in about one fourth of the patients, resection with primary anastomosis or exteriorization of the intestinal loops may be required depending on the intraperitoneal contamination.

Tubercular perforation:[52]

Tubercular perforation is seen mainly in ulcerative type of tuberculosis. Ulcerative tuberculosis is secondary to swallowed tubercle bacilli. Multiple ulcers, lying transversely, develop in the terminal ileum. Serosa is thickened, reddened and covered with tubercles.

Colonic perforation:

The common causes of colonic perforation include

1. Diverticular disease
2. Ischemia: The most common cause of colonic ischemia is due to thrombosis of the inferior mesenteric artery, but in some cases, no specific cause for the ischemia is identified.
3. Abdominal trauma
4. Iatrogenic: Perforation after vascular, urologic, gastrointestinal or gynecologic surgery is the most frequent iatrogenic cause. The incidence of perforation after colonoscopy has been reported to range from 0.03% to 0.65% for diagnostic screening and from 0.073% to 2.1% for therapeutic endoscopies.
5. Crohn's disease and ulcerative colitis.[53]
6. Tumor-Related Perforation: Colonic perforation secondary to a tumor occurs in two different settings. Either a transmural tumor perforates itself or the proximal colon becomes over distended, particularly in case of a competent ileocecal valve. Both conditions may result in diffuse fecal peritonitis with significant morbidity and mortality. In addition, the tumor perforation results in spillage of tumor cells and thus has to be considered as a stage IV tumor[54].

CLINICAL PRESENTATION[55,56]

Clinical features are usually of sudden onset, followed by a distinct intermediate latent interval, which in turn gives place to classical signs and symptoms.

Symptoms of early peritonitis:

- **Pain**

It is the most important and constant finding in patients with acute abdomen. It varies considerably in intensity. It is as a rule that it is most intense in that part of the abdominal wall which lies immediately over the spreading edge of the peritoneal inflammation. When peritoneal inflammation subsides or localizes, pain diminishes in severity and becomes limited to one area of the abdomen.

Pain makes the patient seek medical assistance. The characteristics of pain like the onset, site, type and radiation aids in the diagnosis.

Sudden onset of pain is feature of all perforative peritonitis. In acute appendicitis, diminution of pain may indicate perforation of an obstructive gangrenous appendix. Constant burning pain is a feature of peritonitis and is often seen in perforated peptic ulcer. Sudden pain due to perforation of peptic ulcer usually takes place in the afternoon after a meal.

Since movement aggravates the pain, patient assumes a still posture. Deep inspiration will aggravate pain due to diaphragmatic irritation. A past history of periodic pain is suggestive of peptic ulcer perforation and crampy lower abdominal pain is a feature of tuberculous enteritis, ulcerative colitis and crohn's disease.

- **Vomiting**

Initially vomiting episodes may be less, but as the peritonitis advances, it becomes persistent. Often pain precedes vomiting. Initially the vomitus consists of gastric contents, later it is bile

stained and when the obstruction becomes complete it becomes feculent. Vomitus may rarely contain frank blood in cases of perforation due to gastric ulcer, duodenal ulcer and gastric neoplasm. In early stages of peritonitis vomiting is reflex in origin. Later it is caused by paralytic ileus.

- **Fever**

The temperature is often sub-normal or normal in cases in which onset is sudden.

It tends to rise gradually as true peritonitis supervenes. A rising pulse rate and falling temperature are of the greatest significance. As the disease process advances, the pulse steadily rises and will be bounding. Later it becomes weak and more rapid.

- **Distension of the abdomen**: It may be seen in the later stages where paralytic ileus has already set in and there is peritoneal fluid collection. The distension may be in the upper or lower abdomen in early stages but will be all over the abdomen in late stages. The distension of the abdomen is due to ensuing paralytic ileus and peritoneal fluid collection.

- **Bowel habits:** Absolute constipation is a constant feature of peritonitis. In the early stages, there may be a history of loose stools because of irritation of rectum by pelvic collections. Past history of alternate constipation and diarrhoea are features of tubercular enteritis, carcinoma colon and worm infestation. In cases of ulcerative colitis there will be abrupt explosive severe diarrhoea with bleeding but in crohn's disease most patients have diarrhoea that is usually not bloody. A history of melena will give clue to the diagnosis of peptic ulcer perforation or carcinoma stomach.

- **Other history**: Includes history of drugs particularly NSAIDs and steroids or strong acids ingestion. There may be history of loss of appetite, loss of weight and jaundice in cases of carcinoma with metastasis.

Signs of early peritonitis:

- **Inspection**: The position of the patient in the bed is often characteristic. Patient lies still with the legs drawn up in an effort to relieve tension on the abdominal muscles. There is absence or marked diminution of abdominal respiratory movements. Respiration is shallow, rapid and thoraco-abdominal in nature. Patient may look toxic and dehydrated.
- **Palpation**: Marked abdominal tenderness and guarding will be present. Rigidity may be present in the later stages. Rebound tenderness can be elicited. It may be localized, as in some early cases in which the peritoneal inflammation has involved only a limited area or it may be generalized when the diffusion is extensive.
- **Percussion**: The abdomen is resonant and tympanic because the intestines are filled with gas. Liver dullness is often obliterated due to pneumoperitoneum.
- **Auscultation**: Bowel sounds are diminished or absent due to associated ileus.

INVESTIGATIONS

- **Blood Studies**[57,58,59]

A complete blood count showing Hb%, Haematocrit and WBC counts taken on admission are highly informative. Only a rising or marked leucocytosis especially with the presence of a shift to the left on blood smear is indicative of serious infection. A low white cell count is feature of viral infection such as mesenteric adenitis or gastroenteritis. Serum electrolytes, urea and creatinine are important especially if hypovolemia is expected. ABG should be obtained in patients with hypotension, peritonitis, pancreatitis, ischaemic bowel and septicaemia as unsuspected metabolic acidosis may be the first clue to serious disease.

A raised serum amylase level corroborates a clinical diagnosis of acute pancreatitis. Clotting studies should be done if history is suggestive of a haematological disorder. Recently, acute inflammatory markers like C - reactive protein, Interleukins, Ceruloplasmin and Transferrin are being tested to assess the severity of the infection.

- **Urine Tests**

Dark urine reflects dehydration. Urine ketone bodies may be present in a patient with uncontrolled diabetes mellitus. Routine urine examination can help in assessing any urinary tract infection.

Imaging:

1. **Radiography**[60]

Erect chest radiograph or erect abdomen radiograph:

The presence of free, intra-abdominal gas almost always indicates perforation of a viscus. Free gas can be identified on the erect chest radiograph. As little as 1 ml of free gas can be demonstrated radiographically, on either an erect chest or a left lateral decubitus abdominal radiograph. Small amounts of gas are detectable under the right hemi-diaphragm on erect radiographs, but on the left it can be difficult to distinguish free gas from stomach and colonic gas. There are many circumstances when interpretation of an erect chest radiograph is difficult. There are some situations when the radiologist or clinician may be fooled into thinking that there is a perforation (pseudo-pneumoperitoneum). A lateral decubitus radiograph can resolve the problem by demonstrating gas between the liver and the abdominal wall.

Supine radiograph:

It is also important to be able to recognize the signs of pneumoperitoneum on supine radiographs. In many patients, particularly those who are unconscious, have suffered trauma, are old or are critically ill, perforation may be clinically silent as it is over-shadowed by other serious medical or surgical problems. A supine abdominal radiograph examination may be the only radiograph that can be obtained in these cases. Almost half the patients will have gas in the right upper quadrant adjacent to the liver, lying mainly in the subhepatic space and the hepatorenal fossa (Morrison's pouch). Visualization of both the outer and inner walls of a bowel loop is known as Rigler's sign . The bowel loops then take on a 'ghost-like' appearance. This sign can be misleading if several loops of bowel lie close together. The falciform or umbilical ligaments may be demonstrated by free gas lying on either side. Air can be seen in the fissure for the ligamentum teres.

Signs of a pneumoperitoneum on supine radiograph:

1. Right upper-quadrant gas
    - Perihepatic
    - Subhepatic
    - Morrison's pouch
    - Fissure for ligamentum teres
2. Rigler's[double wall] sign
3. Ligament visualization
    - Falciform [ligamentum teres]
    - Umbilical[inverted V sign] medial and lateral

4. Urachus
5. Triangular air
6. Foot ball or air dome sign
7. Scrotal air [in children]

Conditions simulating a pneumoperitoneum [pseudo-pneumoperitoneum]

- Intestine between liver and diaphragm- Chiladiti's syndrome
- Subphrenic abscess
- Curvilinear Atelectasis in the lung
- Subdiaphragmatic fat
- Diaphragmatic irregularity
- Cysts in pneumatosis intestinalis

Causes of pneumoperitoneum without peritonitis

(i) Silent perforation of viscus that has sealed itself, in:

- Elderly patients
- Patients on steroids
- Unconscious patients
- Patients being ventilated
- Serious medical conditions

(ii) Post operative

- Peritoneal dialysis
- Perforated jejuna diverticulosis
- Perforated cyst in pneumatosis intestinalis

- Tracking down from a pneumomediastinum
- Stercoral ulceration
- Entry of air through the fallopian tubes

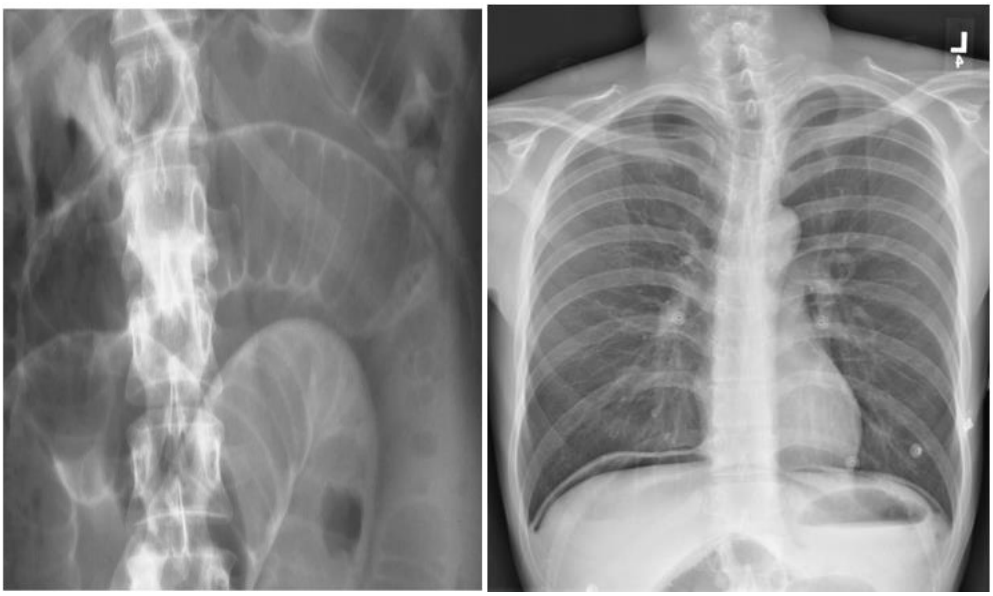


Figure 18: Radiography: Erect chest radiograph or erect abdomen radiograph. Rigler's sign of pneumoperitoneum. The bowel loops have a 'ghost-like' appearance due to gas both inside and outside making the wall more apparent. Air under

❖ **Ultra sound(US) scanning:**

Ultra sound scanning has undoubted value in certain situations such as pelvic peritonitis in females and localized right upper quadrant peritonism.

US plays a role in confirming or excluding specific diagnoses (e.g. subphrenic abscess). The diagnostic accuracy of these modalities has also been affirmed in clinically equivocal cases of acute appendicitis.[43]

- **Computed tomography:**

Discontinuity of the bowel wall may indicate the perforation site. Focal wall thickening may be associated with the perforation of the alimentary tract. This may occur in peptic ulcer disease, trauma, foreign body, iatrogenic event, ischemia, inflammation, appendicitis, diverticulitis and neoplasm. Accurate evaluation of bowel wall thickening can only be performed on the distended bowel loop.[61]

Bowel wall thickening:

> 8 mm in stomach and duodenum,

> 3 mm in jejunum and ileum,

> 6 mm of the appendiceal calibre and

> 5 mm in colon and rectum including soft tissue mass

Upright chest films can detect pneumoperitoneum in only 30% of cases but abdominal CT can demonstrate free air in 100% of cases[61].

CT displays intra and extra-peritoneal free air in amounts too small to be visualized on plain radiography, but it can also recognize the underlying cause and specify the location of the disease.

To assess the distribution of free air, the peritoneal cavity is divided into two-compartments, the supra-mesocolic compartment and the infra-mesocolic compartment, based on the level of transverse mesocolon. In supra-mesocolic compartment, when there was free air in the periportal area, it was defined as periportal free air (PPFA) and the sign was positive.

The “ligamentum teres sign” which is free air confined to the intra-hepatic fissure for ligamentum teres can be seen in the perforation of the duodenal bulb or stomach.[56]

The “falciform ligament sign” is that free air or air-fluid level crossing the midline and accentuating the falciform ligament can be seen more in the perforation of the proximal (stomach, duodenum, jejunum and ileum) GI tract perforation.[62]

When there is free air in the periportal area, it suggests a high probability of perforation in the upper GI tract.

The PPFA sign was the most significant finding in distinguishing upper from lower GI tract perforation. When there is free air in the periportal area, it suggests a high probability of perforation in the upper GI tract.[62]

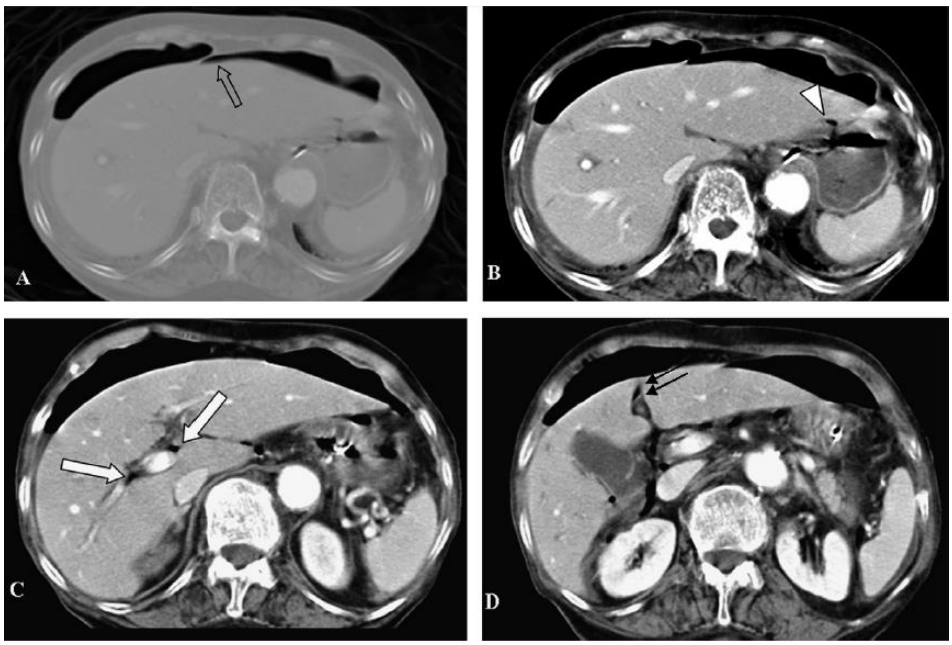


Figure 19: Computed tomography

(A) Contrast-enhanced abdominal CT scan shows that the falciform ligament sign (open arrow) is well demonstrated on the wide-window setting. (B) CT scan shows a mural defect in the upper body of the stomach (arrowhead). (C) CT scan shows the periportal free air sign (arrows). (D) Free air is noted in the fissure for ligamentum teres (double arrows).

❖ DIFFERENTIAL DIAGNOSIS

These can be divided into

1. Intra-abdominal conditions

2. Intra thoracic diseases

3. Metabolic or neurologic conditions

Intra-abdominal conditions

- Acute Appendicitis
- Acute pancreatitis
- Acute cholecystitis
- Acute intestinal obstruction
- Mesenteric ischemia / ruptured aneurysm
- Ruptured ectopic gestation
- Perforated diverticulitis and
- Peritonitis following trauma

Intra-thoracic diseases

- Myocardial infarction, acute pericarditis
- Pneumonia, pleurisy, spontaneous pneumothorax
- Rupture of the esophagus due to emetic abuse

Metabolic and neurologic conditions

- Acute porphyrias, diabetes, uremia, hyperlipidemia, acute poisoning
- Meningitis, multiple sclerosis and neuro-syphilis.

COMPLICATIONS OF PERITONITIS[55]

a) Systemic complications

- Septicemic / endotoxic shock
- Bronchopneumonia / Respiratory failure
- Renal failure
- Bone marrow suppression
- Multisystem failure
- Death

b) Local complications

- Intestinal obstruction
- Paralytic ileus
- Residual or recurrent abscesses - Subphrenic / Paracolic / Pelvic
- Wound infection / Wound dehiscence
- Portal pyaemia.

Treatment

Treatment consists of:

I. **General care of the patient;**

II. **Specific treatment for the cause;**

I. General care of the patient[35]

(i) Correction of circulating volume and electrolyte imbalance.

Patients are frequently hypovolaemic with electrolyte disturbances. The plasma volume must be restored and plasma electrolyte concentrations corrected. Central venous catheterization and pressure monitoring may be helpful in correcting fluid and electrolyte balance particularly in patients with concurrent disease. Plasma protein depletion may also need correction as the inflamed peritoneum leaks large amounts of protein. If the patient's recovery is delayed for more than 7—10 days, intravenous nutrition (total parenteral nutrition) will be required.

(ii) Gastrointestinal decompression.

A nasogastric tube is passed into the stomach and aspirated. Intermittent aspiration is maintained until the paralytic ileus resulting from peritonitis has recovered. Measured volumes of water are allowed by mouth when only small amounts are being aspirated. If the abdomen is soft and not tender, and bowel sounds return, oral feeding may be progressively introduced after removing the nasogastric tube. It is important not to prolong the ileus by missing this stage.

(iii) Antibiotic therapy.

Administration of antibiotics prevents the multiplication of bacteria and the release of endotoxins. As the infection is usually a mixed type, initially parenteral broad-spectrum antibiotics active against aerobic and anaerobic bacteria must be given.

(iv) **A fluid balance chart**

This must be initiated so that daily output by gastric aspiration and urine is known Foley's catheterization for calculating urine output. Additional losses from the lungs, skin and in faeces are estimated, so that the intake requirements can be calculated and administered. Throughout recovery, the haematocrit, serum electrolytes and urea must be checked regularly.

(v) **Analgesia.**

The patient should be nursed in the sitting-up position and must be relieved of pain before and after surgery. Once the diagnosis has been made morphine may be given. If appropriate expertise is available epidural infusion may provide excellent analgesia. Relief from pain allows early mobilization and adequate physiotherapy in the postoperative period which help to prevent basal pulmonary collapse, pneumonia, deep-vein thrombosis and pulmonary embolism.

(vi) **Vital system support.**

Especially if septic shock is present, special measures may be needed for cardiac, pulmonary and renal support. Administration of oxygen postoperatively can help to prevent and mitigate the effects of septic shock, especially acute respiratory distress syndrome (ARDS) which may require a period of mechanical ventilation. If oliguria persists despite adequate fluid replacement, both diuretics and inotropic agents such as dopamine may be needed.

II. Specific treatment of the cause

If the cause of peritonitis is amenable to surgery, such as in perforated appendicitis, diverticulitis, peptic ulcer, gangrenous cholecystitis or in rare cases of perforation of the small bowel, surgery must be carried out as soon as the patient is fit for the procedure. Peritoneal lavage has to be

done with normal saline / normal saline mixed with betadine. Following surgery, tube drains have to be fixed to drain the dependant areas like subhepatic space, pelvis or the paracolic gutters. Breathing exercises or the chest physiotherapy to prevent post operative chest infections. Precautions to prevent general postoperative complications like urinary tract infections, deep vein thrombosis and pulmonary infections have to be followed.

1. **Perforated peptic ulcer**:

In general, the incidence of emergency surgery, hospital admission and mortality for perforated peptic ulcer have remained stable through the last two decades.

In older patients, admission rates for duodenal ulcer perforation have increased and gastric ulcer perforation has decreased in the last decade. Duodenal perforation currently accounts for approximately 75% of peptic ulcer perforation.[63]

Initially, there was concern that simple closure should be reserved for those patients with advanced peritonitis in whom definitive treatment by vagotomy was not advised. The importance of vagotomy has been questioned for more than a decade in the era of superb medical control of acid production and treatment of H pylori.Most surgeons in a recent survey of fellows of the Association of Surgeons of Great Britain and Ireland indicated they no longer perform vagotomy, even in early perforation and good-risk patients.So a repair of perforation by simple closure is readily supported as a definitive surgical care.[64]

- **Duodenal perforation:**

Simple closure is usually the quickest and most appropriate method of dealing with a perforated duodenal ulcer.

Modified graham patch closure for duodenal perforation:[65]

Closure is achieved by the insertion of three or four interrupted, absorbable sutures. Generous bites, which pass through the entire thickness of the gut wall, should be taken. Care must be taken to ensure that they do not catch the posterior wall. Sutures should be inserted in long axis of the gut to avoid narrowing. The closure is then reinforced with an omental onlay patch.

If duodenal induration or edema precludes closure of the defect, then use of a jejunal serosal patch can be helpful. In the unusual circumstances of a large ulcer and significant inflammation, duodenal drainage and pyloric exclusion as described for use in the treatment of traumatic duodenal injuries can be helpful.

A combination of gastrostomy, duodenostomy and jejunostomy tubes would be indicated. Alternatively, a lateral duodenal fistula can be prevented by a Roux-en-Y jejunal "patch" sutured over the defect with a trans-jejunal drain that extends from the duodenum through the jejunal "patch" and exits via a Witzel closure several centimetres downstream in the jejunal limb.[63]

- **Gastric perforation:**

Surgical options include simple closure with absorbable sutures, closure with omental patch, gastrectomy with either Billroth I or II reconstruction and pyloroplasty. Most perforated gastric ulcers are prepyloric. Prepyloric and pyloric ulcers are best treated with distal gastric resection because this avoids the 15% incidence of postoperative gastric obstruction seen with simple closure and also allows histological assessment. If a gastric ulcer is difficult to include in a resection, generous biopsies should be taken to exclude malignancy and the ulcer is closed or patched primarily with omentum.[63]

Laparoscopic and Endoscopic Management of Perforated Duodenal Ulcers:

Laparoscopic closure of perforated duodenal ulcer is a simple and safe procedure. While initial reports of laparoscopic closure of perforated duodenal ulcer demonstrated little difference in comparison with open duodenal ulcer closure, recent data demonstrate that the approach is safer and maintains the benefits of the minimally invasive approach. Specifically, laparoscopic closure of perforated duodenal ulcers has been associated with shorter operating time, less postoperative pain, a shorter postoperative hospital stay and earlier return to normal daily activities than the conventional open repair.[65]

Laparoscopic and endoscopic procedure:

The supra-umbilical port (10 mm) is the camera port. The second port is 5 mm and is just to the right of midline. This port was used for needle and suture. The third port (5 mm) is used for the clamp, dissector and instrument for retrieving the needle and for the suction irrigator. This port will be in the mid-clavicular line. The fourth port (5 mm) is for needle holder and scissor and the position is two fingerbreadths above the umbilicus on the left in the mid-clavicular line. After repair, extensive saline lavage of the abdominal cavity followed by inspection of all quadrants for purulence. Drains are not routinely used. Omentoplasty will be done. Omental plug will be pulled through the ulcer by the endoscope.[64]

Use of the ligamentum teres hepatics or falciform ligament has been described as an alternative to the use of the omentum as a patch. Endoscopic repair with an omental patch would be suitable mainly for perforations on the anterior wall of the stomach. Omentum can also be found in relation to a perforation on the posterior wall, but the procedure may be much more difficult to perform and would therefore not be recommended. In such cases an alternative approach might

be to clip the soft adjacent structures directly to the gastric wall to completely close the perforation.[65]

2. **Jejunal perforation:**

   Primary closure

   Resection and end to end anastomosis.

3. **Ileal perforation:**

   Primary closure

   Wedge resection and closure

   Resection of segment of ileum and anastomosis

   Right hemicolectomy in case of involvement of ileocecal junction.

4. **Appendicular perforation:**

   Emergency laparotomy and appendectomy with irrigation and drainage of the peritoneal cavity is advised.

   Ileocecectomy may be necessary if the inflammation extends to the wall of the caecum. If an abscess cavity is noted on imaging, then image guided drainage can be performed percutaneously or transrectally. Interval appendicectomy can be performed 6 weeks after non operative management.

5. **Colonic perforation:**

Treatment option depends on the etiology.

- Simple suture of the perforation should only be performed after an iatrogenic injury, when the condition of the intestinal wall allows.
- In all other situations primary resection of the septic focus is regarded as the safest approach.

- Tumor-related perforation: Surgical management is indicated in every case and requires not only addressing the site of colonic perforation but also removing the tumor in an oncologically correct fashion[54].

Role of Antimicrobials in Peritonitis:

Antimicrobial trails in peritonitis have revealed that despite marked differences in the antibacterial spectra; substantial differences in the treatment results have been documented. Overall success rate was 84% for Aminoglycoside + Clindamycin, 89% for Aminoglycoside + Metronidazole and 93% for Cephalosporin regimens.[66]

A surgical infection society policy statement on anti infective agents for intra abdominal infections was provided by Bohnen J. M.A and Colleagues.[67] Lau W.Y. and colleagues did a randomized prospective trail on prophylaxis of post appendicectomy sepsis and concluded the Metronidazole and third generation cephalosporin in combination with either Gentamicin/Amikacin to be superior.[68]

Ceftriaxone

Ceftriaxone is a broad-spectrum third generation cephalosporin antibiotic.

The chemical formula of Ceftriaxone sodium is $C_{18}H_{16}N_8Na_2O_7S_3 \cdot 3.5H_2O$.

The bactericidal activity of ceftriaxone results from inhibition of cell wall synthesis. Ceftriaxone is highly stable in the presence of beta-lactamases, both penicillinases and cephalosporinases, of gram-negative and gram-positive bacteria.[69]

Ceftriaxone is active against most strains of the following microorganisms,

Aerobic gram-negative microorganisms:

Acinetobacter calcoaceticus

Enterobacter aerogenes

Escherichia coli

Haemophilus influenzae

Klebsiella pneumoniae

Moraxella catarrhalis

Neisseria gonorrhoeae

Neisseria meningitidis

Proteus vulgaris

Serratia marcescens

Ceftriaxone is also active against many strains of *Pseudomonas aeruginosa.*

Aerobic gram-positive microorganisms:

Staphylococcus aureus

Streptococcus pneumoniae

Streptococcus pyogenes

Viridans group streptococci

Anaerobic microorganisms:

Bacteroides fragilis

Clostridium species

Aerobic gram-negative microorganisms:

Citrobacter diversus

Salmonella and Shigella species

Aerobic gram-positive microorganisms:

Streptococcus agalactiae

Anaerobic microorganisms:

Bacteroides species

Ceftriaxone is indicated for the treatment of the following infections:

INTRA-ABDOMINAL INFECTIONS

LOWER RESPIRATORY TRACT INFECTIONS

ACUTE BACTERIAL OTITIS MEDIA

SKIN AND SUBCUTANEOUS INFECTIONS

URINARY TRACT INFECTIONS

UNCOMPLICATED GONORRHEA

PELVIC INFLAMMATORY DISEASE.

BACTERIAL SEPTICEMIA

BONE AND JOINT INFECTIONS.

MENINGITIS

ADVERSE REACTIONS

LOCAL REACTIONS—pain, induration and tenderness was 1% overall.

HYPERSENSITIVITY—rash (1.7%). Less frequently reported (<1%) were pruritus, fever or chills.

HEMATOLOGIC—eosinophilia (6%), thrombocytosis (5.1%) and leukopenia (2.1%).

GASTROINTESTINAL—diarrhea (2.7%). Less frequently reported (<1%) were nausea or vomiting and dysgeusia.

HEPATIC—elevations of SGOT (3.1%) or SGPT (3.3%). Less frequently reported (<1%) were elevations of alkaline phosphatase and bilirubin.

RENAL—elevations of the BUN (1.2%). Less frequently reported (<1%) were elevations of creatinine and the presence of casts in the urine.

CENTRAL NERVOUS SYSTEM—headache or dizziness were reported occasionally (<1%).

GENITOURINARY—moniliasis or vaginitis were reported occasionally (<1%).

MISCELLANEOUS—diaphoresis and flushing were reported occasionally (<1%).

Dosage in ADULTS: The usual adult daily dose is 1 to 2 grams given once a day (or in equally divided doses twice a day) depending on the type and severity of infection. The total daily dose should not exceed 4 grams.

Metronidazole

Metronidazole is an antibacterial drug with chemical formula of the synthetic nitroimidazole antimicrobial, 2-methyl-5-nitro-1H-imidazole-1-ethanol.

The major route of elimination of metronidazole and its metabolites is via the urine (60% to 80% of the dose), with fecal excretion accounting for 6% to 15% of the dose.

Mechanism of action

Metronidazole exerts antibacterial effects in an anaerobic environment by the following possible mechanism: Once metronidazole enters the organism, the drug is reduced by intracellular electron transport proteins. Because of this alteration to the metronidazole molecule, a concentration gradient is created and maintained which promotes the drug's intracellular transport. Presumably, free radicals are formed which, in turn, react with cellular components resulting in death of the bacteria.

Metronidazole has been shown to be active against most isolates of the following bacteria

Gram-positive anaerobes

Clostridium species

Eubacterium species

Peptostreptococcus species

Gram-negative anaerobes

Bacteroides fragilis group

Fusobacterium species

Protozoal parasites

Entamoeba histolytica

Trichomonas vaginalis

Gram-negative anaerobes

Bacteroides fragilis group

Prevotella species

Metronidazole is indicated for the treatment of the following infections when caused by susceptible organisms mentioned above:

INTRA-ABDOMINAL INFECTIONS[70], including peritonitis, intra-abdominal abscess and liver abscess.

SYMPTOMATIC TRICHOMONIASIS.

ASYMPTOMATIC TRICHOMONIASIS.

AMEBIASIS.

ANAEROBIC BACTERIAL INFECTIONS.

GYNECOLOGIC INFECTIONS.

BACTERIAL SEPTICEMIA

CENTRAL NERVOUS SYSTEM (CNS) INFECTIONS

LOWER RESPIRATORY TRACT INFECTIONS.

Adverse Reactions

Central Nervous System: The most serious adverse reactions reported in patients treated with metronidazole have been convulsive seizures, encephalopathy, aseptic meningitis, optic and peripheral neuropathy

Gastrointestinal: The most common adverse reactions reported have been referable to the gastrointestinal tract, particularly nausea, sometimes accompanied by headache, anorexia and occasionally vomiting

Dermatologic: Erythematous rash and pruritus.

Hematopoietic: Reversible neutropenia (leukopenia); rarely, reversible thrombocytopenia.

Cardiovascular: Flattening of the T-wave may be seen in electrocardiographic tracings.

Hypersensitivity: Urticaria, erythematous rash, Stevens-Johnson Syndrome, toxic epidermal necrolysis.

Renal: Dysuria, cystitis, polyuria, incontinence and a sense of pelvic pressure.

AMIKACIN

Amikacin is a semisynthetic aminoglycoside antibiotic derived from Kanamycin. The molecular formula is C22H47N5O21S2.

Amikacin is active against the following organisms:

Gram-negative

Pseudomonas species

Proteus species

Klebsiella pneumoniae

Enterobacter cloacae

Serratia species

Acinetobacter

Providencia stuartii

Citrobacter freundii

Escherichia coli

Gram-positive

Staphylococcus species

(penicillinase and non-penicillinase producing,

including methicillin resistant strains)

Staphylococcus aureus, including methicillin-resistant strains is the principal Gram-positive organismsensitive to amikacin.

Amikacin is effective in treating bacteraemia, septicaemia including neonatal sepsis and serious infections of the respiratory tract, bones and joints, central nervous system, skin and skin structures (including those resulting from burns), intra-abdominal organs, post-operative infections and complicated and recurrent urinary tract infections.[71]

Adverse Reactions

Amikacin induced hepatotoxicity is not a common side effect, however it may occur.

More common reactions

Auditory and Vestibular: Hearing loss (4%) (permanent in some cases)

Hearing loss is usually manifested initially by diminution of

high-tone acuity.

Biochemical abnormalities: Increased serum urea, decreased creatinine clearance,

elevated serum creatinine, azotaemia.

Genitourinary: Reduced renal function, oliguria

Injection site reactions: Pain at site of intramuscular injection (6%).

Less common reactions

Auditory and Vestibular: Tinnitus, vertigo, dizziness, nystagmus, changes in caloric

testing or electronystagmograms.

Biochemical abnormalities: Casts, cells or protein in the urine, eosinophilia, increase in

AST.

Dermatological: Pruritus, rash

Gastrointestinal: Nausea, vomiting

General: Drug fever

Genitourinary: Renal failure

Haematological: Anaemia

Musculoskeletal: Arthralgia

Nervous system: Paraesthesia, tremor

Adults and Children:

The usual recommended dose of Amikacin is 15mg/kg daily given in two or three equally divided doses.

The effectiveness of antimicrobial therapy in reduction in the incidence of peritoneal infection patient has been established in a number of clinical studies [72]. Inflammation of gut perforation peritonitis due to small bowel, appendix, large bowel, imposes high risk of septic complication [73]. Antimicrobial therapy for patients with inflammation or peritonitis is therapeutic rather than prophylactic because antibiotic is administered after contamination has occurred. In the 1940s the use of penicillin was associated with a 30–40% decrease in mortality rates in penetrating abdominal trauma with peritonitis[74].

Subsequent use of broad-spectrum antimicrobial regimen has been associated with reduced morbidity rates for peritonitis in the range of 4–15%[75]. This reduction was due to one of the most exciting & rewarding microbiological observation in 1970 of the role of human anaerobic endogenous micro flora in abdominal infection. Due to polymicrobial nature of the bacterial flora, broad-spectrum antimicrobial coverage has been considered a necessity[76]. Agents that are directed against aerobic gram negative bacilli includes- Aminoglycosides, IInd and IIIrd generation

Cephalosporins, Monobactams, Carbapenems, Carboxy-Penicillin, Acylapenicillin and either Ampicillin or Ticarcillin combined with β -lactamase inhibitor (i.e. Sulbactam & Clavulanic Acid) [77]. In vitro studies of anaerobic susceptibility demonstrates no resistance to Metronidazole & Chloromphenicol, <1% resistance To Imipenem – Cilastin, Ticarcillin, Clavulanate, Ampicillin – Sulbactum And Cafaperazone – Sulbactam. In vitro resistance rate to Cefoxitin and Clindamycin were 8% and 3% respectively.

Empiric use of Combinations of many antibacterial, were also associated with the emergence of resistant organism as well as serious toxicity and spiraling therapy costs. Despite consensus popularity the "shot gun" approach has been shown to be consistently better than broad-spectrum single agent antimicrobial coverage[78].

In vitro, the synergistic activity of third generation cephalosporins and aminoglycoside was particularly evident with members of enterobacteriacae.

Later on a number of prospective studies comparing Gentamicin & Clindamycin *Vs* single agent therapy with III[rd] generation Cephalosporin in patients with complicated appendicitis have noted treatm associated with β. *Fragilis.*[79]

MATERIALS AND METHODS

MATERIALS AND METHODS

This is a prospective clinical study conducted on 140 consecutive patients who presented to the surgical department of R. L. Jalappa Hospital and Research Centre, Tamaka, Kolar with peritonitis secondary to hollow viscus perforation.

The study period was from December 2015 to June 2017. This was a randomized study and all the patients were divided in two groups.

GROUP A: Patients with all odd serial numbers were included in this group and treated with two antimicrobials(Inj Ceftriaxone 1gm IV BD and Inj Metronidazole 500mg IV TID).

GROUP B: Patients with all even serial numbers were included in this group and treated with three antimicrobials(Inj Ceftriaxone 1gm IV BD , Inj Metronidazole 500mg IV TID and Inj Amikacin 500mg IV BD).

METHODS OF COLLECTION OF DATA:

All patients who presented with features of peritionitis secondary to hollow viscus perforation fulfilling the inclusion criteria were included in the study.

Patient's details like history, clinical examination and investigations were documented using a standard proforma designed for the study.

Pre-operative preparation

All the patients were optimised before surgery by correction of shock, electrolyte imbalance and dehydration . Broad spectrum antibiotic coverage(Ceftriaxone and Metronidazole or Ceftriaxone, Metronidazole And Amikacin) were started preoperatively and continued in the post operative period. Tetanus prophylaxis was given at the time of presentation. Two drugs/ three drugs were initiated depending upon which group the patient is included.

Operative details

At laparotomy, peritoneal contaminated fluid was collected for culture and sensitivity and operative findings such as the site of perforation, degree of peritoneal contamination were recorded. After appropriate surgery was done(Appendicectomy, Closure of perforation/ Grahm's omental patch repair, Resection and Anastomosis(R& A)) thorough wash was given to the peritoneal cavity with normal saline.

Post operatively, the patients were followed up on third, fifth, seventh day and subsequently after one month and following parameters were recorded like development of the local and systemic complications and duration of hospital stay in both the groups.

METHOD OF STATISTICAL ANALYSIS;

Data was entered into Microsoft excel data sheet and was analyzed using SPSS 22 version software. Categorical data was represented in the form of Frequencies and proportions. **Chi-square test** was used as test of significance for qualitative data. Continuous data was represented as mean and SD. **Independent t test** was used as test of significance to identify the mean difference between two quantitative variables.

Graphical representation of data: MS Excel and MS word was used to obtain various types of graphs such as bar diagram.

p value (Probability that the result is true) of <0.05 was considered as statistically significant after assuming all the rules of statistical tests.

Statistical software: MS Excel**,** SPSS version 22 **(**IBM SPSS Statistics, Somers NY, USA) was used to analyze data.

SAMPLESIZE

Sample size was estimated based on the difference in proportion of uneventful recovery in group A and group B. By using the formula

$$\text{Sample size} = \frac{r+1}{r}\frac{(p^*)(1-p^*)(Z_\beta + Z_{\alpha/2})^2}{(p_1 - p_2)^2}$$

r = Ratio of control to cases, 1 for equal number of case and control

p^* = Average proportion exposed = proportion of exposed cases + proportion of control exposed/2

Z_β = Standard normal variate for power = for 80% power it is 0.84 and for 90% value is 1.28. Researcher has to select power for the study.

$Z_{\alpha/2}$ = Standard normal variate for level of significance as mentioned in previous section.

$p_1 - p_2$ = Effect size or different in proportion expected based on previous studies. p_1 is proportion in cases and p_2 is proportion in control.

From the study by Khan S[88]et al p1 = 87.5%, p2 = 70.37% at 80% confidence level and 80% power, with equal ratio in two groups.N = 64 in each group.

Considering non-response rate of 10%, 64 + 6 = 70 patients in each group will be selected.

INCLUSION CRITERIA:

1. Patients with peritonitis secondary to hollow viscus perforation.

2. Patients with age >18years and <70years.

EXCLUSION CRITERIA:

1. Peritonitis secondary to trauma to the abdomen.

2. Peritonitis secondary to gynaecological interventions like D&C.

3. Peritonitis secondary to malignancies and immuno-compromised state

4. Patients allergic to Ceftriaxone, Metronidazole And Amikacin.

5. Tertiary peritonitis.

OBSERVATIONS AND RESULTS

OBSERVATIONS AND RESULTS

A total of 140 patients who presented with peritonitis secondary to hollow viscus perforation, admitted and treated in R. L. Jalappa Hospital and Research Centre attached to Sri Devaraj Urs Medical College, Kolar were studied during the period of December 2015 to June 2017.

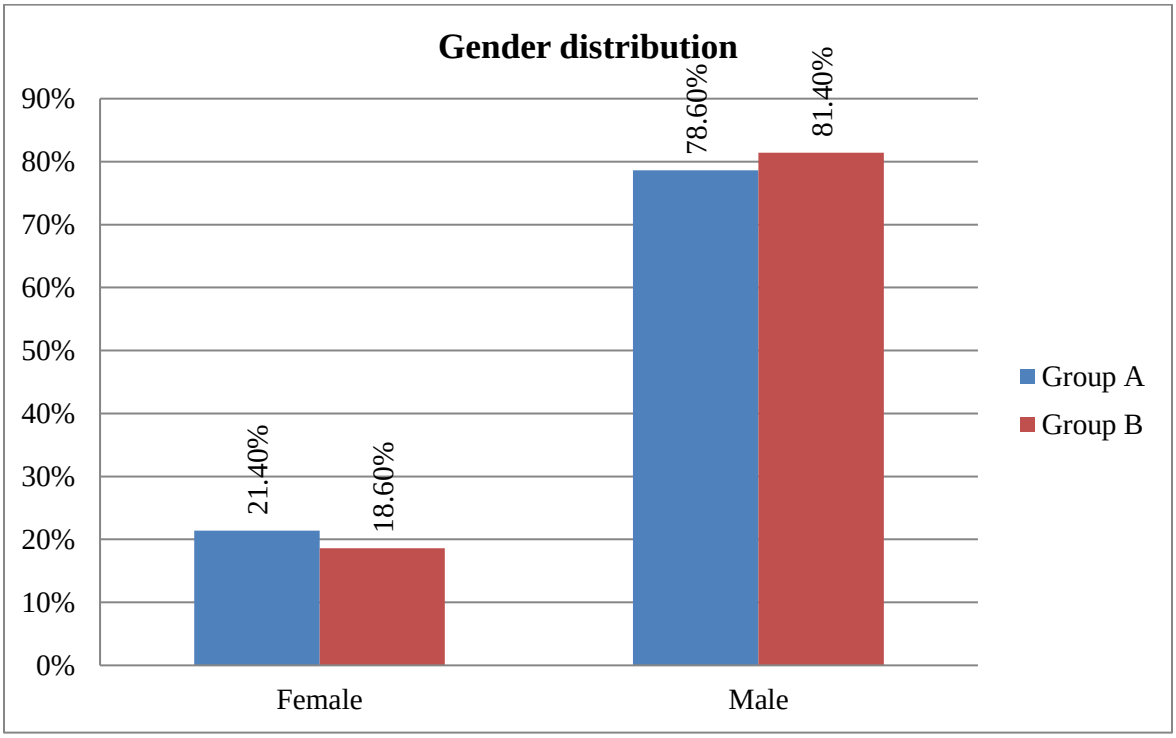
Gender Distribution Comparison Between Two Groups

In our study of 140 patients with peritonitis secondary to hollow viscus perforation, the total number of males were 112(80%) in both the groups(Group A and B). The number of male patients were 55 and 57 accounting for 78.6% and 81.4% in group A and B respectively.

The total number of females were 28(20%) in both the groups(Group A and B). The number of female patients were 15 and 13 accounting for 21.4% and 18.6% in group A and B respectively. There were 6 deaths in the study. These patients had severe form of peritonitis and presented late to the hospital. Males showed higher incidence of hollow viscus perforation in comparison with females.

Table 5: Gender distribution comparison between two groups

		Group			
		Group A		Group B	
		Count	%	Count	%
Gender	Female	15	21.4%	13	18.6%
	Male	55	78.6%	57	81.4%



Graph 1: Bar diagram showing Gender distribution comparison between two groups

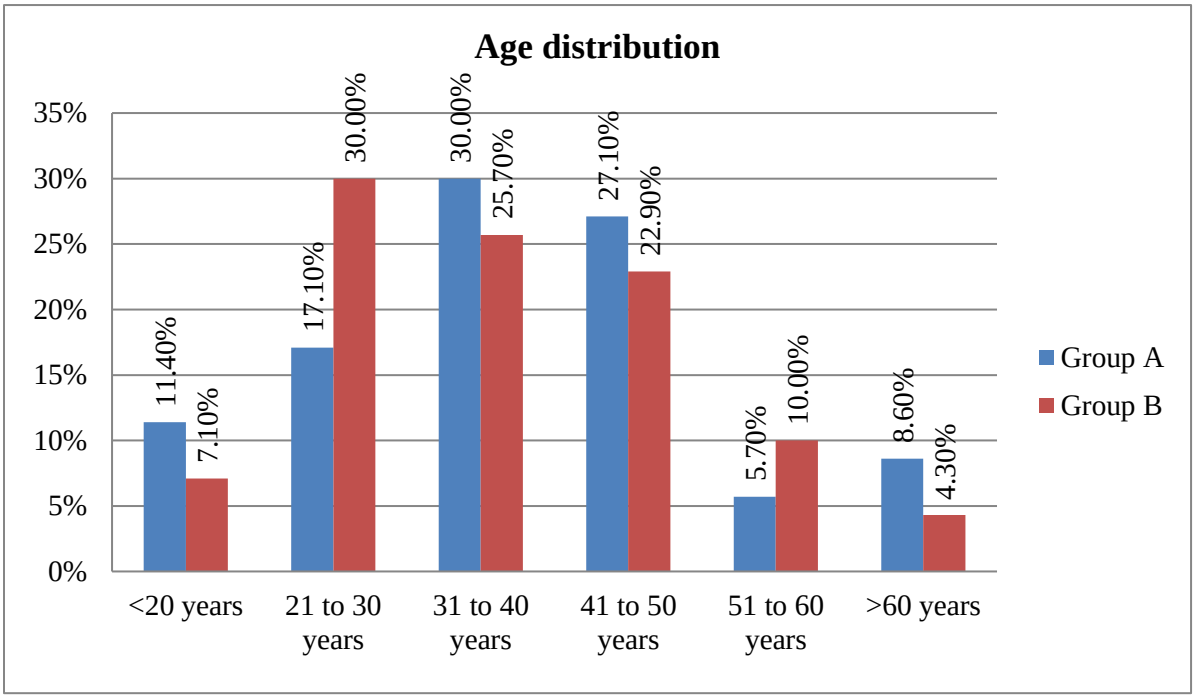
Incidence Of Perforation In Different Age Groups

Patients of the age >18years and <70years were included in the study and the youngest patient in this study is a 19 year old boy who had appendicular perforation and the oldest patient in this study is an old man of 69years who was diagnosed and treated for duodenal perforation. The

mean age of perforation in group A is 38.7 years and the mean age in group B is 37.2 years . More than half of the patients fall between the age group of 21-40, out of which 33 patients were in the age group of 21-30 and 39 patients in the age group of 31-40. This shows that more than half of the perforations are seen in young adults.

Table 6: Age distribution comparison between two groups

		Group			
		Group A		Group B	
		Count	%	Count	%
Age	<20 years	8	11.4%	5	7.1%
	21 to 30 years	12	17.1%	21	30.0%
	31 to 40 years	21	30.0%	18	25.7%
	41 to 50 years	19	27.1%	16	22.9%
	51 to 60 years	4	5.7%	7	10.0%
	>60 years	6	8.6%	3	4.3%
Mean Age		38.7 ± 13.3		37.2 ± 13	



Graph 2: Bar diagram showing Age distribution comparison between two groups

Relation Between Age And The Site Of Perforation

In this study , there were 12 cases of duodenal perforation, 5 cases of pre-pyloric perforation, 14 cases of appendicular perforation and 7 cases of ileal perforations in the age group of 19 to 29 years.

There were 25 cases of duodenal perforation, 07 cases of pre-pyloric perforation, 7 cases of appendicular perforation and 4 cases of ileal perforations in the age group of 30 to 39 years.

There were 20 cases of duodenal perforation, 06 cases of pre-pyloric perforation, 04 cases of appendicular perforation , 1 case of ileal perforation and 1 case of jejunal perforation in the age group of 40 to 49 years.

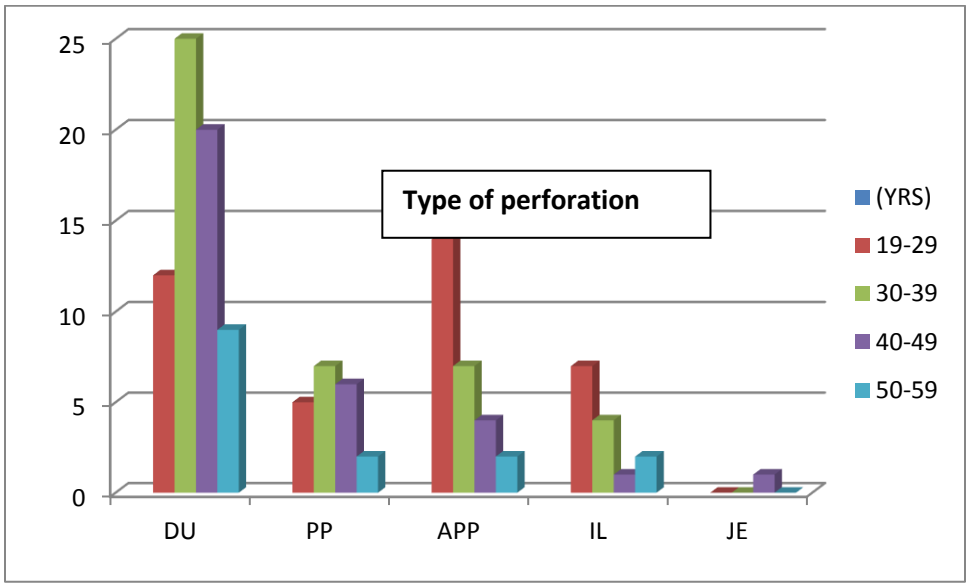
There were 9 cases of duodenal perforation, 2 cases of pre-pyloric perforation, 2 cases of appendicular perforation and 2 cases of ileal perforation in the age group of 50 to 59 years.

There were 7 cases of duodenal perforation, 2 cases of pre-pyloric perforation, 1 case of appendicular perforation and 1 case of ileal perforation in the age group of 60 to 69 years.

More than half of the perforations(n=80) are in the age group of 19 to 39 age group accounting for 57% of the cases. Proximally located perforation like duodenal and pre pyloric perforations are common in the younger age group.

Table 7: Relation Between Age And Site Of Perforation

AGE (YRS)	DU	PP	APP	IL	JE	TOTAL
19-29	12	05	14	07	0	38
30-39	25	07	07	04	0	42
40-49	20	06	04	01	01	33
50-59	09	02	02	02	0	15
60-69	07	02	01	01	0	11
TOTAL	71	21	29	16	01	140



Graph 3: Bar diagram showing relation between Age and site of perforation

Relation Between The Duration Of Symptoms And Type Of Exudate

In our study, around 70 patients(50%) in both the groups(Group A and B) had serous peritoneal fluid at laparotomy.

42 patients who had presented to the hospital with in 12 hours of symptoms had shown serous peritoneal exudate.

4 patients who had presented to the hospital after 72 hours of symptoms had shown fecal peritoneal exudate.

70 patients presented to the hospital within 12 hours and among them 42 had shown serous peritoneal fluid collection, 8 patients had biliary type of exudate, 24 patients had shown purulent exudates.

Around 36 patients presented within 13 to 24 hours and had shown purulent peritoneal fluid collection.

23 patients who presented between 25 to 48 hours and among them 8 patients had biliary type, 11 had purulent collection and 3 patients had shown feculent type of exudates.

2 and 6 patients presented between 49 to 72 hours and more than 72 hours who had shown purulent and feculent peritoneal collection.

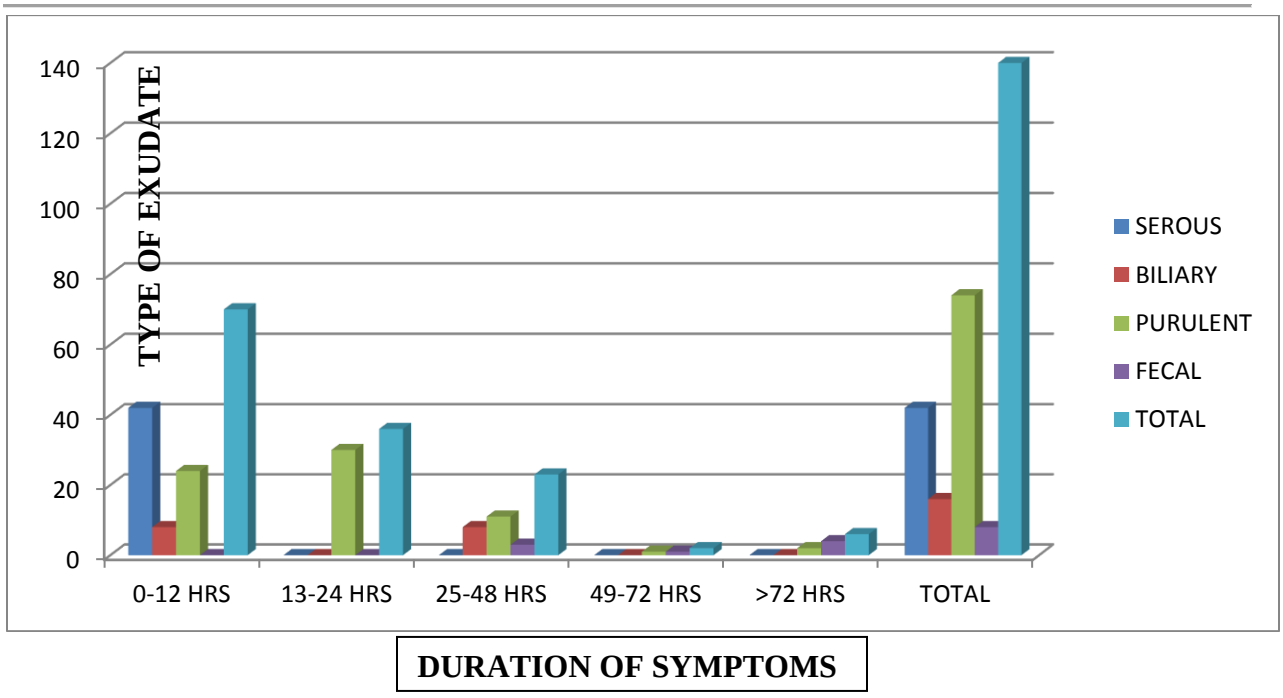
This study shows the more delay in the presentation to the health care centre, the more is the degree of peritoneal contamination.

Majority of the patients who had presented earlier to the hospital i.e., with in 12 hours had shown serous type of exudates

The patients who had late presentation to the hospital had shown contaminated foul smelling type of peritoneal fluid (purulent/ feculent collection)

Table 8: Relation Between Duration Of Symptoms And Type Of Peritoneal Fluid

DURATION OF SYMPTOMS	SEROUS	BILIARY	PURULENT	FECAL	TOTAL
0-12 HRS	**42**	**08**	**24**	**0**	**70**
13-24 HRS	**0**	**0**	**36**	**0**	**36**
25-48 HRS	**0**	**08**	**11**	**03**	**23**
49-72 HRS	**0**	**0**	**01**	**01**	**02**
>72 HRS	**0**	**0**	**02**	**04**	**06**
TOTAL	**42**	**16**	**74**	**08**	**140**



Graph 4: Bar diagram showing relation between duration of symptoms and type of exudate

Comparison Of Type Of Exudate In Two Groups

In both groups, majority had purulent Exudate, 55.7% in Group A and 50% in Group B. There was no significant difference in type of Exudate between two groups.

Table 9: Type of Exudate comparison between two groups

		Group			
		Group A		Group B	
		Count	%	Count	%
Type of Exudate	Biliary	6	8.6%	10	14.3%
	Faecal	6	8.6%	2	2.9%
	Purulent	39	55.7%	35	50.0%
	Serous	19	27.1%	23	32.9%

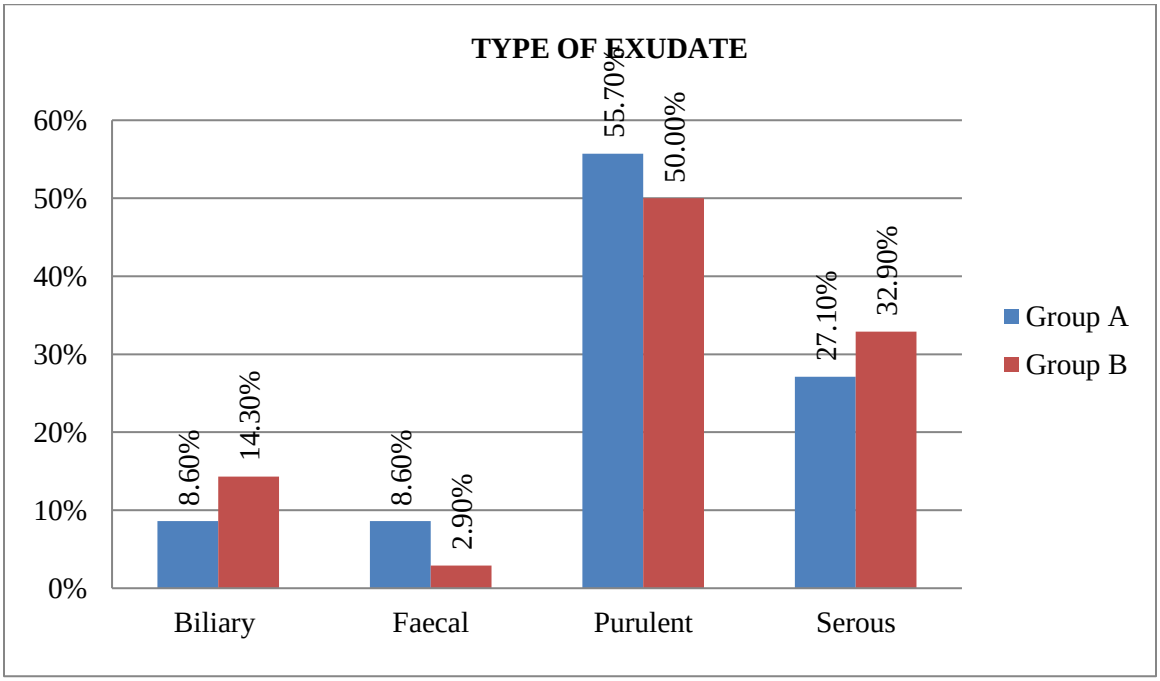


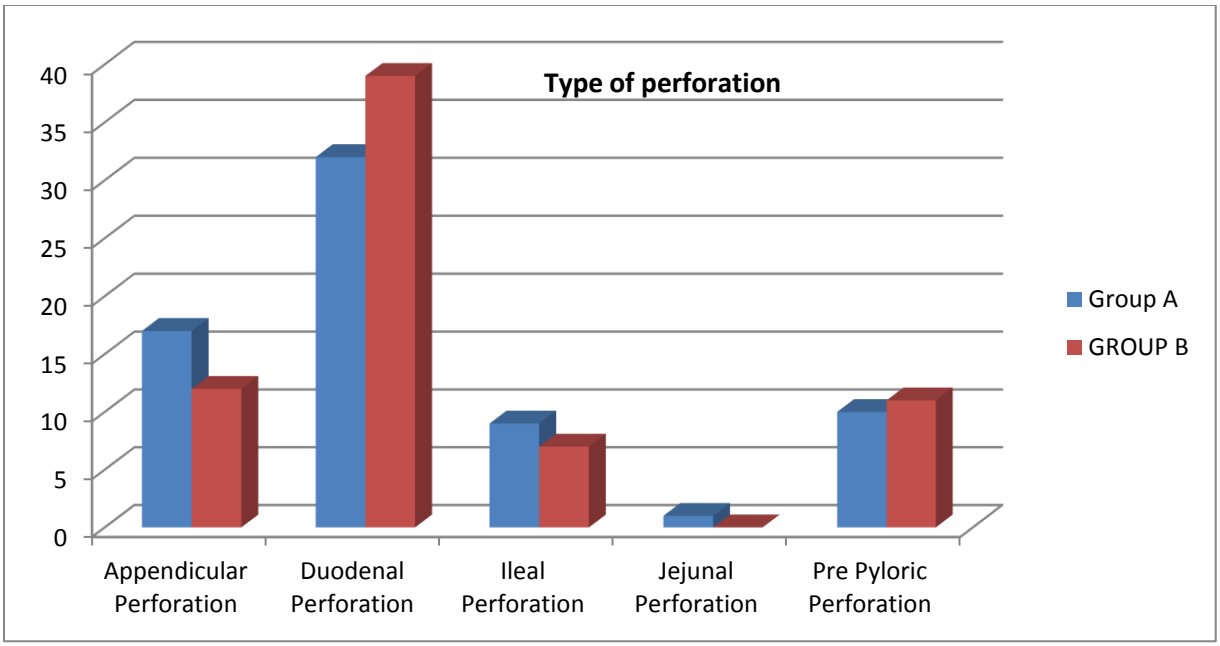
Figure 5: Bar diagram showing comparison of the type of exudate between two groups

Type Of Perforation

Depending on different sites of perforation and the data collected from our study revealed that duodenal ulcer perforation was the commonest being 43.4% in group A and 53.9% in group B. Appendicular perforation was the next commonest accounting for 22.9% in group A and 21.4% in group B, followed by pre pyloric and ileal perforations.

Table 10: Type of perforation comparison between two groups

		Group			
		Group A		Group B	
		Count	%	Count	%
Operative Findings	Appendicular Perforation	17	22.9%	12	21.4%
	Duodenal Perforation	32	43.4%	39	53.9%
	Ileal Perforation	9	12.9%	7	10.0%
	Jejunal Perforation	1	1.4%	0	0.0%
	Pre Pyloric Perforation	10	22.9%	11	21.4%



Graph 6: Bar diagram showing type of perforation comparison between two groups.

Comparison of Post operative complications in both the groups

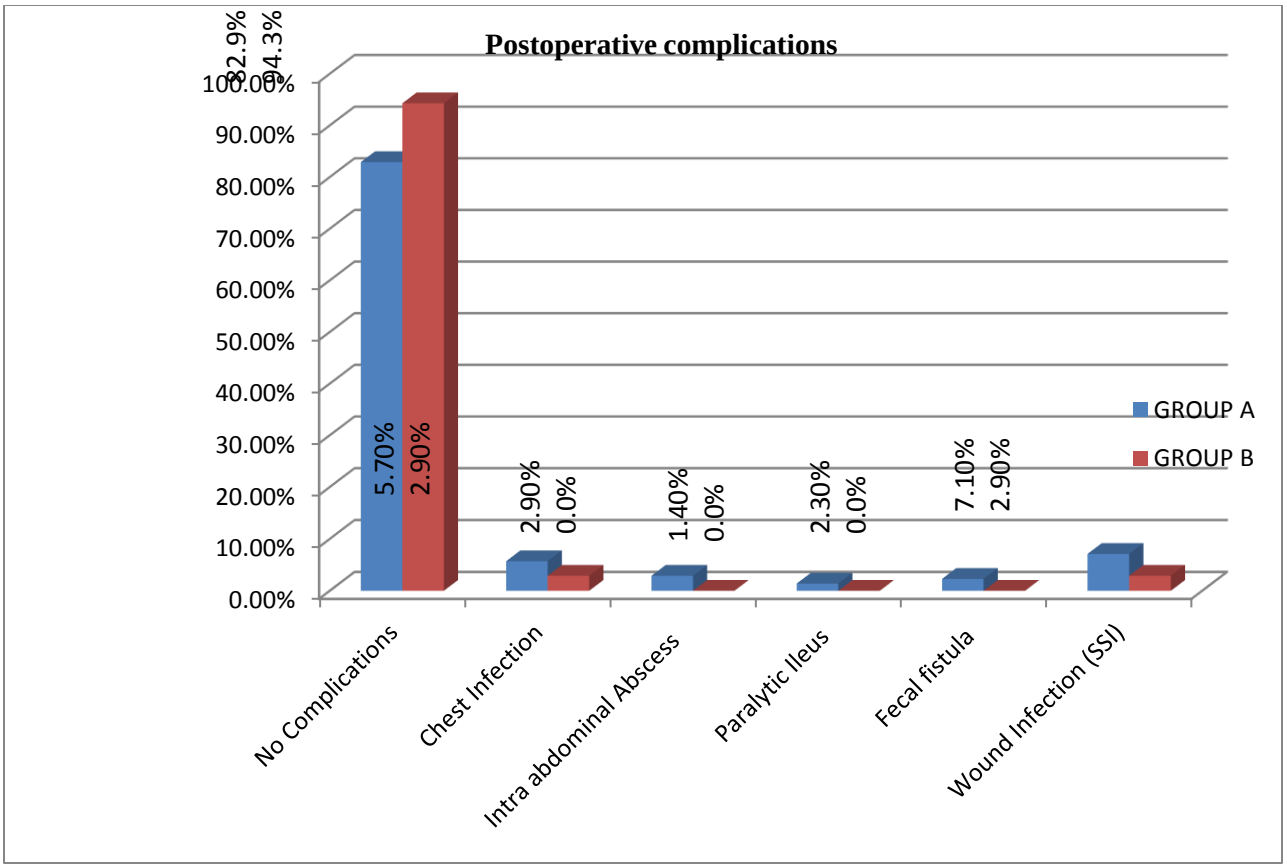
With regards to the postoperative complications(local and systemic),19 cases were detected in both the groups(Group A & B). In Group A, 4 patients developed chest infections accounting for 5.7%, 2 patients developed intra abdominal abscess accounting for 2.9% and the abscess was drained under ultrasound guidance and pig tail catheter was inserted for both patients and the pus was sent for culture and sensitivity and the report was showing the growth of E.coli in 1 patient and Klebsiella in other patient and the organisms were sensitive to ceftriaxone, ciprofloxacin and imepenem and appropriate antibiotic was started and the patients recovered and the catheter was removed when the output has ceased, one patient had developed paralytic ileus who accounted for 1.4%, 3 patients developed fecal fistula who accounted for 2.3% were treated conservatively

and successfully, spontaneous closure of fistula was noted but the patients had a very long duration of hospital stay.5 patients developed surgical site infections(SSI) like wound gaping, stitch abscess accounting for 7.1%.

In Group B, 2 patients developed chest infections accounting for 2.9% and 2 patients developed surgical site infections(SSI) like wound gaping, stitch abscess accounting for 2.9%. There were no complications like intra abdominal abscess, paralytic ileus and fecal fistula in group B patients. Overall, 15 patients who received 2 antimicrobials (Ceftriaxone and Metronidazole) in Group A and 4 patients who received 3 antimicrobials (Ceftriaxone, Metronidazole and Amikacin) had developed post operative compications like chest infections, intraabdominal abscess, paralytic ileus, fecal fistula and surgical site infections. Hence the usage of three drug regimen is more beneficial in treating perforative peritonitis.

Table 11: Comparison of postoperative complications between two groups

		Group			
		Group A		Group B	
		Count	%	Count	%
Complications	No Complications	55	82.9%	66	94.3%
	Chest Infection	4	5.7%	2	2.9%
	Intraabdominal Abscess	2	2.9%	0	0.0%
	Paralytic Ileus	1	1.4%	0	0.0%
	Fecal fistula	3	2.3%	0	0.0%
	Wound Infection (SSI)	5	7.1%	2	2.9%



Graph 7: Bar diagram showing comparison of post operative complications between two groups

Organisms isolated in peritoneal fluid

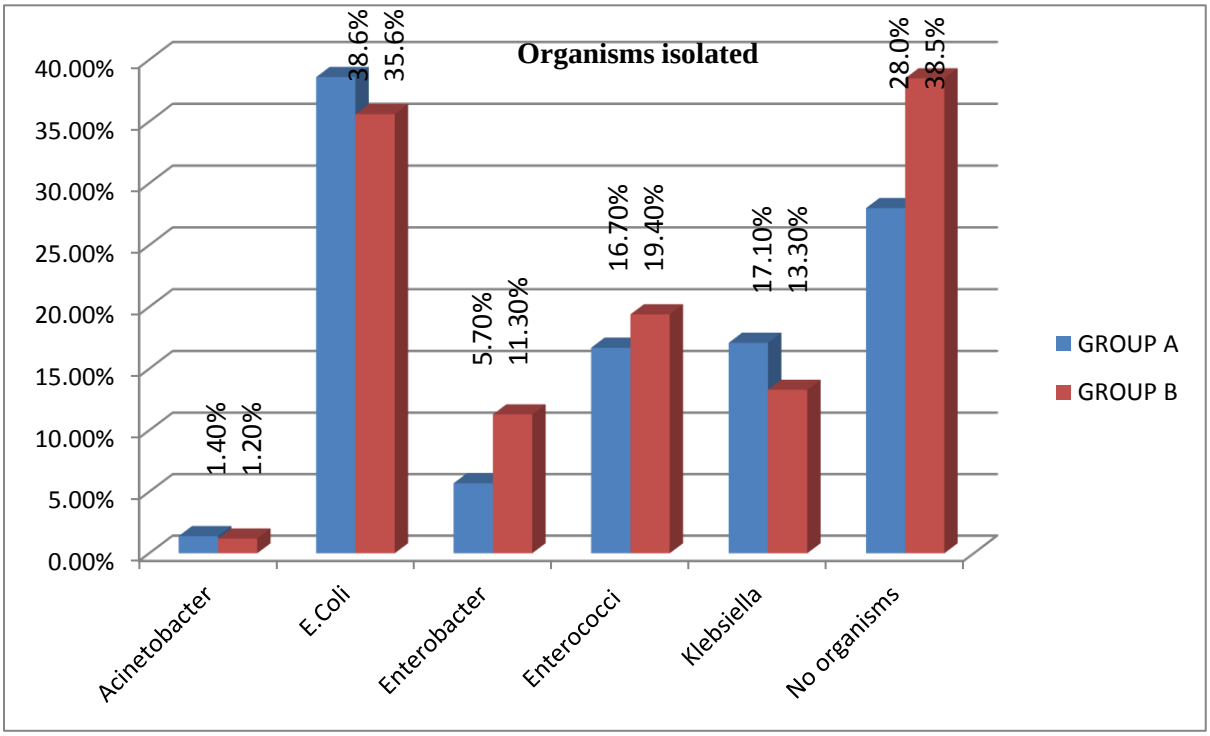
In all the patients at the time of laparotomy, the peritoneal fluid was collected and sent for microscopy and culture and sensitivity and the most common organism isolated in group A is E. coli in 24 cases followed by Klebsiella in 11 cases, where as in group B E.coli is present in 24 cases followed by Enterococci. There was no growth of any organism in 45 patients.

Table No. 8 shows the various bacteria isolated in the two groups, one receiving 2 antimicrobials (Ceftriaxone and Metronidazole) and another receiving 3 antimicrobials (Ceftriaxone, Metronidazole and Amikacin). No growth of organisms was seen in 20 patients in group A and 25 patients belonging to group B.

The organisms grown were E. coli, Enterococci, Acinetobacter, Enterobacter and Klebsiella. However E.coli was the major organism grown accounting for growth in 48 of the patients. There were 24 patients in group A(38.6%) and there were 24 patients who had E.coli isolated in the peritoneal fluid in group B(35.6%). There were 25 patients in group B who did not show growth of any organism in the peritoneal fluid(38.5%) when compared to 20 patients in group A who had negative cultures(28.0%) which is significantly better.

Table 12: Organisms Isolated comparison between two groups

		Group			
		Group A		Group B	
		Count	%	Count	%
Organisms Isolated	Acinetobacter	1	1.4%	1	1.2%
	E.Coli	24	38.6%	24	35.6%
	Enterobacter	4	5.7%	05	11.3%
	Enterococci	10	16.7%	10	19.4%
	Klebsiella	11	17.1%	05	13.3%
	No organisms	20	28.0%	25	38.5%



Graph 8: Bar diagram showing organisms isolated in the peritoneal fluid

Acinetobacter was isolated in the peritoneal sample of 1 and 1 respectively in either of the groups receiving 2 antimicrobials(Ceftriaxone and Metronidazole) or 3 antimicrobials(Ceftriaxone , Metronidazole and Amikacin). Acinetobacter could have been a contaminant carried from the environment to the peritoneal cavity through the instruments used during the surgery.

Similarly, equal number of patients (10 and 10 respectively) in either of the groups had shown Enterococci in the peritoneal fluid cultures.

Klebsiella was isolated in peritoneal fluid cultures in 11 patients belonging to group A and 5 patients belonging to group B.

Enterobacter was grown in the peritoneal fluid cultures in only 4 patients belonging to group A(who had received 2 antimicrobials) and only 5 patients belonging to group B(who had received 3 antimicrobials).

Relation Between The Site Of Perforation And The Type Of Organism Isolated

In this study, 71 patients had duodenal perforation at the time of laparotomy and the most common organism isolated in these patients was E.coli which was grown in 20 patients followed by Enterobacter and Klebsiella.

21 patients had pre pyloric perforation and the most common organism in these patients was E.coli which was cultured in 14 patients followed by Enterococcus.

29 patients had shown appendicular perforation at the time of surgery and 10 patients had shown the growth of E.coli followed by Klebsiella.

16 patients had ileal perforation at the time of laparotomy and the most common organism isolated was E.coli which was seen in 8 patients followed by Enterococcus.

1 patient with jejunal perforation had shown the growth of Enterobacter.

25 patients with duodenal ulcer perforation, 15 patients with pre pyloric perforation and 5 patients with appendicular perforation had shown negative culture of peritoneal fluid.

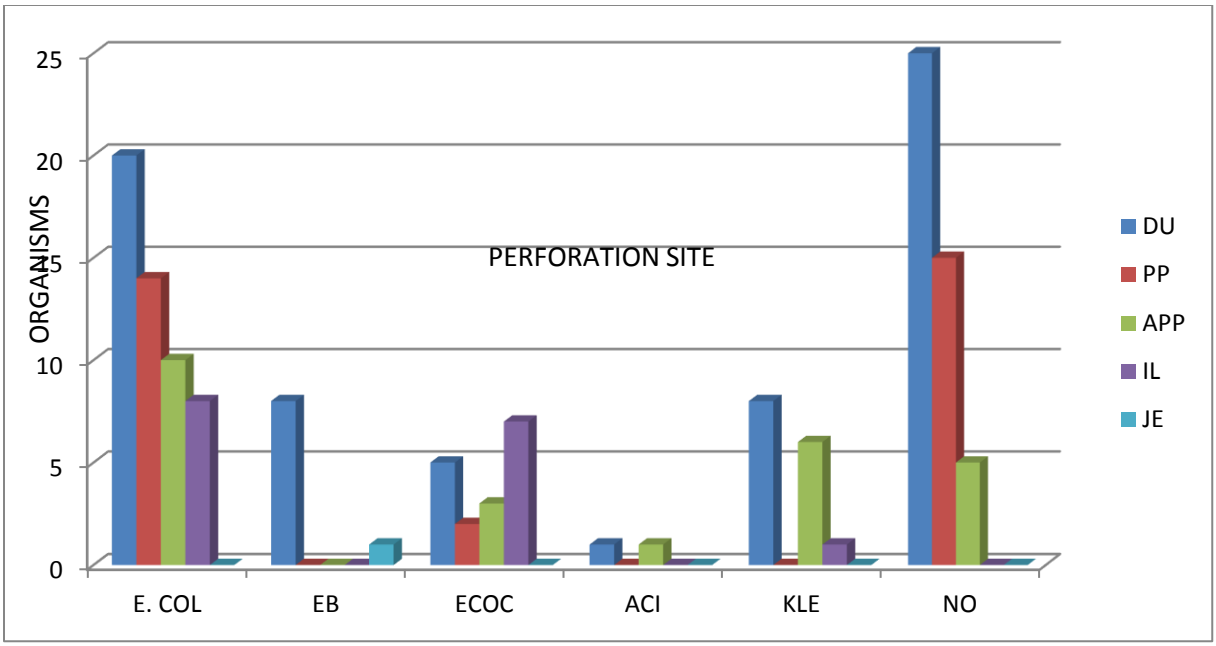
More the distal is the site of perforation in gastrointestinal tract (GIT), more is the degree of peritoneal contamination and bacterial growth.

More the proximal is the site of perforation, less is the degree of peritoneal contamination and bacterial growth provided the patient presents earlier to the hospital.

The patients with lesser degree of peritoneal contamination may be treated with two antimicrobial regimen, but three drug regimen is beneficial in most of the cases because of broad spectrum of activity and also in preventing the post operative hospital stay and complications.

Table 13: Relation Between Site Of Perforation and Type Of Organism Isolated

	TYPE OF ORGANISM ISOLATED						
TOP	E. COL	EB	ECOC	ACI	KLE	NO	TOTAL
DU	**20**	**08**	**05**	**01**	**08**	**25**	**71**
PP	**14**	**00**	**02**	**00**	**00**	**15**	**21**
APP	**10**	**0**	**03**	**01**	**06**	**05**	**29**
IL	**08**	**00**	**07**	**00**	**01**	**00**	**16**
JE	**00**	**01**	**00**	**00**	**00**	**00**	**01**
TOTAL	**48**	**09**	**20**	**02**	**16**	**45**	**140**



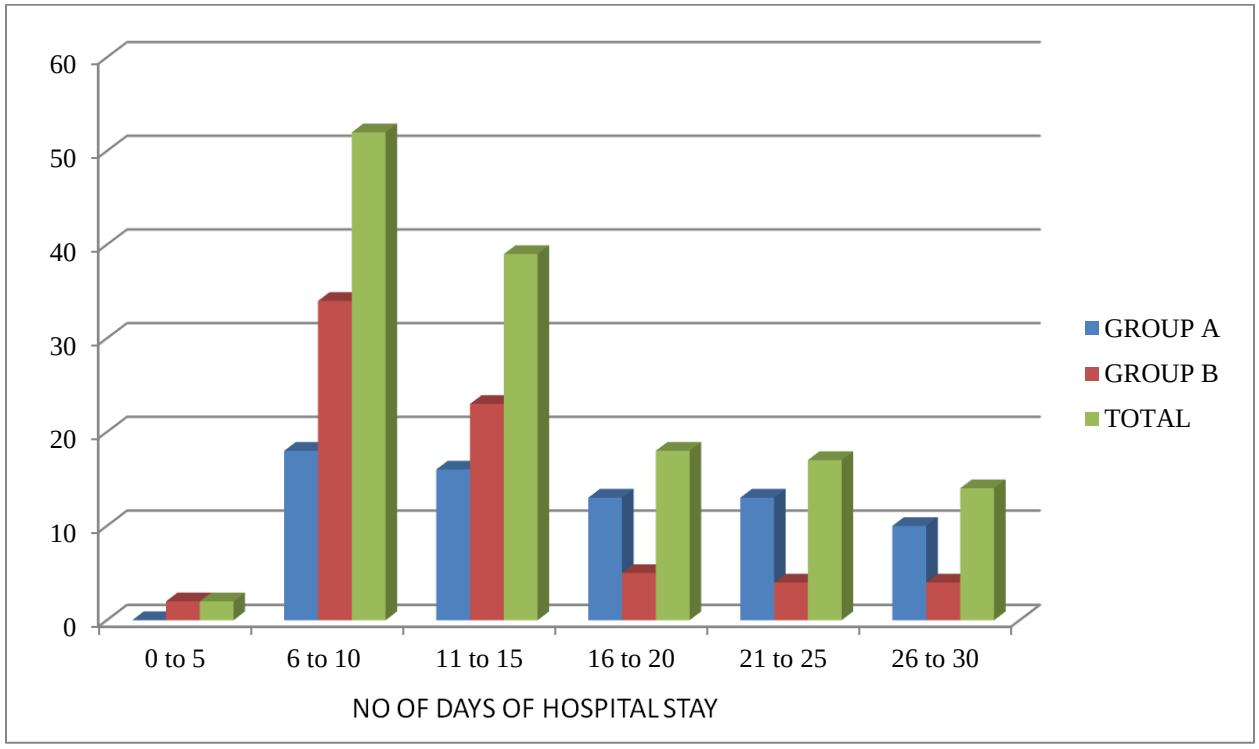
Graph 9: Bar diagram showing relation between the site of perforation and type of organism isolated

Number of days of hospital stay

Duration of hospital stay extended from 5 to 30 days. The maximum number of days of hospital stay is between 06 - 15 days where 52 patients stayed between 6 – 10 days followed by 11 – 15 days which accounted for 39 patients. The mean stay in hospital was 16.3 days for patients who received 2 antimicrobials(Ceftriaxone And Metronidazole) where as it was 12.6 days for patients who received 3 antimicrobials(Ceftriaxone, Metronidazle and Amikacin). The maximum number of days apatient stayed in the hospital is for 29 days who was operated for duodenal perforation and minimal duration of stay of a patient is for 5 days who underwent surgery for appendicular perforation. In our study we found that patients who received 3 antimicrobials(Ceftriaxone , Metronidazole and Amikacin) had shorter stay in the hospital.

TABLE 14:No. OF DAYS OF HOSPITAL STAY

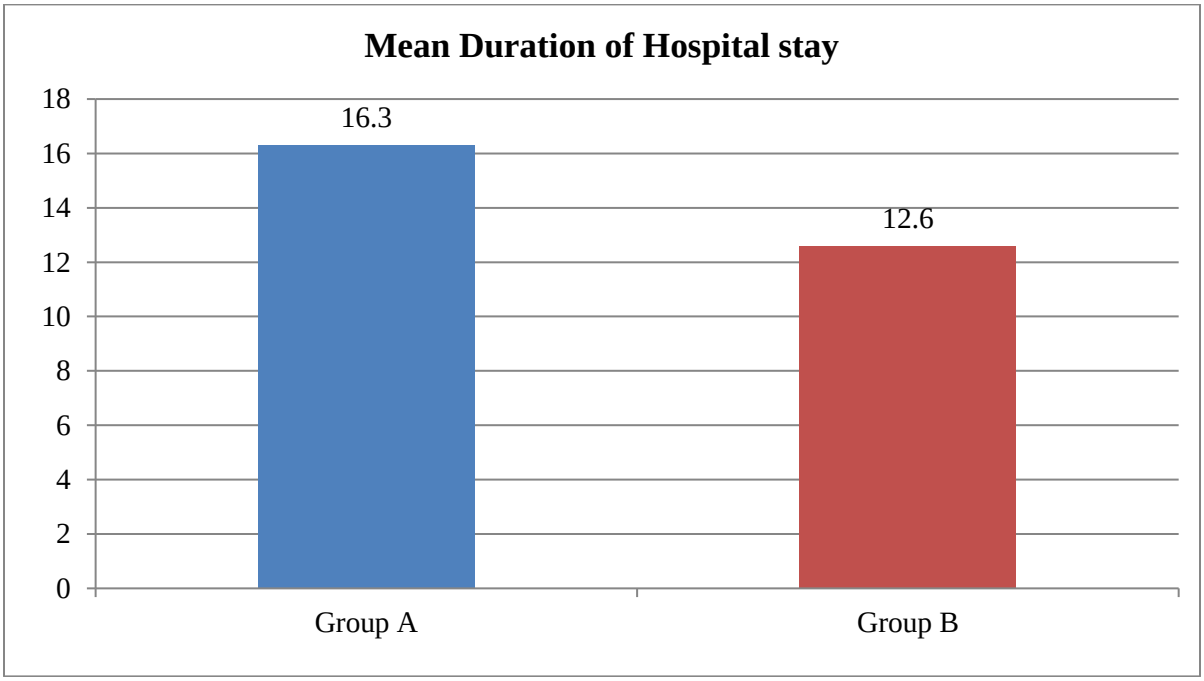
No. of Days	GROUP A	GROUP B	TOTAL
0-5	0	02	02
6-10	18	34	52
11-15	16	23	39
16-20	13	05	18
21-25	13	04	17
26-30	10	04	14
Total	70	70	140



Graph 10: Bar diagram showing number of days of hospital stay in both the groups

Table 15: Comparison of mean duration of hospital stay between two groups

		Duration of Hospital stays (Days)	
		Mean	SD
Group	Group A	16.3	6.2
	Group B	12.6	4.6
P value		<0.001*	



Graph11: Bar diagram showing Mean Duration of Hospital stays comparison between two groups

Comparison of p – values with use of two and three antimicrobials

Overall the number of patients who had developed post operative complications who received three drugs were 4 compared to 15 patients who developed postoperative complications when they received two drugs. Consequently, the mean duration of hospital stay was 12.6 in patients who received three drugs when compared to 16.3 in the patients who received two drugs. Reduction in the number of days of hospital stay and less post operative complications were noted in the patients who received 3 antimicrobials(Ceftriaxone, Metronidazole and Amikacin)(group A) when compared to 2 antimicrobials(Ceftriaxone and Metronidazole)(group B). p – value was significant in patients belonging to group B.i.e. p=0.007(<0.05) when compared to p – value in group patients i.e. p=0.06(>0.05).

Table 16: Comparison of P- values between two groups

GROUPS	P- VALUE
GROUP A	**0.06(not significant)**
GROUP B	**0.007(significant)**

DISCUSSION

DISCUSSION

A total of 140 patients who presented with features of peritonitis secondary to hollow viscus perforation to R. L. Jalappa Hospital and Research Centre, Tamaka , Kolar , from December 2015 to June 2017 were randomized into two groups and studied.

In Group A,patients with all odd serial numbers were included and received 2 antimicrobials(Ceftriaxone and Metronidazole) and in Group B patients with all even serial numbers were taken and received 3 antimicrobials(Ceftriaxone, Metronidazole and Amikacin).

This clinical study was intended to determine the efficacy of two antimicrobials (Ceftriaxone And Metronidazole) and three antimicrobials(Ceftriaxone, Metronidazole and Amikacin)in perforative peritonitis and to compare the clinical outcome of perforative peritonitis with two and three antimicrobials in the terms of reduction in postoperative infections and hospital stay.

In general this study revealed that the maximum number of patients were in the age group of 21-40 with the mean age of 37 years and the youngest patient in this study is a 19 year old boy who had appendicular perforation and the oldest patient in this study is an old man of 69 years who was diagnosed and treated for duodenal perforation. More than half of the patients fall between the age group of 21-40 years, out of which 33 patients were in the age group of 21-30 and 39 patients in the age group of 31-40. This shows that more than half of the perforations are seen in young adults. Male population was affected more than female population because of the irregular

eating or dietary habits, smoking and alcoholism and duodenal ulcer perforation is the commonest followed by appendicular, pre-pyloric, ileal and the rest.

Demography: Age distribution:

The randomized study involved 140 patients of both sexes with perforative peritonitis. Age of the patients in this study ranged from 19years to 69years. The mean age of the patients at the time of admission was 37 years. Maximum number of patients 72(51.4%) were in the age group of 21-40 years, Samir Delibegovic et al and Ashis Ahuja et al stated predominant population from age group 21–40 years. C Ohmann et al study showed predominant population in 50-69 years age group. These findings are similar from our study.

TABLE 17: COMPARISON OF PREDOMINANT AGE GROUP IN PERITONITIS.

Study	**Predominant age group**
Samir Delibegovic et al[80]	21-40 years
Ashis Ahuja et al[1]	21-40 years
C Ohmann et al[81]	50-69years
Our study	21-40 years

Age group with highest incidence of post operative complications

Highest incidence of post operative complications in our study was in the age group of 61-69years. Notash et al[82] also stated complications are more in >60 years of age. C Ohmann et al[81] cited highest mortality in age >70yrs. In our study it was observed that incidence of post operative complications increases with increase in age.

TABLE 18: AGE GROUP WITH MORE POST OPERATIVE COMPLICATIONS

Studies	**Age group with morepost operative complication**
Notash et al[82]	>60 years
C Ohmann et al[81]	>70years
Our study	>60years

SEX DISTRIBUTION:

Current study showed the male preponderance in peritonitis with ratio of male: female as 4:1. Male preponderance was also found in Samir Delibegovicet al[80] with male to female ratio of 3:1, Ajazahamed Malik et al[83] with 2:1 and also in Sharma R, Huttunen et al[84].

TABLE 19: COMPARISON OF MALE TO FEMALE DISTRIBUTIONIN

Studies	M:F ratio
Samir Delibegovicet al[80]	3:1
Ajazahamed Malik et al[83]	2:1
Sharma R, Huttunen et al[84]	2:1
Our study	4:1

PERITONITIS

TABLE 20: SITE OF PERFORATION IN DIFFERENT STUDY GROUP:

	Study	SITE OF PERFORATION		
		Gastroduodenal	Small intestine	Large intestine
1	AjazAhamed Malik et al[83]	30.6%	9.9%	5.9%
2	Notash et al[82]	60%	42.5	-
3	RS Jhobta[85]	65.67%	18.27%	3.7%
4	Nithin Agarwal et al[86]	23%	43%	6%
5	Our study	73.2%	27.2%	-

Current study showed the Gastroduodenal perforations(73.2%) are commoner in peritonitis followed by small intestinal(27.2%) . The observation done in our study is comparable to other studies done in various centres.

More than half of the perforations(n=80) are in the age group of 19 to 39 age group accounting for 57% of the cases. Proximally located perforation like duodenal and pre pyloric perforations are common in the younger age group.

This study shows the more delay in the presentation to the health care centre, the more is the degree of peritoneal contamination. Majority of the patients who had presented earlier to the hospital i.e., with in 12 hours had shown serous type of exudates. The patients who had late presentation to the hospital had shown contaminated foul smelling type of peritoneal fluid (purulent/ feculent collection) .

19 patients had post operative complications ranging from wound gaping, stitch abscess, pneumonia, intra abdominal abscess,paralytic ileus to fecal fistula. 3 patients had fecal fistulas which were treated conservatively and successfully.

More the distal is the site of perforation in gastrointestinal tract(GIT), more is the degree of peritoneal contamination and bacterial growth.

More the proximal is the site of perforation, less is the degree of peritoneal contamination and bacterial growth provided the patient presents earlier to the hospital.

The patients with lesser degree of peritoneal contamination may be treated with two antimicrobial regimen, but three drug regimen is beneficial in most of the cases because of broad spectrum of activity and also in preventing the post operative hospital stay and complications.

The organisms grown were E. coli, Enterococci, Acinetobacter, Enterobacter and Klebsiella. However E.coli was the major and the most common organism grown accounting for growth in 48 of the patients.

Antibiotics should be used prophylactically before contamination has occurred. This is not possible in patients where the infection is already established. In these situations the use of antimicrobial drugs to prevent the growth of bacteria which occurs due to disease / trauma is therapeutic rather than prophylactic.

Combination antibiotic therapy has been used to provide the patient with broad-spectrum coverage against the many potential pathogens encountered in abdominal trauma. Several potential benefits of the clinical use of antibiotic combinations have been advanced. These include expansion of spectrum of either agent alone allowing treatment of polymicrobial infections and prevention of emergence of antibiotic resistant organism, reducing the potential for toxicity with aminoglycosides and other agents with demonstrated in vitro synergistic activity or additive affect; more effective treatment of bacteraemia in neutropenic patient [87].

We have used two antimicrobials(ceftriaxone and metronidazole) in group A and three antimicrobials(Ceftriaxone, Metronidazole and Amikacin) in group B patients. Ceftriaxone is known to be a bactericidal agent against most of aerobic and few anaerobic organisms. Metronidazole is active against most of anaerobic organisms. Amikacin is effective against gram negative organisms and few gram positive organisms like Staphylococcus aureus. In our study treatment with 3 antimicrobials(Ceftriaxone, Metronidazole and Amikacin) in perforative peritonitis is statistically significant in reduction in the postoperative hospital stay and complication in comparison with 2 antimicrobials (Ceftriaxone and Metronidazole).

E. coli was the most common organism isolated in both the groups followed by Enterococci, Klebsiella and the rest.

In our study there were 6 deaths which were due to severity of illness due to delay in seeking medical treatment; all of them were seen by us on 4^{th} – 5^{th} day of onset of severe abdominal pain. The complications due to unhindered pathological process and irreversible damage might have been responsible for death in this patients. This is in contrast to the observation by Atkins and Colleagues[13] who attributed mortality in these series to gross and diffuse peritoneal soiling.

The incidence of post operative complications in this study is less in patients who received treatment with 3 antimicrobials (Ceftriaxone , Metronidazole and Amikacin) and it is statistically significant when compared to treatment with 2 antimicrobials(Ceftriaxone and Metronidazole).

The incidence of hospital stay was less in the patients who received treatment with 3 antimicrobials(Ceftriaxone , Metronidazole and Amikacin) and it is statistically significant when compared to treatment with 2 antimicrobials(Ceftriaxone and Metronidazole).

SUMMARY

SUMMARY

We analyzed 140 patients with perforative peritonitis which were confirmed on emergency laparotomy. Most of the patients in our study group were in the age group 21-40years (51.4%).

The perforations of proximal gastrointestinal tract (gastroduodenal) constituted about 69.2% of all the perforations. Majority of the patients had peptic ulcer perforation which included both prepyloric and duodenal perforations. Site of perforations showed wide variability in different studies. only few patients with duodenal perforation had developed post operative complications. Patients were subjected to emergency exploratory laparotomy after adequate resuscitation. The surgical procedure performed depended upon the operative findings and the surgeon's choice.

The difference in the postoperative hospital stay and complications were studied using 2 antimicrobials (Ceftriaxone and Metronidazole) and 3 antimicrobials (Ceftriaxone , Metronidazole and Amikacin) in perforative peritonitis which were proved surgically.

There was a decrease in the post operative hospital stay and complications in patients where 3 antimicrobials(Ceftriaxone , Metronidazole and Amikacin) were used.

The p – value was significant in patients where 3 antimicrobials (Ceftriaxone , Metronidazole and Amikacin) were used.p=0.007(<0.05).

The p – value was insignificant in patients where 2 antimicrobials (Ceftriaxone and Metronidazole) were used.p=0.06(>0.05)

There were 6 deaths, all of them had severe form of peritonitis and delayed presentation to the hospital.

This study also revealed that men are commonly affected and duodenal ulcer perforation is the commonest site of perforation.

E. coli is the most common organism isolated.

CONCLUSION

CONCLUSION

The clinical and bacteriological study has demonstrated the following:

- ✓ Peritonitis is more common in men compared to women.
- ✓ The common age group is in between 21 -40 years in cases of peritonitis with the mean age of 37 years.
- ✓ Duodenal ulcer perforation is the commonest site of perforation.
- ✓ E. coli is the commonest organism isolated from the peritoneal contamination.
- ✓ Bacterial /peritoneal contamination increases with time.
- ✓ Delayed presentation i.e., more than 12-24 hours increases the degree of contamination.
- ✓ Postoperative complications like surgical site infections, pneumonia intra abdominal abscess are more in the patients with distally situated perforation and who had delayed presentation to the health care centre.
- ✓ So , patients who are elderly, who had delayed presentation to the hospital and distally situated perforation with severe degree of contamination, it is a safe option to consider three antimicrobials for the treatment.
- ✓ 3 antimicrobial usage significantly (<0.05) reduces the post operative complications like surgical site infections, pneumonia intra abdominal abscess when compared to 2 antimicrobial usage.
- ✓ As per the clinical outcome is concerned, there is a significant differences in the both the groups, treatment with 3 antimicrobials is better than 2 antimicrobials.

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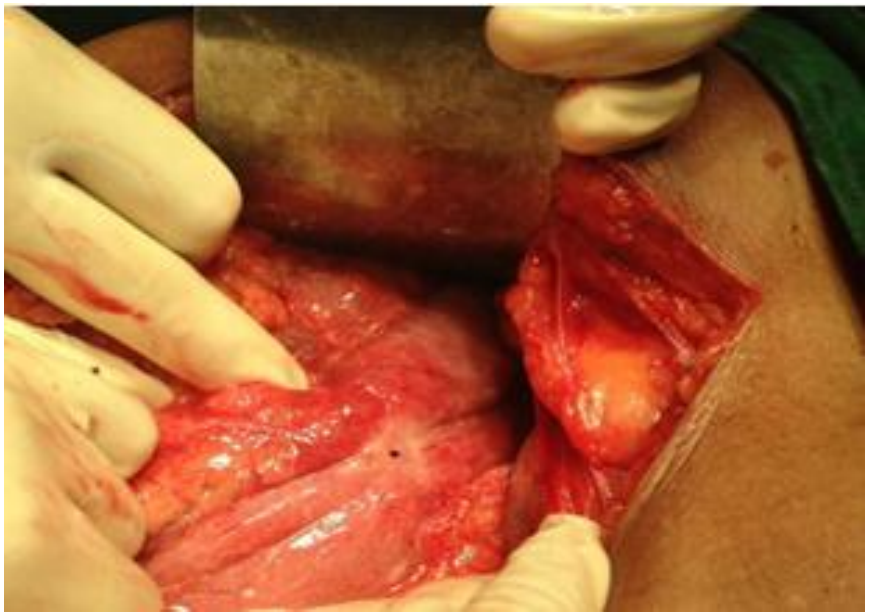
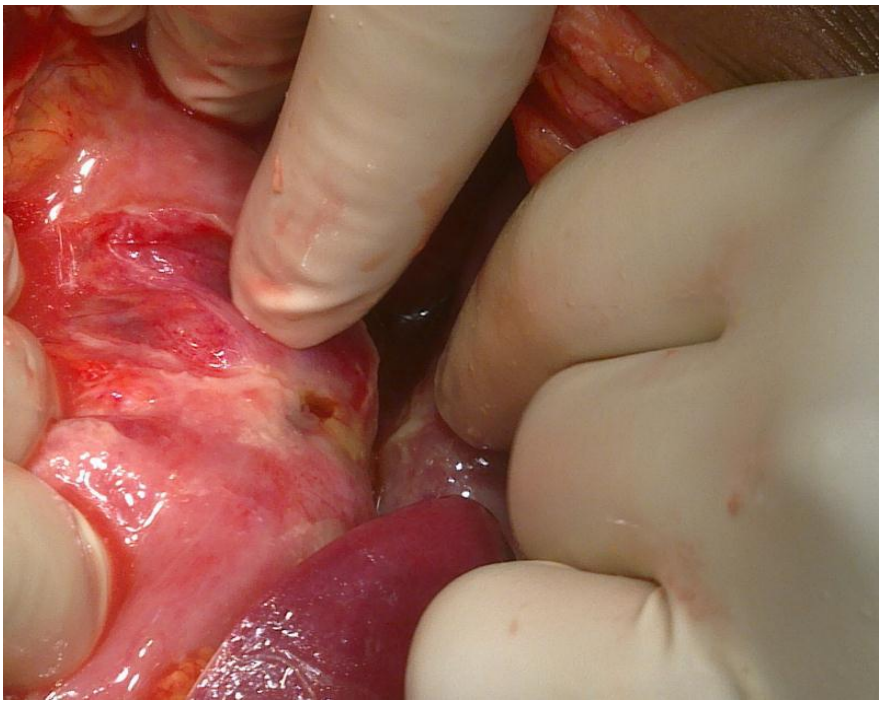
PICTURE GALLERY

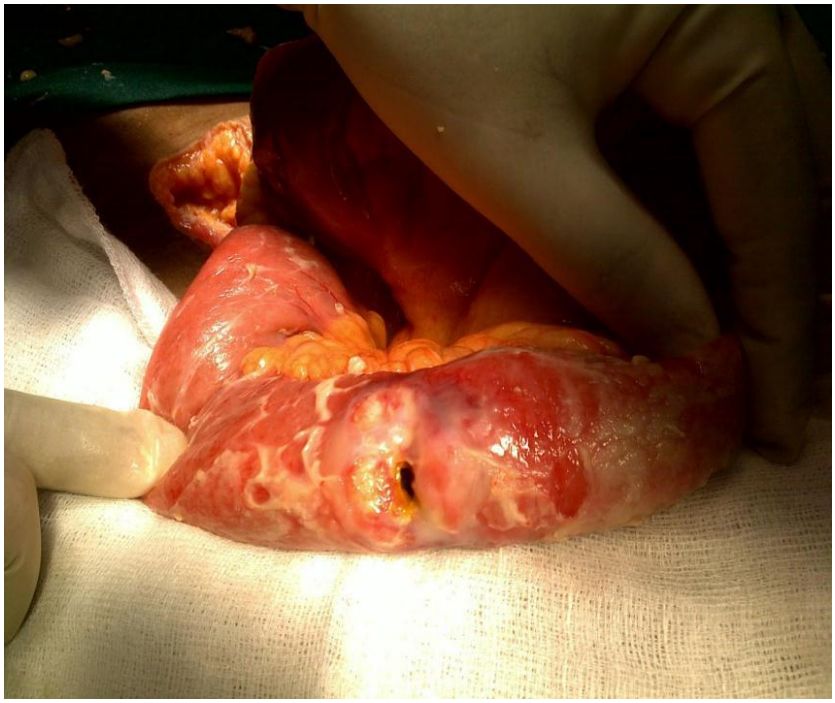
PICTURE GALLERY



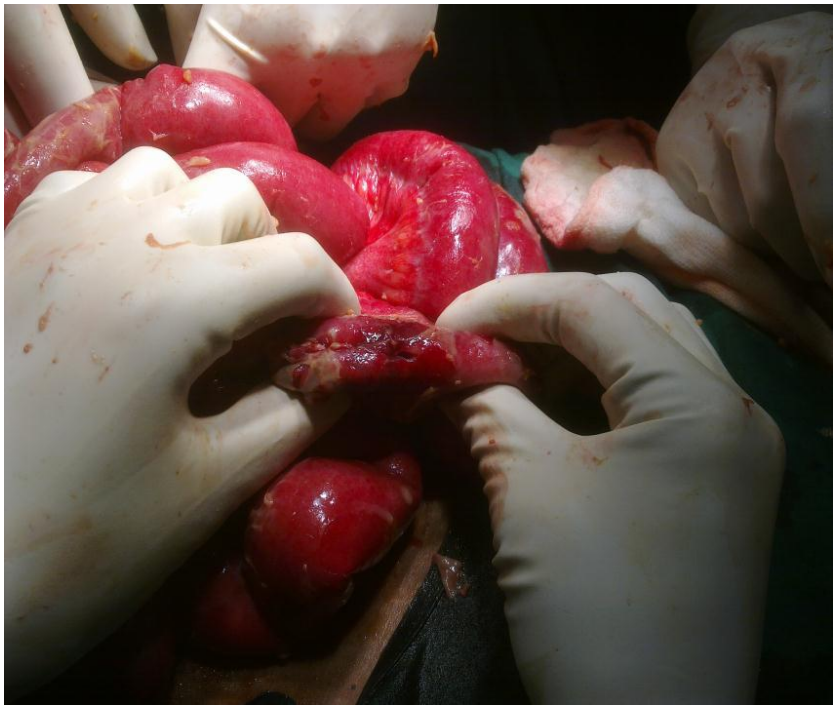
Pre pyloric Perforation



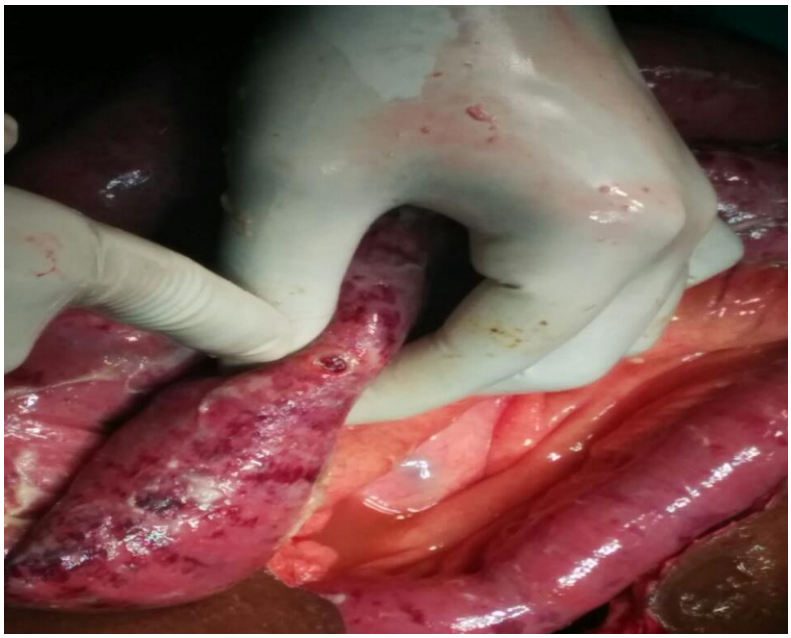
Duodenal perforation



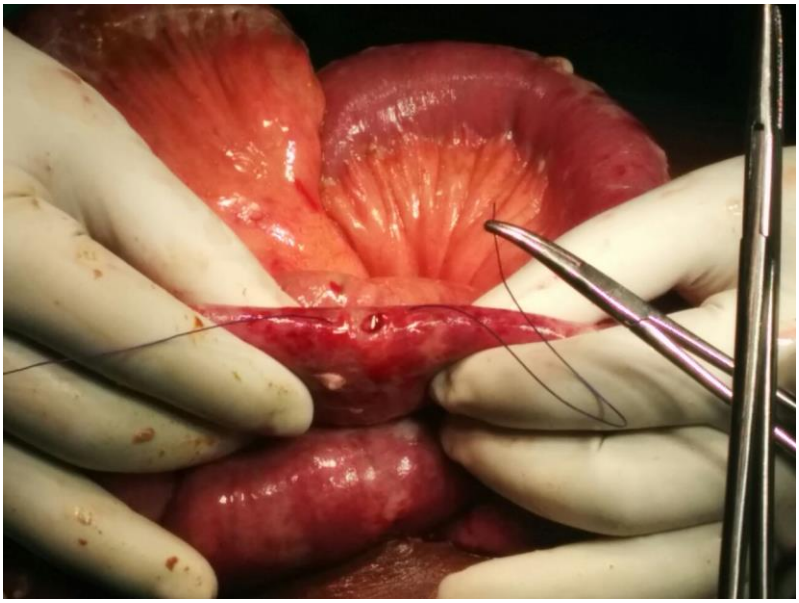
Ileal perforation



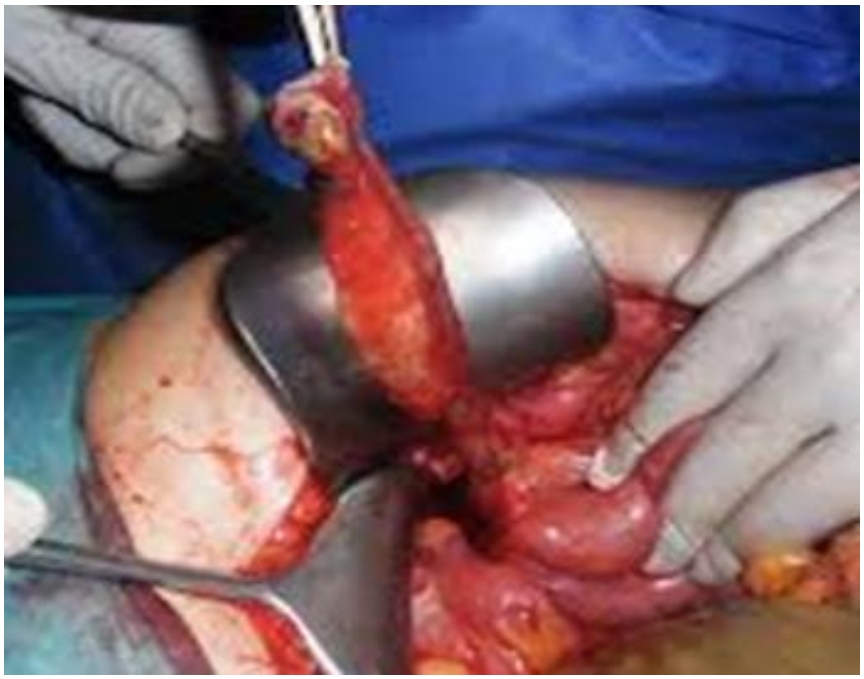
Ileal perforation



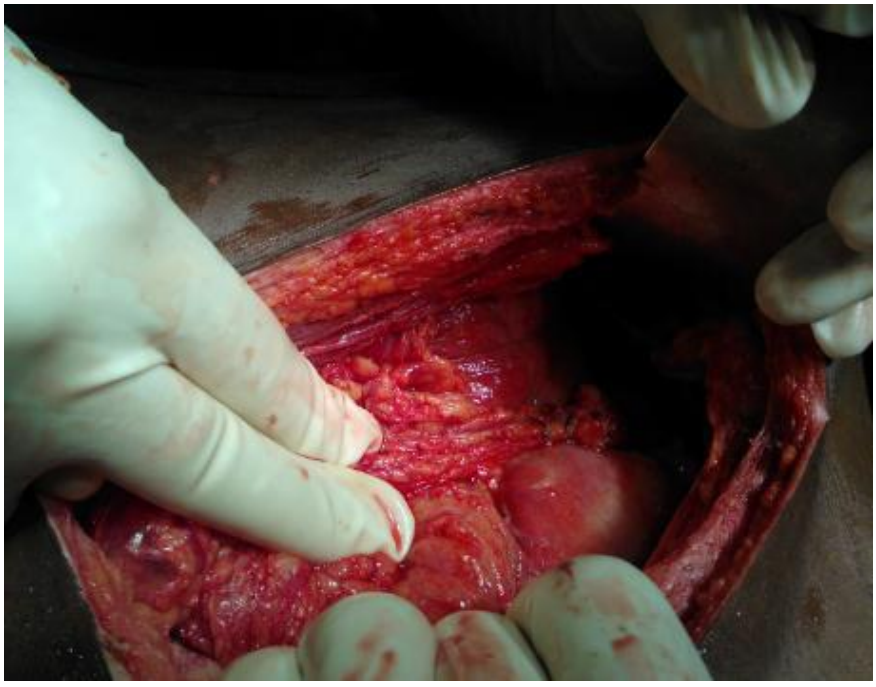
Jejunal perforation



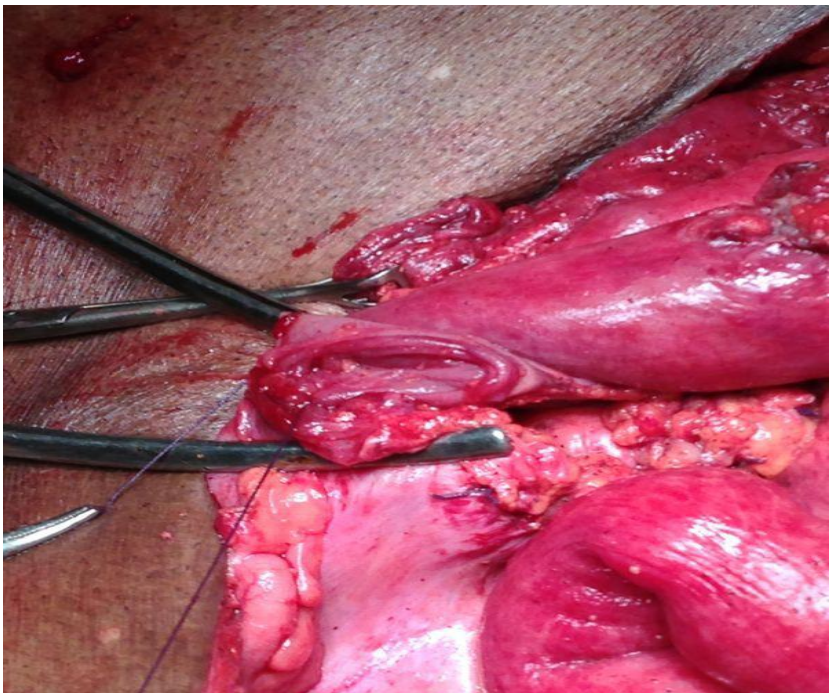
Primary closure of jejunal perforation



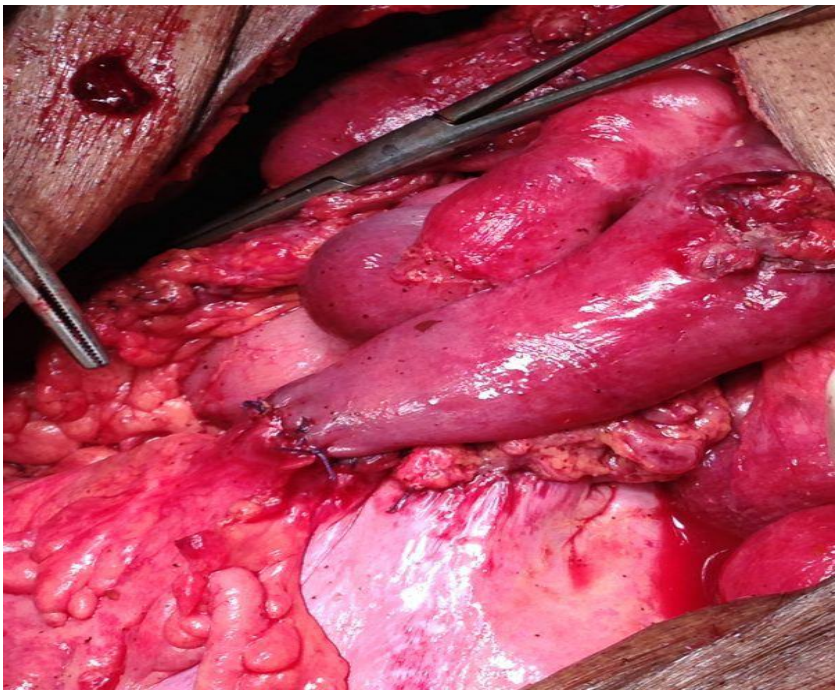
Perforated Appendix



Grahams Omentoplasty repair for duodenal perforation

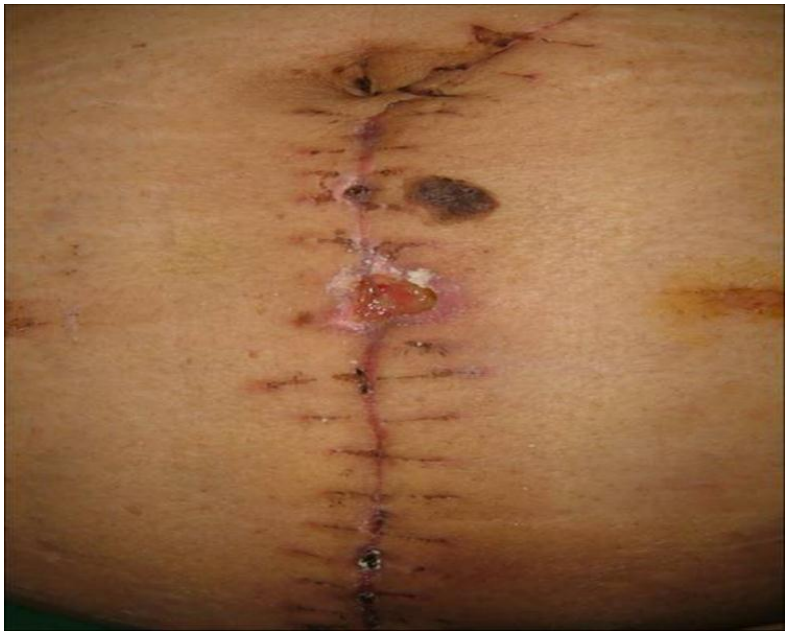


Resection and anastomosis for terminal ileal perforation

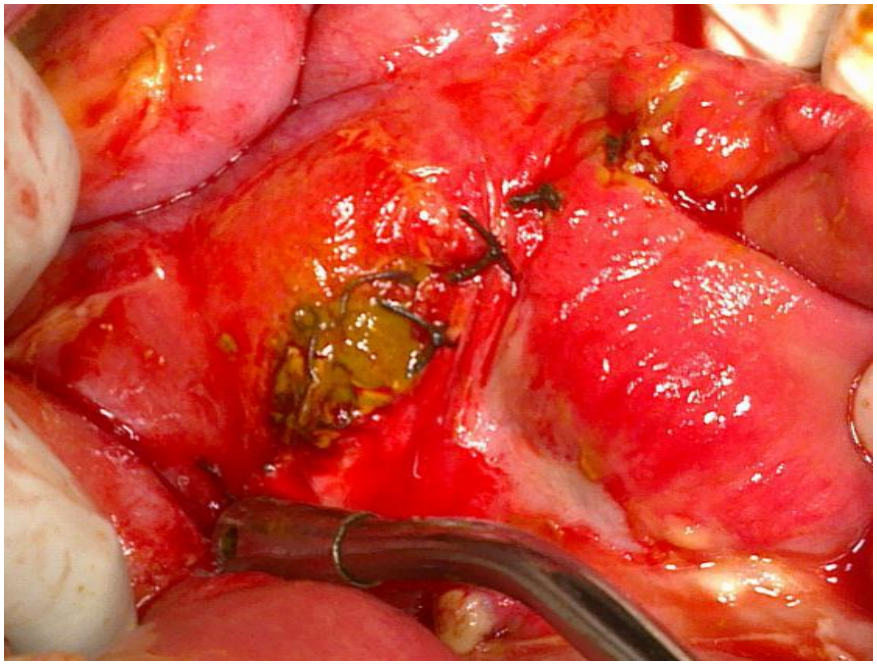


POST OPERATIVE COMPLICATIONS

Wound infection



Anastomotic leak



ANNEXURES

ANNEXURE-1

STANDARD PROFORMA

"CLINICAL EFFICACY OF TWO ANTIMICROBIALS(CEFTRIAXONE AND METRONIDAZOLE) VERSUS THREE ANTIMICROBIALS(CEFTRIAXONE, METRONIDAZOLE AND AMIKACIN) IN PERFORATIVE PERITONITIS"

Particulars of the patients

Name:

Ward:

Age:

OP no.

I.P. no:

Gender:

Unit:

Occupation:

Address:

Date of Admission:

Date of Surgery:

Date of Discharge:

Complaints

1) Pain:

-time of onset

-mode of onset

-site of pain

-migration of pain

-character of pain

-relation to vomiting

-relation to food intake

-aggravating factors

-relieving factors

2) Vomiting:

- onset

-duration

-frequency

-character of onset

-amount

-content

3) Bowels:

-last evacuation

-constipation/normal

-history of passing worms

4) Distension:

-duration

-location

-relation to pain

5) Fever:

-duration

-nature;continuous/intermittent/remittent

-relation to pain

-whether associated with chills and rigors

Previous History

- Of similar complaints

-Haematemesis

- Treatment of peptic ulcer

-Ingestion of drugs

Personal History

-Diet

-Appetite

-Smoking

-Alcohol

-Bowel habits

-Menstrual history

Family History

-Peptic ulcer/Diabetes/Hypertension/TB

General Physical Examination

-Appearance

-Attitude

-Build and nourishment

-Level of consciousness

-Dehydration

-Temperature

-Pulse

-Blood pressure

-Respiration

Local Examination(abdomen)

Inspection

-contours of the abdomen

-distension :uniform/upper/lower

-visible peristalsis

-umbilicus

-operation scars

-hernial orifices

-genitalia

Palpation

-temperature

-tenderness :localized/diffuse/rebound

-muscular rigidity:localized/generalized

-mass

-liver

-spleen

-abdominal girth

Percussion

-obliteration of liver dullness

-shifting dullness

Auscultation

-bowel sounds:present/absent

Other Relevant Examinations:

-Per rectal

empty/loaded

bleeding/mass felt

-Per vaginal

Systemic Examination:

-CVS/CNS/RS

Investigations:

Routine investigations: CBC with blood grouping and typing,BT,CT

RBS, RFT

Serum electrolytes,

HIV,

HbsAg

Chest X ray

widal test

Urine routine,

ECG

Erect Xray abdomen.

USG abdomen and pelvis.

Diagnostic taping of peritoneal fluid

Peritoneal fluid for culture and sensitivity

CT scan abdomen and pelvis as and when required

Pre operative treatment

-Antibiotics(If yes, Drug, dose, frequency , duration)

-Other drugs

-Intravenous fluids

-blood transfusion

-gastric aspiration

Pre medication and Anaesthesia

Operative details

-Type of surgery

-Duration of surgery

-Type of drain

-Type of peritoneal fluid drained

Post operative management

-Iv fluids

-antibiotics

-blood transfusion

-other drugs

-Gastric aspiration

-oral fluids

-removal of drains

Sample for HPE:YES/NO

Histopathological report:

Organism isolated

Antibiotic sensitivity pattern

Post op antibiotics:

-Type, dose, frequency, duration

Complications

Local

-intra abdominal abscess (pelvic abscess, sub-diaphragmatic abscess and subhepatic abscess)

-stitch abscess

-burst abdomen

-paralytic ileus

-faecal fistula

-intestinal obstruction due to adhesions

-incisional hernia

General

-Pulmonary/toxaemia/cardiac/thrombotic/renal/agranulocytosis

Treatment of complications

Follow up in immediate post op period: 3rd , 5th 7th days.

No. of days in hospital

Condition at the time of discharge:

Follow up after 1 month:

ANNEXURE 2

INFORMED CONSENT FORM

"CLINICAL EFFICACY OF TWO ANTIMICROBIALS(CEFTRIAXONE AND METRONIDAZOLE) VERSUS THREE ANTIMICROBIALS(CEFTRIAXONE, METRONIDAZOLE AND AMIKACIN) IN PERFORATIVE PERITONITIS"

I, the undersigned agree to participate in this study and authorize the collection and disclosure of my personal information as outlined in this consent form.

I understand the purpose of the study,the risks and benefits of undergoing surgery for perforative peritonitis and the use of combination of antimicrobial drugs(CEFTRIAXONE AND METRONIDAZOLE or CEFTRIAXONE, METRONIDAZOLE AND AMIKACIN)in such cases and the confidential nature of the information that will be collected and disclosed during the study. The information collected will be used only for research.

I have had the opportunity to ask the questions regarding the various aspects of the study and my questions have been answered to my satisfaction.

I understand that I remain free to withdraw from this study at any time and this will not change my future care

Participation in this study does not involve any extra cost to me.

Subject's name and signature/thumb impression Date:

Name and signature of the witness Date:

Name and signature of the person obtaining consent Date:

ಭಾಗವಹಿಸಲು ಸಮ್ಮತಿ

ನಾನು, ರುಜುಮಾಡಿರುವ, ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ಮತ್ತು ಈ ಒಪ್ಪಿಗೆ ರೂಪದಲ್ಲಿ ಅಂಶಗಳಂತೆ ನನ್ನ ವೈಯಕ್ತಿಕ ಮಾಹಿತಿಯ ಸಂಗ್ರಹಣೆ ಮತ್ತು ಡಿಸ್ಕ್ಲೋಸರ್ ಅಧಿಕೃತಗೊಳಿಸಲು ಒಪ್ಪುತ್ತೀರಿ.

ಈ ಅಧ್ಯಯನದ ಉದ್ದೇಶ, ಬಳಸಲಾಗುತ್ತದೆ ಎಂದು ಕಾರ್ಯವಿಧಾನಗಳು, ಅಧ್ಯಯನ ಮತ್ತು ಅಧ್ಯಯನದ ಸಮಯದಲ್ಲಿ ಸಂಗ್ರಹಿಸಿದ ಮತ್ತು ಬಹಿರಂಗ ನಡೆಯಲಿದೆ ಮಾಹಿತಿಯನ್ನು ಗೌಪ್ಯ ಪ್ರಕೃತಿಯಲ್ಲಿ ನನ್ನ ಒಳಗೊಳ್ಳುವಿಕೆ ಸಂಬಂಧಿಸಿದ ಅಪಾಯಗಳನ್ನು ಮತ್ತು ಲಾಭಗಳನ್ನು ಅರ್ಥಮಾಡಿಕೊಂಡಿದ್ದೇನೆ.

ನಾನು ವಿವಿಧ ಈ ಅಧ್ಯಯನದ ಅಂಶಗಳು ಮತ್ತು ನನ್ನ ಪ್ರಶ್ನೆಗಳಿಗೆ ನನ್ನ ತೃಪ್ತಿಕರ ಉತ್ತರಗಳನ್ನು ಮಾಡಲಾಗಿದೆ ಸಂಬಂಧಿಸಿದ ಪ್ರಶ್ನೆಗಳನ್ನು ಕೇಳಲು ಅವಕಾಶ ಹೊಂದಿದ್ದರು.

ನಾನು ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ಈ ಅಧ್ಯಯನದಿಂದ ಹಿಂತೆಗೆದುಕೊಳ್ಳುವಂತೆ ಮತ್ತು ಈ ನನ್ನ ಮುಂದಿನ ಆರೈಕೆ ಬದಲಾಗುವುದಿಲ್ಲ ಉಚಿತ ಉಳಿಯಲು ಎಂದು ಅರ್ಥ.

ವಿಷಯದ ಹೆಸರು ಮತ್ತು ಸಹಿ / ಹೆಬ್ಬೆಟ್ಟಿನ ಗುರುತು ದಿನಾಂಕ:

ಪೋಷಕ / ಪೋಷಕರು ಹೆಸರು ಮತ್ತು ಸಹಿ ದಿನಾಂಕ:

ಒಪ್ಪಿಗೆ ಪಡೆದ ವ್ಯಕ್ತಿಯ ಹೆಸರು ಮತ್ತು ಸಹಿ ದಿನಾಂಕ:

ANNEXURE-3

PATIENT INFORMATION SHEET

"CLINICAL EFFICACY OF TWO ANTIMICROBIALS (CEFTRIAXONE AND METRONIDAZOLE) VERSUS THREE ANTIMICROBIALS (CEFTRIAXONE, METRONIDAZOLE AND AMIKACIN) IN PERFORATIVE PERITONITIS."

Intra-abdominal infections are among the most difficult infections to diagnose early and treat effectively.Peritonitis resulting from visceral inflammation or perforation is polymicrobial, which contains anaerobic and aerobic nature of bacterial flora. Peritonitis requires draining the abscess, cleaning the peritoneal cavity and eliminating contamination. Antibiotics play a secondary but important role. Combination antibiotic therapy (CEFTRIAXONE AND METRONIDAZOLE orCEFTRIAXONE, METRONIDAZOLE AND AMIKACIN) will be used to provide the patient with broad-spectrum antimicrobial coverage against the many potential pathogens encountered in abdominal trauma. Several potential benefits of the clinical use of antibiotic combinations have been advanced. These include expansion of spectrum of either agent alone allowing treatment of polymicrobial infections and prevention of emergence of antibiotic resistant organism, reducing the potential for toxicity with aminoglycosides and other agents with demonstrated in vitro synergistic activity or additive affect; more effective treatment of bacteraemia in neutropenic patient.

Advantage :1. Broad-spectrum coverage against the many potential pathogens.

Disadvantage :1.Expensive

Principal investigator's details	Surgeon's details:
Dr.Nemalidinne Keerthi	Dr.Mohan Kumar K
Post graduate(08861447665)	Professor
DEPT. OF GENERAL SURGERY	DEPT. OF GENERAL SURGERY

ANNEXURE-4

KEYS TO MASTER CHART

S.N: Serial Number

A: Age

Ip.No:Inpatient Number

G: Gender

ADM:Date Of Admission

DOS:Duration of symptoms in hours

SYMP: symptoms

PR: Pulse Rate

BP: Blood pressure

POD: Pre operative Diagnosis

OF:Operative Finding

TOS: Type of surgery

TOE: Type Of Exudate

O I: Organisms Isolated

RC:Recovery Complete

M:Male

F: Female

H S: Hospital Stay In days

SSI: Surgical Site Infection

DP: Duodenal Perforation

PPP: Prepyloric Perforation

IP: Ileal Perforation

JP: Jejunal perforation

APP: Appendicular Perforation

Cx:Ceftriaxone

Me:Metronidazole

Am:Amikacin

BI:Biliary Exudate

PU: Purulent Exudate

FE: Faecal Exudate

SE: Serous Exudate

COML:Complications

OCM: Outcome

E.coli: Escherichia coli

RC:Recovery Complete

IAA: Intra Abdominal Abscess

D: Death

PNEU: Pneumonia

F F: Fecal Fistula

Par IL: Paralytic Ileus

NC: No Complications

AP: Abdominal Pain

AD:abdominal distension

C: Constipation

D: diarrhea

F: fever

V: vomiting

P2HVP: peritonitis secondary to hollow viscus perforation

EX&GOPR: exploratory laparotomy and grahams omental patch repair

EX&R&A: exploratory laparotomy and resection and anastomosis

EX&APPEND: exploratory laparotomy and appendectomy

EX&PC: exploratory laparotomy and primary closure

KLE: klebsiella

EBAC: enterobacter

ECOC: enterococcus

ACI: Acinetobacter

NG: no growth

CX : Ceftriaxone

ME: Metronidazole

AM: Amikacin

MASTER CHART

S.N	IP.NO	A	G	DOA	SYMP	DOS	PR	BP	POD	OF	TOS	OI	TOE	COML	HS	OCM	DRUGS
1	218721	36	M	12/12/2015	AP,V,F,C	12	100	100/70	P2HVP	DP	EX&GOPR	E.COLI	PU	NC	10	RC	CX, ME
2	278234	38	M	25/12/2015	AP,F,AD	14	96	110/60	P2HVP	IP	EX&R&A	ECOC	PU	SSI	4	RC	CX, ME ,AM
3	238099	43	M	28/12/2015	AP,F,V	10	112	100/60	P2HVP	PPP	EX&GOPR	NG	SE	PNU	15	RC	CX, ME
4	218010	30	M	6/1/2016	AP,V	12	108	100/70	P2HVP	DP	EX&GOPR	NG	PU	NC	14	RC	CX, ME ,AM
5	242581	34	M	9/1/2016	AP,V,F,AD	16	104	110/80	P2HVP	DP	EX&GOPR	KLE	BI	SSI	18	RC	CX, ME
6	250488	33	M	11/1/2016	AP,F,V	12	110	100/60	P2HVP	DP	EX&GOPR	NG	PU	NC	5	RC	CX, ME, AM
7	257761	25	F	20/2/2016	AP,C	18	100	90/60	P2HVP	PPP	EX&GOPR	ECOC	SE	NC	10	RC	CX, ME
8	278617	37	M	27/2/2016	AP,D	16	110	100/70	P2HVP	DP	EX&GOPR	E.COLI	PU	NC	6	RC	CX, ME , AM
9	260300	19	F	27/2/2016	AP,V,F	12	98	100/60	P2HVP	APP	EX&APPEND	NG	SE	NC	14	RC	CX, ME
10	263270	37	M	6/3/2016	AP,V,F	14	110	90/60	P2HVP	DP	EX&GOPR	EBAC	PU	NC	8	RC	CX, ME,AM
11	373172	38	M	19/3/2016	AP,F,C	24	110	100/60	P2HVP	DP	EX&GOPR	ECOC	FE	PR IL	18	RC	CX, ME
12	273612	39	M	5/4/2016	AP,F	18	96	110/80	P2HVP	DP	EX&GOPR	E.COLI	SE	NC	8	RC	CX, ME,AM
13	277742	44	M	16/4/2016	AP,F,V	16	98	100/60	P2HVP	IP	EX&R&A	E.COLI	PU	NC	12	RC	CX, ME
14	288647	37	M	13/5/2016	AP,F,AD	20	110	100/70	P2HVP	DP	EX&GOPR	ECOC	PU	NC	10	RC	CX, ME , AM
15	288706	22	F	14/5/2016	AP,F,D	20	112	100/60	P2HVP	PPP	EX&GOPR	KLE	SE	NC	16	RC	CX, ME
16	289147	20	M	15/5/2016	AP,F,C	24	110	100/70	P2HVP	DP	EX&GOPR	E.COLI	BI	SSI	10	RC	CX, ME , AM
17	293802	35	F	27/5/2016	AP,F	12	100	110/60	P2HVP	APP	EX&APPEND	NG	PU	NC	21	RC	CX, ME
18	294119	30	M	31/5/2016	AP,F	12	100	100/70	P2HVP	DP	EX&GOPR	NG	PU	NC	10	RC	CX, ME , AM
19	297668	27	M	4/6/2016	AP,F,V	50	96	110/60	P2HVP	DP	EX&GOPR	ECOC	PU	SSI	20	D	CX, ME
20	300092	45	F	11/6/2016	AP,AD	10	112	100/60	P2HVP	DP	EX&GOPR	NG	SE	PNU	10	RC	CX, ME , AM
21	313386	19	M	15/7/2016	AP,V,AD	12	108	100/70	P2HVP	PPP	EX&GOPR	NG	PU	NC	14	RC	CX, ME
22	313501	35	M	15/7/2016	AP,F,V	16	104	110/80	P2HVP	DP	EX&GOPR	KLE	BI	NC	8	RC	CX, ME , AM
23	322482	27	M	6/8/2016	AP,V	12	110	100/60	P2HVP	DP	EX&GOPR	NG	PU	NC	12	RC	CX, ME
24	343174	38	M	16/9/2016	AP,AD	18	100	90/60	P2HVP	PPP	EX&GOPR	ECOC	SE	NC	10	RC	CX, ME , AM
25	333821	55	M	17/9/2016	AP,D	16	110	100/70	P2HVP	DP	EX&GOPR	E.COLI	PU	NC	12	RC	CX, ME
26	340440	25	F	18/9/2016	AP,F,C	12	98	100/60	P2HVP	DP	EX&GOPR	NG	SE	NC	8	RC	CX, ME , AM
27	346182	47	M	30/9/2016	AP,F,V	30	110	90/60	P2HVP	APP	EX&APPEND	E.COLI	PU	NC	18	RC	CX, ME
28	346183	30	M	30/9/2016	AP,V,F	24	110	100/60	P2HVP	DP	EX&GOPR	EBAC	FE	NC	8	RC	CX, ME , AM
29	347572	28	M	3/10/2016	AP,V,F	18	96	110/80	P2HVP	JP	EX&GOPR	E.COLI	SE	NC	14	RC	CX, ME
30	351806	24	M	14/10/2016	AP,F,C	16	98	100/60	P2HVP	DP	EX&GOPR	E.COLI	PU	NC	9	RC	CX, ME , AM
31	354577	38	F	29/10/2016	AP,F	20	110	100/70	P2HVP	IP	EX&R&A	EBAC	PU	NC	14	RC	CX, ME

32	399113	54	F	10/11/2016	AP,F,V	20	112	100/60	P2HVP	DP	EX&GOPR	KLE	SE	NC	8	RC	CX, ME , AM
33	401473	35	F	13/11/2016	AP,F,AD	24	110	100/70	P2HVP	DP	EX&GOPR	E.COLI	BI	SSI	20	RC	CX, ME
34	239731	33	M	16/11/2016	AP,F,D	12	100	110/60	P2HVP	DP	EX&GOPR	NG	PU	NC	9	RC	CX, ME , AM
35	242998	45	M	18/11/2016	AP,F,C	12	100	100/70	P2HVP	PPP	EX&GOPR	NG	PU	NC	10	RC	CX, ME
36	248051	55	M	21/11/2016	AP,F	14	96	110/60	P2HVP	DP	EX&GOPR	ECOC	PU	NC	10	RC	CX, ME , AM
37	252226	50	M	22/11/2016	AP,V,F,C	10	112	100/60	P2HVP	PPP	EX&GOPR	NG	SE	PNU	15	RC	CX, ME
38	254626	41	M	25/11/2016	AP,F,AD	12	108	100/70	P2HVP	DP	EX&GOPR	NG	PU	NC	10	RC	CX, ME , AM
39	257052	45	M	26/11/2016	AP,F,V	16	104	110/80	P2HVP	DP	EX&GOPR	KLE	BI	SSI	18	RC	CX, ME
40	262475	35	M	28/11/2016	AP,V	12	110	100/60	P2HVP	DP	EX&GOPR	NG	PU	NC	10	RC	CX, ME , AM
41	268099	22	M	30/11/2016	AP,V,F,AD	18	100	90/60	P2HVP	PPP	EX&GOPR	ECOC	SE	NC	10	RC	CX, ME
42	269819	36	M	1/12/2016	AP,F,V	16	110	100/70	P2HVP	DP	EX&GOPR	EBAC	PU	NC	10	RC	CX, ME , AM
43	269390	50	F	3/12/2016	AP,C	12	98	100/60	P2HVP	APP	EX&APPEND	NG	SE	NC	14	RC	CX, ME
44	299685	24	M	3/12/2016	AP,D	14	110	90/60	P2HVP	DP	EX&GOPR	EBAC	PU	NC	10	RC	CX, ME , AM
45	299613	25	M	4/12/2016	AP,F,V	24	110	100/60	P2HVP	PPP	EX&GOPR	ECOC	FE	NC	18	RC	CX, ME
46	316099	37	M	6/12/2016	AP,AD	18	96	110/80	P2HVP	DP	EX&GOPR	E.COLI	SE	NC	10	RC	CX, ME , AM
47	316104	19	M	8/12/2016	AP,V,AD	16	98	100/60	P2HVP	DP	EX&GOPR	E.COLI	PU	NC	12	RC	CX, ME
48	318701	42	M	9/12/2016	AP,F,V	20	110	100/70	P2HVP	DP	EX&GOPR	ECOC	PU	NC	10	RC	CX, ME , AM
49	324777	45	M	11/12/2016	AP,V	20	112	100/60	P2HVP	PPP	EX&GOPR	KLE	SE	NC	16	RC	CX, ME
50	339516	49	M	13/12/2016	AP,AD	34	110	100/70	P2HVP	DP	EX&GOPR	E.COLI	BI	NC	10	RC	CX, ME , AM
51	342171	40	M	14/12/2016	AP,D	12	100	110/60	P2HVP	DP	EX&GOPR	NG	PU	NC	21	RC	CX, ME
52	348633	21	F	16/12/2016	AP,F,C	12	100	100/70	P2HVP	PPP	EX&GOPR	NG	PU	NC	10	RC	CX, ME , AM
53	351511	36	M	17/12/2016	AP,F,V	28	96	110/60	P2HVP	DP	EX&GOPR	ECOC	PU	SSI	20	RC	CX, ME
54	354406	30	M	19/12/2016	AP,F,V	10	112	100/60	P2HVP	PPP	EX&GOPR	NG	SE	PNU	10	RC	CX, ME , AM
55	371417	57	M	20/12/2016	AP,AD	12	108	100/70	P2HVP	APP	EX&APPEND	NG	PU	NC	14	RC	CX, ME
56	373365	21	M	21/12/2016	AP,V,AD	36	104	110/80	P2HVP	DP	EX&GOPR	KLE	BI	NC	14	RC	CX, ME , AM
57	376117	31	M	23/12/2016	AP,F,V	12	110	100/60	P2HVP	PPP	EX&GOPR	NG	PU	NC	12	RC	CX, ME
58	381446	45	M	25/12/2016	AP,V	28	100	90/60	P2HVP	DP	EX&GOPR	ECOC	SE	NC	10	RC	CX, ME , AM
59	381725	45	M	26/12/2016	AP,AD	16	110	100/70	P2HVP	DP	EX&GOPR	E.COLI	PU	NC	12	RC	CX, ME
60	384666	30	F	27/12/2016	AP,D	12	98	100/60	P2HVP	APP	EX&APPEND	KLE	SE	NC	14	RC	CX, ME , AM
61	408949	65	M	28/12/2016	AP,V,F,C	12	100	100/70	P2HVP	PPP	EX&GOPR	NG	PU	NC	10	RC	CX, ME
62	221146	20	M	30/12/2016	AP,F,AD	26	96	110/60	P2HVP	DP	EX&GOPR	ECOC	PU	NC	14	RC	CX, ME ,AM

63	221173	69	M	30/12/2016	AP,F,V	10	112	100/60	P2HVP	DP	EX&GOPR	NG	SE	PNU	15	RC	CX, ME
64	226484	59	M	31/12/2016	AP,V	12	108	100/70	P2HVP	APP	EX&APPEND	E.COLI	PU	NC	14	RC	CX, ME ,AM
65	229451	65	M	31/12/2016	AP,V,F,AD	16	104	110/80	P2HVP	DP	EX&GOPR	KLE	BI	NC	18	RC	CX, ME
66	241034	56	M	1/1/2017	AP,F,V	12	110	100/60	P2HVP	DP	EX&GOPR	E.COLI	PU	NC	12	RC	CX, ME, AM
67	243445	46	F	3/1/2017	AP,C	18	100	90/60	P2HVP	PPP	EX&GOPR	ECOC	SE	NC	10	RC	CX, ME
68	211247	56	F	4/1/2017	AP,D	26	110	100/70	P2HVP	IP	EX&R&A	E.COLI	PU	NC	12	RC	CX, ME , AM
69	273691	63	F	5/1/2017	AP,V,F	12	98	100/60	P2HVP	APP	EX&APPEND	NG	SE	NC	14	RC	CX, ME
70	291877	59	M	6/1/2017	AP,V,F	28	110	90/60	P2HVP	IP	EX&R&A	E.COLI	PU	NC	15	RC	CX, ME , AM
71	295411	45	M	8/1/2017	AP,F,C	24	110	100/60	P2HVP	DP	EX&GOPR	ECOC	FE	NC	18	RC	CX, ME
72	311226	69	M	9/1/2017	AP,F	28	96	110/80	P2HVP	APP	EX&APPEND	E.COLI	SE	NC	14	RC	CX, ME , AM
73	323085	20	M	11/1/2017	AP,F,V	16	98	100/60	P2HVP	DP	EX&GOPR	E.COLI	PU	NC	12	RC	CX, ME
74	332471	62	M	12/1/2017	AP,F,AD	36	110	100/70	P2HVP	IP	EX&R&A	ECOC	PU	NC	14	RC	CX, ME , AM
75	366162	48	F	14/1/2017	AP,F,D	20	112	100/60	P2HVP	PPP	EX&GOPR	KLE	SE	NC	16	RC	CX, ME
76	389741	45	M	14/1/2017	AP,F,C	36	110	100/70	P2HVP	DP	EX&GOPR	E.COLI	BI	NC	20	RC	CX, ME , AM
77	222516	50	F	15/1/2017	AP,F	12	100	110/60	P2HVP	APP	EX&APPEND	E.COLI	PU	NC	21	RC	CX, ME
78	257174	47	M	16/1/2017	AP,F	12	100	100/70	P2HVP	PPP	EX&GOPR	E.COLI	PU	NC	10	RC	CX, ME , AM
79	231027	50	M	17/1/2017	AP,F,V	3	96	110/60	P2HVP	DP	EX&GOPR	ECOC	PU	NC	20	RC	CX, ME
80	244011	68	F	19/1/2017	AP,AD	10	112	100/60	P2HVP	PPP	EX&GOPR	NG	SE	NC	15	RC	CX, ME , AM
81	276643	20	M	20/1/2017	AP,V,AD	12	108	100/70	P2HVP	APP	EX&APPEND	E.COLI	PU	NC	21	RC	CX, ME
82	278008	21	M	21/1/2017	AP,F,V	36	104	110/80	P2HVP	DP	EX&GOPR	KLE	BI	NC	18	RC	CX, ME , AM
83	279219	33	F	23/1/2017	AP,V	12	110	100/60	P2HVP	PPP	EX&GOPR	E.COLI	PU	NC	22	RC	CX, ME
84	279670	26	M	25/1/2017	AP,AD	36	100	90/60	P2HVP	PPP	EX&GOPR	ECOC	SE	NC	10	RC	CX, ME , AM
85	296209	42	M	26/1/2017	AP,D	36	110	100/70	P2HVP	IP	EX&R&A	E.COLI	PU	NC	23	RC	CX, ME
86	296369	20	M	27/1/2017	AP,F,C	12	98	100/60	P2HVP	APP	EX&APPEND	NG	SE	NC	14	RC	CX, ME , AM
87	315622	60	M	28/1/2017	AP,F,V	30	110	90/60	P2HVP	IP	EX&R&A	E.COLI	PU	NC	24	RC	CX, ME
88	304423	50	M	29/1/2017	AP,V,F	34	110	100/60	P2HVP	PPP	EX&GOPR	ECOC	FE	NC	18	RC	CX, ME , AM
89	324427	45	M	30/1/2017	AP,V,F	40	96	110/80	P2HVP	APP	EX&APPEND	E.COLI	SE	NC	22	RC	CX, ME
90	325873	44	M	31/1/2017	AP,F,C	76	98	100/60	P2HVP	APP	EX&APPEND	E.COLI	PU	NC	12	RC	CX, ME , AM
91	329428	34	M	1/2/2017	AP,F	36	110	100/70	P2HVP	DP	EX&GOPR	ECOC	PU	NC	24	RC	CX, ME
92	329939	20	M	1/2/2107	AP,F,V	78	112	100/60	P2HVP	PPP	EX&GOPR	KLE	PU	NC	16	D	CX, ME , AM
93	341798	36	M	2/2/2017	AP,F,AD	36	110	100/70	P2HVP	DP	EX&GOPR	E.COLI	BI	NC	24	RC	CX, ME
94	338992	27	M	3/2/2017	AP,F,D	12	100	110/60	P2HVP	APP	EX&APPEND	E.COLI	PU	NC	21	RC	CX, ME ,

																	AM
95	356858	46	M	5/2/2017	AP,F,C	12	100	100/70	P2HVP	PPP	EX&GOPR	E.COLI	PU	NC	10	RC	CX, ME
96	374474	25	F	6/2/2017	AP,F	80	96	110/60	P2HVP	PPP	EX&GOPR	ECOC	PU	NC	20	D	CX, ME , AM
97	385606	50	M	7/2/2017	AP,V,F,C	10	112	100/60	P2HVP	PPP	EX&GOPR	E.COLI	SE	PNU	22	RC	CX, ME
98	341616	35	F	8/2/2017	AP,F,AD	12	108	100/70	P2HVP	APP	EX&APPEND	E.COLI	PU	NC	14	RC	CX, ME , AM
99	213512	42	M	9/2/2017	AP,F,V	26	104	110/80	P2HVP	DP	EX&GOPR	KLE	BI	NC	22	RC	CX, ME
100	219654	47	F	10/2/2017	AP,V	12	110	100/60	P2HVP	PPP	EX&GOPR	E.COLI	PU	NC	12	RC	CX, ME , AM
101	223288	65	M	11/2/2017	AP,V,F,AD	36	100	90/60	P2HVP	DP	EX&GOPR	ECOC	SE	NC	10	RC	CX, ME
102	236086	23	M	13/2/2017	AP,F,V	80	110	100/70	P2HVP	IP	EX&R&A	E.COLI	PU	NC	12	D	CX, ME , AM
103	247811	34	M	16/2/2017	AP,C	12	98	100/60	P2HVP	APP	EX&APPEND	KLE	SE	NC	24	RC	CX, ME
104	238964	60	F	17/2/2017	AP,D	72	110	90/60	P2HVP	IP	EX&PC	E.COLI	PU	NC	26	RC	CX, ME , AM
105	224411	30	M	18/2/2017	AP,F,V	50	110	100/60	P2HVP	APP	EX&APPEND	ECOC	FE	NC	26	RC	CX, ME
106	266411	28	M	20/2/2017	AP,AD	72	96	110/80	P2HVP	PPP	EX&GOPR	E.COLI	PU	NC	14	RC	CX, ME , AM
107	266781	25	M	22/2/2017	AP,V,AD	10	98	100/60	P2HVP	DP	EX&GOPR	NG	PU	NC	28	RC	CX, ME
108	273732	48	M	24/2/2017	AP,F,V	72	110	100/70	P2HVP	IP	EX&PC	ECOC	PU	FF	14	RC	CX, ME , AM
109	274146	39	M	26/2/2017	AP,V	10	112	100/60	P2HVP	APP	EX&APPEND	NG	SE	NC	29	RC	CX, ME
110	275503	45	M	27/2/2017	AP,AD	72	110	100/70	P2HVP	DP	EX&GOPR	E.COLI	BI	FF	22	RC	CX, ME , AM
111	280998	55	F	28/2/2017	AP,D	12	100	110/60	P2HVP	APP	EX&APPEND	E.COLI	PU	NC	26	RC	CX, ME
112	287393	23	F	1/3/2017	AP,F,C	12	100	100/70	P2HVP	PPP	EX&GOPR	E.COLI	PU	NC	10	RC	CX, ME , AM
113	257492	29	M	5/3/2017	AP,F,V	10	96	110/60	P2HVP	IP	EX&PC	ECOC	PU	NC	26	RC	CX, ME
114	295890	24	M	7/3/2017	AP,F,V	10	112	100/60	P2HVP	PPP	EX&GOPR	E.COLI	SE	NC	15	RC	CX, ME , AM
115	306697	65	M	10/3/2017	AP,AD	12	108	100/70	P2HVP	IP	EX&PC	ACI	PU	NC	26	RC	CX, ME
116	309070	48	M	11/3/2017	AP,V,AD	12	104	110/80	P2HVP	DP	EX&GOPR	KLE	BI	NC	23	RC	CX, ME , AM
117	318211	19	M	14/3/2017	AP,F,V	12	110	100/60	P2HVP	IP	EX&PC	E.COLI	PU	NC	26	RC	CX, ME
118	326689	16	M	16/3/2017	AP,V	12	100	90/60	P2HVP	DP	EX&GOPR	ECOC	SE	NC	10	RC	CX, ME , AM
119	331925	38	M	18/3/2017	AP,AD	6	110	100/70	P2HVP	DP	EX&GOPR	NG	PU	NC	27	RC	CX, ME
120	338574	49	M	20/3/2017	AP,D	12	98	100/60	P2HVP	APP	EX&APPEND	NG	SE	NC	14	RC	CX, ME , AM
121	344770	37	M	23/3/2017	AP,V,AD	12	110	100/70	P2HVP	DP	EX&GOPR	NG	FE	NC	10	RC	CX, ME
122	353005	46	M	26/3/2017	AP,F,V	12	100	100/60	P2HVP	DP	EX&GOPR	NG	SE	NC	10	RC	CX, ME , AM
123	359034	34	M	27/3/2017	AP,V	10	110	100/70	P2HVP	APP	EX&APPEND	NG	PU	NC	7	RC	CX, ME
124	356092	42	M	29/3/2017	AP,AD	12	98	110/60	P2HVP	DP	EX&GOPR	EBAC	PU	NC	14	RC	CX, ME , AM
125	367456	36	M	31/3/2017	AP,D	11	110	100/70	P2HVP	IP	EX&R&A	NG	SE	NC	6	RC	CX, ME

126	368693	35	F	1/4/2017	AP,F,C	12	110	110/60	P2HVP	DP	EX&GOPR	E.COLI	BI	NC	24	RC	CX, ME , AM
127	300484	20	F	4/4/2017	AP,F,V	10	96	100/60	P2HVP	APP	EX&APPEND	NG	PU	NC	8	RC	CX, ME
128	300529	21	M	5/4/2017	AP,V,F	12	98	100/70	P2HVP	DP	EX&GOPR	NG	PU	NC	10	RC	CX, ME , AM
129	300889	22	M	6/4/2017	AP,V,F	12	110	110/80	P2HVP	IP	EX&R&A	ECOC	PU	NC	8	RC	CX, ME
130	315972	31	M	8/4/2017	AP,F,C	10	112	100/60	P2HVP	DP	EX&GOPR	NG	SE	NC	15	RC	CX, ME , AM
131	277409	35	M	10/4/2017	AP,F	6	110	90/60	P2HVP	DP	EX&GOPR	NG	PU	NC	8	RC	CX, ME
132	280112	24	M	12/4/2017	AP,F,V	8	100	100/70	P2HVP	PPP	EX&GOPR	NG	BI	NC	26	RC	CX, ME , AM
133	280043	20	F	14/4/2017	AP,V,AD	76	100	100/60	P2HVP	DP	EX&GOPR	E.COLI	PU	FF	7	RC	CX, ME
134	277749	40	M	20/4/2017	AP,F,V	10	96	90/60	P2HVP	DP	EX&GOPR	NG	SE	NC	10	RC	CX, ME , AM
135	285079	27	M	26/4/2017	AP,V	8	112	100/60	P2HVP	DP	EX&GOPR	NG	PU	NC	6	RC	CX, ME
136	288275	21	M	30/4/2017	AP,AD	8	108	110/80	P2HVP	PPP	EX&GOPR	NG	SE	NC	14	RC	CX, ME , AM
137	234675	35	M	16/5/2017	AP,D	12	104	100/60	P2HVP	DP	EX&GOPR	NG	FE	IAA	8	RC	CX, ME
138	231607	40	M	26/5/2017	AP,F,C	12	110	100/70	P2HVP	APP	EX&APPEND	NG	SE	NC	10	RC	CX, ME , AM
139	228343	35	M	1/6/2017	AP,F,V	78	100	100/60	P2HVP	DP	EX&GOPR	EBAC	PU	IAA	10	RC	CX, ME
140	217885	35	M	2/6/2017	AP,V,F	12	110	100/70	P2HVP	APP	EX&APPEND	NG	PU	NC	10	RC	CX,ME, AM