"Comparison of Modified APACHE II scoring and Mannheim's peritonitis index in prognosis of peritonitis"

By

Dr. P. Naveen

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Under the Guidance of

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Dr. P NAVEEN

ABSTRACT

INTRODUCTION:

Acute generalized peritonitis due to underlying hollow viscus perforation is a critical & life-threatening condition. It is often associated with significant morbidity and mortality.[1]

Categorizing patients into different risk groups would help prognosticate the outcome, select patients for intensive care and determine operative risk, thereby helping to choose the nature of the operative procedure, e.g. damage control vs. definitive procedure.[2] The mortality of intra-abdominal infection is related mainly to the severity of the patient's systemic response and his premorbid physiologic reserves, estimated best using the Acute Physiology and Chronic Health Evaluation II (APACHE-II) scoring system.[4]

Various authors have reported APACHE II to be a better system for prognostication of the outcome of patients with peritonitis, while others concluded that MPI provides a more reliable means of risk evaluation.[2]

OBJETIVES OF THE STUDY:

To compare the efficacy of Modified APACHE II scoring and Mannheims Peritonitis Index (MPI) in predicting the outcome of patients with peritonitis secondary to hollow viscous perforation.

METHODOLOGY:

SOURCE OF DATA:

A prospective clinical study was conducted on 80 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 from December 2013 to June 2015.

INCLUSION CRITERIA:

All patients diagnosed to have peritonitis secondary to hollow viscus perforation

EXCLUSION CRITERIA:

1. Patients less than 16 years of age.
2. Post-operative peritonitis.
3. Gynaecological causes of peritonitis.
4. Spontaneous bacterial peritonitis.
5. Peritonitis secondary to ventriculo-peritoneal shunts.
6. Blunt and penetrating abdominal injuries.

APACHE II and Mannheim Peritonitis Index (MPI) scoring systems were assigned to all the patients in order to calculate their individual risk of mortality and survival at the time of admission.

RESULTS:

Highest mortality was seen in the age group of 41-50years and 61-70years (37.5%) and in cases with gastric (37.5%), unknown(25%) and colonic(12.5%) perforations. Mortality was observed more in males (n=5) compared to females(n=3). Patients with longer duration of peritonitis had a higher mortality rate. Patients also developed post-operative complications like surgical site infections(42.5%), respiratory(22.5%) and sepsis(17.5%). Mean apache II scores in survivors were 7.5±5.3 and in non survivors 19.7±4.7. A mean MPI score of 15.86±6.57 was seen

among survivors and a mean MPI score of 32.13±4.67 was seen among non-survivors.

Age over 50 years, longer duration of perforation, extent of peritoneal contamination and associated medical illness adversely affect the prognosis of patients in perforative peritonitis. Delayed presentation had an important adverse effect on both mortality & morbidity. The type and extent of peritoneal contamination seem to have a bearing on mortality. Patients with diffuse peritonitis and with fecal contamination did worse.

CONCLUSION:

As per our analyses APACHE II and MPI both had good sensitivity and specificity. Both the scoring systems were accurate, sharp and reliable in predicting outcome. In all these aspects APACHE II was found to be better than MPI in prediction. An efficient scoring system is one which is accurate and sharp in predicting the prognosis and also reliable i.e., which can be reproduced if needed to stratify the patients to risk category. This will help us to divert the resources of the hospital for appropriate patient care and in decisions like transfer of patients to intensive care unit, the choice of more effective antibiotics and treatment modality.

Keywords: Peritonitis; Mannheim Peritonitis Scoring; Perforation; Prognosis.

LIST OF ABBREVATIONS:

AIDS	Acquired Immunodeficiency Syndrome
APACHE II	Acute Physiological and Chronic Health Evaluation
APS	Acute Physiological Score
ASA	American Society of Anesthesiologists
AUC	Area Under Curve
BP	Blood pressure
CAPD	Chronic Ambulatory Peritoneal Dialysis
CI	Confidence Interval
CMV	Cytomegalovirus
CNS	Central Nervous System
COAD	Chronic Obstructive Airway Disease
CT	Computed Tomography
ERCP	Endoscopic Retrograde Cholangiopancreatography
FiO2	Fraction of inspired oxygen
GCS	Glasgow Coma Score
K	Potassium
MEFV	Mediterranean fever
MPI	Mannheim Peritonitis Index
MR	Mortality Rate
N	Total number of patients

Na+	Sodium
NSAID	Non-Steroidal Anti Inflammatory Drug
NS	Non Survivors
PaO_2	Partial pressure of oxygen
PBP	Primary Bacterial Peritonitis
PCO_2	Partial pressure of carbon-dioxide
PEEP	Positive End Expiratory Pressure
PIA	Peritonitis Index Altona
POSSUM	Physiological and Operative Severity Score for Enumeration of Mortality and Morbidity
PPFA	Periportal Free Air
PULP	Peptic Ulcer Perforation score
ROC	Receiver Operative Curve
SD	Standard Deviation
WBC	White Blood Cells

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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 & life-threatening condition. It is a common surgical emergency in most of the general surgical units across the world. It is often associated with significant morbidity and mortality.[1]

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.

High-risk patients require timely and aggressive treatment especially in severe peritonitis. To select them reasonably well, evaluation through a prognostic scoring system is the approach of choice. 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 includes` patient-related factors, disease-specific factors, diagnostic and therapeutic interventions. Categorizing patients into different risk groups would help prognosticate the outcome, select patients for intensive care and determine operative risk, thereby helping to choose the nature of the operative procedure, e.g. damage control vs. definitive procedure.[2]Various scoring systems have been used to assess the prognosis and outcome of patients with peritonitis. Those used include the Acute Physiological and Chronic Health Evaluation score (APACHE II)(1985), the Mannheim Peritonitis Index (MPI)(1983), the

Peritonitis Index Altona (PIA), The Sepsis Severity Score(1983), and the Physiological and Operative Severity Score for Enumeration of Mortality and Morbidity (POSSUM).[3]

The mortality of intra-abdominal infection is related mainly to the severity of the patient's systemic response and his premorbid physiologic reserves, estimated best using the Acute Physiology and Chronic Health Evaluation II (APACHE-II) scoring system.[4]

The Mannheim peritonitis index (MPI) emerged as a reliable marker for assessing the severity and prognosis of intra-abdominal infection with sensitivity and specificity comparable to APACHE II score which has been adopted as the gold standard by Surgical Infection Society. This score was designed specifically for peritonitis and it combines preoperative and operative data and is easy to apply.[3]

Various authors have reported APACHE II to be a better system for prognostication of the outcome of patients with peritonitis, while others concluded that MPI provides a more reliable means of risk evaluation.[2]

AIMS AND OBJECTIVES

To compare the efficacy of Modified APACHE II scoring and Mannheims peritonitis Index (MPI) in predicting the outcome of patients with peritonitis secondary to hollow viscous perforation.

REVIEW OF LITERATURE

The first scientific theory of this disease was developed by Hippocrates and the Koic School of Medicine, the first clinical description of this disease: "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 furred, the skin shiny. The patient avoids all movements and breathes are 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".[5]

B.C. 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.

Galen performed several abdominal procedures as a surgeon to the Roman gladiators, he also observed and described the anatomy of the small intestine.

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.

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

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

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

Wegner in 1877, was the first to perform experimental peritoneal lavage.

Putnam in 1922 studied the properties of the peritoneal membrane.

Kriege in 1892 did the first successful closure of perforated gastric ulcer.[6]

Johann Von Mikulicz-Radecki in 1880, operated successfully on a patient with perforated gastric ulcer; 4 years later he exteriorized a perforated sigmoid colon.

A perforated typhoid ulcer was closed with a suture in 1885.[7]

Earlier, in the first half of the century, the Paris Clinical School developed the modern physical examination and stressed the correlation between clinical findings and pathologic processes.[7]

Introduction of anesthesia by Horace Wells and Thomas G Morton, in 1846 in Boston was the most important development.[8]The first anesthesia was accomplished by Ether, but 1 year later, Simpson of Edinburgh introduced chloroform.[9] Although laparotomy quickly became a relatively safe operation, there were fatalities that could not be explained readily.

Georg Wegener in Berlin was the first to conduct a series of logical experiments about the physiology of the peritoneal cavity. His results were reported to the German Surgical Society in 1876.[7] The current therapy of peritonitis was summarized by Martin Kirschner[10] 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.

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 constitutes 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 similar manner, cells of the visceral layer of lateral plate mesoderm form the visceral layer of the serous membranes covering the abdominal organs, lungs, and heart.[11]

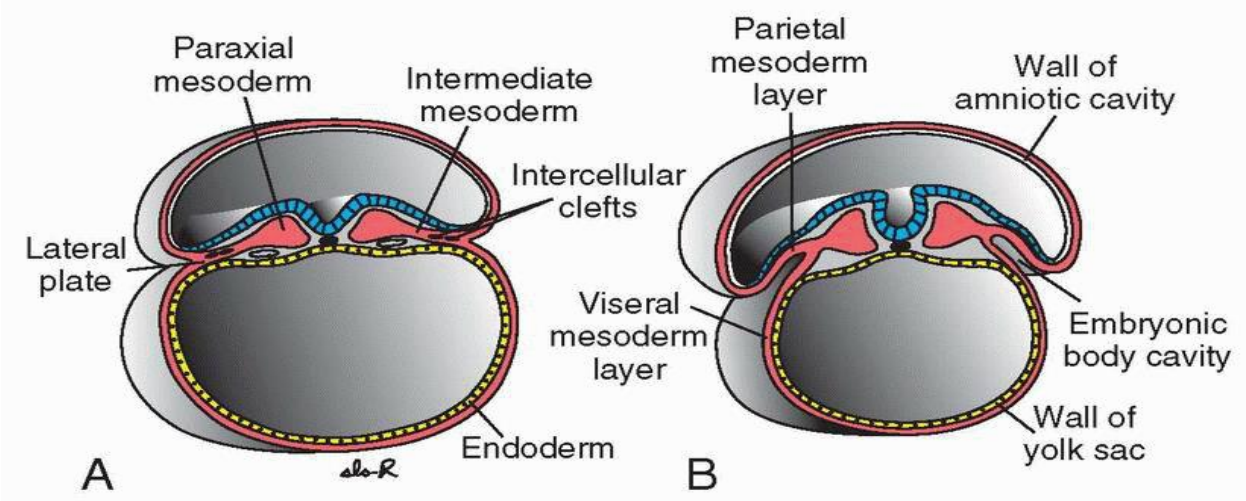


Figure 1: Embryology of peritoneum. A. Transverse section through an embryo of approximately 19 days B. Section through an embryo of approximately 20 days[11].

Formation of the Peritoneal Ligaments and Mesenteries[12]

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 gastrosplenicomentum, the splenorenal ligament, the greater omentum, and the mesenteries of the small and large intestines.

Formation of the Lesser and Greater Peritoneal Sacs[12]

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[12]

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.

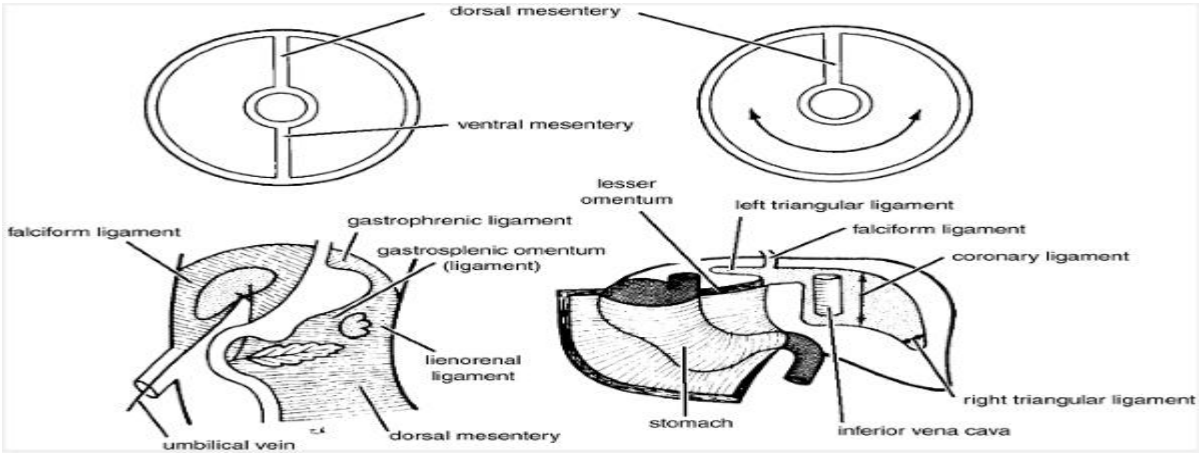


Figure 2: Ventral and dorsal mesenteries and the organs that develop within them

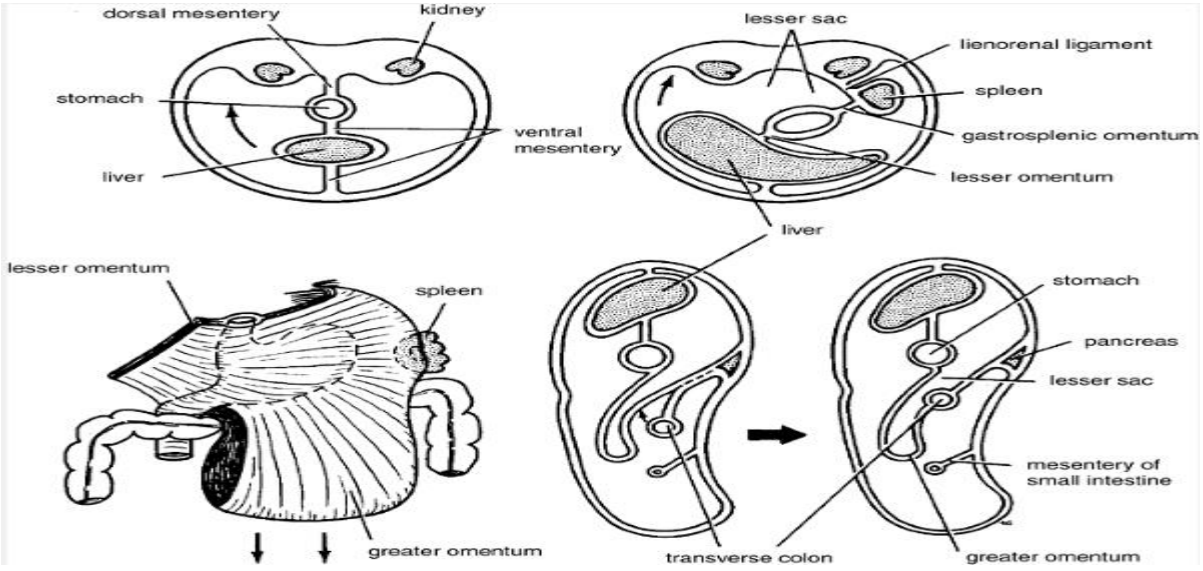


Figure 3: Rotation of the stomach and the formation of the greater omentum and lesser sac.

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. Consequently, the more superiorly placed abdominal organs (spleen, liver, part of the kidneys, and stomach) are protected by the thoracic cage. The greater pelvis (expanded part of the pelvis superior to the pelvic inlet) supports and partly protects the lower abdominal viscera (part of the ileum, cecum, and sigmoid colon).[13]

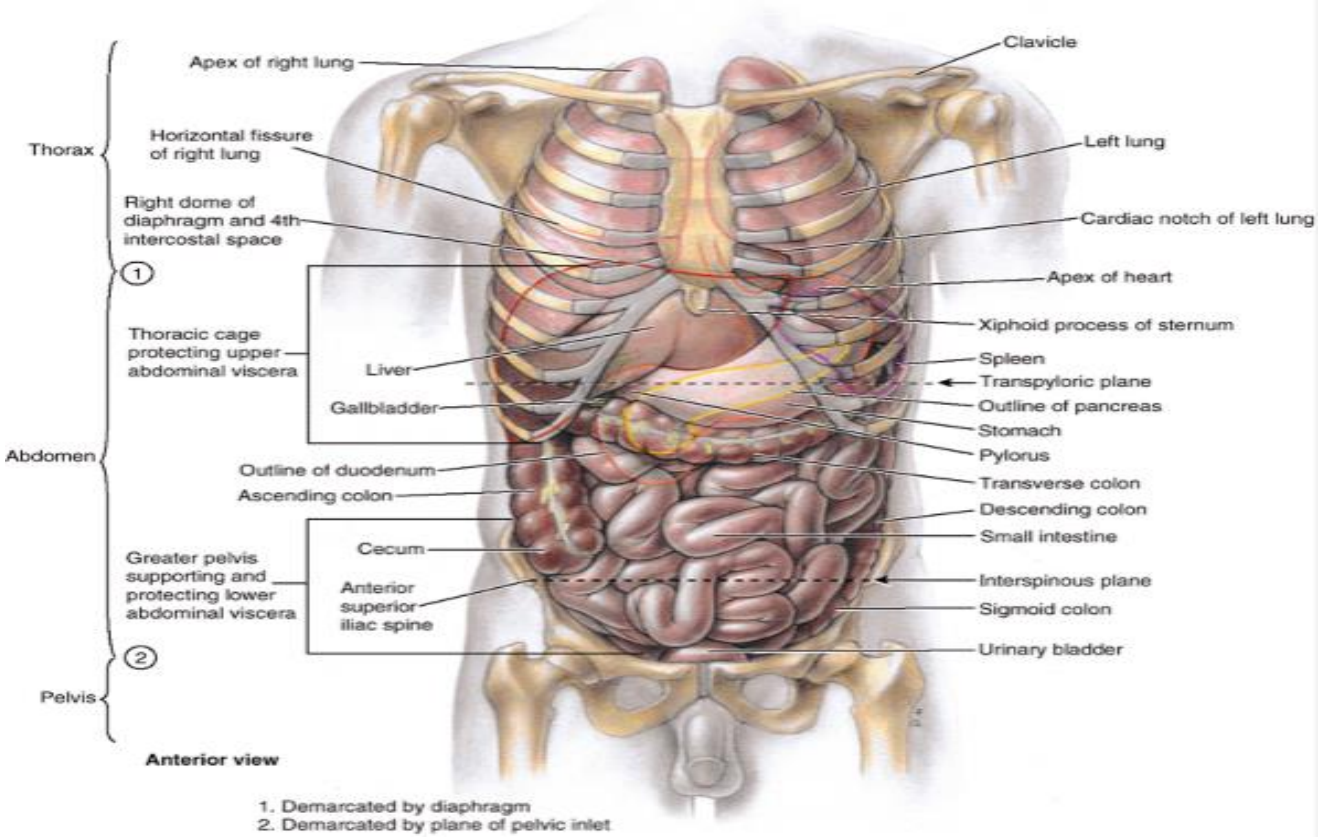


Figure 4: Overview of viscera of thorax and abdomen in situ[13].

In summary, the abdominal cavity is[13]

- 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, the surface area of its lining membrane (2 m^2 in an adult) being nearly equal to that of the skin. It can be 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 viscera 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.[6]

Table 1: Parts of the Peritoneum[6]

Omenta	Greater omentum
	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[6]

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[6]

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[6]

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.[6]

Spaces in the peritoneum:

The peritoneal cavity is subdivided into interconnected compartments or spaces by 11 ligaments and mesenteries.[14]

The peritoneal ligaments or mesenteries include the[14]

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.

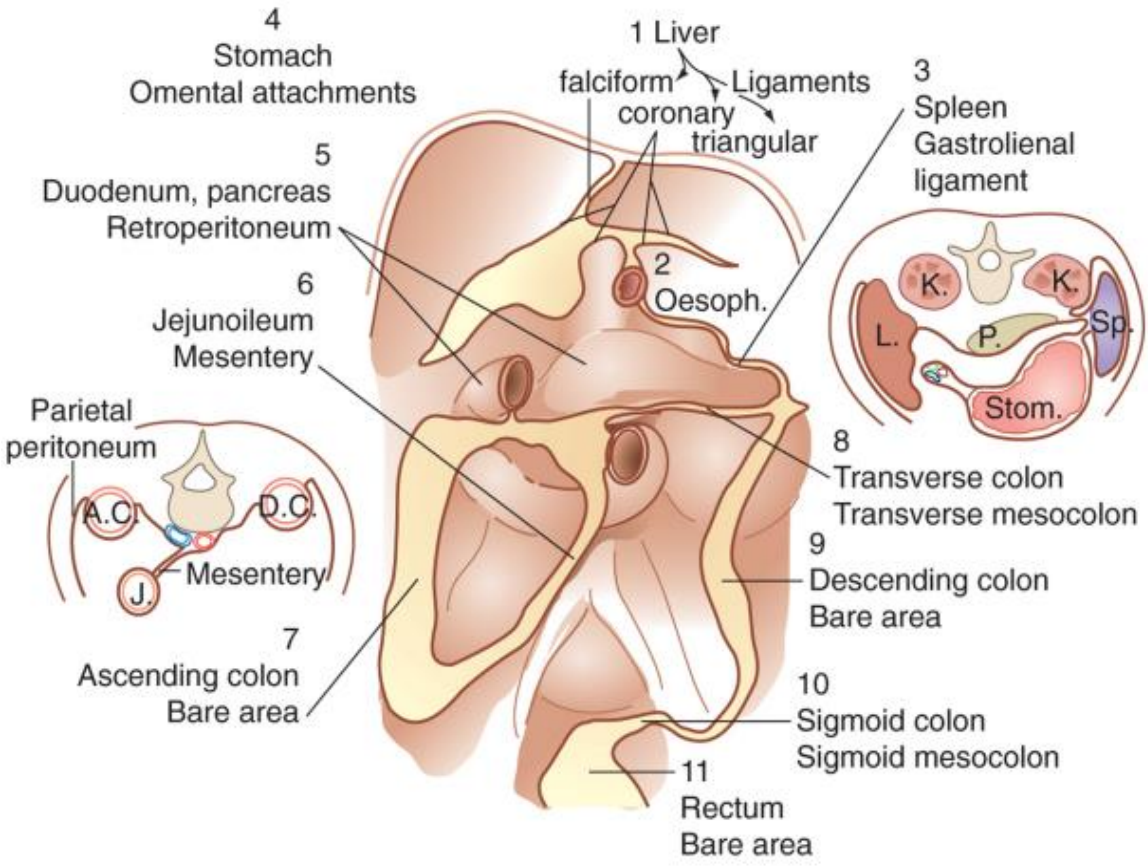


Figure 5: Peritoneal ligaments and mesenteric reflections in the adult. [14]

Peritoneal recesses, Spaces, and Gutters

These ligaments partition the abdomen into nine potential spaces:[14]

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[14]. These attachments partition the abdomen into nine potential spaces and are represented in figure no 6.

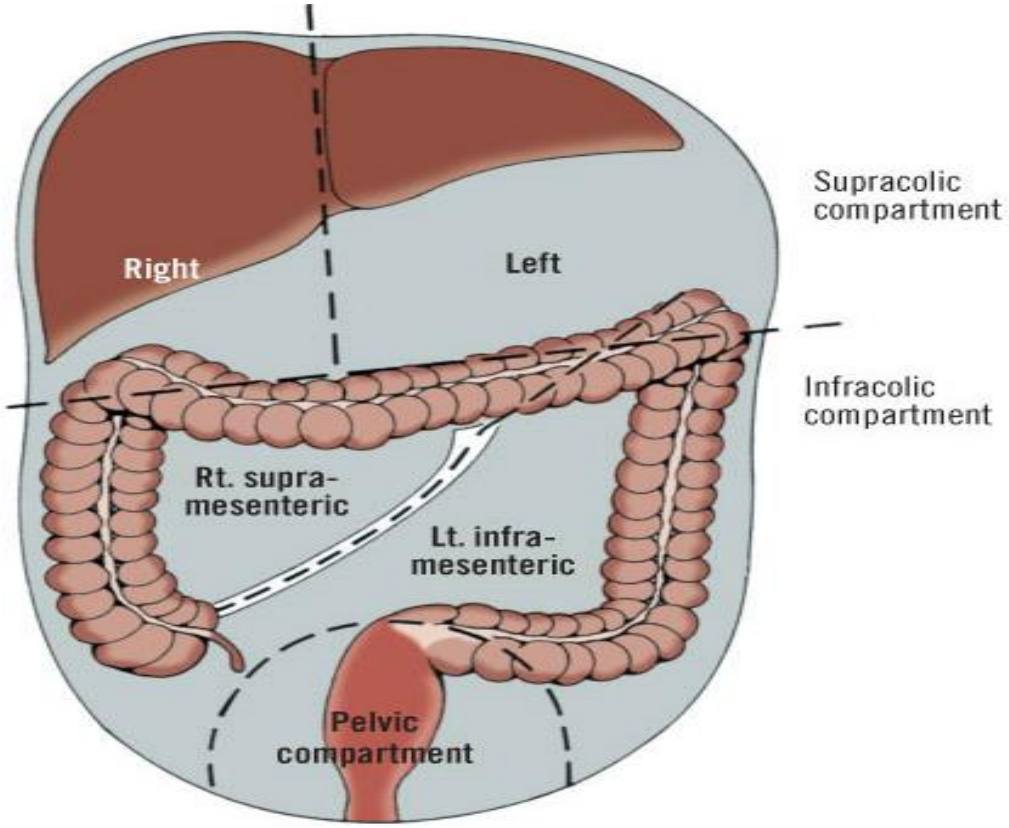


Figure 6: Spaces in the peritoneum:

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: 7 and 8). The greater sac is the main compartment and extends from the diaphragm down into the pelvis.

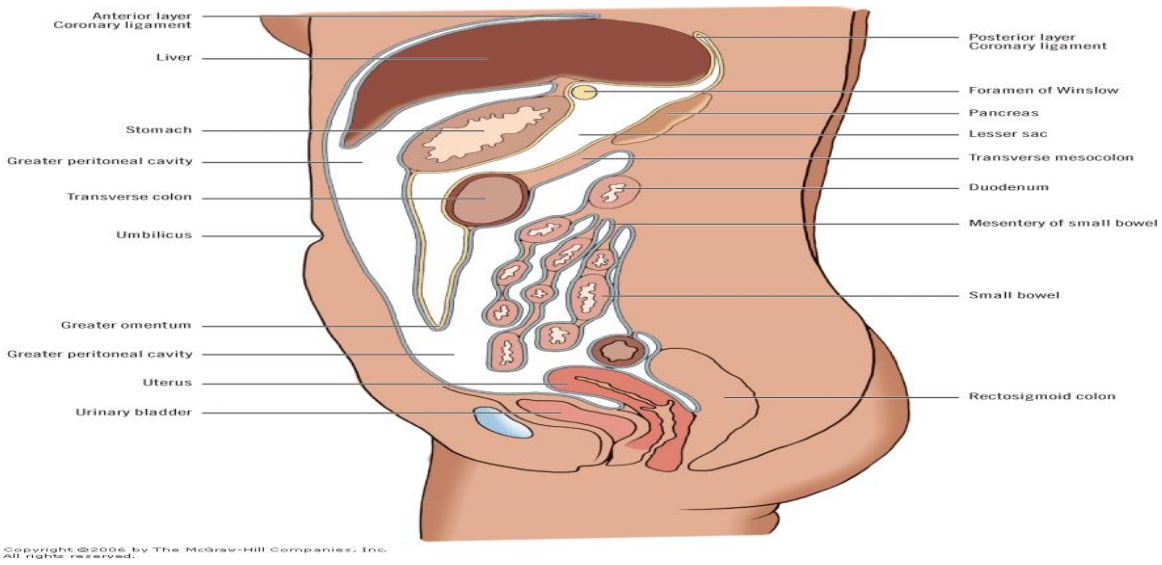


Figure 7: Vertical disposition of the peritoneum (abdominopelvic cavity).[6]

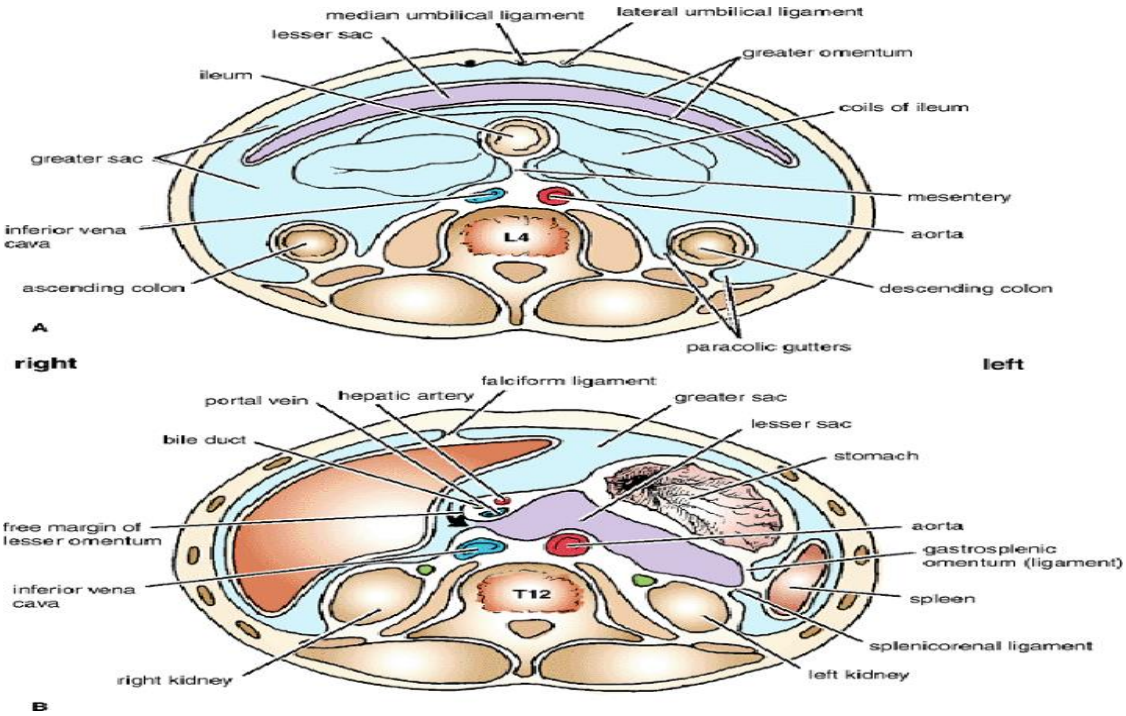


Figure 8: Transverse sections of the abdomen showing the arrangement of the peritoneum[12]

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 (the main part of the peritoneal cavity) through the opening of the lesser sac, or epiploic foramen (Foramen of Winslow).

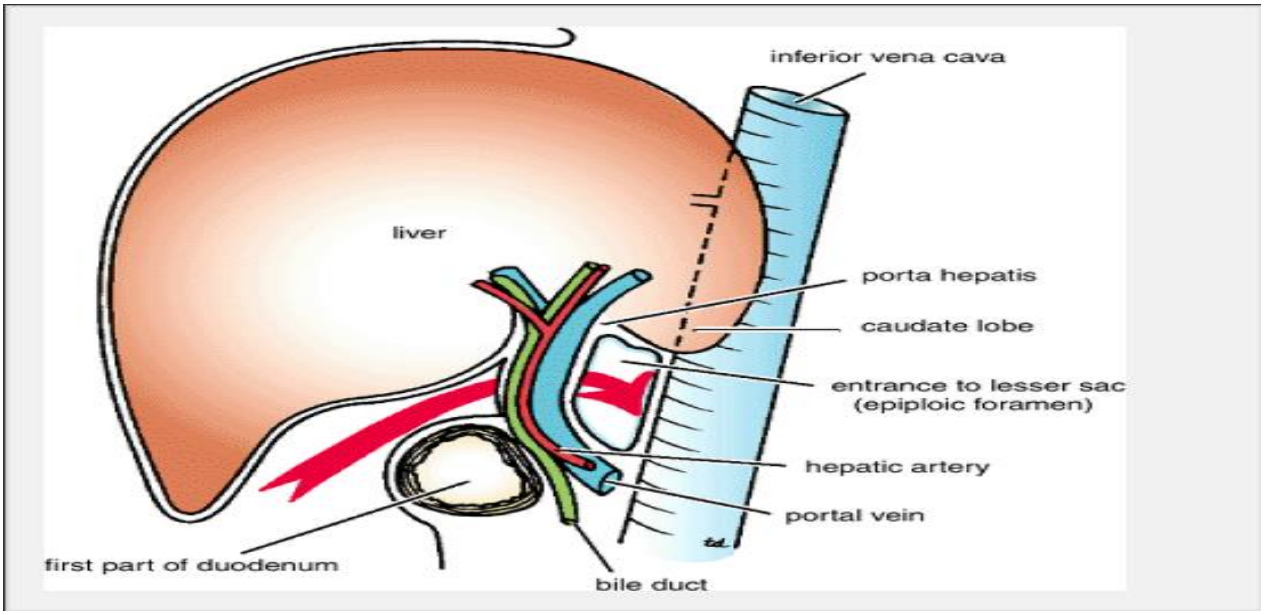


Figure 9: lesser sac

Duodenal Recesses

Close to the duodenojejunal junction, there may be four small pocketlike pouches of peritoneum called the superior duodenal, inferior duodenal, paraduodenal, and retroduodenal recesses as depicted in figure 10.

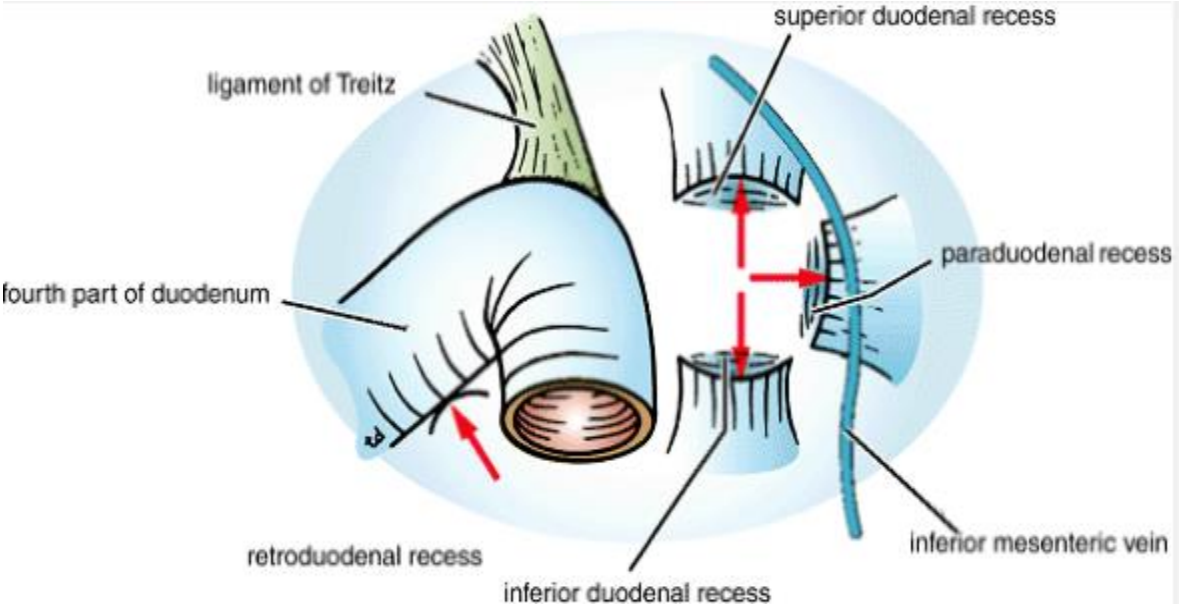


Figure 10: Peritoneal recesses forming the paraduodenal recess.

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. 11).

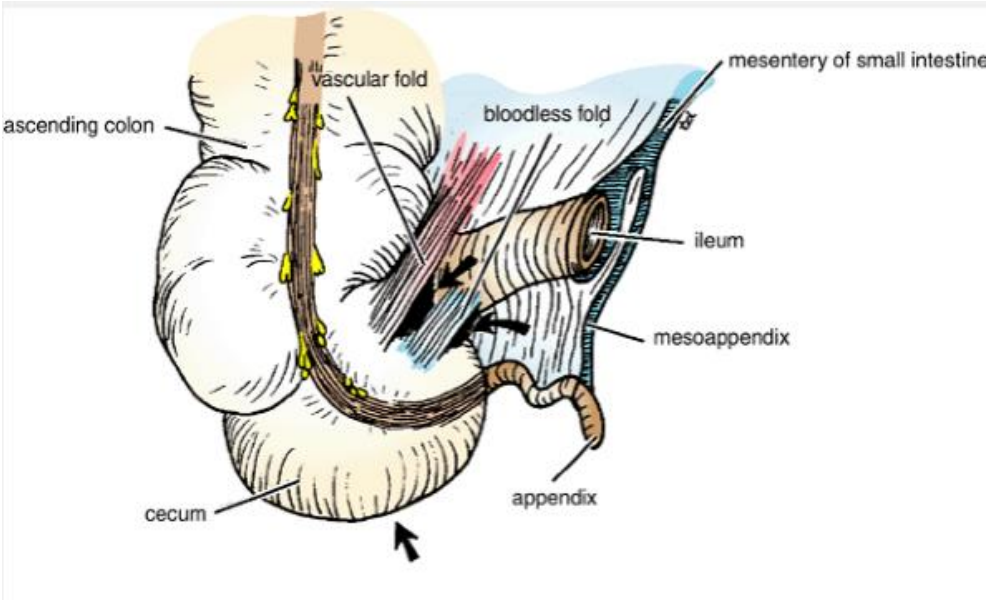


Figure 11: cecal recess

Inter-sigmoid Recess

The inter-sigmoid recess is situated at the apex of the inverted, V-shaped root of the sigmoid mesocolon (Fig. 12); its mouth opens downward.

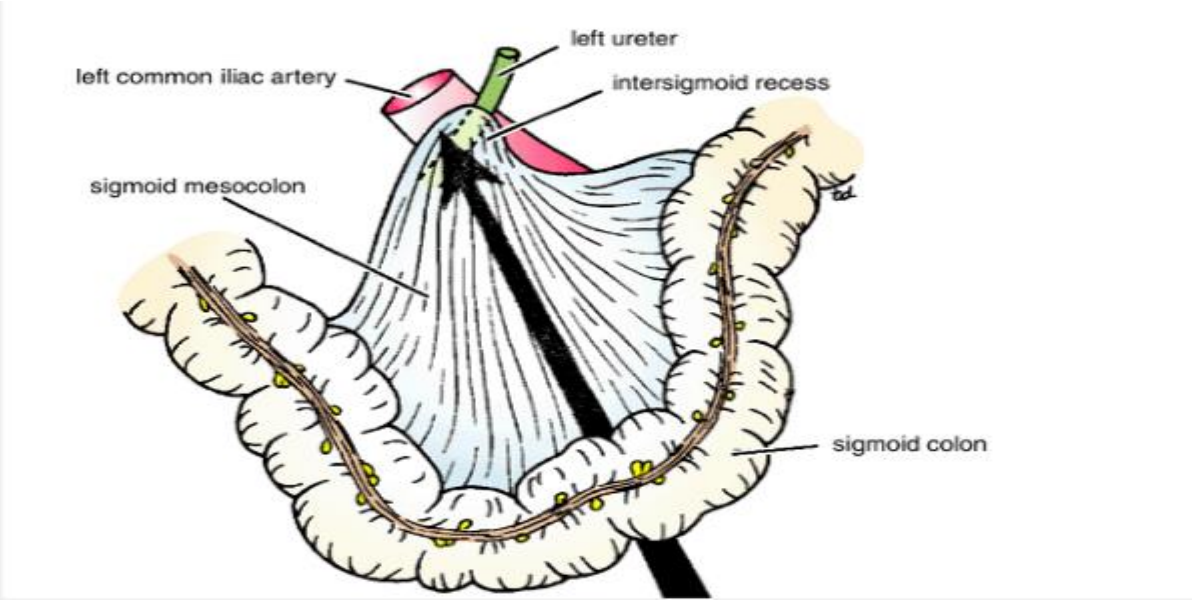


Figure 12: Inter sigmoid recess

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 sites for the 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).[15]

Peritoneal spread of disease[6]

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

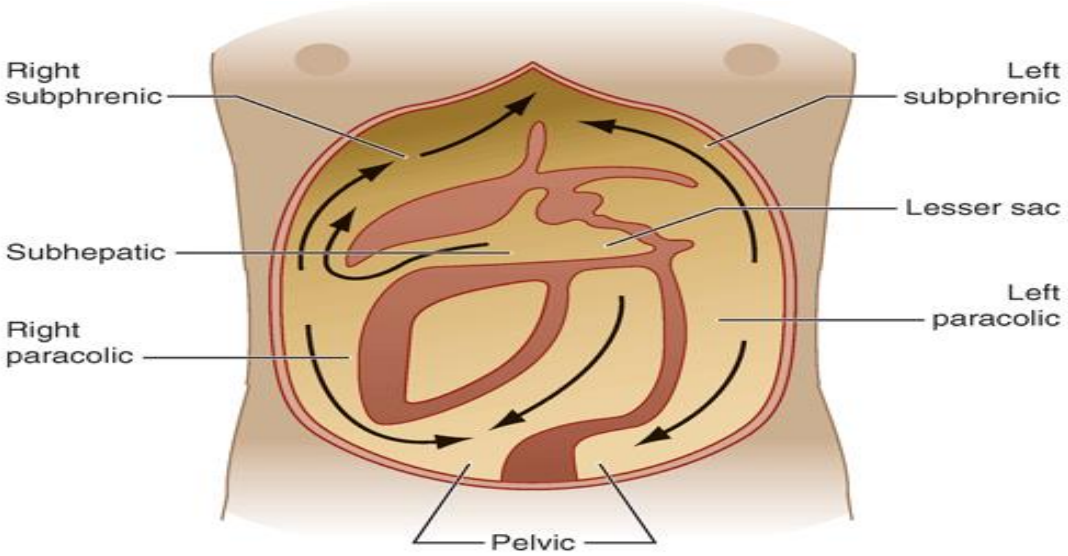
- Location of the source and the rate of fluid production
- Pressure differences in the abdomen
- Mesenteric partitions and peritoneal fossae
- Position of the body in relation to gravity

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.[16]

The circulation of fluid and potential areas for abscess formation is shown in figure 13 and 14). 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.[15]

Figure 13: peritoneal spread of disease[15]



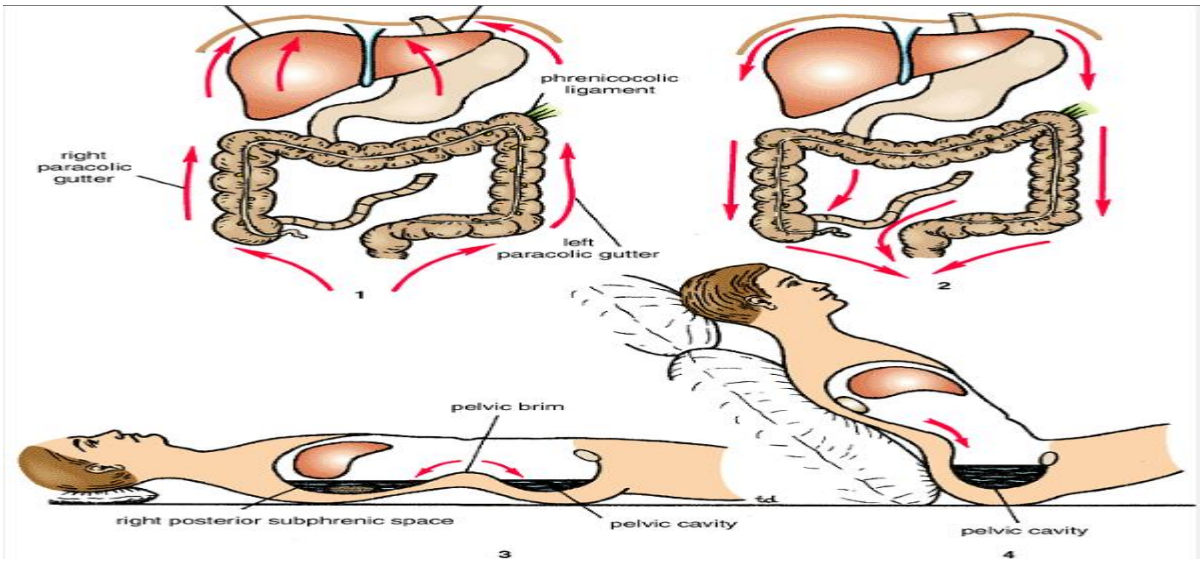


Figure 14: Direction of flow of the peritoneal fluid. 1. Normal flow upward to the subphrenic spaces. 2. Flow of inflammatory exudate in peritonitis. 3. The two sites where inflammatory exudate tends to collect when the patient is nursed in the supine position. 4. Accumulation of inflammatory exudate in the pelvis when the patient is nursed in the inclined position.

Intraperitoneal and Retroperitoneal Relationships[12]

The terms intraperitoneal and retroperitoneal are used to describe 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 good examples of intraperitoneal organs. Retroperitoneal organs lie behind the peritoneum and are only partially covered with visceral peritoneum. 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: [13]

- Intra peritoneal organs are almost completely covered with visceral peritoneum (e.g., the stomach and spleen). Intra peritoneal organs have conceptually, if not literally, invaginated into the closed sac, like pressing your fist into an inflated balloon.
- Extra peritoneal, retroperitoneal, and sub peritoneal organs are outside the peritoneal cavity and are only partially covered with peritoneum (usually on just one surface).

The peritoneal cavity is within the abdominal cavity and continues inferiorly into the pelvic cavity. The peritoneal cavity is completely closed in males; however, there is a communication pathway in females to the exterior of the body through the uterine tubes, uterine cavity, and vagina. This communication constitutes a potential pathway of infection from the exterior.[13]

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.[17]

Factors influencing diaphragmatic uptake of fluid and particles[18].

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[14]:

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.[14]

Paths to peritoneal infection[17]**:**

- 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[19]

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 stomata in the diaphragmatic peritoneum and is absorbed into lymphatic lacunae. 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. Phospholipid surfactants produced by the peritoneal mesothelial cells 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 are Escherichia coli, aerobic and anaerobic streptococci, and bacteroides. Less frequently Clostridium welchii is found; still less frequently staphylococci or Klebsiella pneumoniae (Friedländer's bacillus).[17]

Source of peritonitis:

Stomach and duodenum are the major source of peritonitis.[6]

Figure 15: Source of peritonitis in 567 patients.[7]

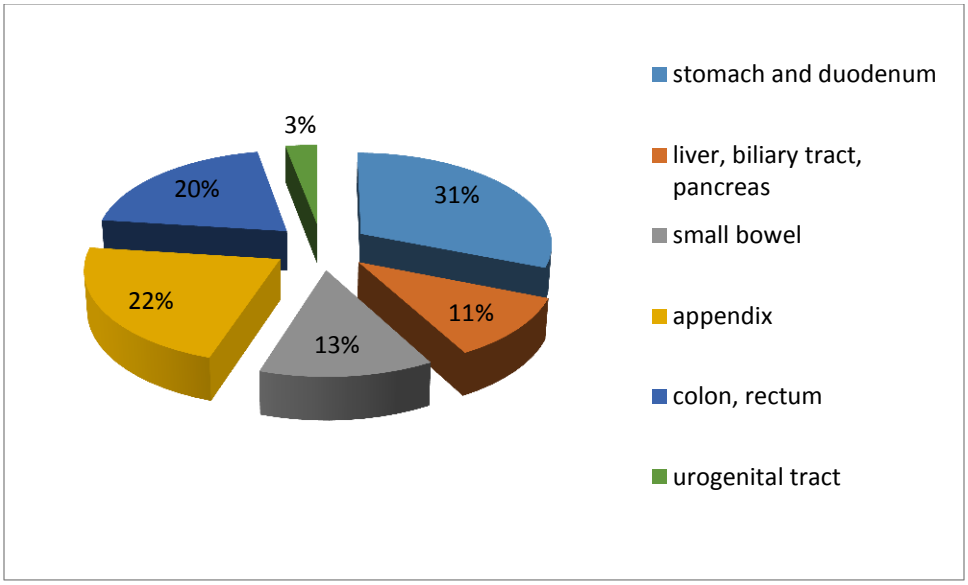
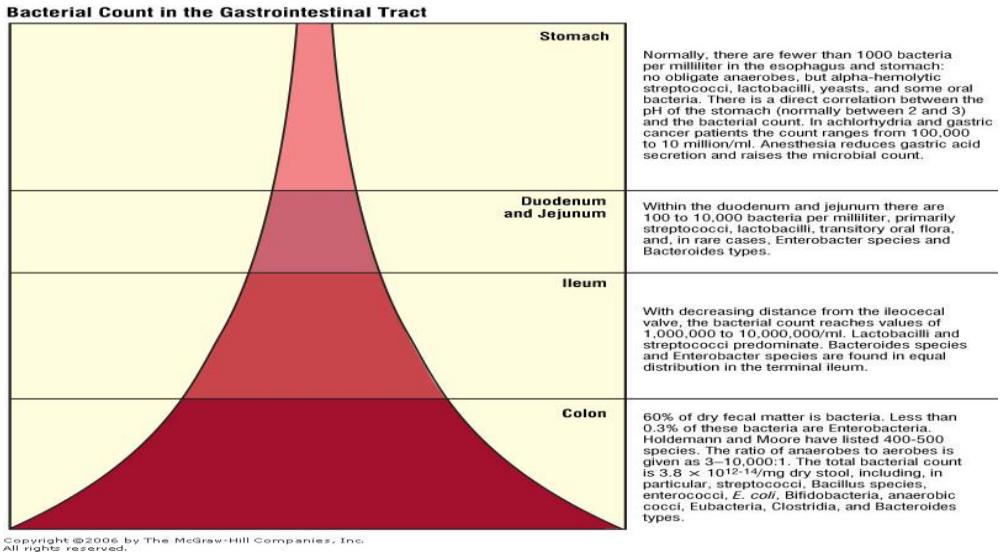


Figure 16: Bacterial count in gastrointestinal tract



Microorganisms in peritonitis[17]

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.[15]

Secondary peritonitis

Secondary peritonitis develops when bacteria contaminate the peritoneum as a result of spillage from an intraabdominal viscus. The organisms found almost always constitute a mixed flora in which facultative gram-negative bacilli and anaerobes predominate, 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 only 10^{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.[15]

Factors favouring localization of peritonitis[17]

a. Anatomical:

Transverse colon and Transverse mesocolon deters the spread of infection from supracolic to infracolic compartment of peritoneal cavity. When supracolic compartment 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.

b. 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 reddened 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 serous inflammatory exudates rich in leukocytes and plasma proteins that soon 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[17]

- 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 (‘immune deficiency’) may result from drugs (e.g. steroids), disease (e.g. AIDS) or old age.

Sequelae leading to multiorgan failure[20]

Sepsis is the major risk factor in the development of multiorgan failure syndrome (MOFS). OFS 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 105 / mm^3 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.[4]

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[4]		
1	Primary peritonitis Diffuse bacterial peritonitis in the absence of disruption of intraabdominal hollow viscera	A. Spontaneous peritonitis in children
		B. Spontaneous peritonitis in adults
		C. Peritonitis in patients with CAPD
		D. Tuberculous and granulomatous peritonitis
2	Secondary peritonitis Localized (abscess) or diffuse peritonitis originating from a defect in abdominal viscus	A. Acute perforative peritonitis 1. Gastrointestinal perforation 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[4]

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.[17]

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 cheek- 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:[15] (PBP)

In adults, primary bacterial peritonitis (PBP) occurs most commonly in conjunction with cirrhosis of the liver (frequently as a result of alcoholism). 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.[15]

SECONDARY PERITONITIS:

Secondary peritonitis develops when bacteria contaminate the peritoneum as a result of spillage from an intraabdominal viscous. 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.[15]

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[21].

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[22].

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 a third of gastric perforations are due to gastric carcinomas.The overall mortality rate is relatively high (~20-40%), largely because of complications such as septic shock and multi organ failure.[23]

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 tomach.[22]

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.[24]

The options for surgical treatment of perforated duodenal ulcer are simple patch closure, patch closure and HSV, or patch closure and V+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[25]

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 fevers 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. Abscesses can also form in the retroperitoneum due to perforation of a retrocecal appendix, or in the liver from hematogenous spread of infection through the portal venous system. An intraperitoneal abscess could fistulize to the skin, resulting in an enterocutaneous 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[24]

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.[24] 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[22]

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 pathologic 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 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 titers 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:[26]

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.[27]
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[28].

CLINICAL PRESENTATION[29,30]

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 faeculent. 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**[31,32,33]

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**[34]

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'ssign . 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

(i) Peritoneal dialysis

(ii) Perforated jejuna diverticulosis

*(iii)*Perforated cyst in pneumatosis intestinalis

(iv) Tracking down from a pneumomediastinum

(v) Stercoral ulceration

(vi) Entry of air through the fallopian tubes

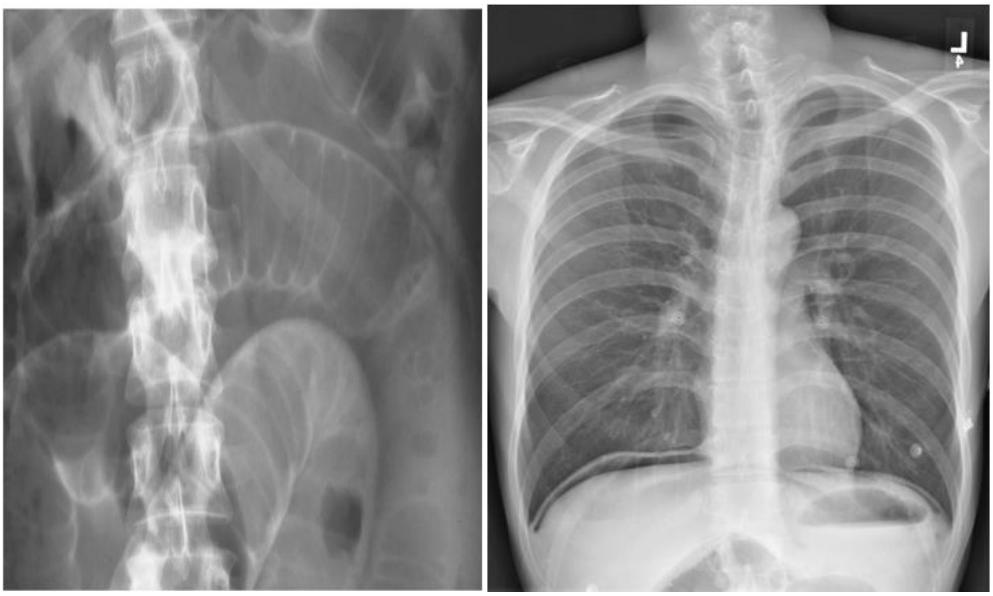


Figure 17: Radiography: Erect chest radiograph or erect abdomen radiograph

a. 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.
b. Air under diaphragm

❖ **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.[19]

❖ **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.[35]

Bowel wall thickening:

> 8 mm in stomach and duodenum,

> 3 mm in jejunum and ileum,

> 6 mm of the appendiceal caliber 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[35].

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.[30]

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.[36]

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.[36]

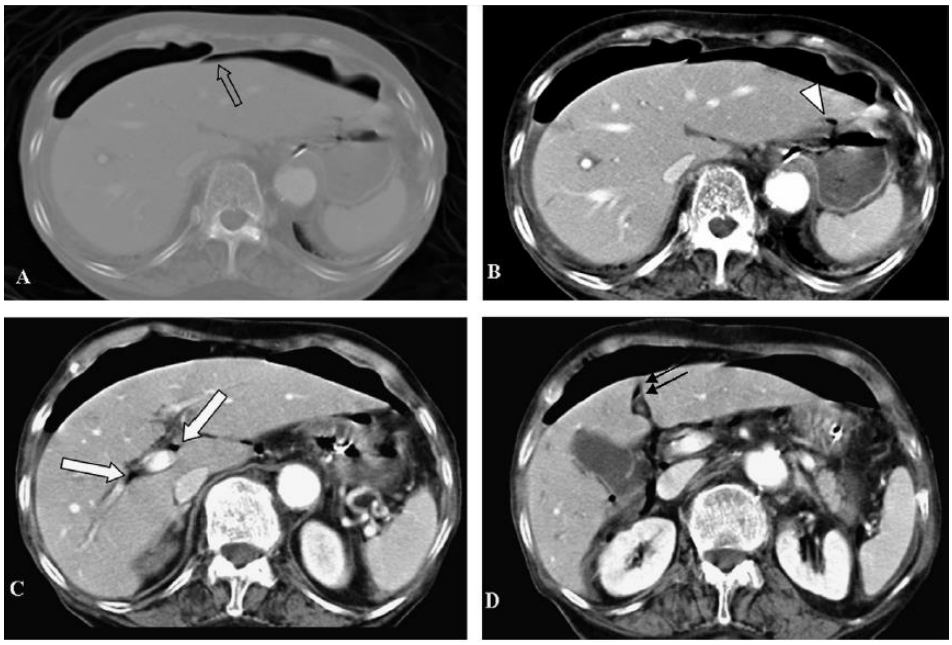


Figure 18: Computed tomography

(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. Other intra-abdominal conditions

2. Intra thoracic diseases and

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[29]

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[12]

(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. 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 canhelp 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.

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.[37]

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.[38]

- **Duodenal perforation:**

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

Modified graham patch repair:[39]

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 on lay 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 transjejunal drain that extends from the duodenum through the jejunal "patch" and exits via a Witzel closure several centimeters downstream in the jejunal limb.[37]

- **Gastric perforation:**

Surgical options include simple closure with biopsy, excision and closure, and resection. 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 histologic 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.[37]

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.[39]

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 midclavicular 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 midclavicular 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.[38]

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.[39]

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. **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[28].

❖ ***What is the pre-requisite for scoring systems?***

Early diagnosis, control of sepsis and management of primary cause is very important in the management of peritonitis. Various scoring systems have been developed that aid in stratifying the patients into various risk groups, predicting the prognosis and choosing the appropriate line of management as well as identifying the cases that need intensive care[3]. Early prognostic evaluation of patients with peritonitis is done to select high-risk patients for intensive management and also to provide a reliable objective classification of severity and operative risk. The prognosis and outcome of peritonitis depends on the interaction of many factors, including patient-related factors, disease-specific factors, and diagnostic and therapeutic interventions.

Categorizing patients into different risk groups would help prognosticate the outcome, select patients for intensive care and determine operative risk, thereby helping to choose the nature of the operative procedure, e.g. damage control vs. definitive procedure[12].

Several scoring systems that have been commonly used to assess the prognosis and outcome of peritonitis include -

- The Acute Physiological and Chronic Health Evaluation score (APACHE II),
- Mannheim Peritonitis Index (MPI),
- The Peritonitis Index Altona (PIA),

- The Sepsis Score, and
- The Physiological & Operative Severity Score for Enumeration of Mortality & Morbidity (POSSUM).
- Boey's score
- Hacetteppe score
- Reiss Index and fitness score
- ASA score
- Sickness assessment score
- Hardman index
- Cleveland clinic colorectal cancer model

The multifaceted nature of surgical infections, the complexity of management and ICU care, make evaluation of new diagnostic and therapeutic advances in this field challenging. Scoring systems provide objective descriptions of the patient's condition at specific points in the disease process and aid our understanding of these problems[40].

MANNHEIM PERITONITIS INDEX (MPI)

The Mannheim peritonitis index was initially described by Wacha H *et al.,* in 1987. It was derived from a retrospective study of 1,253 patients with peritonitis. The index predicts the individual risk of death. The overall mortality was 24%, average age 56.4 years, and proportion of females 49.2%. A score exceeding the average mortality (index>26) was defined as severe peritonitis. The reclassification method was used to estimate the prognostic accuracy of the statistically based index. The index showed good specificity (79%), sensitivity (84%) and accuracy (81%). 17 possible risk factors were identified, 8 of which were of prognostic relevance

and are currently employed widely for predicting mortality from peritonitis. The information is collected at the time of admission and during laparotomy.

Table 5: Mannheim peritonitis index scoring system

Risk factor	**Points**
Age>50yrs	5
Female sex	5
Organ failure	7
Malignancy	4
Preoperative duration of peritonitis>24h	4
Origin of sepsis not colonic	4
Diffuse generalized peritonitis	6
Exudates	
Clear	0
Cloudy, purulent	6
Fecal	12
Definitions of organ failure	
Kidney	Creatinine level >177umol/L Urea>167mmol/L Oliguria<20ml/h
Lung	PO_2<50mmHg PCO_2>50mmg
Shock	Hypodynamic or hyperdynamic
Intestinal obstruction	Paralysis >24h or complete mechanical obstruction

Billing A *et al.,*[41]conducted a meticulous study on MPI and analyzed the data.

Each risk factor is given a weightage to produce a score used for prognostic purposes.

- Maximal score is 47
- The cut-off point taken was a score of 26. Patients with higher values being classified as non-survivors.
- Patients were divided into 3 categories of severity. MPI < 21, 21-29 and >29.
- A linear correlation was found between the mean index score and the mean mortality rate.

Benefits of MPI

- ✓ Easy to apply
- ✓ Pre and intraoperative risk determination can be done.
- ✓ Likely outcome can be anticipated and apt management decided.
- ✓ Patient with fewer score can be treated with customary minimal risks, while patient with high score may need aggressive and intensive approach with critical care monitoring.
- ✓ It is peritonitis specific index and seems to be best for statistical studies and for comparison of clinical trials.

Detriments of MPI

- × This index does not include the probability of eradicating the source of inflammation.
- × Score is calculated only once; post-operative complications may hinder the results.
- × The index assigns peritonitis originating from colon to be a low risk. Since most of the colonic perforations are usually secondary to malignancy, this may not be applicable uniformly.
- × It does not at all consider the underlying physiological derangement of the patients, which is important in the acute classification or categorization of the patients who need intensive supportive care.

APACHE-II SCORE

This score was predominantly designed for ICU patients described by Knaus*et al.*[42]. In 1984, Meakins and associates used this score to evaluate patients with peritonitis. It comprises of 2 parts: First concerns with acute physiology while the second is centered on chronic health evaluation.

Distinct correlation between mortality rate and increase in score was noted. This system even though correctly measures severity of illness; is cumbersome in surgical practice and does not give any indication regarding management modalities of patient.

The Acute Physiological Score (APS) is based upon 12 physiological variables. These values were scored in accordance with abnormally high or low range. The score ranged from 0 to 4 on each side of the normal value. Zero score represents a normal value; an increase to 4 indicates the extreme end of high or low abnormal levels. Chronic Health Points (CHP) were added if the patient had a history of severe organ system insufficiency or was immunocompromised; points were assigned as follows: 2 for elective postoperative patients and 5 for non-operative or emergency postoperative patients.

The APACHE II Score was then calculated by the formula:

APACHE II score = APS + Age points + CHP

TABLE:6 APACHE II SCORING

Calculation of Acute Physiology and Chronic Health Evaluation II									
Score	**4**	**3**	**2**	**1**	**0**	**1**	**2**	**3**	**4**
Rectal temperature,°C	>41	39.0–40.9		38.5–38.9	36.0–38.4	34.0–35.9	32.0–33.9	30.0-31.9	<29.9
Mean blood pressure, mmHg	>160	130–159	110–129		70–109		50–69		<49
Heart rate (ventricular response)	>180	140–179	110–139		70–109		55–69	40–54	<39
Respiratory rate (non ventilatedos ventilated)	>50	35–49		25–34	12–24	10–11	6–9		<5
Arterial pH	>7.70	7.60–7.69		7.50–7.59	7.33–7.49		7.25–7.32	7.15–7.24	<7.15
Oxygenation									
If FI_{O2}> 0.5, use (A - a) D_{O2}	>500	350–499	200–349		<200				

If F_{O2}<0.5, use Pa_{O2}					70	61–70		55–60	<55
Serum sodium, mMol/L	>180	160–179	155–159	150–154	130–149		120–129	111–119	<110
Serum potassium, mMol/L	>7.0	6.0–6.9		5.5–5.9	3.5–5.4	3.0–3.4	2.5–2.9		<2.5
Serum creatinine, mg/100ml	>3.5	2.0–3.4	1.5–1.9		0.6–1.4		<0.6		
Hematocrit(%)	>60		50–59.9	46–49.9	30–45.9		20–29.9		<20
WBC count, 10^3/mL	>40		20–39.9	15–19.9	3–14.9		1–2.9		<1

Glasgow Coma Score

Eye Opening	**Verbal (Nonintubated)**	**Verbal (Intubated)**	**Motor Activity**
4—Spontaneous	5—Oriented and talks	5—Seems able to talk	6—Verbal command
3—Verbal stimuli	4—Disoriented and talks	3—Questionable ability to talk	5—Localizes to pain
2—Painful stimuli	3—Inappropriate words	1—Generally unresponsive	4—Withdraws to pain
1—No response	2—Incomprehensible sounds		3—Decorticate
	1—No response		2—Decerebrate
			1—No response

Points Assigned to Age and Chronic Disease as Part of the APACHE II Score

Age, Years	**Score**
<45	0
45–54	2
55–64	3
65–74	5
75	6

Benefits

✓ The APACHE II scores correlate well with mortality and are effective in the prediction of outcome.

✓ It considers the acute physiology of the patient, and can be completed before surgery.

✓ It is very useful in the acute stratification of the patients into risk groups and in predicting which patients can be considered for more extensive procedures.

Demerits

× The score does not consider the aetiology of peritonitis or the nature of peritoneal contamination, which has an important bearing on the outcome.

× Furthermore, the score is not as simple as the MPI; it is more extensive and needs lab support.

SEPSIS SCORE OF ELEBUTE AND STONER

It was described in 1983 by Elebute EA and Stoner HB, primarily for district general hospitals, for monitoring patients suffering from peritonitis and for grading the severity of sepsis. Clinical features of the septicemia were divided into 4 classes for which an independent degree of severity was attributed on an analogue scale. The attributes were local effects of tissue infection, pyrexia, secondary effects of sepsis and laboratory data. The system produces a number which indicates the severity of sepsis and which varies with the patient's condition. This system could be useful in comparing patients with sepsis and studies on such patients in different centers[43].

The possible range of score is 0 to more than 45. This system was examined in more detail by Dominioni*et al*. [44], conducted a study based on the sepsis score and CRP and described the

sepsis scores range from 10 to >30 and its overall accuracy for predicting mortality around 84%.

Benefits

- ✓ This system could be useful in comparing patients with sepsis and studies on such patients in different centers.
- ✓ Since, this was primarily designed for district hospitals, it is more appropriate for our rural population set up.
- ✓ It includes a comprehensive clinical work up, hence is more sensitive.
- ✓ Laboratory investigations are minimal.
- ✓ It can be used either as a single one time score or can be used to monitor critical patients and score tabulated on regular basis.

Detriments

- × Attributes are calculated subjectively. More chances of observer bias and variations.
- × No direct attempt to score "septic shock", hence it provides indirect evidence for sepsis syndrome.

❖ **POSSUM SCORING**

This is denoted as a Physiological and Operative Severity Score for the enumeration of Mortality and morbidity. This system has been devised from both a retrospective and prospective analysis.

Physiological score (to be scored at the time of surgery) (Table no: 7)

Score				
	1	2	4	8
Age	<60	61-70	>71	
Cardiac signs	No failure	Diuretic,digoxin, antianginal or hypertensive therapy	Peripheral oedema; warfarin therapy Borderline cardiomegaly	Raised jugular venous Pressure Cardio**m**egaly
Respiratory history	No dyspnoea	Dyspnoea on exertion	Limitingdyspnoea (one flight)	Dyspnoea at rest (rate > 30/min)
Chest radiograph		Mild COAD	Moderate COAD	Fibrosis or consolidation
Blood pregsure (systolic)mmHg	110-130	131-170;100-109	>171; 90-99	<89
Pulse (beats/min)	50-80	81-100, 40-49	101-120	>121, <39
Glasgow coma score	15	12-14	9-11	<8
Haemoglobin	13-16	11.5-12.9	10.0-11.4	<9.9
White cell count (x10'*/1)	4-10	10.1-20.0;3.1-4.0	>20.1; <3.0	

Urea (mmol/l)	<7.5	7.6-10.0	10.1-15.0	>15.1
Sodium (mmol/l)	>136	131-135	126-130	<125
Potassium (mmol/l)	3.5-5.0	3.2-3.4 5.1-5.3	2.9-3.1 5.4-5.9	<2.8 >6.0
Electrocardiogram	Normal		Atrial fibrillation rate 60-90	other abnormal rhythm or 2-5 ectopics/min Q waves or ST/T wave changes

Operative score

POSSUM is not a specific scoring systems for individual disease states or for intensive care. However, it may provide an efficient indicator of the risk of morbidity and mortality in the general surgical patient.

POSSUM may be used as an adjunct to surgical audit. It does not affect the decision to operate. It could theoretically assist in the direction of resuscitative efforts[45].

MULTIPLE ORGAN FAILURE SCORING [(46,47,48)]

Since organ failure and dysfunction ultimately evolve in patients with sepsis, organ function is monitored routinely in intensive care unit patients. This multi-organ failure scoring was described by Goris *et al* [47]in 1985. It grades patients on three point scale and takes into

consideration dysfunction of the pulmonary, cardiovascular, hepatic, renal, nervous, hematological and gastro-intestinal systems; however, in a recent revision, gastro-intestinal and nervous systems have been removed.

In 2002, Goris *et al* [48] suggested a revised MOF score that did not include the GI and nervous system failures. GI failure lacked a clear definition, its incidence was low, and its occurrence was rarely associated with poor outcome. Also, a valid assessment of mental function is difficult in intensive care patients who are receiving sedation and assisted ventilation.

Multiple organ failure score

Table no: 8

Organ	Normal function	Organ dysfunction	Organ failure
Points	0	1	2
Lung	No mechanical ventilation	mechanical ventilation with PEEP <10 and FiO2<0.4	mechanical ventilation with PEEP >10 or FiO2>0.4
Heart	Normal blood pressure	Bpsyst>100mmHg with low dose of vasoactive drugs[a]	BP syst<100mmHg and/or high dose of vasoactive drugs[b]
Kidney	Serum creatinine<2mg/dL	>2mg/dL	Hemodialysis or peritoneal dialysis
Liver	Normal AST and bilirubin	AST >25 units /L Bilrubin>2mg/dL	AST >50 units /L Bilrubin>6mg/dL
Blood	Normal counts	Leukocytes>30000/µ L Platelets <50000/µ L	Leukocytes 60000/micro L or

			<2500 micro /L
GI tract	Normal	Stress ulcer Acalculous cholecystitis	Bleeding ulcer, necrotizing enterocolitis and/or pancreatitis
CNS	Normal	Diminished responsiveness	Severely disturbed responsiveness Diffuse Neuropathy

a.Dopamine hydrochloride<10µg/kg/min or nitroglycerine<20µg/kg/min or volume loading

b.Dopamine hydrochloride>10µg/kg/min or nitroglycerine>20µg/kg/min; GI Gastrointestinal

SAPS II[49]

SAPS II is a severity of disease classification system described in 1993 by Le Gall JR *et al.*

Its stands for "Simplified Acute Physiology Score", and is one of several ICU scoring Systems[49]. It includes only 17 variables: 12 physiology variables, age, and type of admission (scheduled surgical, unscheduled surgical, or medical), and three underlying disease variables (acquired immunodeficiency syndrome, metastatic cancer, and hematologic malignancy). The SAPS II, based on a large international sample of patients, provides an estimate of the risk of death without having to specify a primary diagnosis.

In 1986 John Boey*et al*, [50]described the risk stratification in perforated duodenal ulcer. Three criteria's namely major medical illness, preoperative shock, and long standing perforation (more than 24 hours) were assessed. 0 points was assigned if no risk factor was present and scores to 1 to 3 were applied depending on number of risk factors present. It was concluded that definitive

surgery (vagotomy and drainage) can be securely performed if no risk factors are present. If any of the risk factors is present, it is preferable to do simple closure of the perforation. If all 3 risk factors are present, the outcome was poor whether patient was operated or treated conservatively. Non operative treatment deserves reevaluation in patients with all three risk factors because of their uniformly dismal outcome after operation.

- **Peptic Ulcer Perforation score (Pulp score)[51]:**

Table no:9 Assignment of points according to the Peptic Ulcer Perforation score

	Variables	Points
1	Age > 65 years	3
2	Co-morbid active malignant disease or AIDS	1
3	Co-morbid liver cirrhosis	2
4	Concomitant use of steroids	1
5	Shock on admission*	1
6	Time from perforation to admission > 24 h	1
7	Serum creatinine > 130 mmol/l	2
8	ASA Score ASA 2 ASA 3 ASA 4 ASA 5	 1 3 5 7

Total PULP score: 0–18

*Shock on admission is defined as blood pressure < 100 mmHg

and heart rate > 100 beats per min.

ASA, American Society of Anaesthesiologists.

- **Jabalpur prognostic scoring system for peptic perforation[52](2003)**

Table 10: Jabalpur prognostic scoring system for peptic perforation

Factor	Score						
	0	1	2	3	4	5	6
P-O interval(hours)	<24	25-72	73-96	97-120	>120	-	-
Mean systolic BP(mmHg)	70-109	-	50-69or110-129	130-159	<49or>160	-	-
Heart rate	70-120	-	55-59or110-139	40-54or40-179	<39or>180	-	-
Ser	0.6-1.4	-	1.5-1.9	2.0-3.4	>3.5	-	-
Age	<45	-		55-64	-	65-74	>75

MATERIALS AND METHODOLOGY

A prospective clinical study was conducted on 80 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 2013 to June 2015. Study population consisted of 80 consecutive patients with peritonitis secondary to hollow viscus perforation which were confirmed on emergency laparotomy.

INCLUSION CRITERIA:

All patients diagnosed to have peritonitis secondary to hollow viscus perforation

EXCLUSION CRITERIA:

1.Patients less than 16 years of age.

2. Post-operative peritonitis

3. Gynaecological causes of peritonitis.

4. Spontaneous bacterial peritonitis.

5. Peritonitis secondary to ventriculo-peritoneal shunts.

6. Blunt and penetrating abdominal injuries.

Diagnosis of peritonitis due to hollow viscus perforation was made by:

- History:

  Symptoms, onset of presenting illness and duration of illness noted.

- Patient details suggestive of chronic health disorders such as cardiac, respiratory, renal, liver failure and immunodeficiency disorders noted.

- Clinical examination

  Presence of guarding, rigidity, tenderness on palpation and obliteration of liver dullness of the abdomen were noted.

- Radiologically: air under diaphragm.

- At the time of admission:

1. Vital parameters noted:

   Heart rate, Blood pressure, Mean arterial pressure, Respirator rate, Temperature

2. Investigations

   - Hematocrit
   - Total WBC count
   - Blood - urea
   - Serum creatinine
   - Serum Na+
   - Serum K+
   - PaO_2
   - Arterial pH
   - Chest x-ray

- Plain x-ray abdomen - erect
- Abdominal paracentesis

- Proforma filled.
- Intra operative findings noted

All the patients were subjected to emergency exploratory laparotomy. The surgical procedure performed depended upon the operative findings and the surgeon's choice, as no guidelines could be laid down due to the varied etiology with peritonitis due to hollow viscus perforation.

- Etiological factors were studied.

Two systems, APACHE II and Mannheim Peritonitis Index (MPI) scoring systems were assigned to all the patients in order to calculate their individual risk of mortality and survival at the time of admission.

Mannheim Peritonitis index(1983):

The MPI analyzes 8 prognostically significant factors. Points were given to each factor as given in table 9. Points were added for each factor present and the MPI score was calculated by adding these points as given in table 9.

Mannheim peritonitis index scoring system

Risk factor	**Points**
Age>50yrs	5
Female sex	5
Organ failure	7
Malignancy	4
Preoperative duration of peritonitis>24h	4
Origin of sepsis not colonic	4
Diffuse generalized peritonitis	6
Exudates	
Clear	0
Cloudy, purulent	6
Fecal	12
Definitions of organ failure	
Kidney	Creatinine level >177umol/L Urea>167mmol/L Oliguria<20ml/h
Lung	PO_2<50mmHg PCO_2>50mmg
Shock	Hypodynamic or hyperdynamic
Intestinal obstruction	Paralysis >24h or complete mechanical obstruction

APACHE II

APACHE II scores were calculated as per the method of Knaus. Acute physiological and chronic health evaluation includes The Acute Physiological Score(APS), age points and chronic health score. APS is based upon 12 physiological variables.

Calculation of Acute Physiology and Chronic Health Evaluation II [40]									
Score	**4**	**3**	**2**	**1**	**0**	**1**	**2**	**3**	**4**
Rectal temperature,°C	>41	39.0–40.9		38.5–38.9	36.0–38.4	34.0–35.9	32.0–33.9	30.0-31.9	<29.9
Mean blood pressure, mmHg	>160	130–159	110–129		70–109		50–69		<49
Heart rate (ventricular response)	>180	140–179	110–139		70–109		55–69	40–54	<39
Respiratory rate (non ventilatedos ventilated)	>50	35–49		25–34	12–24	10–11	6–9		<5
Arterial pH	>7.70	7.60–7.69		7.50–7.59	7.33–7.49		7.25–7.32	7.15–7.24	<7.15

Oxygenation									
If FI_{O2}> 0.5, use (A - a) D_{O2}	>500	350–499	200–349		<200				
If F_{O2}<0.5, use Pa_{O2}					70	61–70		55–60	<55
Serum sodium, mMol/L	>180	160–179	155–159	150–154	130–149		120–129	111–119	<110
Serum potassium, mMol/L	>7.0	6.0–6.9		5.5–5.9	3.5–5.4	3.0–3.4	2.5–2.9		<2.5
Serum creatinine, mg/100ml	>3.5	2.0–3.4	1.5–1.9		0.6–1.4		<0.6		
Hematocrit(%)	>60		50–59.9	46–49.9	30–45.9		20–29.9		<20
WBC count, 10^3/mL	>40		20–39.9	15–19.9	3–14.9		1–2.9		<1

Glasgow Coma Score

Eye Opening	**Verbal (Nonintubated)**	**Verbal (Intubated)**	**Motor Activity**
4—Spontaneous	5—Oriented and talks	5—Seems able to talk	6—Verbal command
3—Verbal stimuli	4—Disoriented and talks	3—Questionable ability to talk	5—Localizes to pain
2—Painful stimuli	3—Inappropriate words	1—Generally unresponsive	4—Withdraws to pain
1—No response	2—Incomprehensible sounds		3—Decorticate
	1—No response		2—Decerebrate
			1—No response

Points Assigned to Age and Chronic Disease as Part of the APACHE II Score

Age, Years	**Score**
<45	0
45–54	2
55–64	3
65–74	5
75	6

Chronic Health Points: If the patient has a history of severe organ system insufficiency or is immunocompromised as defined below, assign points as follows:

a. for non-operative or emergency postoperative patients - 5 points
b. for elective postoperative patients - 2 points

***Definitions*:** organ insufficiency or immunocompromised state must have been evident **prior** to this hospital admission and confirm to the following criteria:

- **Liver** – biopsy proven cirrhosis and documented portal hypertension; episodes of past upper GI bleeding attributed to portal hypertension; or prior episodes of hepatic failure/encephalopathy/coma.
- **Cardiovascular** – New York Heart Association Class IV.
- **Respiratory** – Chronic restrictive, obstructive, or vascular disease resulting in severe exercise restriction (i.e., unable to climb stairs or perform household duties; or documented chronic hypoxia, hypercapnia, secondary polycythemia, severe pulmonary hypertension (>40 mmHg), or respirator dependency.
- **Renal** – receiving chronic dialysis.
- **Immunocompromised** – the patient has received therapy that suppresses resistance to infection (e.g., immunosuppression, chemotherapy, radiation, long term or recent high dose steroids, or has a disease that is sufficiently advanced to suppress resistance to infection, e.g., leukemia, lymphoma, AIDS).

Patients were divided into survived and expired, and scores were compared between groups. All the patients were followed up till the hospital stay. Mortality was defined as death occurring

during hospital stay. The value of each scoring system was tested in prognosticating the outcome of patients.

Statistical analysis

APACHE II and MPI scores were tested by quantitative methods based on statistical criteria.

The following statistical tests were done to know the ability to predict outcome.

1. **Accuracy or discriminative ability:**

The accuracy of the test depends on how well the test separates the group being survivors and non survivors. Accuracy is measured by the area under the ROC curve. The area measures discrimination, that is, the ability of the test to correctly classify those who survived or not.

Accuracy explains What is the percentage of correct predictions in the group of survivors (specificity), what is the percentage of correct predictions in the group of non-survivors (sensitivity), what are the differences between these as measured by the area under the receiver-operator characteristic (ROC) curve. A rough guide for classifying the accuracy of a diagnostic test is the traditional academic point system:

- 0.9-1.0 = excellent
- 0.8-0.9 = good
- 0.7-0.8 = fair
- 0.6-0.7 = poor
- 0.5-0.6 = fail

Decision matrices were formed that compared predicted events with events that occurred. Subsequently, sensitivity was plotted against specificity for different cut-off points, which gave ROC curves. The difference between areas under two ROC curves was calculated using the trapezoidal rule, a conservative estimate for the standard deviations, and Kendall's are to measure the correlation between the areas.

2. **Sharpness**

What is the degree of confidence associated with the predictions for example, do most of the predictions for survival or death exceed a certain value (> 0.9)? The distribution of scoring systems, is a measure for sharpness. Sharpness was estimated measuring the proportion of high probabilities for one of the outcome categories (death or survival). Predicted probabilities of death in between (> 0.9 and <0.1) designated as "not sharp".

3. **Distribution of scores**

4. **Reliability**

How good is the agreement between predicted and observed mortality? To test reliability (calibration), 10 equidistant intervals were drawn on a probability scale of 0 to 1. The predicted death rate (sum of the individual probabilities for each interval) was compared with the observed mortality (number of actual deaths for each equidistant interval), and the agreement between observed and predicted events was compared.

RESULTS

Our study was conducted on patients admitted to R.L.Jalappa Hospital and Research Centre, Kolar, Karnataka. The study period was from December 2013 to June 2015.

A total of 80 cases of peritonitis secondary to hollow viscus perforation after confirming on emergency laparotomy were included.

AGE DISTRIBUTION

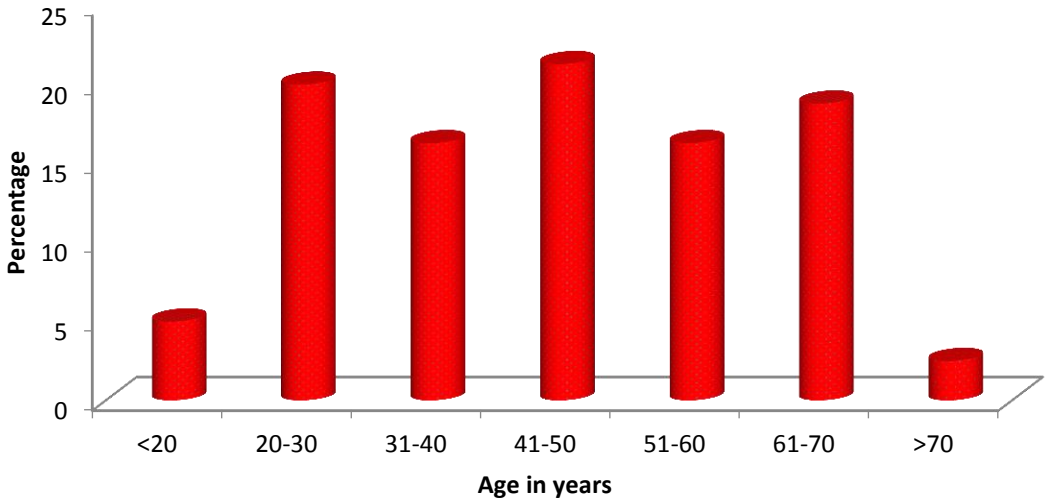
Age of the patients in this study ranged from 16years to 75years. The mean age of the patients at the time of admission was 45.55 years(SD 16.43).

Maximum number of patients 17(21.3%) were in the age group of 41-50years, followed by 20% (n= 16) in age group of 21-30years, 18.8% (n=15) in 61-70years, 16.3% (n=13) in both 31-40years and 51-60years. 5% (n=4) of cases were in the age group of <20years, 2.5% (n=2) cases in >70years, 5.33% (n=8) in age group of more than 70years as depicted in the table and graph.

TABLE NO: 11

Age in years	No. of patients	%
<20	4	5.0
20-30	16	20.0
31-40	13	16.3
41-50	17	21.3
51-60	13	16.3
61-70	15	18.8
>70	2	2.5
Total	80	100.0

GRAPH NO 1: AGE DISTRIBUTION



STATUS OF MORTALITY BY AGE GROUPS:

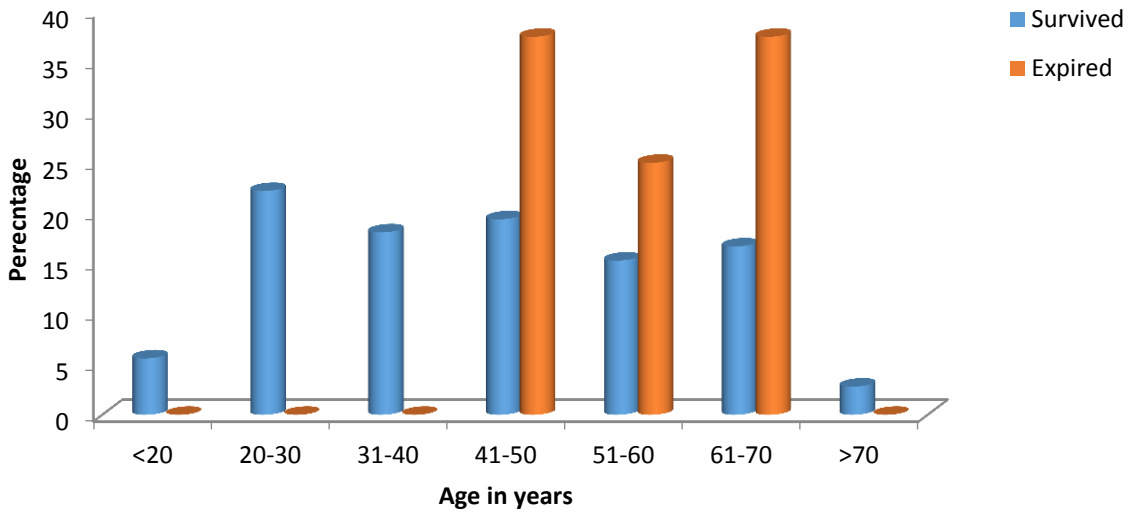
Highest mortality is in the age group of 41-50years and 61-70years (37.5%). There were 3 patients in each age group. The next highest mortality (25%) is seen in age group of 51-60years. Other age groups did not have any mortality. Mortality rate of 20% (3 of 15 patients) seen in age group of 61-70years. Similarly 17.64% (3 of 17patients) of mortality rate between 41-50years, 15.38%(2 of 13 patients) between 51-60years and is depicted in table no 11. Thus in our study mortality rate is more in the middle and older age group and with increase in age as depicted in the table and graph.

TABLE 12: STATUS OF MORTALITY BY AGE GROUPS

Age in years	Outcome		Total
	Survived	Expired	
<20	4(5.6%)	0(0%)	4(5%)
20-30	16(22.2%)	0(0%)	16(20%)
31-40	13(18.1%)	0(0%)	13(16.3%)
41-50	14(19.4%)	3(37.5%)	17(21.3%)
51-60	11(15.3%)	2(25%)	13(16.3%)
61-70	12(16.7%)	3(37.5%)	15(18.8%)
>70	2(2.8%)	0(0%)	2(2.5%)
Total	72(100%)	8(100%)	80(100%)

P=0.314, Not significant, Fisher Exact test

GRAPH NO:2 STATUS OF MORTALITY BY AGE GROUPS



STATUS OF MORTALITY BY GENDER:

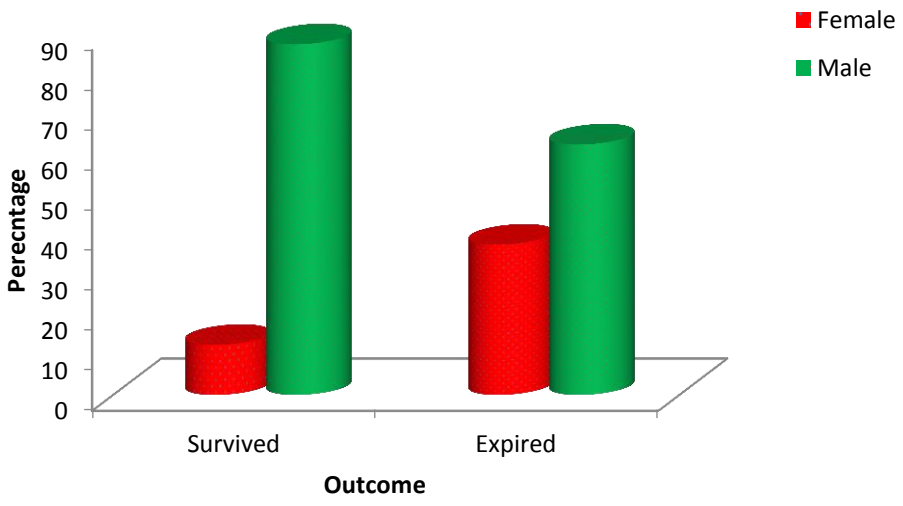
Out of 72 patients who had survived 63(87.5%) were males and 9(12.5%) were females. Out of 8 patients who had expired 5(62.5%) were males and 3(37.5%) were females. This is depicted in the table and graph. Thus in our study mortality was observed more in males.

TABLE NO:13 STATUS OF MORTALITY BY GENDER

Gender	Outcome		Total
	Survived	Expired	
Female	9(12.5%)	3(37.5%)	12(15%)
Male	63(87.5%)	5(62.5%)	68(85%)
Total	72(100%)	8(100%)	80(100%)

P=0.060+, significant, Chi-Square test

GRAPH NO: 3 STATUS OF MORTALITY BY GENDER



STATUS OF MORTALITY DEPENDING ON SITE OF PERFORATION:

Mortality rate and rate of survival according to the site of perforation is depicted in the table.

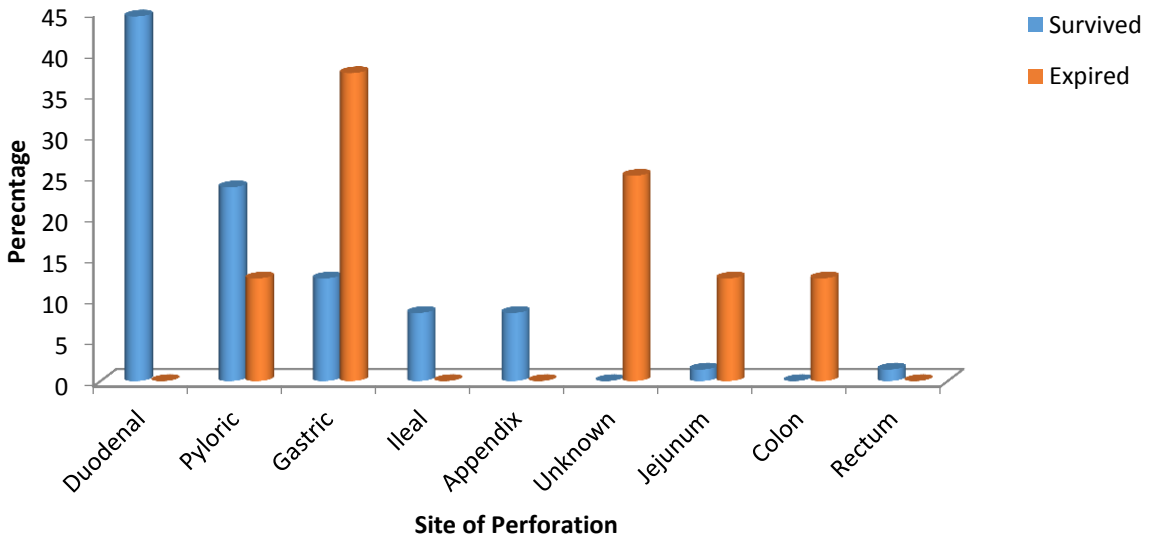
TABLE NO: 14 STATUS OF MORTALITY DEPENDING ON SITE OF PERFORATION

Site of Perforation	Outcome		Total
	Survived	Expired	
Duodenal	32(44.4%)	0(0%)	32(40%)
Pyloric	17(23.6%)	1(12.5%)	18(22.5%)
Gastric	9(12.5%)	3(37.5%)	12(15%)
Ileal	6(8.3%)	0(0%)	6(7.5%)
Appendix	6(8.3%)	0(0%)	6(7.5%)
Unknown	0(0%)	2(25%)	2(2.5%)
Jejunum	1(1.4%)	1(12.5%)	2(2.5%)
Colon	0(0%)	1(12.5%)	1(1.3%)
Rectum	1(1.4%)	0(0%)	1(1.3%)
Total	72(100%)	8(100%)	80(100%)

P<0.001**, significant, Fisher Exact test

In the study group of 80 patients, majority of the patients had duodenal perforation (40%). Highest survival rate was seen among duodenal perforation 32 of 32(100%) and the highest mortality was seen among patients with gastric, unknown and colonic perforations as shown in the graph.

GRAPH NO: 4 STATUS OF MORTALITY DEPENDING ON SITE OF PERFORATION



STATUS OF MORTALITY IN RELATION TO TIME OF PRESENTATION:

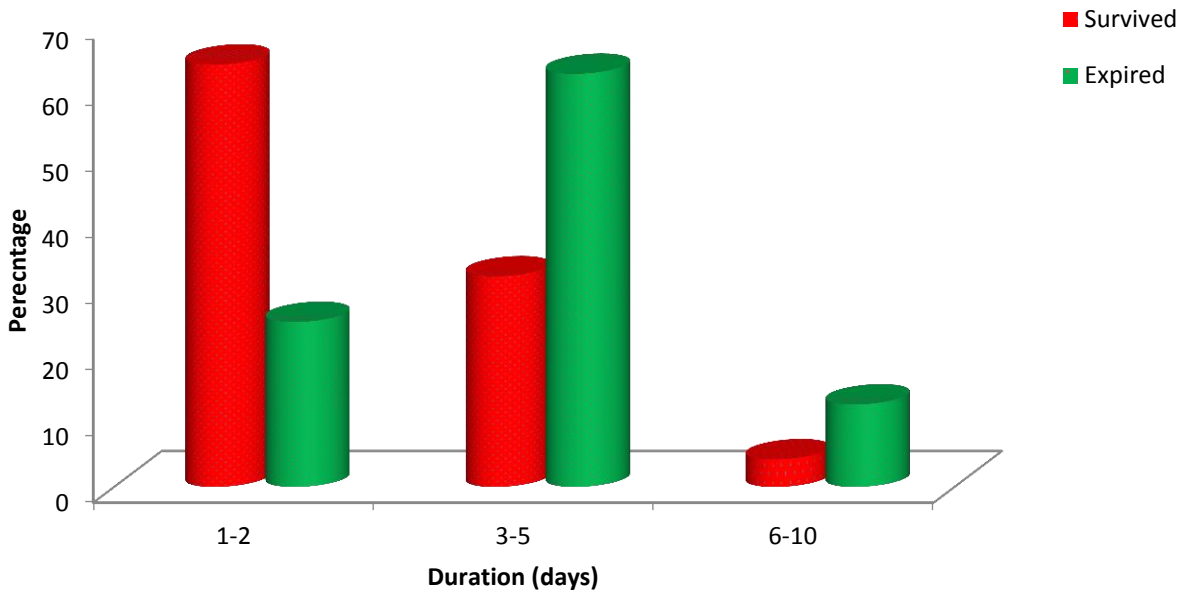
The time of presentation of patients ranged from < 24 hours to 10 days. Most of the patients presented within 1-2 days. Mortality increased correspondingly with delay in presentation to the hospital. It was 25% for 1-2days, 62.5% for 3-5 days and 12.5% for 6 to 10 days. Delayed presentation was usually seen in cases of peritonitis secondary to appendicular perforation which had better prognosis compared to other hollow viscus perforation presenting late. This above data is depicted in the table and graph below.

TABLE NO: 15 STATUS OF MORTALITY IN RELATION TO TIME OF PRESENTATION

Duration (days)	Outcome		Total
	Survived	Expired	
1-2	46(63.9%)	2(25%)	48(60%)
3-5	23(31.9%)	5(62.5%)	28(35%)
6-10	3(4.2%)	1(12.5%)	4(5%)
Total	72(100%)	8(100%)	80(100%)

P=0.106, Not significant, Fisher Exact test

GRAPH NO: 5 STATUS OF MORTALITY IN RELATION TO TIME OF PRESENTATION



COMPLICATIONS IN RELATION TO OUTCOME OF PATIENTS:

Patients with higher APACHE II score and Mannheims Peritonitis Index(MPI) had more associated complications. 34(42.5%) patients had SSI with a p value of 1.000, a total of

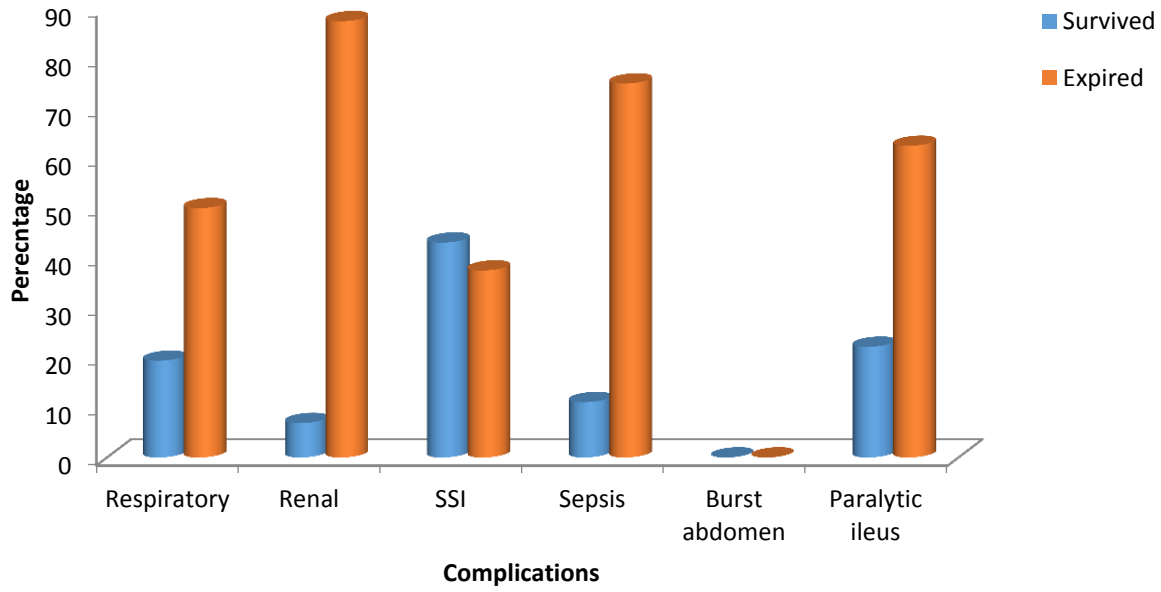
18(22.5%) patients had respiratory complications with a P value of 0.071, a total of 12(15%) patients had renal complications with a P value of <0.001, 21(26.3%) patients had paralytic ileus and none of the patients had burst abdomen. This is depicted in the table and graph below.

TABLE NO: 16

Complications	Outcome		Total (n=80)	P value
	Survived (n=72)	Expired (n=8)		
Respiratory	14(19.4%)	4(50%)	18(22.5%)	0.071+
Renal	5(6.9%)	7(87.5%)	12(15%)	<0.001**
SSI	31(43.1%)	3(37.5%)	34(42.5%)	1.000
Sepsis	8(11.1%)	6(75%)	14(17.5%)	<0.001**
Burst abdomen	0(0%)	0(0%)	0(0%)	1.000
Paralytic ileus	16(22.2%)	5(62.5%)	21(26.3%)	0.026*

Chi-square test/ Fisher Exact test

GRAPH NO: 6



APACHE II score:

APACHE II score was assigned to all patients. Mean apache II scores in survivors were 7.5±5.3 and in non survivors 19.7±4.7. Of the 72 survivors, with mean of 7.5, 8 patients who died had a mean of 19.5, and again the difference between groups were significant (p<0.0001).

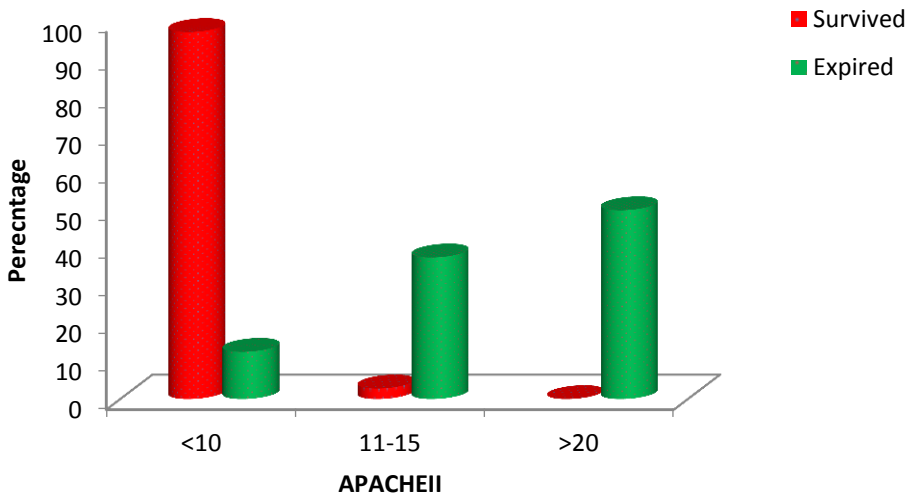
Based on APACHE II scores patients were divided into 3 groups with scores of <10, 11-20 and >20. The number of patients scoring less than 10 was 71(88.8%) of the study group. One patient with less than a score of 10 expired. 5 patients had scores in range of 11-20, 2 survived and 3 expired. 4 patients had scores more than 20 and all 4 patients expired. These are shown in table and graph below.

TABLE 17: APACHE II DISTRIBUTION IN RELATION TO OUTCOME OF PATIENTS STUDIED

APACHEII	Outcome		Total
	Survived	Expired	
<10	70(97.2%)	1(12.5%)	71(88.8%)
11-15	2(2.8%)	3(37.5%)	5(6.3%)
>20	0(0%)	4(50%)	4(4.9%)
Total	72(100%)	8(100%)	80(100%)

P<0.001**

GRAPH NO: 7 APACHE II DISTRIBUTION IN RELATION TO OUTCOME OF PATIENTS STUDIED



MANNHEIMS PERITONITIS INDEX (MPI):

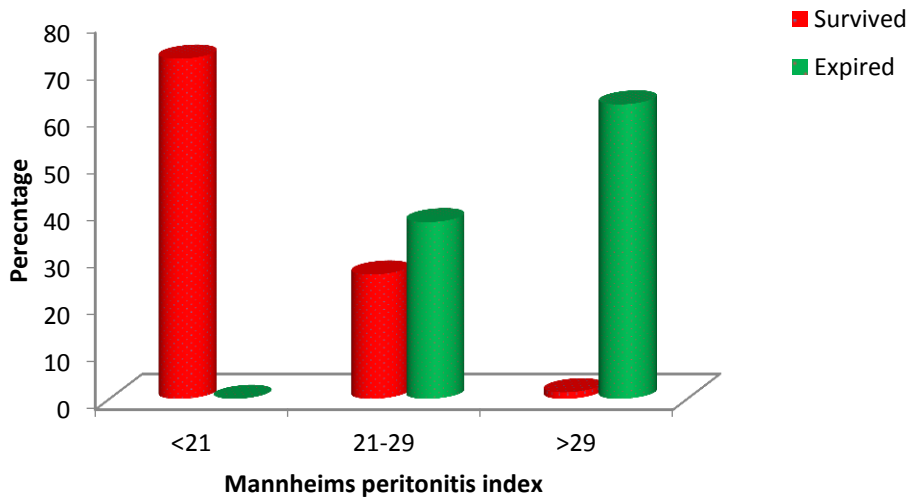
Based upon their MPI score, the patients were divided into three groups, MPI scores of less than 21, 21-29 and more than 29. None of the 52 patients with score <21 had mortality. 22 patients scored in range of 21-29 with mortality rate of 13.63%. 5 of 6 patients(MR=83.72) died who scored >29 as shown in table and graph below. Mean MPI score among survivors was 15.86±6.57 and in non-survivors was 32.13±4.67.

TABLE 18: MANNHEIMS PERITONITIS INDEX (MPI) IN RELATION TO OUTCOME OF PATIENTS STUDIED

Mannheims peritonitis index	Outcome		Total
	Survived	Expired	
<21	52(72.2%)	0(0%)	52(65%)
21-29	19(26.4%)	3(37.5%)	22(27.5%)
>29	1(1.4%)	5(62.5%)	6(7.5%)
Total	72(100%)	8(100%)	80(100%)

P<0.001**, significant, Fisher Exact test

GRAPH NO: 8 MANNHEIMS PERITONITIS INDEX (MPI) IN RELATION TO OUTCOME OF PATIENTS STUDIED



Accuracy or discriminative ability:

Receiver operative characteristic curve

ROC curve was drawn by plotting sensitivity against specificity for different cut off points.

The ROC curves that related sensitivity to specificity for different cut-off points were shown in graph no 9.

GRAPH NO 9: RECEIVER OPERATIVE CURVES FOR APACHE II AND MPI

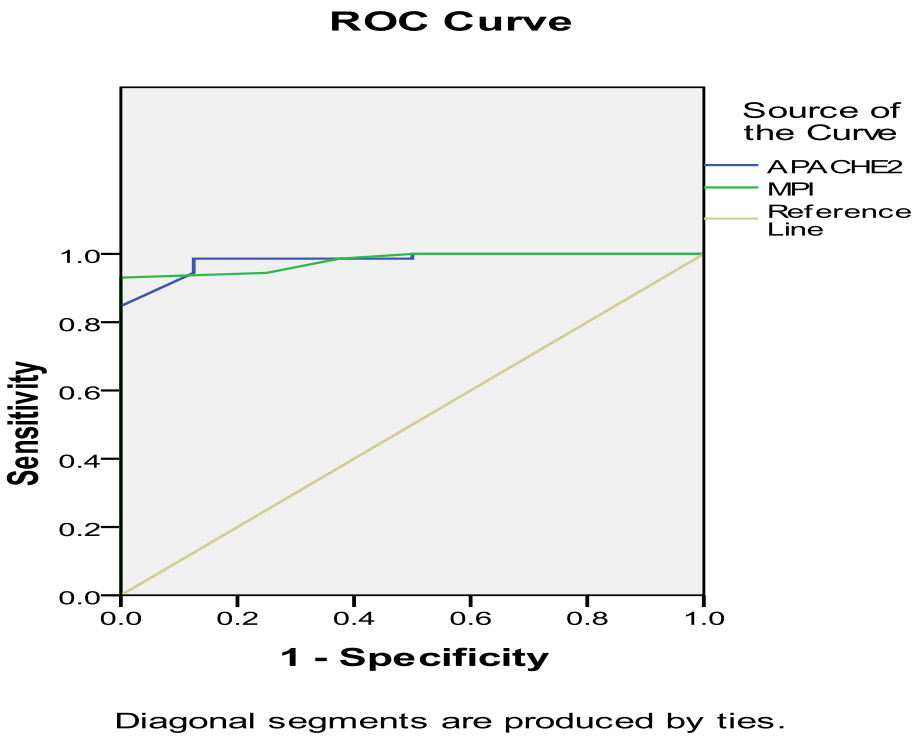


TABLE: 19 AREA UNDER ROC CURVE IN APACHE II AND MPI

Area Under the Curve	
Test Result Variable(s)	Area
MPI	.979
APACHE2	.982

The area below the curve was 0.982 for APACHE II and 0.979 for MPI showing that APACHE II is significantly better than MPI ($p < 0.01$).The APACHE II curve showed that it discriminated better than the MPI. The sensitivity of APACHE II was superior to MPI at any given point of specificity. This difference was maintained across the entire range of values.

TABLE 20- DISTRIBUTION OF APACHE II AND MPI AMONG SURVIVORS AND NON-SURVIVORS

Score	Survivors n=72	Non survivors n=8	P value
APACHEII	4.78±2.63	15.38±4.65	<0.0001
MPI	15.86±6.57	32.13±4.67	<0.0001

Distribution of APACHE II and MPI among survivors showed mean apache score of 4.78±2.63 and mean MPI score of 15.86±6.57 which was found statistically significant(P<0.0001), and non survivors had mean APACHE II score of 15.38±4.65and mean MPI score of 32.13±4.67 and was statistically significant (P>0.0001).

TABLE 21: SHARPNESS OF APACHE II AND MPI

	<0.1 (sharp)	0.1-0.9 (Not sharp)	>0.9 (sharp)
APACHE II	72	7	1
MPI	72	5	3

The distribution of scores, a measure for sharpness of the predictions, is shown in table 21. The distribution of APACHE II scores with low score values had low probabilities of death(< 0.1)

for 72 of the 80 patients, (90%). In addition, APACHE II assigned a high risk of death (p >0.9) to 1 of 80 patients (1.2%) of patients. But 7 patients (8.8%) were assigned a moderate risk *(*>0.1 and < 0.9) of death indicating that its predictions were "not sharp" in these cases.

The distribution of MPI scores with low score values had low probabilities of death (< 0.1) for 72 of the 80 patients, (90%). MPI assigned a high risk of death (p > 0.9) to 3 of 80patients (3.75%) of patients. But 5 patients (6.25%) were assigned a moderate risk (>0.1 and < 0.9) of death indicating that its predictions were "not sharp" in these cases.

MPI and APACHE II both were sharp in prediction. But MPI is sharper than APACHE II.

Reliability:

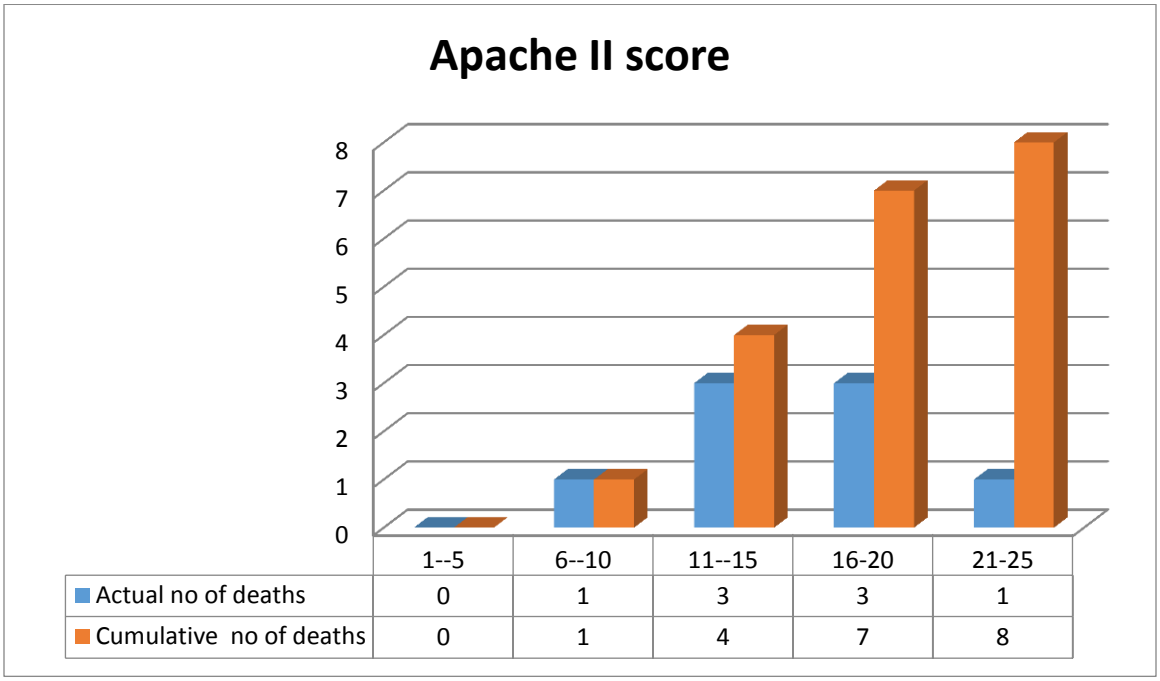
Reliability (calibration) of probabilities was investigated by comparing observed and cumulative no of deaths as shown in table.

TABLE 22: ASSOCIATION BETWEEN APACHE II TOTAL SCORE AND PROBABILITY OF DEATH

APACHE II total score	Actual no of deaths	Cumulative no of deaths	Proportion of deaths	Probability of death
1-5	0	0	0.00	0.00
6-10	1	1	0.125	0.125
11-15	3	4	0.375	0.50
16-20	3	7	0.375	0.875
21-25	1	8	0.125	1.00
Total	8		1.00	

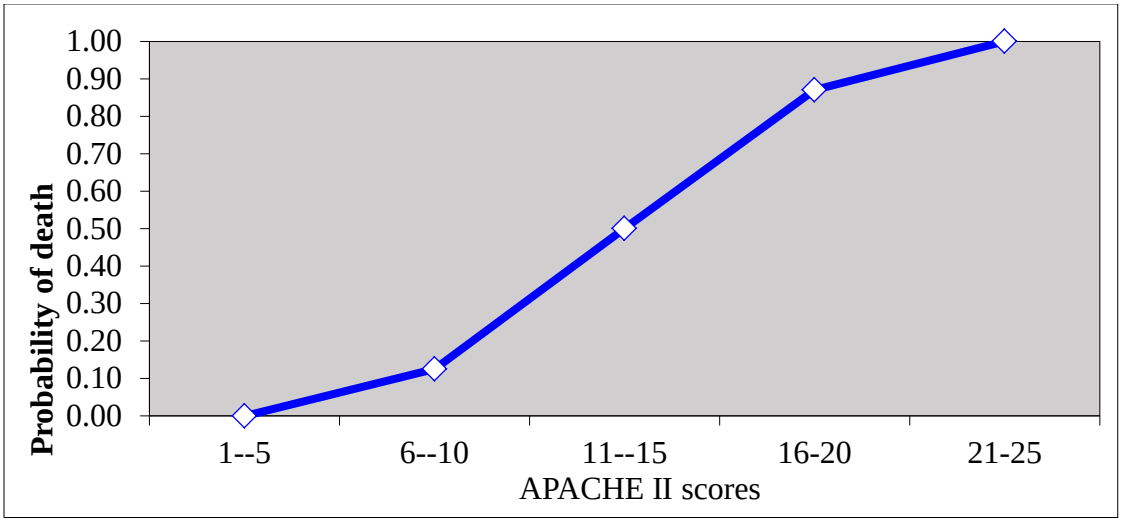
APACHE II scores for 1 to 15 there were no deaths and expected number of deaths was also zero, and for 6-10, actual number of death was equal to expected number of deaths. With scores of 16 to 20 actual number of death was 3 as expected number of death was 7 with probability of 0.875 indicating it is reliable. For scores 21-25 actual number of death was 1 where as expected number of deaths was 8 with probability of 1.00.

GRAPH NO 10: APACHE II SCORE AND COMPARISON OF ACTUAL AND CUMULATIVE NO OF DEATHS.



	1--5	6--10	11--15	16-20	21-25
Actual no of deaths	0	1	3	3	1
Cumulative no of deaths	0	1	4	7	8

GRAPH NO 11: ASSOCIATION BETWEEN APACHE II TOTAL SCORE AND PROBABILITY OF DEATH



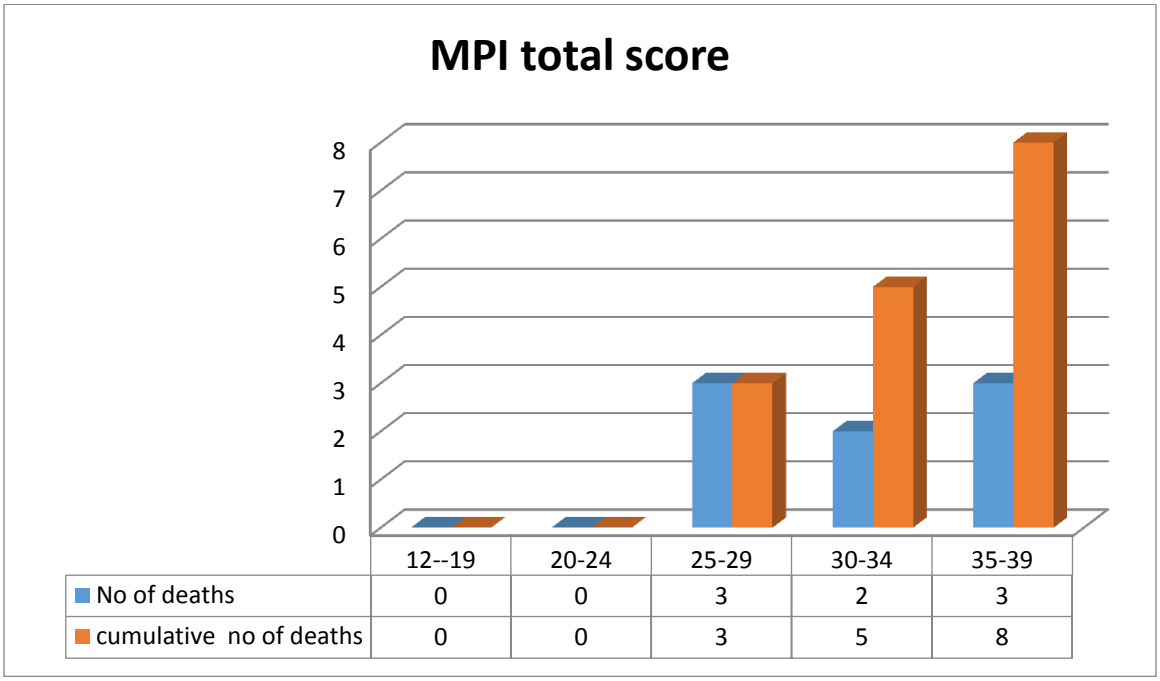
ASSOCIATION BETWEEN MPI TOTAL SCORE AND PROBABILITY OF DEATH:

TABLE 23: ASSOCIATION BETWEEN MPI TOTAL SCORE AND PROBABILITY OF DEATH

MPI total score	No of deaths	cumulative no of deaths	Proportion of deaths	Cumulative proportion of deaths
12-19	0	0	0.00	0.00
20-24	0	0	0.00	0.00
25-29	3	3	0.375	0.375
30-34	2	5	0.25	0.625
35-39	3	8	0.375	1.00
Total	8		1.00	

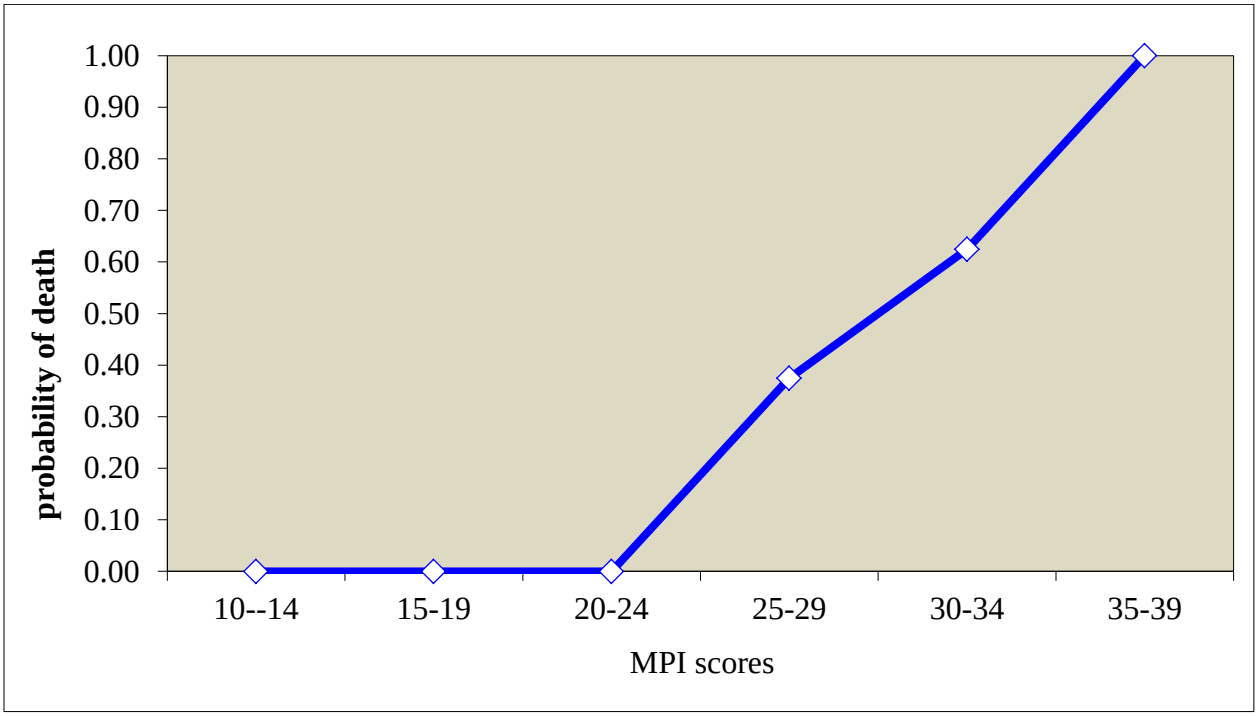
MPI scores from 12 to 24, there were no deaths and expected number of deaths was also 0. With scores of 25 to 29 actual number of death was 3 and was equal to expected number of death. For scores 30-34 actual number of death was 2 where as expected number of deaths was 5 with probability of 0.65. For scores 35-39 actual no of deaths was 3 and expected number of deaths was 8 with probability of 1.00.

GRAPH NO 12: MPI SCORE AND COMPARISON OF ACTUAL AND CUMULATIVE NO OF DEATHS.



	12--19	20-24	25-29	30-34	35-39
No of deaths	0	0	3	2	3
cumulative no of deaths	0	0	3	5	8

GRAPH NO 13: ASSOCIATION BETWEEN MPI TOTAL SCORE AND PROBABILITY OF DEATH



DISCUSSION

The fundamental difficulty in prediction of outcome in patients with peritonitis is the incidence of unpredictable complications. Unforeseen events may occur that influence the course of the disease. Furthermore, the diversity and individuality of biological reactions may prevent accurate prediction in quite a large proportion of the patients. In this respect we must find out whether for these reasons prediction is simply not possible in most patients or whether the prediction instruments are faulty or inadequate data are used.

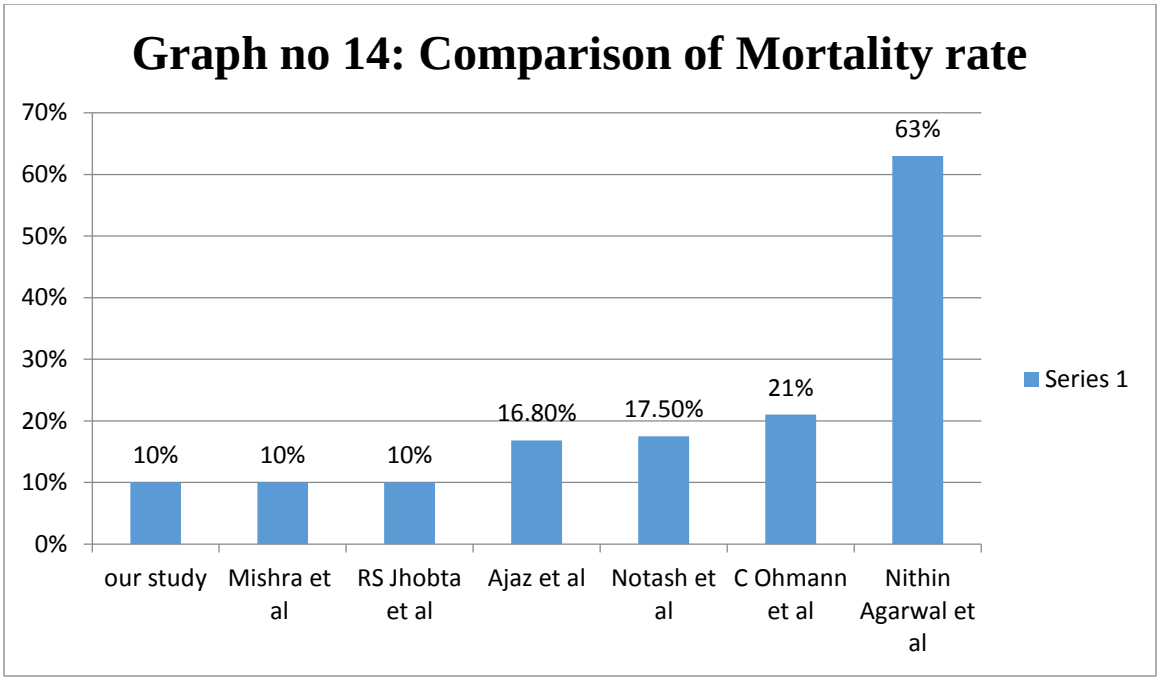
Peritonitis and mortality:

In hospital, mortality rate due to peritonitis remains high. In the current study, the in hospital mortality rate was 28%, most of them were due to septicemia.

The hospital mortality rate according to other studies ranged from 10% in Mishra et al and Jhobta et al and reaching up to 63 per cent in case of Nithin Agarwal et al as in table 24 and graph 14. In all these studies septicemia is the main cause of death.

TABLE 24: MORTALITY RATE IN VARIOUS STUDIES

	Study	Mortality rate
1.	Our study	10%
2	Mishra et al [53]	10%
3	RS Jhobta et al[54]	10%
4	Ajaz et al[2]	16.8%
5	Notash et al[55]	17.5%
6	C Ohmann et al[56]	21%
7	Nithin Agarwal et al[57]	63%



Demography: Age distribution:

The prospective study involved 80 patients of both sexes with secondary peritonitis. Age of the patients in this study ranged from 16years to 75years. The mean age of the patients at the time of admission was 45.55 years(SD 16.43).Maximum number of patients 17(21.3%) were in the age group of 41-50years, 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 50-69years age group. These findings are different from our study.

TABLE 25: COMPARISON OF PREDOMINANT AGE GROUP IN PERITONITIS.

Study	Predominant age group
Samir Delibegovic et al[58]	21-40 years
Ashis Ahuja et al[1]	21-40 years
C Ohmann et al[56]	50-69years
Our study	41-50 years

Age group with highest mortality

Highest mortality in our study was in the age group of 61- 70years. Notash et al[55] also stated mortality(58.8%) being more in >60 years of age C Ohmann et al[56] cited highest mortality in age >70yrs with 37%. In our study it was observed that mortality rate increases with increase in age.

TABLE 26: AGE GROUP WITH HIGHEST MORTALITY

Studies	Age group with highest mortality
Notash et al[55]	>60 years
C Ohmann et al[56]	>70years
Our study	>60years

SEX DISTRIBUTION:

Current study showed the male preponderance in peritonitis with ratio of male: female as 5.6:1. Male preponderance was also found in Samir Delibegovicet al[58] with male to female ratio of 3:1,

Ajazahamed Malik et al[2] with 69:32 and also in Sharma R, Huttunen et al[59]. In our study mortality rate was observed more among males (62.5%) than females (37.5%).

Etiological Spectrum of perforation:

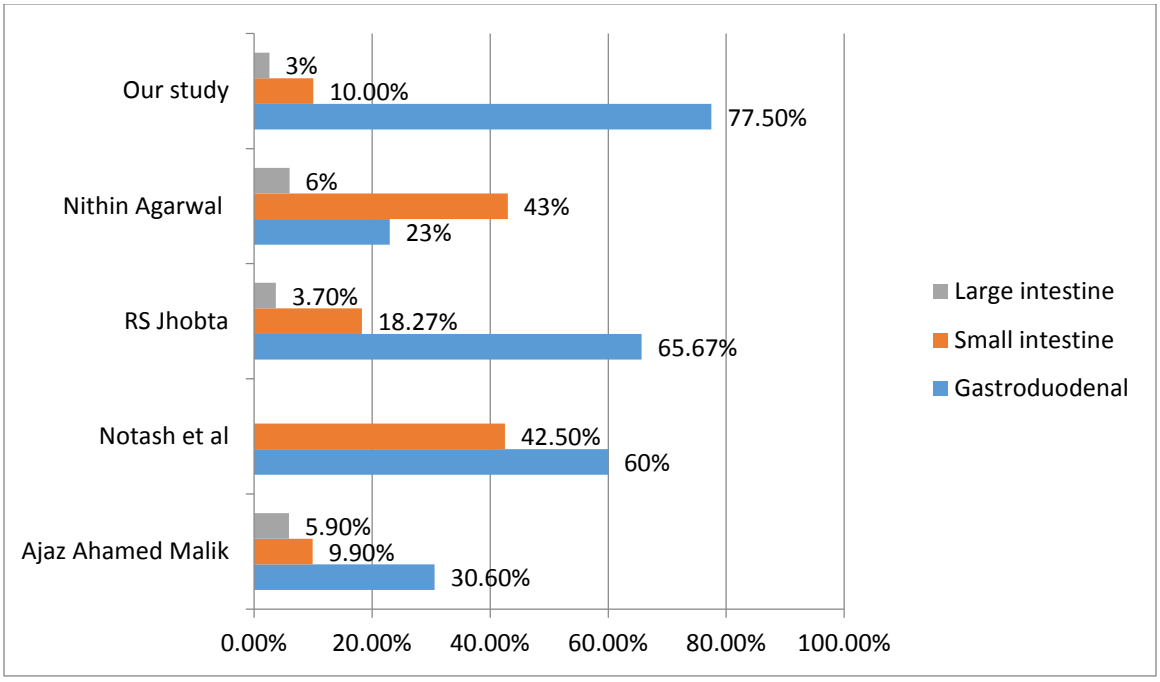
Site of peroration show a wide variability in different studies as shown in table 27 and graph 15.The perforations of proximal gastrointestinal tract were six times as common as perforations of distal gastrointestinal tract as has been noted in earlier studies from India, which is in sharp contrast to studies from developed countries like United States, Greece and Japan which revealed that distal gastrointestinal tract perforations were more common.[54]

Gastroduodenal perforations were most common site of etiology for perforation. But many studies had small intestine as most common site.

TABLE 27: SITE OF PERFORATION IN DIFFERENT STUDY GROUP:

	Study	SITE OF PERFORATION		
		Gastroduodenal	Small intestine	Large intestine
1	AjazAhamed Malik et al[2]	30.6%	9.9%	5.9%
2	Notash et al[55]	60%	42.5	
3	RS Jhobta[54]	65.67%	18.27%	3.7%
4	Nithin Agarwal et al[57]	23%	43%	6%
5	Our study	77.5%	10%	2.6%

GRAPH 15: SITE OF PERFORATION IN DIFFERENT STUDY GROUP



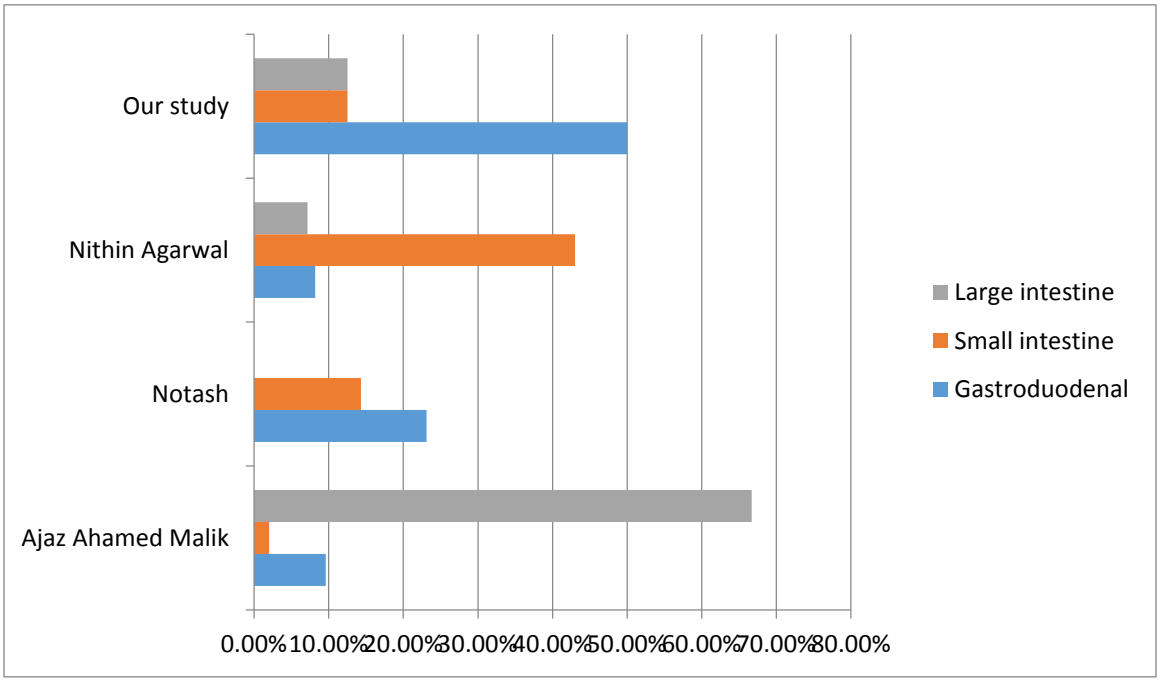
Site specific mortality:

Overall mortality rate in peritonitis due to hollow viscus perforation in our study was 10%. The individual mortality according to etiology showed highest with gastroduodenal perforation (50%) as seen in Notash et al study , but Ajaz found highest mortality in large intestine perforation as shown in table no 28 and graph no 16. Most of the study showed maximum mortality with colonic perforation.

TABLE 28: COMPARING SITE SPECIFIC MORTALITY RATE

	Study	Site specific mortality rate		
		Gastroduodenal	Small intestine	Large intestine
1	AjazAhamed Malik et al[2]	9.6%	2%	66.7%
2	Notash et al[55]	23.1%	14.3%	
4	Nithin Agarwal et al[57]	8.2%	43%	19.2%
5	Our study	50%	12.5%	12.5%

GRAPH 16: SITE SPECIFIC MORTALITY



APACHE II Score:

All the patients were assigned APACHE II score. APACHE II score in our study was from 0 to 30, with the average of 5.84(SD 4.291) points. None of the patients (n-14)with scores more than 20 survived (MR-100%). This finding was consistent with all the other studies .There was 100% mortality in patients whose score was >20 in Ajaz et al,Horiuchi et al andAshish Ahuja studies.

In other studies, different values of scores were reported for the dead patients as shown in the table 29.

TABLE 29: APACHE II SCORE WITH 100% MORTALITY IN VARIOUS STUDIES

	Various World-wide Studies	Apache II scores with 100% mortality
1	our study	>20
2	Ajaz et al[2]	>20
3	Horiuchi et al[60]	>20
4	Ashish Ahuja[1]	>20
5	Samir Delibegovic et al[58]	>28
6	Chen et al[61]	>40
7	Edward et al[62]	>22

MPI score

Our study had MPI score ranging from 10 to 38, the overall mean was 17.49(SD 8.052). None of the patients(n-22) with scores >31 survived. Similarly various studies showed 100% mortality with varied scores as shown in table 30.

TABLE 30: MPI SCORE WITH 100% MORTALITY IN VARIOUS STUDIES

	Studies	MPI scores with 100% mortality
1	our study	>31
2	Ajaz et al[2]	>29
3	Notash et al[55]	>21
4	C Ohmann et al[56]	>30

Accuracy or discriminative ability:

The area under ROC curve measures discrimination, that is, the ability of the scoring system to correctly classify survivors and non survivors. The area below the curve was 0.982 for APACHE II in our study and was consistant with Samir Delibegovic et al study (0.96) implying that it has an excellent discriminative ability where asMishra et al (0.82) and C Ohmann et al (0.87) showed good accuracy. AUC for MPI in our analysis was 0.979 which was consistant with Notash et al (0.97) and Samir Delibegovic et al(0.90) implying excellent discriminative ability but Mishra et al with AUC of 0.85 showed good accuracy where asC Ohmann et al (AUC-0.79) had fair accuracy. Our analysis resulted in APACHE II being more accurate than MPI as with C Ohmann et al and Samir Delibegovic et al where as MPI had better discriminative ability than APACHE IIin Mishra et al study as shown in table 31.

TABLE 31: COMPARISON OF AREA UNDER ROC CURVE IN VARIOUS STUDIES

	Study	Area under ROC curve in APACHE II	Area under ROC curve in MPI
1	our study	0.982	0.979
2	Mishra et al[53]	0.82	0.85
3	Notash et al[55]		0.97
4	C Ohmann et al[56]	0.87	0.79
5	Samir Delibegovic et al[58]	0.96	0.90

Distribution of APACHE II and MPI

Distribution of APACHE II and MPI among survivors showed mean apache score of 4.78±2.63 and mean MPI score of 15.86±6.57 which was found statistically significant(P<0.0001), and non survivors had mean APACHE II score of 15.38±4.65 and mean MPI score of 32.13±4.67 and was statistically significant (P>0.0001).

Thus APACHE II scores were consistent with survivors having lower scores and non-survivors high scores. Similarly MPI scores were also consistent with low scores among survivors and higher scores among non survivors.

TABLE 32: DISTRIBUTION OF APACHE II AND MPI AS IN OTHER STUDIES

Studies	Score	Survivors n=72	Non survivors n=08	P value
Our study	APACHE II	4.78±2.63	15.38±4.65	<0.0001
	MPI	15.86±6.57	32.13±4.67	<0.0001
Notash et al[55]	MPI	19.4(6.7)	33.1(4.8)	<0.0001
A Horiuchi et al[60]	APACHE II	10.4(3.84)	19.3(2.87)	0.00003
	MPI	25.1(4.68)	28.6(5.95)	0.141

n:no of patients

Scoring systems as cited in various other studies are compared in Table 32.

Mean APACHE was lower in survivors than in non-survivors in our analysis and in study by A Horiuchiet al[60]which was statistically significant with P value <0.0001 in both the studies.

Mean MPI was lower in survivors than in non-survivors in our analysisandNotashet al[55] and had statistically significant difference with P value <0.0001 in both the studies. Whereas in Horiuchiet al[60] analyses mean MPI scores among survivors did not vary much from non survivors and was not statistically significant. Thus APACHE II score distribution was significantly better among survivors and non survivors than MPI score distribution.

SHARPNESS

Sharpness is the degree of confidence associated with the predictions- for example, do most of the predictions for survival or death exceed a certain value (> 0.9).

We can conclude from our study that both APACHE II and MPI are sharp in predicting outcome, but MPI is sharper in prediction than APACHE II.

Prediction of sharpness as cited in other studies are listed in table 33.

TABLE 33: SHARPNESS SHOWING COMPARISON WITH OTHER STUDIES

		<0.1 (sharp)	0.1-0.9 (Not sharp)	>0.9 (sharp)
APACHE II	Our study	72 (90%)	7(8.8%)	1(1.2%)
	Samir Delibegovic et al[58]	71(48.9%)	64(44%)	10
	C. Ohmann et al[56]	68(25%)	201(74.1%)	2
MPI	Our study	72 (90%)	5(6.25%)	3 (3.75%)
	Samir Delibegovic et al[58]	0	145(100%)	0
	C. Ohmann et al[56]	164(60.5%)	101(37.2%)	6

Most of the patients in our study (90%) and Samir Delibegovic et al found APACHE II as sharp predictor of outcome as most of low score values had low probabilities of death in both the studies. In addition, APACHE II assigned a high risk of death ($p > 0.9$) to only 1 of 80 patients but in C. Ohmann et al study APACHE II predictions were "not sharp"(74.1%).

MPI was also found to be sharp in predicting outcome in our study which is concurrent with C Ohmann et al. In Samir Delibegovic et al study MPI was not at all sharp as all 145 patients were in moderate risk category(0.1-0.9).

Thus there is varying opinion regarding sharpness of scoring systems in literature.

Reliability of scoring systems:

We analysed Reliability (calibration) of probabilities by comparing observed and predicted death rates (Fig. both APACHE II and MPI scoring systems observed and predicted death rates showed no significant difference). Thus in our analyses both APACHE II and MPI were reliable in predicting prognosis in perforative peritonitis patients.

C Ohmann et al cited that only for APACHE I1 there were no significant differences between observed and predicted death rates, which indicates reliable predictions (goodness of fit). In the middle range (probability of death0.2 and < 0.8) the reliability was good. At the extreme end, probabilities indicated a higher expected death rate than was actually observed. The MPI was not reliable (with differences between expected and observed death rates for small and high probabilities), with higher expected than observed death rates for all probabilities greater than 0.2. In summary, only the APACHE II produced reliable predictions, and the probabilities derived from the MPI score cannot be relied on.[56]

Samir Delibegovic et al have found that the highest rate of correlation between the observed and the expected mortality rate was in APACHE II system thus APACHE II exhibited the best predictive power.

CONCLUSION

Peritonitis secondary to hollow viscus perforation is most common in young males in their prime age. In hospitals, mortality rate for perforative peritonitis remains high in spite of advances in investigation, improved treatment modality, better inpatient care and advanced hospital resources.

Modified APACHE II and MPI scoring predicts mortality which was significant irrespective of the etiology. As per our analyses APACHE II and MPI both had good sensitivity and specificity. Both scoring systems were accurate, sharp and reliable in predicting outcome. But in all these aspects APACHE II was found to be better than MPI in prediction.

Modified APACHE II score considers physiological adversities of the disease which can be used easily and effectively to identify high risk patients for intensive care. Whereas MPI score has the advantage of being easier to calculate with very minimum basic investigations and was specifically designed as a scoring system for peritonitis. The draw back with MPI is that it needs operative findings to complete the scoring.

An efficient scoring system is one which is accurate and sharp in predicting the prognosis and also reliable i.e., which can be reproduced if needed to stratify the patients to risk category. This will help us to divert the resources of the hospital for appropriate patient care and in decisions like transfer of patients to intensive care unit, the choice of more effective antibiotics

and treatment modality. By comparing expected against observed outcome the score can be used to monitor quality of patient care.

These scoring systems are most effective in predicting out come in perforative peritonitis and will be valuable in a tertiary care centre where there is availability of all diagnostic tools and also resources for effective management in terms of ICU care and surgical management of perforative peritonitis.

SUMMARY

Even at this present age mortality due to secondary peritonitis remains one of the major causes of death in surgical wards. We analyzed 80 patients with perforative peritonitis which were confirmed on emergency laparotomy. Mortality rate as cited in various studies ranged from 10% to 60%, our study had only 10% of mortality rate. Most of the patients in our study group were almost equally distributed between 20-70years (92.7%).

The perforations of proximal gastrointestinal tract constituted about 77.5% 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. Mortality rate was highest with colonic perforation (100%) consistent with other studies and all cases with duodenal perforation survived. Patients were subjected to emergency exploratory laparotomy after adequate resuscitation. The surgical procedure performed depended upon the operative findings and the surgeon's choice.

There are several scoring systems available for the estimation of severity of the disease and prognosis in peritonitis patients. Most widely used and accepted is APACHE II scoring system. We evaluated two such scoring systems APACHE II and MPI.

Each patient was assigned both APACHE II score and MPI score.

APACHE II score in our study was from 0 to 30, with the average of 5.84(SD 4.291) points. They were divided into three groups, those with scores <10, 10-20 and >20. One patient with a score of less than 10 expired and 37.5% mortality was seen with scores between 11-20. None of the patients (n=4) with scores more than 20 survived. Survivors had low APACHE II score with

mean of 4.78±2.639 whereas non survivors had higher score with mean of 15.38±4.658 which was statistically significant.

MPI score ranged from 6 to 38, the overall mean was 17.49(SD 8.052). Based upon their MPI score, the patients were divided into three groups according to MPI scores of less than 21, 21-29 and more than 29. None of the patients (n=52) with scores less than 21 expired. We observed 37.5% mortality rate with scores 21-29 and 62.5% mortality with scores >29. Even MPI scores showed low values among survivors with mean 15.86±6.570 and higher values among non survivors with mean 32.13±4.673.

APACHE II and MPI were accurate in predicting the outcome. Accuracy i.e. discriminative ability of the scoring system is measured by area under receiver operative curve. APACHE II with AUC 0.982 was found more accurate than MPI with AUC of 0.979.

In our study, the predictions resulting from APACHE II and MPI were reliable; indicating that risk groups can be defined naturally by probability intervals. We analyzed Reliability (calibration) of probabilities by comparing observed and predicted death rates, both APACHE II and MPI scoring systems observed and predicted death rates showed no significant difference. Thus both APACHE II and MPI are reliable scoring systems.

APACHE II and MPI scores in our analysis were sharp predictor of mortality. The distribution of APACHE II scores has low score values and low probabilities of death (<0.1)72% in our study indicating that its predictions were "sharp" for most cases. The distribution of MPI scores with low score values and low probabilities of death (< 0.1), 72% of the patients had probabilities of death less than 0.10, thus even MPI scores in our study was a sharp predictor of mortality. According to our analysis both Modified APACHE II and MPI scoring predict mortality, which

was significant irrespective of the etiology. APACHE II and MPI both had good sensitivity and specificity. Both the scoring systems were accurate, sharp and reliable in predicting outcome. In all these aspects APACHE II was found to be better than MPI in prediction.

These scoring systems are most effective in predicting out come in perforative peritonitis and will be valuable in a tertiary care centre where there is availability of all diagnostic tools and also resources for effective management in terms of ICU care and surgical management of perforative peritonitis.

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ANNEXURES

PROFORMA

PROGNOSTIC FACTORS IN PERITONITIS DUE TO HOLLOW VISCUS PERFORATION

CASE NO :

1. Name :
2. Age :
3. Sex :
4. I.P.No: O.P.No:
5. Religion :
6. Occupation :
7. Address :
8. DOA : DOD: Date of Surgery: Date of death:
9. CHIEF COMPLAINTS:

a. Pain abdomen – Duration :

b. Vomiting – Duration :

c. Distension of abdomen – Duration :

d. Fever- Duration :

e. Others :

10. HISTORY OF PRESENTING ILLNESS

a. Pain--

- Duration -
- Mode of onset -
- Site -
- Character -
- Shifting / Radiation / Referred -
- Aggravating factors -
- Relieving factors -
- Variations –

b. Vomiting-

- Duration -
- Character - Projectile / non projectile / effortless
- Frequency –
- Vomitus -

∗ Quantity -

∗ Colour -

∗ Contents -

• Relation with Pain -
c. Distension of abdomen -
• Duration -
• Location –
• Onset -
• Progressive / non progressive –
• Association with pain –
• Others –
d. Fever -
• Duration -
• Character–
• Associated with chills & rigors- yes / no
e. Others -
11. PAST HISTORY:
• DM / HTN / TB
• Any chronic illness
• Any h/o Haematemesis / Malaena
• Any Drug intake/ Medications
• Others
12. PERSONAL HISTORY
• Appetite Diet
• Sleep
• Bowel / Bladder
• Any h/o Smoking
• H/o Alcohol consumption
13. MENSTRUAL HISTORY :
• Age of menarche :
• LMP :
• Menstrual cycles :
• Others :
14. FAMILY HISTORY :
15. GENERAL PHYSICAL EXAMINATION
• Built and nourishment :
• Level of Consciousness :
• Appearance :
• Vital Data:
a. Temperature
b. Pulse
c. BP
d. RR

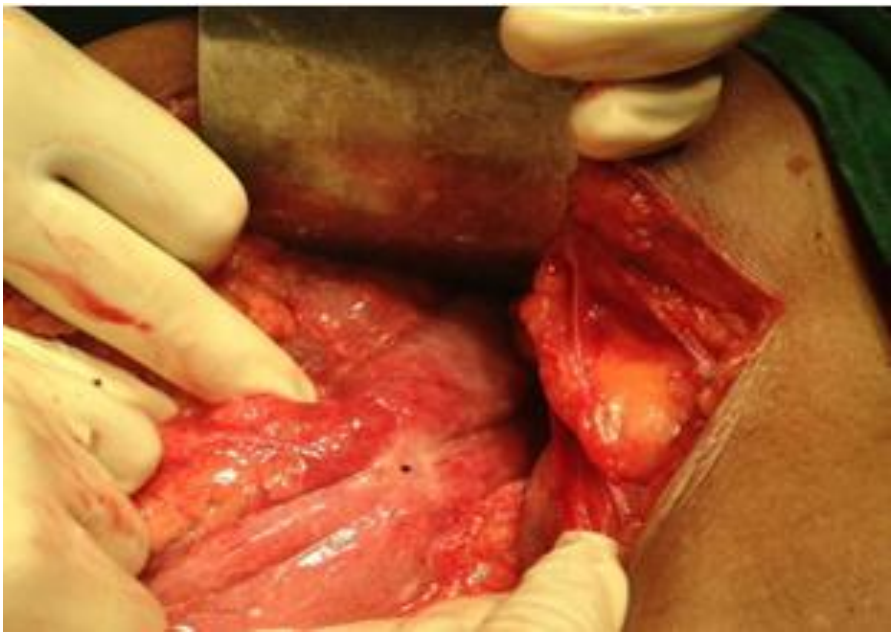
e. Spo2
f. Pallor ( ) / Icterus ( ) / Clubbing ( ) / Cyanosis ( ) /
Lymphadenopathy ( ) / Edema ( )
• Others
16. EXAMINATION OF ABDOMEN:
a. Inspection
• Umbilicus : (a) position (b) shape
• Contour of abdomen :
• Skin over abdomen :
• Movements :
• Visible peristalsis :
• Pulsations :
• Hernial orifices
b. Palpation:
• Local rise of temp – present / absent
• Tenderness - Localised / Generalised
• Guarding – present / absent
• Rigidity - present / absent
• Hernial orifices
c. Percussion
• Shifting dullness -
• Fluid Thrill -
• Obliteration of liver dullness -
d. Auscultation
• Bowel Sounds - Normal / Decreased / Increased / Absent
17. OTHER SYSTEMS EXAMINATION:
• RS -
• CVS -
• CNS -
18. INVESTIGATIONS:
- ABG

• Routine Blood
a. Hb %
b. TLC DLC
c. PLT
d. BT - CT -
e. RBS & FBS / PPBS
f. Blood Urea
g. Serum Creatinine

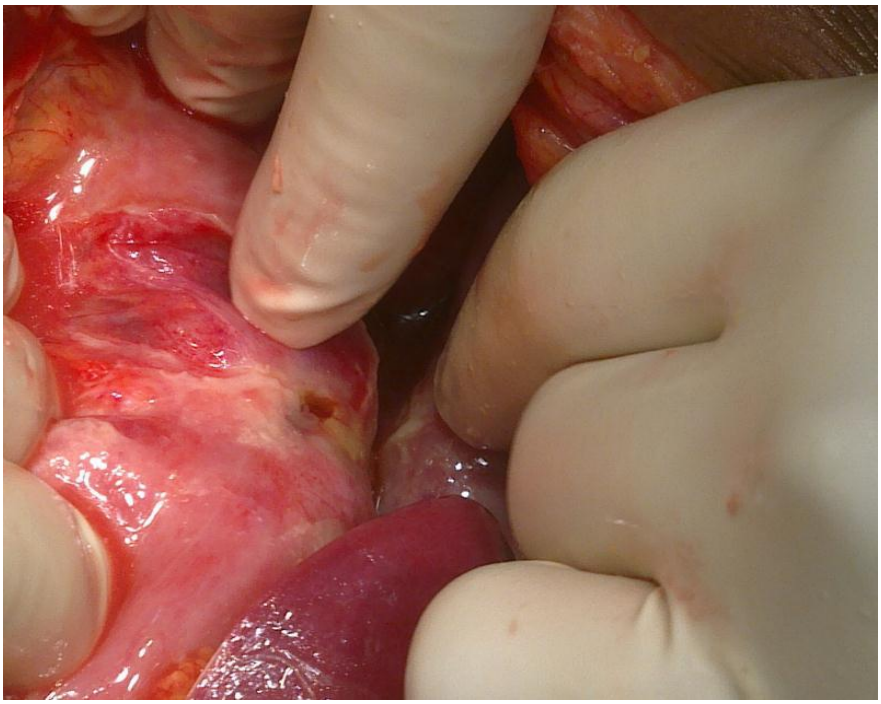
h. LFT
i. WIDAL
j. Serum Electrolytes – Na, K.
k. Serum Amylase
• Urine Routine -Albumin – present /absent
-Sugar - present / absent
Microscopy -
Culture -
• Stool examination –
• Radiological - Erect abdomen X-ray / Left Lateral
• Chest X ray –
• USG abdomen
• ECG
• Diagnostic tap of Peritoneal fluid -
a. Color:
b. Smell :
• Peritoneal culture
• Biopsy from edge of the perforation-
• Others -
19. DIAGNOSIS: peritonintis secondary to .....Perforation
20. SCORING SYSTEM: APACHE II score - . MPI score .
21. SURGICAL PROCEDURE:
22. PER OP FINDINGS
23. POST-OPERATIVE COURSE:

PICTURE GALLERY

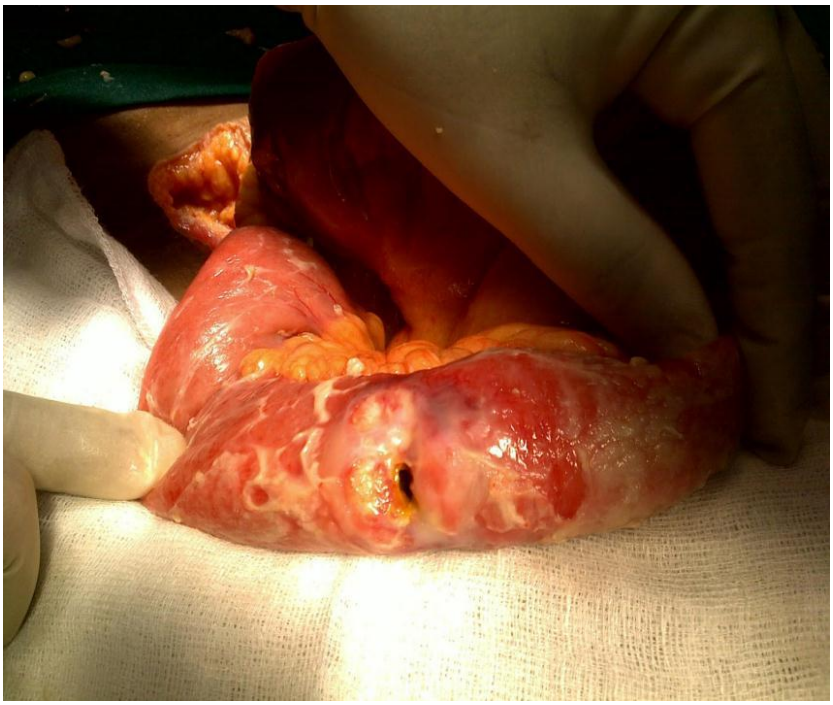
Pre pyloric Perforation



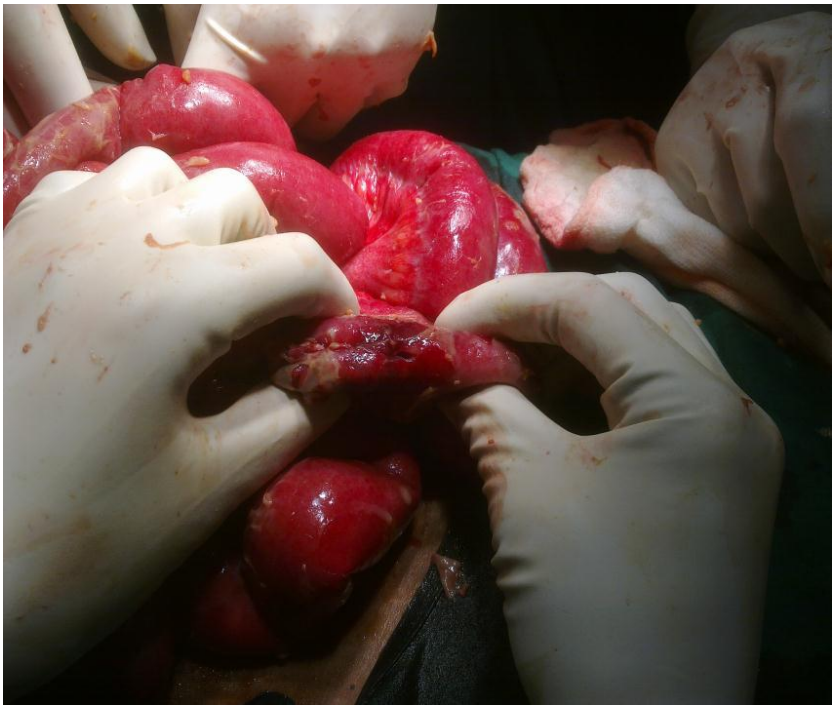
Duodenal perforation



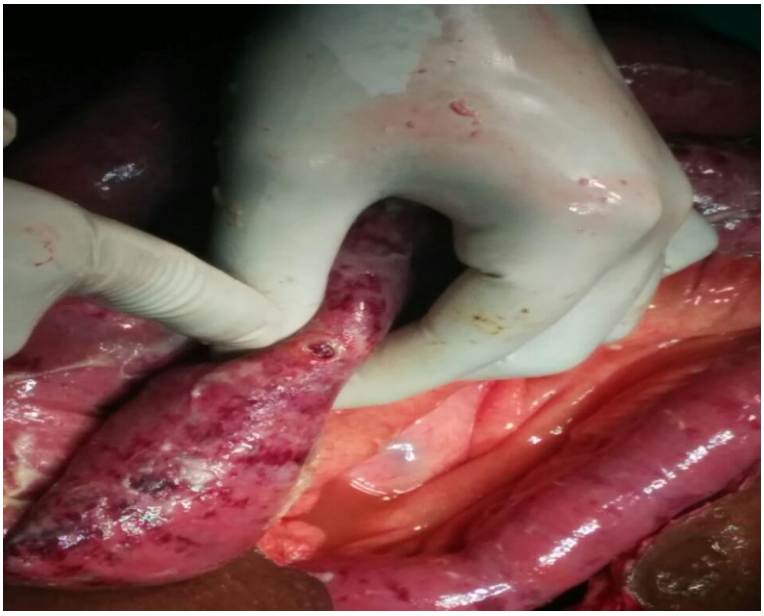
Ileal perforation



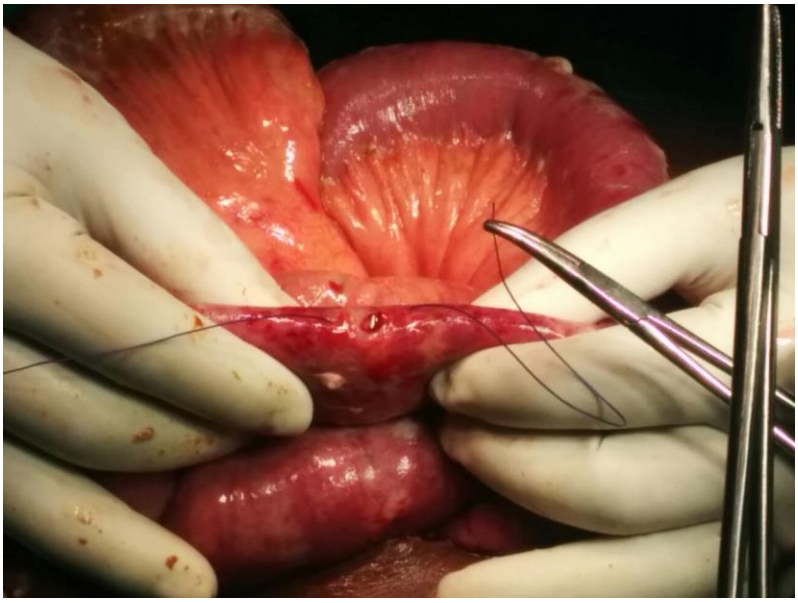
Ileal perforation



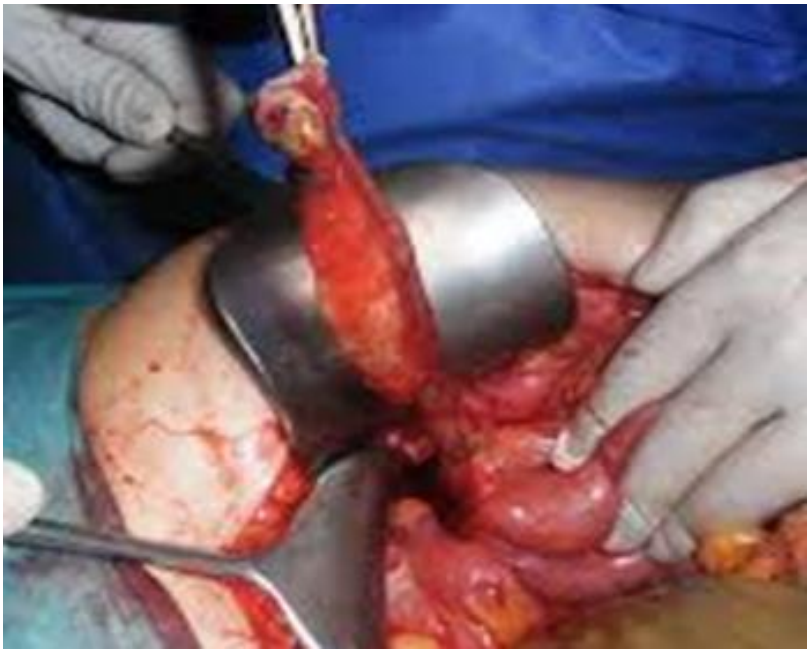
Jejunal perforation



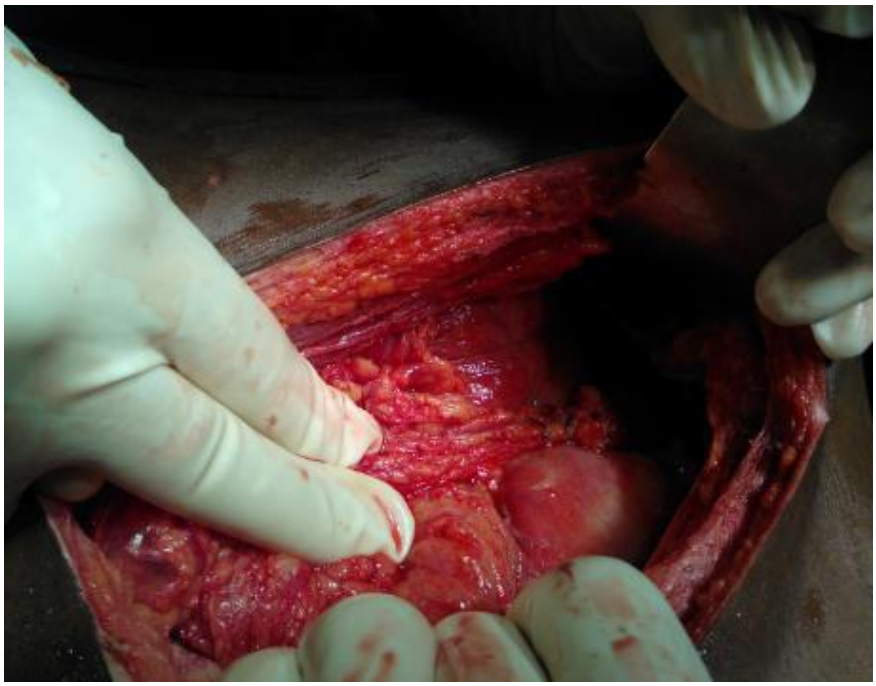
Primary closure of jejunal perforation



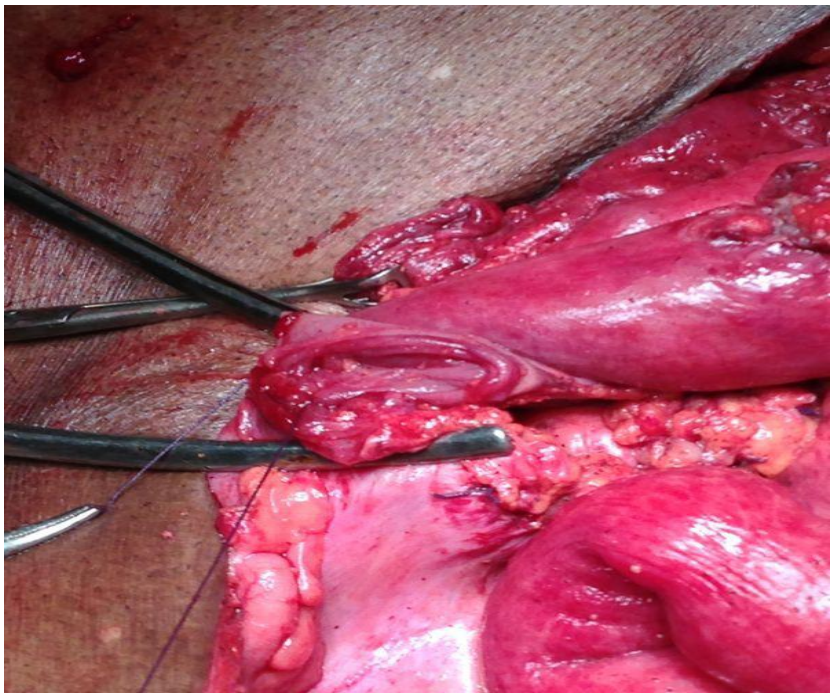
Perforated Appendix at the tip

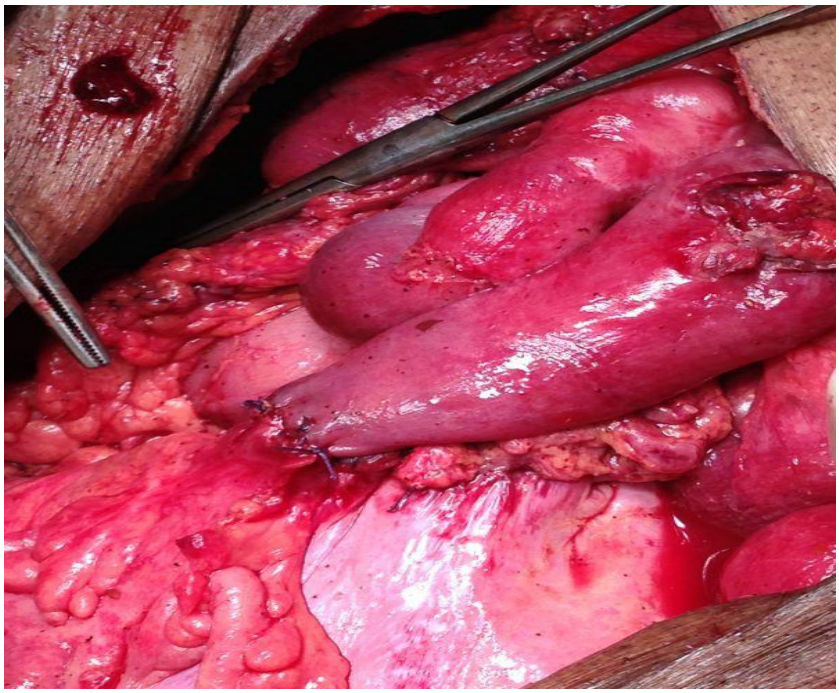


Grahams Omentoplasty repair for duodenal perforation



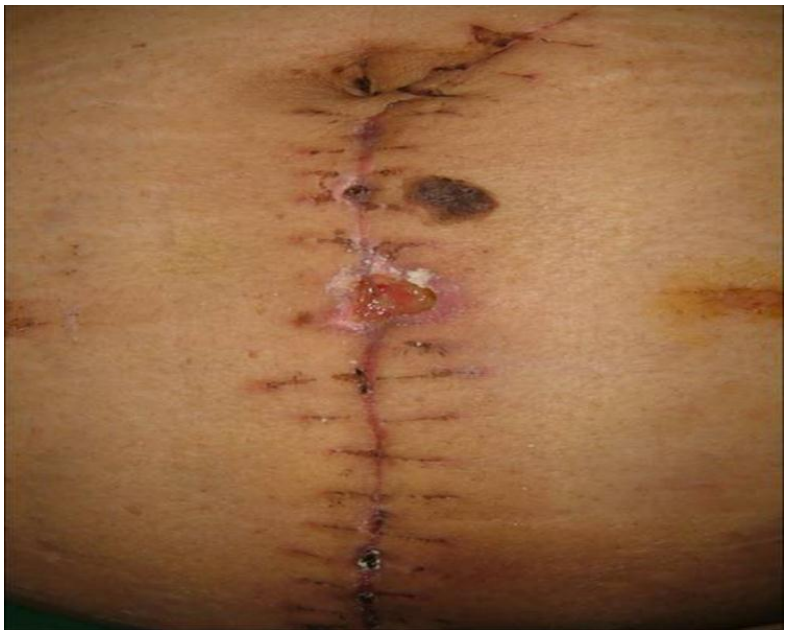
Resection and anastomosis for terminal ileal perforation



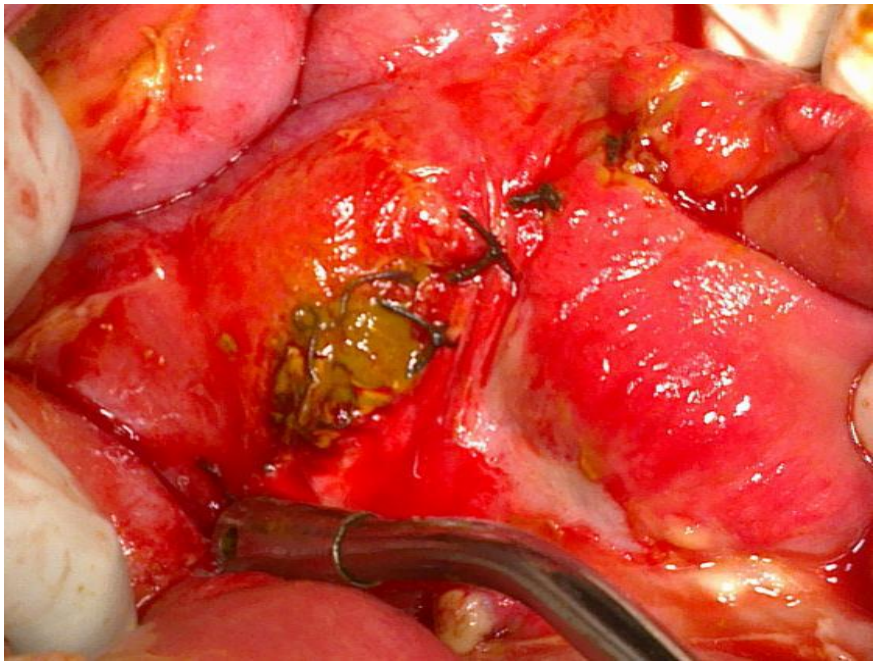


POST OPERATIVE COMPLICATIONS

Wound infection



Anastomotic leak



INFORMED CONSENT FORM

If you agree to participate in the study your information will be collected(as per proforma) from you or a person responsible for you or both. We will collect the treatment and relevant details from your hospital record. This information collected will be used for only dissertation and publication. This study has been reviewed by the institution ethical committee. There is no compulsion to agree to this study. The care you will get will not change if you don't wish to participate. You are required to sign/ provide thumb impression only if you voluntarily agree to participate in this study.

I understand that I remain free to withdraw from the study at any time and this will not change my future care. I have read or have been read to me and understood the purpose of the study, the procedure that will be used, the risk and benefitsassociated with my involvement in the study and the nature of information that will becollected and disclosed during the study. I have had the opportunity to ask my questions regarding various aspects of the study and my questions are answered to my satisfaction. I the undersigned agree to participate in this study and authorize the collection and disclosure of my personal information for dissertation.

Subject name: DATE:
Signature/ thumb print:

Parents/ guardians name: DATE:
Signature/ thumb print:

Signature of person taking consent: DATE

KEY TO MASTER CHART

M - Male

F - Female

IP No - In patient number

APACHE II - Acute Physiological and Chronic Health Evaluation II

MPI - Mannheims Peritonitis Index

SSI - Surgical Site Infection

Master Chart

NAME	AGE	SEX	IP NO	SITE OF PERFORATION	DURATION	ORGAN FAILURE	EXUDATE	APACHE2	MPI	COMPLICATIONS						OUTCOME
										RESPIRATORY	RENAL	SSI	SEPSIS	BURST ABDOMEN	PARALYTIC ILEUS	
Sonappa	68	M	399383	duodenal	3 days	-	-	5	21	+	-	-	-	-	-	improved
Syed Ameer	65	M	2954	gastric	1 day	-	-	5	17	+	-	-	+	-	-	improved
Krishnappa	45	M	398463	duodenal	2 days	-	-	2	16	-	-	-	-	-	-	improved
Venkataravanappah	60	M	393767	duodenal	1 day	-	-	2	19	-	-	-	-	-	-	improved
Akbar	50	M	38772	colon	1 day	-	+	12	27	-	-	-	+	-	-	expired
Nagaraj	32	M	397938	gastric	1 day	-	-	2	6	-	-	-	+	-	-	improved
Parvathamma	28	F	21261	duodenal	3 days	-	-	6	21	+	-	+	-	-	-	improved
Akbar	17	M	396750	duodenal	1 day	-	-	11	21	-	-	+	-	-	+	improved
Sri Ramappa	55	M	401148	prepyloric	1 day	-	-	8	16	-	-	-	-	-	-	improved
Ganesh	23	M	20662	duodenal	3 days	-	-	7	14	-	-	-	-	-	-	improved
Venkateshappa	70	M	403002	duodenal	2 days	-	-	1	15	-	-	-	+	-	-	improved
Shivanna	70	M	25690	gastric	6days	-	+	8	27	+	+	+	-	-	+	improved
Lakshmidevamma	30	F	389269	ileal	3 days	-	+	5	31	-	-	+	-	-	-	improved
somappa	85	M	7089	ileal	5 days	-	+	6	25	-	-		+	-	+	improved
Rangappa	58	M	401470	ileal	1day	-	-	2	11	-	-	+	-	-	-	improved
Sri Ramappa	50	M	389777	pyloric	2 days	-	-	3	10	-	-	-	-	-	-	improved
Shekar	16	M	7183	appendix	3days	+	+	6	23	-	+	-	-	-	-	improved
Ganesh. G	55	M	991995	not known	3days	+	+	17	28	-	+	-	+	-	-	expired
Ambrish	23	M	6932	duodenal	1 day	-	-	3	6	-	-	+	-	-	+	improved
Vasantha	25	F	396197	pyloric	1 day	-	+	6	20	-	-	-	-	-	-	improved
Kempanna	70	M	591	jejunum	1 day	-	-	9	24	-	-	+	-	-	+	improved
Rajan Babu	65	M	8036	pyloric	2 days	-	-	2	15	-	-	-	-	-	-	improved
Manjunath	25	M	36971	gastric	1 day	-	-	2	6	-	-	-	-	-	-	improved
Yellappa	50	M	16284	duodenal	1 day	-	-	5	15	-	-	-	-	-	-	improved
Naramma	50	F	38844	unknown	1 day	-	-	24	37	-	+	-	+	-	-	expired
Venkataravanappa	65	M	12639	duodenal	1 day	-	+	3	11	-	-	+	-	-	-	improved
Chikkanarayanappa	65	M	12721	duodenal	2 days	-	+	2	21	+	-	-	-	-	-	improved
Muniyappa	65	M	14434	duodenal	1 day	-	-	2	11	-	-	-	-	-	+	improved
Srinivas	40	M	46622	pyloric	2 days	-	-	6	15	-	-	-	-	-	-	improved
Sri Ramappa	55	M	401148	pyloric	1 day	-	-	3	10	-	-	-	-	-	-	improved
Munireddy	55	M	18283	pyloric	3 days	+	+	2	28	-	+	+	-	-	+	improved
Suresh	26	M	5455	gastric	3 days	-	-	6	15	-	-	-	-	-	-	improved
Lagumanna	60	M	168513	pyloric	1 day	-	+	3	17	-	-	+	-	-	-	improved
Venkateshappa	55	M	26557	jejunum	10 days	+	+	17	38	+	+	+	+	-	+	expired
Chalapathi	32	M	28073	gastric	1 day	-	-	2	20	-	-	-	-	-	-	improved
Yerappa	45	M	51209	pyloric	3 days	-	-	8	24	-	-	-	-	-	-	improved
Fayaz khan	32	M	6167	gastric	1 day	-	-	3	6	-	-	-	-	-	-	improved
Shiva kumar	47	M	25675	pyloric	2 days	-	-	4	10	-	-	+	-	-	-	improved
Thimmarayappa	23	M	29925	gastric	2 days	-	-	1	10	-	-	-	-	-	-	improved
Rathnamma	56	F	56345	duodenal	3 days	-	-	8	21	+	-	+	-	-	+	improved
Soma reddy	39	M	23467	duodenal	1 day	-	-	3	10	-	-	-	-	-	-	improved
Nayappa	45	M	12678	ileal	3 days	-	+	8	26	-	-	+	-	-	+	improved
Nagaraj	40	M	31744	ileal	2 days	-	+	6	26	-	-	-	+	-	+	improved
Venkateshappa	38	M	43613	gastric	1 day	-	-	2	6	-	-	-	-	-	-	improved
Gangalappa	30	M	16547	duodenal	3 days	-	-	2	14	-	-	-	+	-	-	improved
Gowramma	48	F	65743	duodenal	2 days	-	-	6	16	-	-	-	-	-	-	improved
Muniyamma	65	F	40704	gastric	3 days	+	-	14	27	-	+	+	-	-	+	expired
Nagaraj	28	M	46481	duodenal	3 days	-	-	3	10	-	-	-	-	-	-	improved
Chandramma	22	F	46737	pyloric	7 days	-	+	3	21	+	-	+	+	-	-	improved
Narayanappa	65	M	34578	pyloric	2 days	-	-	6	16	+	-	-	-	-	+	improved

Erappa	65	M	50950	gastric	3 days	+	+	8	32	+	+	+	+	-	+	expired
Ramappa	55	M	75839	duodenal	1 day	-	-	2	6	-	-	-	-	-	-	improved
Chinappa	55	M	187601	pyloric	2 days	-	+	7	15	+	-	+	-	-	-	improved
Narayanamma	45	F	36507	pyloric	4 days	+	+	17	37	+	+	-	+	-	+	expired
Rama Rao	48	M	48612	duodenal	3 days	-	-	7	17	-	-	+	-	-	-	improved
Byroji Rao	69	M	45912	pyloric	3 days	-	-	6	25	-	-	+	-	-	-	improved
Chan Pasha	45	M	25440	rectum	1 day	-	+	5	28	-	-	-	+	-	-	improved
Tabresh Pasha	36	M	41770	appendix	3 days	-	+	6	11	-	-	+	-	-	+	improved
Venkataswamy	35	M	23737	duodenal	1 day	-	-	3	6	-	-	-	-	-	-	improved
Venkataravanappa	45	M	19870	duodenal	5 days	+	+	8	20	+	-	+	-	-	+	improved
Vemanna	35	M	1027	appendix	3 days	-	+	3	14	-	-	+	-	-	-	improved
Channabasavaiah	65	M	4025	duodenal	1 day	-	-	3	11	+	-	+	-	-	-	improved
Akram	18	M	390923	ileal	4 days	-	+	6	28	-	-	+	-	-	+	improved
Krishnappa	45	M	20313	duodenal	1 day	-	+	2	10	-	-	-	-	-	-	improved
Kumar	50	M	391667	pyloric	1 day	-	-	15	18	+	-	+	-	-	-	improved
Manjunatha	33	M	1582	duodenal	1 day	-	-	5	16	-	-	+	-	-	-	improved
Manjunath V	30	M	20098	duodenal	7 days	+	+	9	23	-	+	+	-	-	+	improved
Manjunathchari	35	M	13938	duodenal	2 days	-	+	7	14	-	-	-	-	-	-	improved
Nanjamma	60	F	12471	duodenal	1 day	-	-	8	23	-	-	+	-	-	-	improved
Parvathamma	45	F	397795	appendix	3 days	-	+	5	11	-	-	+	-	-	-	improved
Narayanaswamy	32	M	30709	appendix	4 days	+	+	6	10	-	+	+	-	-	-	improved
Pilappa	65	M	395660	gastric	3 days	+	-	14	31	+	+	-	-	-	+	expired
Prabhavathi	30	F	33910	duodenal	1 day	-	-	3	10	-	-	-	-	-	-	improved
Redappa	30	M	18141	duodenal	3 days	+	-	3	14	-	-	+	-	-	+	improved
Rizwan R	25	M	51488	pyloric	2 days	-	-	6	16	-	-	-	-	-	-	improved
Sagarsindhya	19	M	19064	duodenal	1 day	-	-	3	6	+	-	+	-	-	-	improved
Sonnappa	45	M	10870	duodenal	2 days	-	-	7	15	-	-	-	-	-	-	improved
Srinivas	27	M	390882	duodenal	1 day	-	-	2	6	-	-	-	-	-	-	improved
Velu KT	56	M	166049	pyloric	2 days	-	+	6	16	+	-	-	-	-	-	improved
Nanjappa	75	M	2365	appendix	3 days	-	+	3	10	-	-	+	-	-	-	improved