# "PREDICTION OF OUTCOME OF PATIENTS WITH PERFORATIVE PERITONITIS ON THE BASIS OF APACHE III SCORING SYSTEM"

**By**

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**Dissertation submitted to**


In partial fulfillment for the degree of

**MASTER OF SURGERY**

**IN**

**GENERAL SURGERY**

**Under the guidance of**

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**VIJAYAPUR – 586103**

**2017**

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*I hereby declare that this dissertation entitled* ***"PREDICTION OF OUTCOME OF PATIENTS WITH PERFORATIVE PERITONITIS ON THE BASIS OF APACHE III SCORING SYSTEM*** *"is a bonafide and genuine research work carried out by me under the guidance of* ***DR. M.B PATIL,****Professor, Department of General Surgery, Shri. B.M. Patil Medical College, Hospital and Research center, Vijayapur.*

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## ***ACKNOWLEDGEMENT***

*On completion of my post graduation journey and this scientific document, I would like to acknowledge the immense help received from my mentors in the Department of General Surgery.*

*With privilege and respect I would like to express my gratitude and indebtedness to my Guide,**Dr. M.B PATIL ,**for hisconstant inspiration, extensive encouragement and loving support, which he rendered in pursuit of my post-graduate studies and in preparing this dissertation.*

*I am forever grateful to Professors,**Dr. TejaswiniVallabha, Dr.AravindPatil, Dr.B.B.Metan,Dr.M.S.KottenavarDr.VijayaPatil** for their guidance and encouragement provided to me, to achieve new heights professionally over my course period.*

*I am grateful to Associate Prof.Dr.Basavaraj.Narasangi, Dr.Hemanth.Kumar, Dr.Girish .Kulloli for their guidance, encouragement and inspiration.*

*I am thankful to Dr. Dayanand..S.Biradar, Dr.Vikram.Sindagikar, Dr. Deepak. Chavan, Dr. S.S.Patil, Dr. Shailesh.Kannur, Dr. Surekha.Rathod, Dr. Harshavardhan. Biradar for their great help.*

*I am extremely thankful to Prof. Dr.S P Guggarigoudar, Principal, of B.L.D.E.U'S Shri.B.M. Patil Medical College, Hospital and Research Centre, VIJAYAPUR, for permitting me to utilize resources in completion of my work.*

*I also thank MrMohd. Shahnawaz Lecturer Statistics, for his guidance during my dissertation work.*

*My thanks to one and all staff of Library, GENERAL SURGERY Department and Hospital for their co-operation in my study.*

*I am thankful to my seniors, Dr.Ashrith.I.M, Dr.Sunil.K, Dr.Rakshith, Dr.Bharath, Dr.Ravi.I, Dr.Anikethan.V, Dr.Anand.S, Dr.Jadesh, Dr. Umesh, Dr. Rohith, Dr. Mallikarjun, Dr. Abhilash for their advice, suggestions and co-operation in my journey.*

*I would also like to thank my colleaguesDr. Suhas t Dr.AhmedFaraz Patel, Dr. Anup.Kubsad,Dr. Varun Kumar Damera, Dr.MrinalKumar,Dr.Kruti for their help and co-operation.*

*I would also like to thank my juniors Dr.Balakrishna, Dr.Vijaykumar, Dr.Surya, Dr.Santhosh, Dr. Manoj, Dr.Harsh,Dr. Ritesh for their support and co-operation.*

*I am deeply indebted to my parent's keeni raja reddy and sujatha for their blessings, which helped me to complete this dissertation. .*

*Last but not the least; I convey my heartfelt gratitude to all my patients, without whose co-operation, this study would be incomplete.*

**DR. KEENI DILIP REDDY**

# ABSTRACT

**Background & Objectives:**

This was a prospective study to predict the outcome of patients with perforative peritonitis on the basis of APACHE III scoring system.

**Methods:**

This study consists of 100 selected cases patients admitted with features suggestive of perforative peritonitis condition in B.L.D.E.U s Shri.B.M.Patil Medical College, Hospital and Research Centre, Vijayapur from October 2014 to June 2016. All the patients were assigned APACHE III scoring system.

**Results:**

In this study mortality rate was 21%. Highest mortality was in the age group of 51-60(30.83%). Lowest mortality (1) is seen in age group of 21-30years, In this study mortality increased with increasing age. In this study mortality was observed more among male sex (17.9%), Indicating male sex is a prognostic factor. The most common diagnosis in this study was duodenal perforation (29.4%). All patients were divided into 3 groups with scores <60,31-60 and >60. Mortality was 4% in patients with score<30, mortality was 5.4% in patients with score 30-60, and mortality was 47% in patients with score >60. In this study mortality was observed more among patients with higher APACHE III score >60. Mean duration of hospital stay in this study was 12.3 among non-survivors and 18.3 days among survivors. Most common postoperative complication in this study among patients with higher APACHE III score was wound infection (23%) followed by burst abdomen (18%), septicemia (11%) and fecal fistula (8%). Patients with higher APACHE III score in this study had lesser duration of stay in hospital due

tomortality and more risk of postoperative complications. Mean APACHE III score among non-survivors was 56.6 and among survivors were 50.4. Using ROC analysis the area under curve was found to be 0.955 correlation of APACHE III score and predicted death rate showed perfect correlation in patients with score <60. (P <. 001).In patients with score >60 observed death rate was higher than predicted death rate in this study.

**Interpretation and Conclusion:**

Patients with lower APCHE III scores have more favorable prognosis than patientswith higherAPCHE III score.Patients with higher APACHE III score had more risk of postoperative complications. In patients with higher APACHE III score>60, predicted mortality did not correlate with observed hospital mortality in this study. Thus it was concluded from this study that APACHE III score was reliable in predicting mortality for patients with score < 60.And APACHE III score, as measured before the treatment of perforation peritonitis correlates significantly with the outcome of disease in respect to both morbidity and mortality.

**Keywords: Peritonitis, APACHE, Prognosis, and Outcome.**

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# INTRODUCTION

Peritoneum inflammation, called peritonitis, presents most commonly due to localized or generalized infection caused from various probable factors. Secondary peritonitis is the most common & follows an intraperitoneal source usually from perforation of hollow viscera. Acute generalized peritonitis coming forth due to underlying hollow viscus perforation is a critical & life-threatening medical 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 agreater degree, the physiologic compromise it inflicts and affects the outcome.

High-risk patients require timely & aggressive treatment especially in severe peritonitis & to select them reasonably well; evaluation through prognostic scoring is an approach of choice. Early prognostic evaluation is desirable to be able to select high-risk patients for more aggressive treatment especially in severe peritonitis.[1]

Various scoring systems have been used to assess the prognosis and outcome of 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).[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.[3]

The APACHE prognostic scoring system for measuring severity of illness in critically ill patients was developed in 1981 by William A Knaus.[4]APACHE - II introduced in 1985 was a simplified modification of original APACHE. APACHE - II was further refined to APACHE - III in 1991. It is important for surgeons to develop at least a rudimentary knowledge of scoring system for perforation peritonitis, as it will play an increasing role to explain the prognosis of the disease.

Various authors have reported that recently introduced APACHE 111 scoring system is superior to established but older APACHE 11 scoring system[5]

5 points increase in APACHE III score (range 0 – 299) is independently associated with a statistically significant increase in the relative risk of hospital death[6].

## AIMS AND OBJECTIVES

1. To predict the outcome of patients with perforative peritonitis on the basis of APACHE III scoring system.

## REVIEW OF LITERATURE

### History

Peritonitis was recognized as a universal fatal condition from the earliest of times. Monographs and review articles on peritonitis are found almost exclusively in the surgical literature. An historical perspective of the slow unraveling of the pathology, microbiology, and evolution of the treatment is best appreciated in "The peritoneum" by Hertzler[7] (1919), "Infections of the peritoneum" by Steinberg(1944),and reviews by Hedberg and Welch and Hauet al[8]. Kennedy (1951) found the incidence of perforation in carcinomatous ulcer to be at least 16.7 % of all gastric perforation and 5.4 % of all gastro-duodenal perforations.

The importance of correct diagnosis and treatment of gastroduodenal perforation is gradually increasing due to high incidence of mortality of 10-20% (Bryne) and gradual increase in the incidence of perforation every year.

Jamieson (1955) reported that the incidence of perforation increased three fold between 1924 and 1958.

Portis and Jaffo(1936) found the occurrence rate of perforation to be 14% of all ulcer patients.

Georg Wegener in Berlin was the first to conduct a series of logical experiments about the physiology of the peritoneal cavity. His results, reported to the German Surgical Society in 1876.[9]The current therapy of peritonitis at the time was summarized by Martin Kirschner[11] in 1926. His therapeutic principles are valid to this day, and his article represents a hallmark in the therapy of intraperitoneal infections.

**Its conclusions, briefly, are:**

1. The operative procedure and the anesthesia should be conducted as gently as possible.
2. Every patient with acute diffuse peritonitis should be operated on immediately unless there is an absolute contraindication to surgery.
3. The incision should be made over the focus of infection. If there is any doubt, a median laparotomy should be performed. The incision should be long enough to allow easy excess to the infectious focus.
4. The most important aim of surgery is the secure elimination of the source of infection. This should be done by the simplest possible procedure.
5. Exudate and debris found in the peritoneal cavity are removed by irrigation with normal saline solution.
6. The free peritoneal cavity cannot be drained, and drains should not be used. Only if secure elimination of the infectious focus is impossible is drainage indicated.

## ANATOMY OF PERITONEUM AND PERITONEAL CAVITY

### Embryology

Intra embryonic mesoderm differentiates into paraxial mesoderm, intermediate mesoderm, and lateral plate mesoderm by 3rd week of embryogenesis. 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 mesothelium 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.[13]

**Formation of the Peritoneal Ligaments and Mesenteries**[19]

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

**Formation of the Lesser and Greater Peritoneal Sacs**[19]

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**[19]

The spleen is developed in 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 ofthe lesser sac extends inferiorly between the anterior and the posterior layers of the fold of the greater omentum.

## SURGICAL ANATOMY[5,7-10]

The peritoneum is a continuous, glistening and slippery transparent serous membrane. It lines the abdominopelvic cavity and invests the viscera. The peritoneum consists of two continuous layers the: the parietal peritoneum, which lines the internal surface of the abdominopelvic wall, and the visceral peritoneum, which invests viscera such as stomach and intestines. Both layers of peritoneum consist of mesothelium, a layer of simple squamous epithelial cells[10].

The parietal peritoneum is served by the same blood and lymphatic vasculature and the same somatic nerve supply as is the region of the wall it lines. Like the overlying skin, the peritoneum lining the interior of the body wall is sensitive to pressure, pain, and heat and cold, and lacerations. Pain from the parietal peritoneum is generally well localized, except for the on the inferior surface of the central part of diaphragm, where innervations is provided by phrenic nerve, irritation here is often referred to the C3-C4 dermatomes over the shoulder[10].

The visceral peritoneum and the organs it covers are served by the same blood and lymphatic vasculature and visceral nerve supply. The visceral peritoneum is insensitive to touch, heat and cold, and lacerations; it is stimulated primarily by stretching and chemical irritation. The pain produced is poorly localized, being referred to dermatomes of spinal ganglia providing sensory fibres, particularly to midline portions of these dermatomes. Consequently, pain from the foregut derivatives is usually experienced in the epigastric region, that from midgut derivatives in the umbilical region, and that from hindgut derivatives in the pubic region[10].

The peritoneal cavity is within the abdominal cavity and continues inferiorly into pelvic cavity. The peritoneal cavity is a potential space of capillary thinness

between the partial and visceral layers of peritoneum. It contains no organs but contains a thin film of peritoneal fluid, which is composed of water, electrolytes, and other substances derived from interstitial fluid in adjacent tissues. Peritoneal fluid lubricates the peritoneal surfaces, enabling the viscera to move over each other without friction and allowing the movements of digestion. In addition to lubricating the surfaces of the viscera, the peritoneal fluid contains leukocytes and antibodies that resist infection.

Lymphatic vessels, particularly on the inferior surfaces of the diaphragm, absorb the peritoneal fluid. 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 exterior.

**Table 1: Parts of the Peritoneum[11]**

| | |
|---|---|
| Omenta | Great 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[11]**

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.

**Innervations of the Peritoneum[11]**

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, as 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.[12]

**Spaces in the peritoneum:**

The peritoneal cavity is subdivided into interconnected compartments or spaces by 11 ligaments and mesenteries.[13]

The peritoneal ligaments or mesenteries include the[13]

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

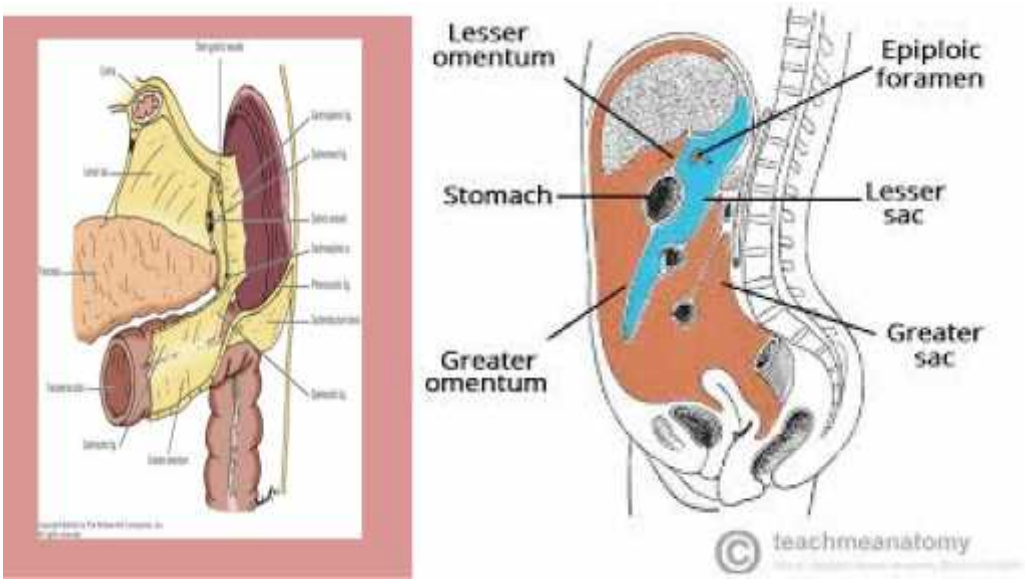


**Figure 1: Peritoneal ligaments and mesenteric reflections in the adult.** [13]

**Peritoneal Recesses, Spaces, and Gutters**

These ligaments partition the abdomen into nine potential spaces:[13]

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..These attachments partition the abdomen into nine potential spaces and are represented in figure no 2.

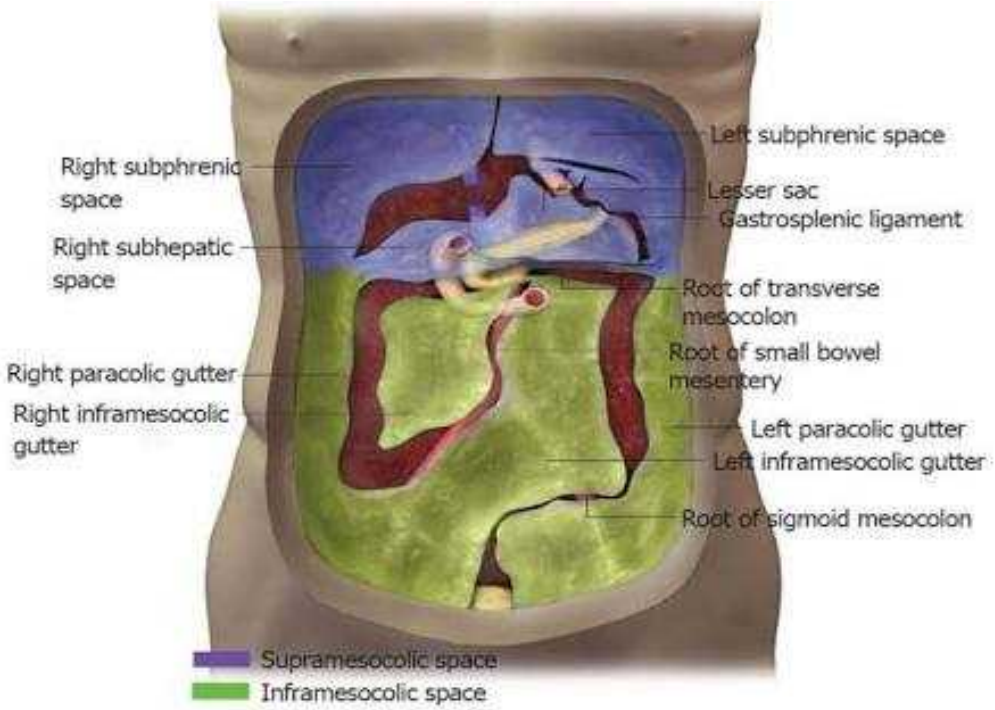


**Figure 2: Spaces in the peritoneum:**

**Greatersac:**

The peritoneal cavity is the largest cavity in the body and is divided into two parts: the greater sac and the lesser sac (fig: 3). The greater sac is the main compartment and extends from the diaphragm down into the pelvis.

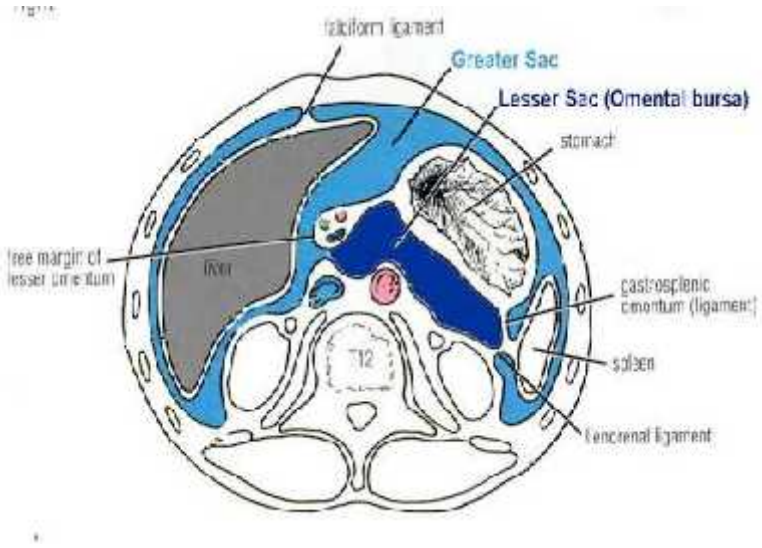


**Figure 3 : GREATER AND LESSER SAC**

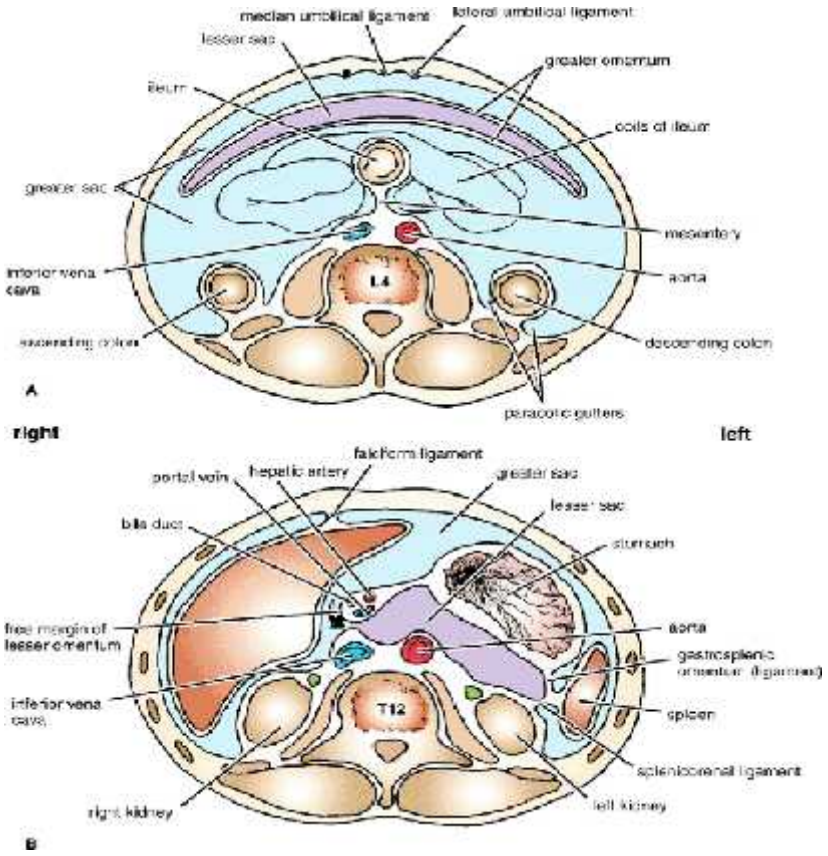


**Figure 4 (A): Vertical disposition of the peritoneum (abdominopelvic cavity)**

**Figure 4(B): Transverse sections of the abdomen showing the arrangement of the peritoneum**

**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 spleen and the gastrosplenicomentum and splenicorenal ligament form the left margin of the sac. 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.

**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 (fig: 4)

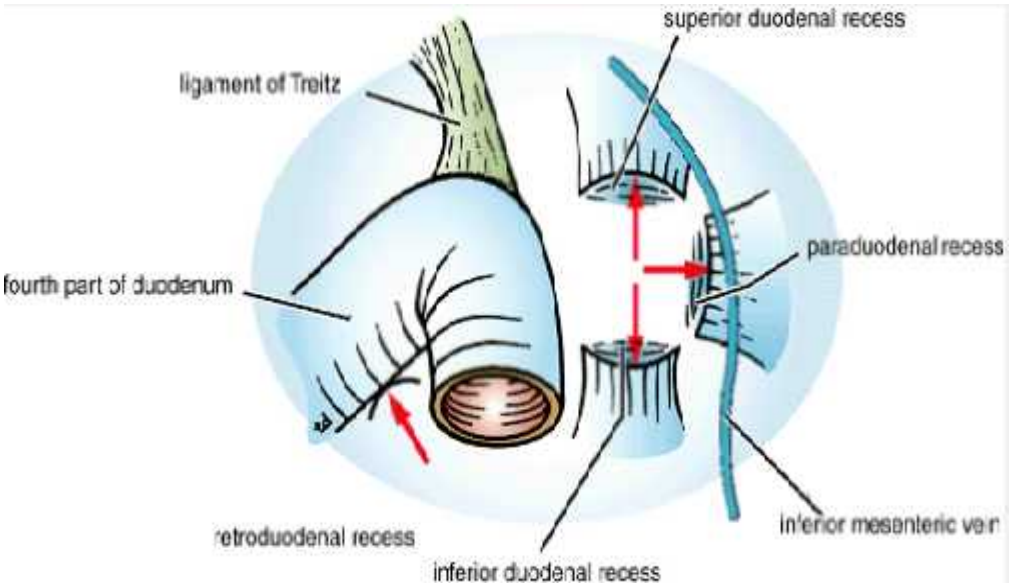


**Figure 5: Peritoneal recesses forming the Para duodenal recess.**

**Cecal Recesses**

Folds of peritoneum close to the cecum produce three peritoneal recesses called the superior ileocecal, the inferior ileocecal, and the retrocecalrecesses (fig: 6)

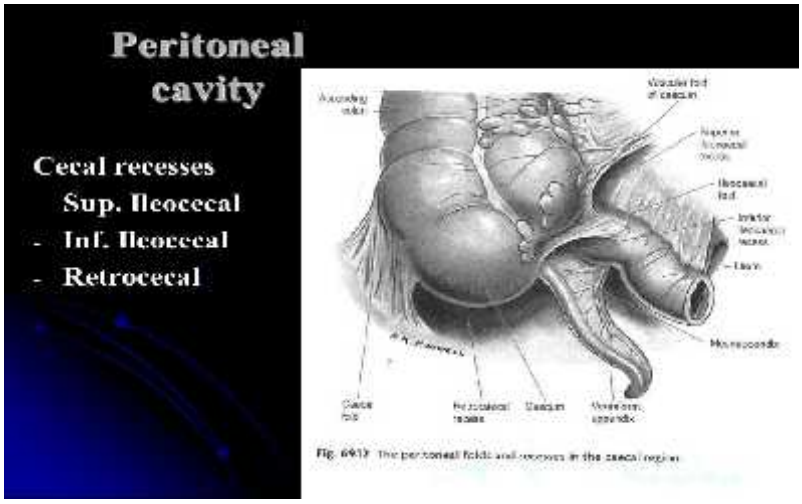


**Figure 6: Cecal recess**

**Intersigmoid Recess**

The intersigmoid recess is situated at the apex of the inverted, V-shaped root of the sigmoid mesocolon (Fig. 7); its mouth opens downward.

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The intersigmoid recess is situated at the apex of the inverted, V-shaped root of the sigmoid mesocolon its mouth opens downward.

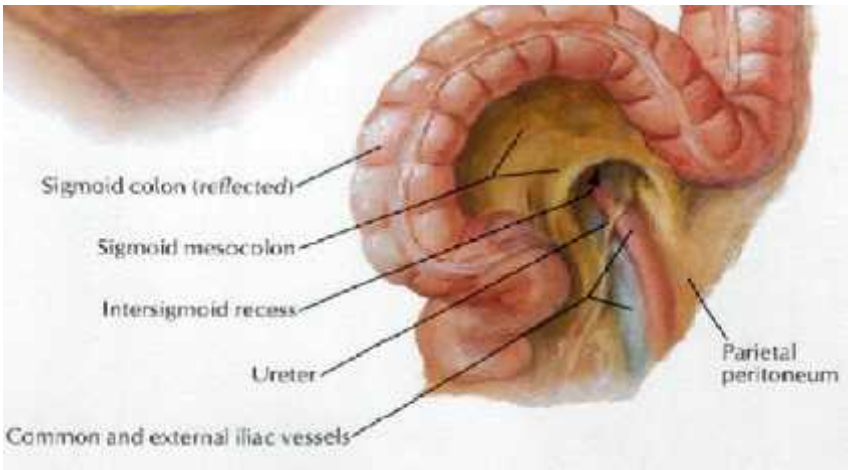



**Figure 7: Inter sigmoid recess**

**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
- <300 white blood cells per microliter (WBCs, generally mononuclear cells).[14]

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 may be rapid.[15]

The circulation of fluid and potential areas for abscess formation is shown in Fig 8 and 9. Some compartments collect fluid or pus more often than others. These compartments include the pelvis (the lowest portion), the sub phrenic spaces on the right and left sides, and Morrison's pouch, which is a poster superior extension of the sub hepatic spaces and is the lowest part of the paravertebral groove when a patient is recumbent. The falciform ligament separating the right and left sub phrenic spaces appears to act as a barrier to the spread of infection; consequently, it is unusual to find bilateral sub phrenic collections.[14]

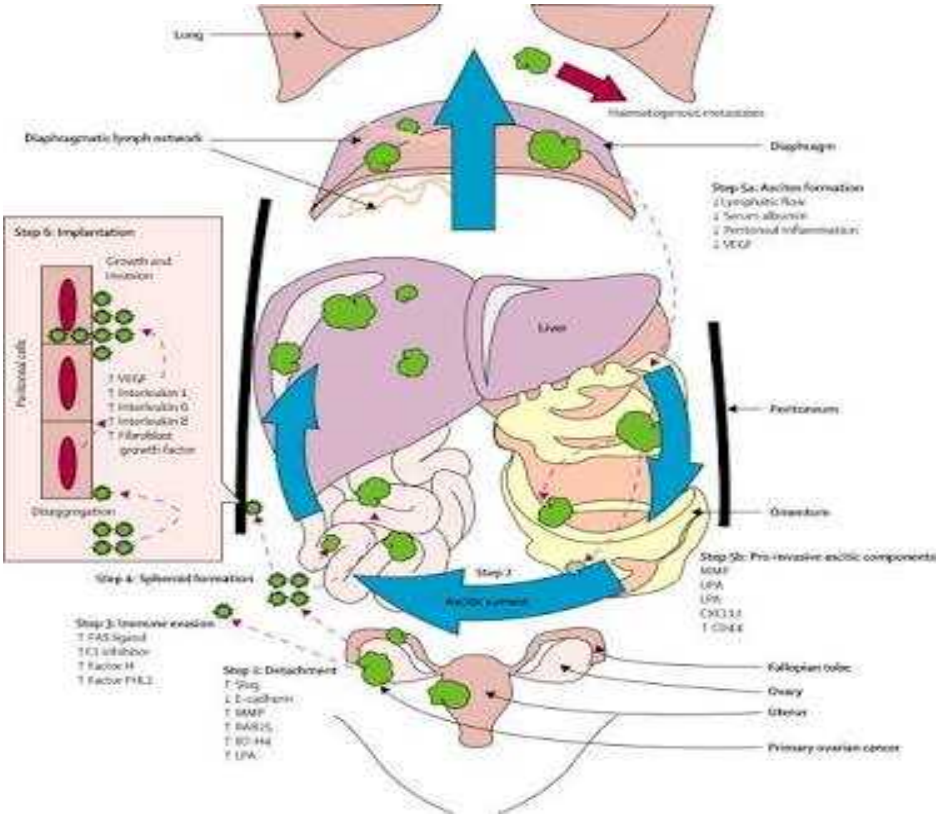



**Figure 8: peritoneal spread of disease**[14]

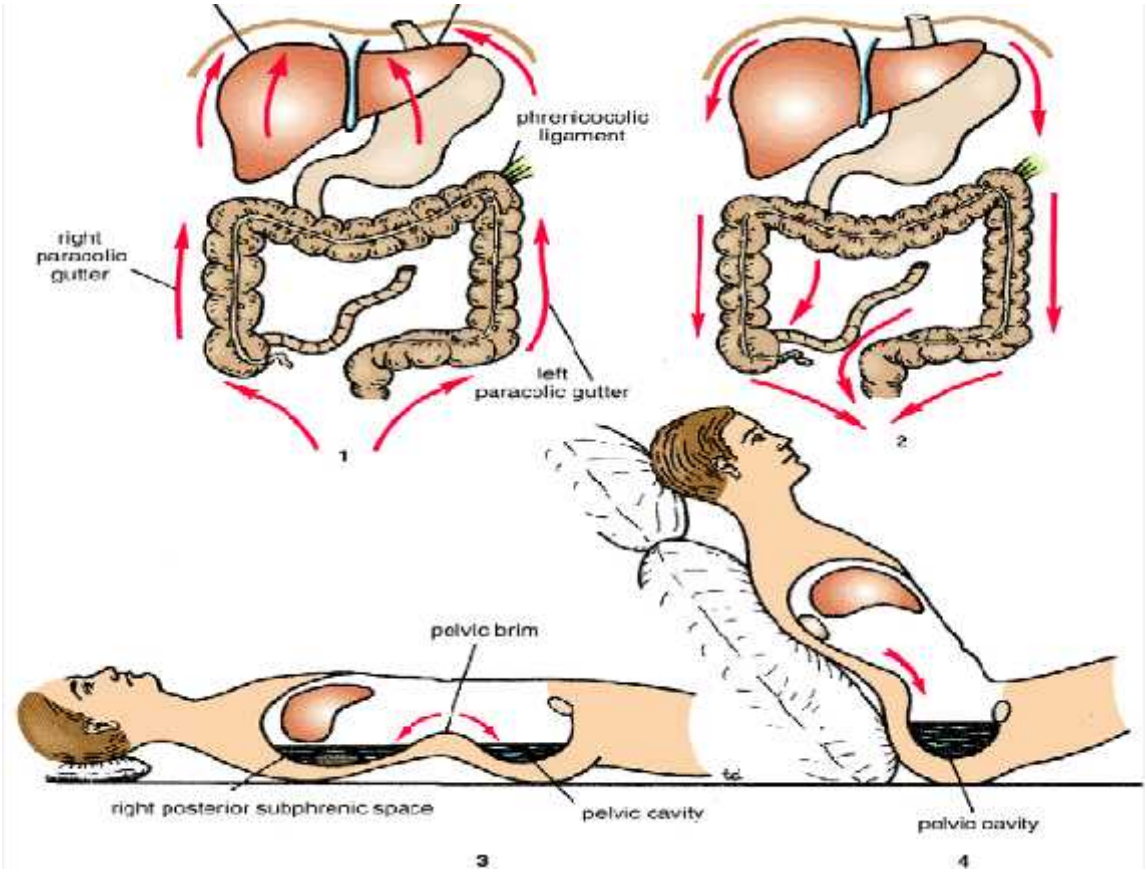



**Figure 9: Direction of flow of the peritoneal fluid.**

1. Normal flow upward to the sub phrenic 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.

**FUNCTIONS OF PERITONIUM**

The peritoneal membrane provides lubrication for the loops of intestine by secreting a highly viscous fluid. Peritoneal macrophages release pro inflammatory mediators that promote migration of the leukocytes into the peritoneal cavity from the surrounding microvasculature.

Degranulation of peritoneal mast cells releases histamine and other vasoactive products, causing local vasodilation and the extravasation of protein-rich fluid containing complement and immunoglobulin's into the peritoneal space. Protein within the peritoneal fluid opsonizes bacteria, which, along with activation of the complement cascade, promotes neutrophil and macrophage, mediated bacterial phagocytosis and destruction. Bacteria become sequestered within fibrin matrices, thereby promoting abscess formation and limiting the generalized spread of the infection.

## PATHOPHYSIOLOGY

Peritonitis and intra-abdominal infection are not synonymous. Peritonitis denotes inflammation of the peritoneum from any cause. It may be regarded as the localized equivalent of the systemic inflammatory response seen after any trigger of inflammation, which recently has been described as systemic inflammatory response syndrome[16].

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

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

**Peritonitis:**

Peritonitis is simply defined as inflammation of the peritoneum and may be localized or generalised.[17]

**Stages of peritonitis:**

**Stage 1: Stage of peritonism**

i.e. irritation of the peritoneum. It is due to leakage of gastric juice into the peritoneal cavity (chemical peritonitis). This stage usually lasts for about six hours. On examination there will be little change in the pulse, respiration and temperature. Tenderness and muscle guard are constantly present over the site of perforation. Great importance should be led on the diagnosis of this condition at this stage as chance 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. The intensity of the symptoms dwindles although the fire is still burning under the ashes. Symptoms are relieved but signs are there and 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 rectovesical or recto uterine pouch. Straight x ray in sitting position will show air under the diaphragm in 70% of the cases.

**Stage 3: Stage of diffuse peritonitis**

It indicates that the patient has gone a step further towards the grave. The pinched and anxious face, sunken eyes and hollow cheek- the so called facieshippocritica, 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:**[14]

In adults, primary bacterial peritonitis (PBP) occurs most commonly in conjunction with cirrhosis of the liver (frequently the 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 erythematous, and lymphedema as well as in patients with no underlying disease. The cause of PBP has not been established definitively but is believed to involve hematogenous spread of organisms in a patient in whom a diseased liver and altered portal circulation result in a defect in the usual filtration function. 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.[14]

**Secondary peritonitis:**

Secondary peritonitis develops when bacteria contaminate the peritoneum as a result of spillage from an intraabdominalviscus. 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.[14]

**Phases of Peritonitis**[18]

**Phase I:**

Rapid removal of contaminants from the peritoneal cavity into the systemic circulation occurs because contaminated peritoneal fluid moves cephalad in response to pressure gradients generated by the diaphragm. The lymph flows into the main lymphatic ducts via the substernalnodes. The resultant septicemia predominantly involves Gram-negative facultative anaerobes and is associated with high morbidity.

**Phase II:**

There are 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. Peritoneal mesothelial cells are also potent secretors of pro-inflammatory mediators, including interleukin-6, and IL-8, monocyte chemo attractant protein-1, macrophage inflammatory protein-1α and tumor necrosis factor-α.2 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:**

Fibrinousexudates traps microbes within its matrix and promote local phagocytic effectors mechanisms. It is an attempt by host defenses to localize infection It also serves to promote the development of abscesses. Regulation of the formation and degradation of fibrin 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**[19]

Typically primary peritonitis is a mono microbial, aerobic infection. The presence of obligate anaerobes or a mixed flora, suggest secondary peritonitis. The later represents a polymicrobial infection, after a spontaneous or traumatic breach in a microorganism containing viscous or because of a postoperative breakdown of intestinal anastomosis.

The number and type of bacteria increases progressively down the GI tract. Proximally it contains a sparse aerobes (coliforms) and oral anaerobic flora (<104), with the stomach and duodenum normally sterile. However, disease of stomach (eg carcinoma, gastric outlet obstruction) or acid reducing drugs may results in its colonization. Distally the colon contains largest concentration of bacteria – in 1 gram of stool up to 1012 obligate anaerobes and 108 facultative anaerobes (formally aerobes).Postoperative state, administration of systemic and luminal antibiotics and

the invasive environment of the intensive care unit may drastically modify patients ecology resulting in colonization of foregut with peculiar microorganisms(fungi, coagulase negative staphylococci and gram negative bacteria of low pathogenicity). These are the organisms that may be found in tertiary peritonitis in intensive care unit infection or in multiple organ failure.

**Microorganisms in peritonitis[19]**

- Escherichia coli
- Streptococci
- Bacteroides
- Clostridium
- Klebsiellapneumoniae

Other sources

- Chlamydia trachomatis
- Neisseria gonorrhoeae
- Haemolytic streptococci
- Staphylococcus
- Streptococcus pneumoniae
- Mycobacterium tuberculosis and other spp
- 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.[14]

**Secondary peritonitis**

Secondary peritonitis develops when bacteria contaminate the peritoneum as a result of spillage from an intraabdominalviscus. 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/g of feces but only $10^{8}$ aerobes/g; 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.[14]

Factors favouring localization or generalization of peritonitis[17] –

Localization

- Fibrinous Exudates
- Anatomical compartmentalization of peritoneum -Greateromentum (Adheres to inflamed peritoneum) Generalization
- Sudden visceral perforation
- Violent peritonitis
- Virulent infecting organisms
- Injudicious handling
- Immunocompromized state.

## ETIOLOGY

**Causes of peritoneal inflammation.**[20]

**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[20] | | |
|---|---|---|
| 1 | Primary peritonitis<br>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. Tuberculousand granulomatous peritonitis |
| 2 | Secondary peritonitis<br>Localized (abscess) or diffuse Peritonitis<br>Originating from a defect in abdominalviscus | A. Acute perforation peritonitis<br>1. Gastrointestinal perforation<br>2. Intestinal ischemia<br>3. Pelviperitonitis and other forms |
| | | B. Postoperative peritonitis<br>1. Anastomotic leak<br>2. Accidental perforation and Devascularization |
| | | C. Post-traumatic peritonitis<br>1. After blunt abdominal trauma<br>2. After penetrating abdominal Trauma |
| 3 | Tertiary peritonitis<br>Peritonitis like syndrome<br>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[21]**

| Acute peritonitis | Chronic (sclerosing) peritonitis |
|---|---|
| • Primary (spontaneous)<br>• Secondary<br>• Acute suppurative<br>• Granulomatous<br>• Chemical (aseptic)<br>• Interventional<br>• Traumatic<br>• Drug-induced | • Infectious<br>• Drug-induced<br>• Chemical<br>• Foreign-body<br>• Carcinomatosis |

**Secondary peritonitis due to hollow viscus perforation:**

**Perforated Gastric Ulcer**

The mortality rate of perforated gastric ulcer is 15–20%. Perforated gastric ulcers can pose a diagnostic challenge in a few clinical situations. Posterior gastric ulcer perforation is characterized by the insidious onset of upper abdominal pain and delayed presentation. A high index of suspicion regarding marginal and remnant gastric ulceration in these patients is important.[22]

**PHOTO 1: DUODENAL PERFORATION**

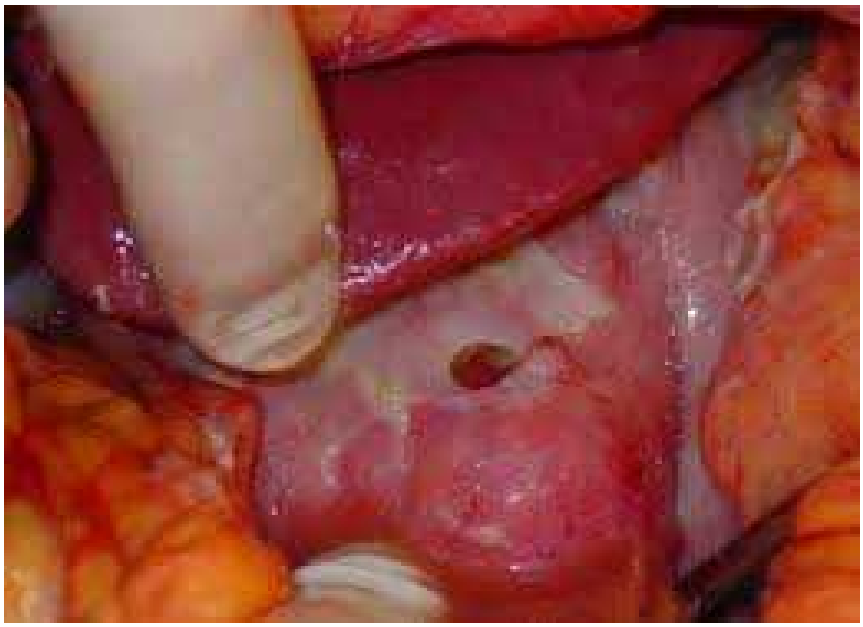
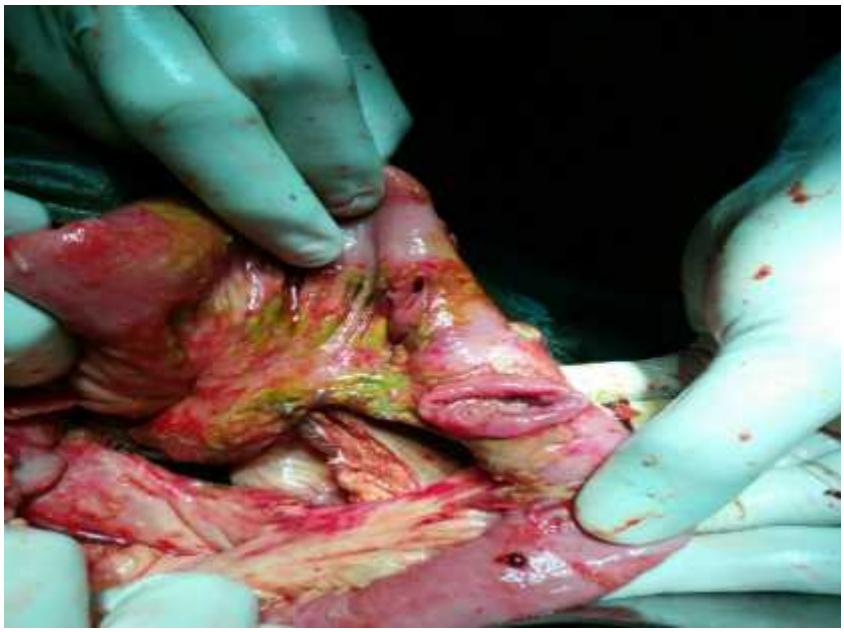


**PHOTO 2: MULTIPLE ILEAL PERFORATION**



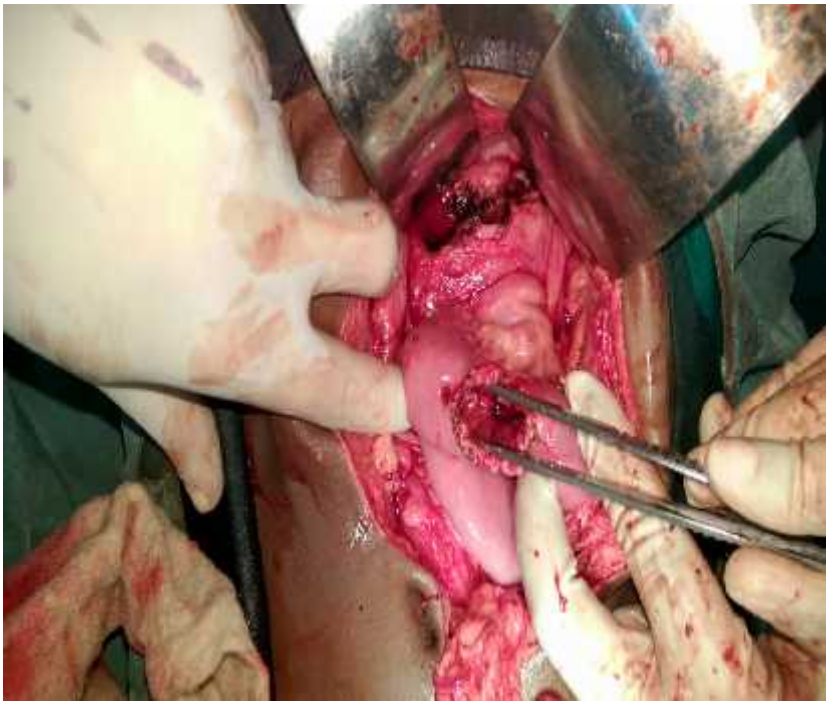


**PHOTO 3: GAINT ILEAL PERFORATION**

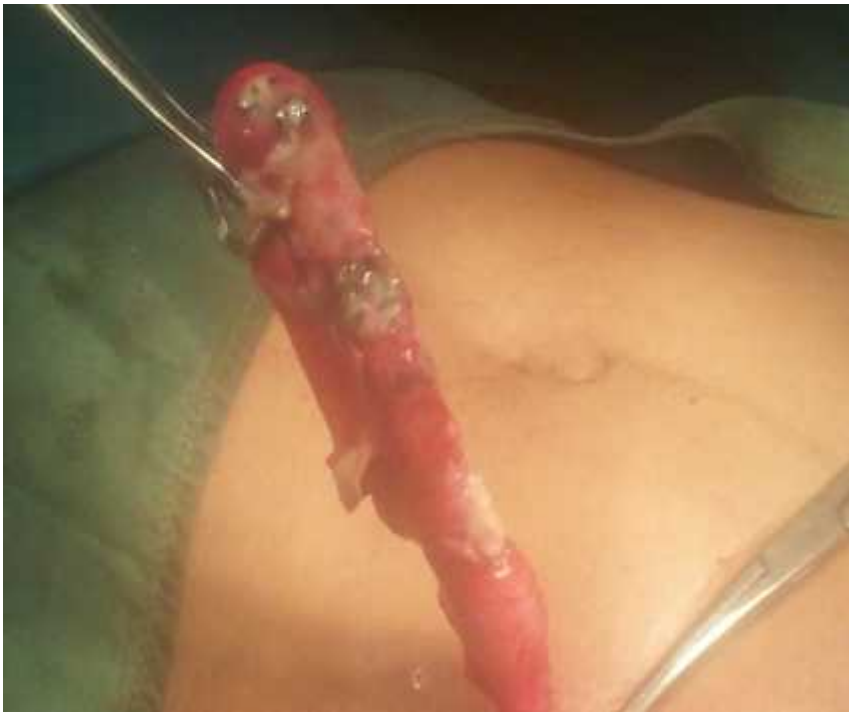


**PHOTO 4: APPENDICULAR PERFORATION**

**Small Bowel Perforation**

Perforated Duodenal Ulcer:

Duodenal perforation currently accounts for approximately 75% of peptic perforation. The mean prevalence of *H. pylori* infection in patients with perforated peptic ulcer is approximately 60% compared with 90–100% in uncomplicated ulcer disease. In addition to *H. pylori* and NSAID use, smoking and alcohol consumption are also associated with perforated peptic ulcer.[22]

Giant duodenal perforation should be restricted to such large defects, where omentopexy may be deemed unsafe, and other options may be thought to be necessary.[22]

Other etiologies of small bowel perforation include

1. Infections (especially tuberculosis, typhoid, and CMV),
2. Crohn's disease,
3. Ischemia,
4. Drugs (e.g., potassium- and NSAID-induced ulcers),
5. Radiation-induced injury,
6. Meckel's and acquired diverticula,
7. Neoplasms (especially lymphoma, adenocarcinoma, and melanoma),
8. Foreign bodies.

Among iatrogenic injuries, duodenal perforation during ERCP with endoscopic sphincterotomy is the most common. Patients who have undergone Billroth II gastrectomy are at increased risk of duodenal perforations as well as free jejunal perforations during ERCP. Manifestations of such a contained duodenal perforation following ERCP can resemble those of ERCP-induced pancreatitis, including hyperamylasemia[23].

**Typhoid ulcers perforation:**

**Histology: in the edges of ulcers,**

I. Typhoid: Marked proliferation of reticuloendothelial cells of the lymphoid follicles was seen. The reticulum cells, reticuloendothelial cells and imigrant macrophages in the resultant aggregation contained large cells having an abundant pink cytoplasm often containing bacteria, cellular debris and red cells. Erythrophagocytosis was prominent and virtually marked the lesion as being due to enteric fever.

II. Non-specific ulcers: Large number of polymorphonuclear cells with cellular debris was seen. There was no evidence of large macrophages.[24]

Evert and Black andGoehrs et al from Mayo Clinic gave a theory of sub mucosal vascular embolization as the cause of nonspecific perforations. Many workers proved, clinically and experimentally, that enteric-coated tablets of potassium chloride lead to nonspecific ulcers in the distal ileum, which perforated easily[24].

Perforation of typhoid ulcer usually occurs during the third week and is sometimes the first sign of the disease. The ulcer is parallel to the long axis of the gut and is ususlly situated in the distal ileum.[25]

**Tubercular perforation:[25]**

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:**

1. Diverticular disease
2. Ischemia: The most common cause of colonic ischemia is 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, digestive, or gynecologic surgery was 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.[26]
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 overdistended, particularly in the 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 a stage IV tumor[22].

**Special forms of peritonitis**

**A. Biliary peritonitis:**

**Causes of bile peritonitis[17]:**

- Perforated cholecystitis
- Postcholesystectomy
- Cystic duct stump leak
- Leakage from an accessory duct in the gallbladder bed
- T-tube drain dislodgement

- Following other operations/procedure

  Leaking duodenal stump postgastrectomy

  Leaking biliary enteric anastomosis

  Leakage around percutaneous placed biliary drains

- Following liver trauma

**B. Chemical (aseptic) peritonitis[27]**

Aseptic peritonitis refers to the peritoneal inflammation from substances other than bacteria, but bacterial contamination and overgrowth soon follow. A perforated peptic ulcer provides the most severe and common form of chemical peritonitis with gastric juice and bile contaminating the peritoneal cavity. Blood in the peritoneum is also a cause of peritoneal irritation after slow bleeding (e.g. a ruptured graafianfollicle or following splenic injury) rather than from a catastrophic hemorrhagic event such as ruptured aneurysm where the primary pathology itself overshadows the peritoneal irritation. Meconium and urine may also precipitate chemical peritonitis.

**Sterile peritonitis: there are five forms.[28]**

1. Blood e.g. ruptured ovarian cysts, leaking aortic aneurysm.[28]
2. Urine e.g. intraperitoneal rupture of the bladder.[28]
3. Meconium is a sterile mixture of epithelial cells, mucin, salts, fats and bile, which is formed when the fetus begins to swallow amniotic fluid. Meconium peritonitis develops late in intrauterine life or in the perinatal period when meconium enters the peritoneal cavity through an intestinal perforation.[28]
4. Bile.
5. Pancreatic juice e.g. due to acute pancreatitis, trauma. Pancreatitis may be the cause of a diagnostic (but unnecessary) laparotomy in patients who do not exhibit a raised concentration of amylase in serum.[28]

6. **Interventional peritonitis:**[29]

Acute peritonitis may be precipitated through colonoscopic perforation of a diverticulum or inadvertent perforation of the oesophagogastric junction during oesophageal dilatation.

Peritonitis may follow abdominal surgery where bowel and gastric contents, blood, and urine escape into the abdominal cavity following anastomotic dehiscence. In patients with renal failure treated by continuous ambulatory peritoneal dialysis, a permanent indwelling catheter in the abdominal cavity provides a portal of entry for exogenous bacteria despite the use of stringent aseptic techniques during dialysis exchanges.

**7. Traumatic peritonitis**[29]

Penetrating wounds of the abdomen without visceral injury may provide a route for exogenous bacterial contamination. Penetration of a visceral organ may precipitate the spillage of visceral contents into the peritoneal cavity. Several blunt trauma may disrupt intra-abdominal organs directly or indirectly through disruption of their vascular supply.

**8. Drug-induced peritonitis**[29]

Warfarin anticoagulation can cause peritoneal irritation and peritonitis through leakage from a spontaneous retroperitoneal haematoma. The symptoms of acute peritonitis have also been described during treatment with the antituberculous agent, isoniazid.

**c) Foreign-body granulomatous peritonitis**

Foreign-body granulomatous peritonitis was not uncommon in patients who had undergone laparotomy in the days before the introduction of 'talc-free' surgical gloves. At laparotomy, multiple nodules are seen over the parietal and visceral

peritoneum, mimicking malignant deposits with dense adhesions between loops of bowel. Treatment consists of lysis of adhesions and the diagnosis is confirmed by histology where birefringent granules are observed. Administration of steroids is ineffective in preventing intestinal adhesions.

Other forms of chronic peritonitis which may form dense adhesions include the adhesive form of tuberculous peritonitis and carcinomatosis.[29]

**d) Chlamydial peritonitis:**

Fitz-Hugh–Curtis syndrome can occur following pelvic inflammatory disease and is characterized by right hypochondrial pain, pyrexia and a hepatic rub.

**e) Familial Mediterranean fever [periodic peritonitis]:**

Arab, Armenian and Jewish population races are primarily affected. Mutations in he MEFV [Mediterranean fever] gene appear to cause the disease. This gene produces a protein called pyrin, which is expressed mostly in neutrophils but whose exact function is not known.

They present with mild pyrexia, polymorphoneuclear leukocytosis and occasionally pain in the thorax and joints. This duration of attack is 24-72hrs, when it is followed by complete remission, but exacerbations recur at regular intervals. At operation, which may be necessary to exclude other causes[should be avoided if possible], peritoneum is inflamed, particularly in the vicinity of the spleen and the gallbladder. There is no evidence that the interior of these organs is abnormal. Colchicine's therapy is used during attacks and to prevent recurrent attacks.[17]

**f) Peritonitis Associated With Chronic Ambulatory Peritoneal Dialysis** .[13]

Peritoneal dialysis associated peritonitis patients present with abdominal pain, fever, and cloudy peritoneal dialysate containing more than 100 leukocytes/$mm^3$, with more than 50% of the cells being neutrophils. Gram stain detects organisms in only

about 10% to 40% of cases. About 75% of infections are due to gram-positive organisms, with *Staphylococcus epidermidis* accounting for 30% to 50% of cases. *S. aureus*, gram-negative bacilli, and fungi are also important causes of dialysis-associated peritonitis.

Peritoneal dialysis–associated peritonitis is treated by the intraperitoneal administration of antibiotics, most commonly a first-generation cephalosporin. Recurrent or persistent peritonitis requires removal of the dialysis catheter and resumption of hemodialysis.

**g) Chronic (sclerosing) peritonitis[27]**

Classically, sclerosing peritonitis is characterized by dense adhesions, especially between loops of small bowel, and in the most extreme cases the entire small bowel and even the large intestine and liver are cocooned in a dense adhesive membrane of fibrous tissue. Treatment consisted of surgical stripping of the cocoon of fibrous tissue from the underlying intestine. The classic description of sclerosing peritonitis relates to the b-adrenoceptor blocker, practolol. Sclerosing peritonitis made reappearance in patients undergoing continuous ambulatory peritoneal dialysis. The sclerosis was due to the chlorhexidine and alcohol solution used to sterilize the connectors of the CAPD catheters at the time of peritoneal dialysis 'exchanges'. Other mechanisms include the presence of acetate in the dialysate and recurrent CAPD peritonitis with fibrin deposition over the peritoneal membrane.

**h) Tuberculous peritonitis[25]**

I. **Acute tuberculousperitonitis:**Tuberculous peritonitis sometimes resembles closely acute peritonitis. Tubercules are seen scattered over the peritoneum and greater omentum. Early tubercles are greyish and translucent. They soon undergo caseation, and appear white or yellow and are then less difficult to

distinguish from carcinoma. A portion of the diseased omentum is removed for histological confirmation of the diagnosis and the wound closed without drainage.

II. **Chronic tuberculousperitonitis:**The condition presents with abdominal pain (90 per cent of cases), fever (60 per cent), loss of weight (60 per cent), ascites (60 per cent), night sweats (37 per cent) and abdominal mass (26 per cent).

**Infection originates from:**

- Tuberculous mesenteric lymph nodes
- A tuberculouspyosalpinx
- Tuberculosis of the ileocaecal region
- Blood-borne infection from pulmonary tuberculosis, usually the 'miliary' but occasionally the 'cavitating' form.

**Varieties of tuberculous peritonitis:**

There are four varieties of tuberculous peritonitis: ascitic, encysted, fibrous and purulent.

- **Ascitic form:**

Studded with tubercules in the peritoneal cavity it becomes filled with pale, straw-coloured fluid. On abdominal palpation a transverse solid mass can often be detected. This is rolled-up greater omentum infiltrated with tubercules.

- **Encysted form:**

A localised intra-abdominal swelling is produced which gives rise to difficulty in diagnosis. For these reasons laparotomy is often performed, and if an encapsulated collection of fluid is found, it is evacuated and the abdomen is closed. Late intestinal obstruction is a possible complication.

- **Fibrous form**

Fibrous (syn. plastic) form is characterized by the production of widespread adhesions, which cause coils of intestine, especially the ileum, to become matted together and distended.

On examination, the adherent intestine with omentum attached, together with the thickened mesentery, may give rise to a palpable swelling or swellings.

- **Purulent form**

The purulent form is rare. When it occurs, usually it is secondary to tuberculoussalpingitis. Amidst a mass of adherent intestine and omentum, tuberculous pus is present. Sizeable cold abscesses often form, and point on the surface, commonly near the umbilicus, or burst into the bowel.

**CLINICAL MANIFESTATIONS[30]**

The clinical manifestations of peritonitis are fluid shifts and metabolic disturbance. The heart rate and respiratory rate initially increase as a result of volumetric, intestinal, diaphragmatic, and pain reflexes. Metabolic acidosis and the increased secretion of aldosterone, antidiuretic hormone and catecholamine's subsequently alter cardiac output and respiration.

Protein is broken down and hepatic glycogen is mobilized as the body enters a highly catabolic state. Paralytic ileus develops, leading to pro-found sequestration of fluid and loss of electrolytes and protein-rich exudate. Gross abdominal distension causes diaphragmatic elevation, with resultant atelectasis and pneumonia. Multiple-organ failure, coma and death will follow if peritonitis persists and fails to localize.

**DIAGNOSIS / PRESENTATION:**

Pain is the most common symptom and may be localized or diffuse; it is usually constant and of a sharp, pricking character. A visceral perforation causes a

sudden, severe pain that is usually first appreciated in the area of the perforation, but may become more generalized as peritoneal contamination spreads. The pain will be referred to the ipsilateral shoulder tip if the diaphragmatic peritoneum is involved.

Anorexia, malaise, nausea and vomiting are common associated features. Constipation is usually present, unless a pelvic abscess develops (which can cause diarrhea)

**EXAMINATION:**

General: a patient with peritonitis is pale, drawn andanxious; the eyes are sunken because of dehydration. Regular observations will show signs of systemic inflammatory response syndrome or, at worst, septic shock, hypovolaemic shock or multiple-organ failure.

Abdomen: The patient will lie supine and relatively motionless with shallow respiratory excursions. The knees are flexed and drawn up in order to reduce tension in the abdominal wall. In diffuse peritonitis, spasm of the abdominal musculature will result in board like rigidity and failure of the abdomen to move with respiration.

Abdominal palpation exacerbates the pain and therefore should be undertaken carefully and gently. It will show tenderness, guarding and rebound tenderness; the site of maximum tenderness is usually related to the site of pathology. Guarding will initially be voluntary, before becoming an involuntary reflex as inflammation progresses.

Specific pathognomonic signs of disease may be clinically evident (e.g. Rovsing's sign in acute appendicitis).

**Digital rectal examination:** will elicit anterior tenderness in pelvic peritonitis.

**Auscultation:** will confirm increasing ileus as bowel sounds diminish and eventually cease.

**INVESTIGATION**

**Hematological tests**:

- ✓ Full blood count will demonstrate a leukocytosis.
- ✓ Urea, serum creatinine andserum electrolytes will confirm dehydration and acute renal failure;
- ✓ Blood grouping and typing – laparotomy may be indicated and therefore cross matched blood will be required.[30]
- ✓ Liver function tests and serum amylase – a high concentration of amylase in serum is diagnostic of acute pancreatitis, but a moderately elevated concentration can be caused by other intra-abdominal catastrophes (e.g. perforated duodenal ulcer).

**Imaging:**

**I.** Radiography [32]

**Erect chest radiograph or erect abdomen radiograph:** Free gas should be identified on the erect chest radiograph. As little as 1 ml of free gas can be demonstrated radio graphically, 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 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:**

The signs of pneumoperitoneum on supine radiographs are important to be identified. 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, frequently portable, 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 sub hepatic 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.

**Signs of a pneumoperitoneum on supine radiograph:**

1. Right upper-quadrant gas
    - Sub hepatic
    - Per hepatic
    - Morrison's pouch
    - Fissure for ligamentumteres
2. Ligament visualization
    - Falciform [ligamentumteres]
    - Umbilical [inverted V sign] medial and lateral
3. Rigler's [double wall] sign
4. Urachus
5. Triangular air
6. Foot ball or air dome sign
7. Scrotal air [in children]

**Conditions simulating a pneumoperitoneum [pseudo-pneumoperitoneum]**

- Curvilinear atelectasis in the lung
- Subdiaphragmatic fat
- Cysts in pneumatosisintestinalis
- Diaphragmatic irregularity
- Intestine between liver and diaphragm- chiladiti's syndrome
- Sub phrenic abscess

**Causes of pneumoperitoneum without peritonitis**

I. Silent perforation of viscus that has sealed itself, in:
   - Patients on steroids
   - Patients being ventilated
   - Unconscious patients
   - Serious medical condition
   - The aged

II. Perforated jejunal diverticulosis

III. Perforated cyst in pneumatosisintestinalis

IV. Tracking down from a pneumomediastinum

V. Post operative

VI. Peritoneal dialysis

VII. Stercoral ulceration

VIII. Vaginal tubal entry of air

Arterial blood gas reflects a metabolic acidosis, often preceded by a low arterial carbon dioxide tension caused by hyperventilation

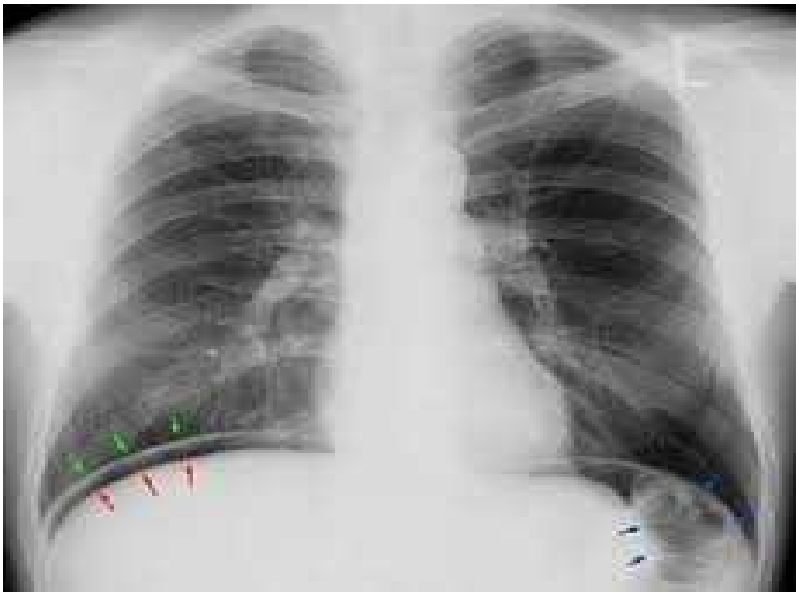


**Photo 5:**

Radiography: **erect chest radiograph or erect abdomen radiograph**

- **Ultra sound scanning:**

US play roles in confirming or excluding specific diagnoses (e.g. sub phrenic abscess). The diagnostic accuracy of these modalities has also been affirmed in clinically equivocal cases of acute appendicitis.[21]

- **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.[33]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 and including soft tissue mass), [33]

Upright chest, films can detect pneumoperitoneum in only 30% of cases but abdominal CT can demonstrate free air in 100% of cases .[33]

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

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

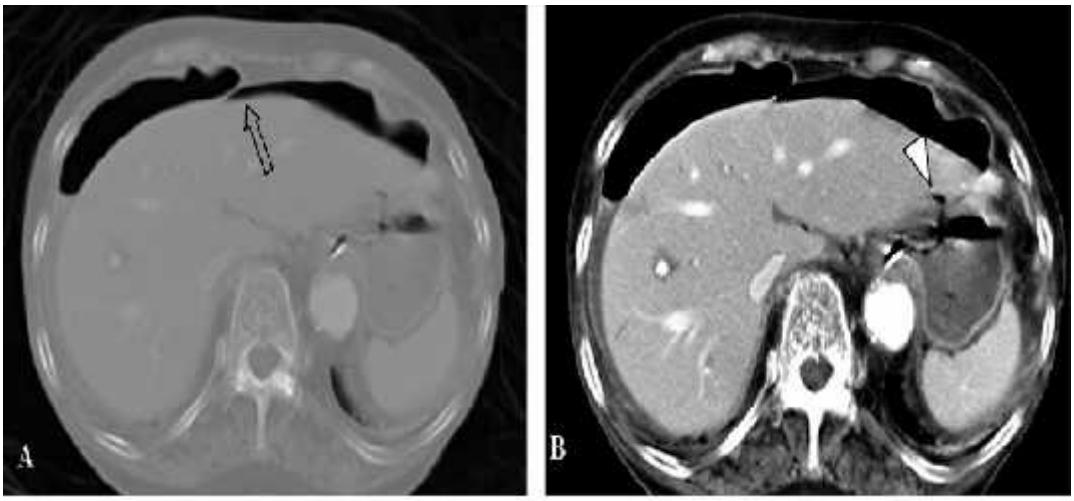
The “ligamentumteres sign” which is free air confined in the intra-hepatic fissure for ligamentumterescan be seen in the perforation of the duodenal bulb or stomach.[34]

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.[34]

When there is free air in the periportal area, it suggests a high probability of perforation in the upper GI tract.[34]

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.[34]

**Photo 6: computed tomography**




**Treatment**

**Treatment consists of:**

1. **General care of the patient**[35]
2. **Specific treatment for the cause**

**Correction of circulating volume and electrolyte imbalance**.

The plasma volume must be restored and the 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 feeding ('hyper alimentation' or 'total parenteral nutrition') is required.

1. **A fluid balance chart** Must be started 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 seen to have been administered. Throughout recovery, the hematocrit and serum electrolytes and urea must be checked regularly.

2. **Gastrointestinal decompression[35].** 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. It is important not to prolong the ileus by missing this stage.
3. **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 adult 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.
4. **Antibiotic therapy.** Administration of antibiotics prevents the multiplication of bacteria and the release of endotoxins. As the infection is usually a mixed one, initially parenteral broad-spectrum antibiotics active against aerobic and anaerobic bacteria should be given.
5. **Analgesia.** The patient should be nursed in the sitting-up position and must be relieved of pain before and after operation. Once the diagnosis has been made morphine may be given, and continued as necessary. If appropriate expertise is available epidural infusion may provide excellent analgesia. Freedom from pain allows early mobilization and adequate physiotherapy in the postoperative period which help to prevent basal pulmonary collapse, deep-vein thrombosis and pulmonary embolism.

2. **Specific treatment of the cause**

1. **Perforated peptic ulcer**: Duodenal perforation currently accounts for approximately 75% of peptic perforation.[36]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 definitive surgical care.[37]

- **Duodenal perforation:** Simple closure is usually the quickest and most appropriate method of dealing with a perforated duodenal ulcer.

**Modified Graham patch repair:[38]**

Generous bites, which pass through the entire thickness of the gut wall, should be taken with three or four interrupted, absorbable sutures. 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 an omental or jejunalserosal 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 then 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.[36]

- **Gastric perforation:**

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.[36]

**Laparoscopic and Endoscopic Management of Perforated Duodenal Ulcers:**

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 safe 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.[38]

**Laparoscopic and endoscopic procedure:**

The supraumbilical port (10 mm) was the camera port. The second port was 5 mm and was just to the right of midline. This port was used for needle and suture. The third port (5 mm) was used for the clamp, dissector, and instrument for retrieving the needle and for the suction irrigator Note that this port is in the midclavicular line. The fourth trocar (5 mm) was 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 and all quadrants were inspected for purulence. Drains were not routinely used. The omentoplasty was applied. The omental plug pulled through the ulcer by the endoscope.[37]

**Experimental Endoscopic repair of gastric perforation with omental patch and clips:**

A perforation was made in the anterior gastric wall between the angulus and the greater curvature in 2 steps: mucosal resection and complete perforation (muscularispropria and serosa).

In the first step (mucosal resection), a transparent plastic cap used for variceal band ligation was fitted to the distal end of the endoscope. Mucosa was aspirated into the cap and the snare was released and closed. Suction was then discontinued but the grasped mucosa remained within the closed snare and was subsequently resected by using electrosurgical cutting current with the generator output set at 40 W.

The second step (complete perforation of the muscularispropria and serosa) was performed in the same manner as that described above for mucosal resection except that a cap with a smaller inner diameter (8 mm) was used.

The omentum was pulled into the gastric cavity. Once the omentum was visible through the perforation, the easiest method of pulling it into the stomach was simple aspiration. However, sometimes it was necessary to catch the omentum within the abdominal cavity with the polypectomy snare or a biopsy forceps Continuous traction on the polypectomy snare allowed greater control and mobilization of the omentum and permitted performance of the clipping procedure with the endoscope in any position. Endoscopic clips were placed under endoscopic guidance so as to attach the omental patch to the muscularispropria of the stomach wall. Four to 7 clips (mean, 6) were needed to complete the procedure.

Use of the ligamentumtereshepatis 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.[38]

1. **Jejunal perforation:**

Primary closure Resection and end-to-endanastomosis.

2. **Ileal perforation:**

Primary closure

Wedge resection and closure

Resection of segment of ileum and anastomosis

Right hemicolectomy Incase of involvement of ileocecal junction.

3. **Colonic perforation:**

Treatment option depends on the etiology.

- Simple suture of the perforation should only be performed after 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.

**Prognostic scoring systems**

Prognostic scores are based on numerical weighting of clinical variables. The score calculated for an individual patient may, for example, be used to estimate the probability of death. There are several applications for prognostic scores in peritonitis. In clinical trials they are used to define risk, to compare treatment, to define inclusion and exclusion criteria, and to measure outcome in trials that do not involve comparisons.

- **The Sepsis Severity Score by Stevens (1983)**

This score includes variables from major body systems; respiratory, cardiovascular, renal, hepatic, renal, hematological, gastrointestinal and nervous. Applied to 30 patients, the scores reflected prognosis. Like the system by Elebute and Stoner, it was criticized for lack of clear definitions, objectivity and validation in a large patient population.[39]

- **Mannheim peritonitis index (1983)**

Mannheim peritonitis index, which was developed by Wacha and Linder in 1983. It was developed based on the restrospective analysis of data from 1,253 patients with peritonitis, in which 20 possible risk factors were considered. Of these only 8 proved to be of prognostic relevance and were entered into MPI, classified according to their predictive power.[40]

- **APACHE**

**APACHE I (1978):[41]**

In 1978, the developers of APACHE recognized the need to collect quality information on patients in the ICU and to use that information to improve outcomes. This led to the development of the Acute Physiology, Age and Chronic Health Evaluation system, known by its acronym, APACHE.

APACHE classification is composed of two parts a physiological score representing the degree of acute illness including 34 parameters and preadmission health evaluation **APACHEII (1985):[44]**

In 1983 Knaus while leading a team of critical care experts developed a scoring system based on 32 variables, named acute physiologic and chronic health evaluation, APACHE, for patient stratification in the intensive care unit. Meakins and associates applied it to patients with intraabdominal infection and found strong

correlation with mortality. The original APACHE score did not enjoy widespread popularity because it was too dependent on intensive care facilities. In1985, APACHE II, a less ICU- dependent version, with 12 variables, age and chronic health status, was developed, without loss of effectiveness. In 1987, the Surgical Infection Society [SIS] adopted APACHE II the standard for stratification of intraabdominal infection ahead of scores designed specifically for sepsis, because, at that time, it had been prospectively validated in large patient populations. Modifications such as the mode of score implementation, standard definition criteria and outcome measures for intraabdominal infection were approved by SIS.[39]

indicating health status before acute illness.

**APACHE III (1991) [43]**

- APACHE-II was further refined to APACHE-III in 1991, with five new variables-
- 1) Blood urea nitrogen
- 2) Urine output
- 3) Serum bilirubin
- 4) Serum albumin
- 5) Glucose

APACHE III is a severity-adjusted methodology that predicts outcomes for critically ill adult patients. In order to predict these outcomes, the APACHE methodology looks at 27 variables for each patient. These variables include the patient's diagnosis, age, vital signs, and laboratory values. This data, in conjunction with a few other pieces of information about the patient's history, is combined to predict the mortality of hospitalized patients. The APACHE III score provides ***relative***

risk stratification for acutely ill hospitalized adults when used within a *single* medical or surgical diagnostic category[42]

**APACHE IV[44]**

There were several changes made in this new version of APACHE IV. The first excluded patients transferred from another ICU from receiving predictions.

The second change involved measuring previous length of stay as a continuous rather than an integer variable.

Third change included a variable for designating whether a patient's Glasgow Coma Score could not be assessed due to sedation. The most important change involved the new categorization of disease groups. Based on the frequency of selected diagnoses and their mortality rate, the existing 94 groups were expanded to 116.

The APACHE III score is a component in the APACHE IV predictive equations that include the score, the patient's length of stay in the hospital prior to ICU admission, the patient's exact ICU admission disease classification (there are 116 specific diagnostic category classifications), the patient's chronic health conditions, the patient's origin immediately prior to ICU admission and a measure of practice patterns to provide probability estimates for various outcomes on a daily basis. The APACHE IV predictive equations reference the current 131,988 ICU patient database and the risk predictions are calculated for patients selected by similar criteria.

- **Possum score**[46]

**Table 5: Possum score**

| Score | | | | |
|---|---|---|---|---|
| | 1 | 2 | 4 | 8 |
| Age | <60 | 61-70 | 71-80 | >80 |
| Cardiac signs | No failure | Diuretic, digoxin, antianginal Or hypertensive therapy | Peripheral edema; Warfarin therapy Borderline cardiomegaly | Raised jugular venous Pressure Cardio**m**egaly |
| Respiratory history | No dyspnea | Dyspnea on exertion | Limitingdyspnoea (One flight) | Dyspnea at rest (Rate> 30/min) |
| Chest radiograph | | Mild COAD | Moderate COAD | Fibrosis or consolidation |
| Blood pressure (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 |
| Hemoglobin | 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 ectopic/min Q waves or ST/T wave changes |

- **THE MULTIPLE ORGAN FAILURE SCORE (1988)** [45]

**Table 6: The multiple organ failure score**

| 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 Bilirubin>2mg/dL | AST >50 units /L Bilirubin>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 Acalculouscholecystitis | Bleeding ulcer, necrotizing enter colitis 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

- **PEPTIC ULCER PERFORATION SCORE (PULP SCORE) [47]:**

**Table 7: 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<br>ASA 2<br>ASA 3<br>ASA 4<br>ASA 5 | <br>1<br>3<br>5<br>7 |

Total PULP score: 0–18

- **Jabalpur prognostic scoring system for peptic perforation[47] (2003)**

**Table 8: Jabalpur prognostic scoring system for peptic perforation[47]**

| 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 | 40-54 or 40-179 | <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 survey of patients with acute generalized peritonitis due to hollow viscus perforation was carried out in general surgical wards of our institute during the period starting from October 2014 to June 2016.

Study population consisted of 100 consecutive patients with perforativeperitonitis which were confirmed on emergency laparotomy.

**INCLUSION CRITERIA:**

1. Peritonitis secondary to hollow viscus perforation.
2. Age group more than 15yrs.
3. Non-traumatic perforative peritonitis.

**EXCLUSION CRITERIA:**

1. Perforation secondary to abdominal trauma.
2. Primary peritonitis.
3. Post op peritonitis due to anastomotic leak, etc.
4. Age group less than 15yrs.
5. Perforative peritonitis patients managed conservatively.

**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.
- Radio logically: gas 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 viscusperforation.

- Etiological factors were studied.

APACHE 111 scoring system was assigned to all the patients in order to calculate their individual risk of mortality and survival at the time of admission.

**APACHE III (1991)** [48]

- APACHE -III was introduced to address some of the flaws of APACHE-II. Although APACHE-III resembles APACHE-II, it includes new variables such as prior treatment location and the disease requiring ICU admission.
- In APACHE-III scoring. Patients age and chronic health history are worth up to 47 points
- Within 24hours of ICU admission, 17 physiologic variables are measured and may add up to a maximum of additional 252 points
- The resulting total score, in combination with prior treatment location and principal diagnosis provides a predicted mortality.

**Benefits of APACHE-III scoring system**

- Saves lives by better management of critically ill individuals
- Reduces incidence of complications
- Evaluates and improves ICU performance
- Optimizes ICU resource allocation.

## APACHE III SCORING SYSTEM

| **Parameter** | **Value range** | Points | **Parameter** | **Value range** | **Points** |
|---|---|---|---|---|---|
| Core temperature (°C) | 0-32.9 | 20 | Plasma bilirubin (μmol/L) | 0-34 | 0 |
| | 33.0-33.4 | 16 | | 35-51 | 5 |
| | 33.5-33.9 | 13 | | 52-85 | 6 |
| | 34.0-34.9 | 8 | | 86-135 | 8 |
| | 35.0-35.9 | 2 | | 136 plus | 16 |
| | 36.0-36.9 | 0 | | | |
| | 40 or more | 4 | | | |
| Heart (r/min) | 0-39 | 8 | Urine volume (mL/24 h) | 0-399 | 15 |
| | 40-49 | 5 | | 400-599 | 3 |
| | 50-99 | 0 | | 600-899 | 7 |
| | 100-109 | 1 | | 900-1499 | 5 |
| | 110-119 | 5 | | 1500-1999 | 4 |
| | 120-139 | 7 | | 2000-3999 | 0 |
| | 140-154 | 13 | | 4000 plus | 1 |
| | 155 or more | 17 | | | |
| Mean blood pressure (mmHg) | 0-39 | 23 | Plasma Creatinine (μmol/L) (if no acute | 0-43 | 3 |

| | | | | | |
|---|---|---|---|---|---|
| | 40-59 | 15 | renal failure) or in ARF (< 410 mL urine vol/24 h) | 44-132 | 0 |
| | 60-69 | 7 | | 133-171 | 2 |
| | 70-79 | 6 | | 172 or more | 7 |
| | 80-99 | 0 | | | |
| | 100-119 | 4 | | | |
| | 120-129 | 7 | | 0-132 | 0 |
| | 130-139 | 9 | | 133 or more | 10 |
| | 140 or more | 10 | | | |
| Respiratory (r/min) (zero points for 6-12/min rate if on ventilation) | 0-5 | 17 | Arterial $PO_2$ (kPa) (Inspired $O_2$ < 50%) or alveolar/arterial $PO_2$ difference kPa (Pa-$PaO_2$) (Inspired $O_2$ > 50%) | 0-6.66 | 15 |
| | 6-11 | 8 | | 6.67-9.32 | 5 |
| | 12-13 | 7 | | 9.33-10.6 | 2 |
| | 14-24 | 0 | | 10.7 plus | 0 |
| | 25-34 | 6 | | | |
| | 35-39 | 9 | | 0-13.2 | 0 |
| | 40-49 | 11 | | 13.3-33.2 | 7 |
| | 50 or more | 18 | | 33.3-46.5 | 9 |
| | | | | 46.6-66.6 | 11 |
| | | | | 66.7 and over | 14 |
| White cell count (× $10^9$/L) | 0-0.9 | 19 | Age (yr) | 0-44 | 0 |
| | 1.0-2.9 | 5 | | 45-59 | 5 |
| | 3.0-19.9 | 0 | | 60-64 | 11 |
| | 20.0-24.9 | 1 | | 65-69 | 13 |
| | 25 or more | 4 | | 70-74 | 17 |

| | | | | | |
|---|---|---|---|---|---|
| | | | | 75 or more | 24 |
| Hematocrit (%) | 0-49.9 | 0 | Chronic health evaluation (do not score in elective surgery patients) | Cirrhosis Immunosuppression Leukemia Multiple myeloma | 4 |
| | 50 or more | 3 | | | 10 |
| | | | | | 10 |
| | | | | | 10 |
| Plasma sodium (mmol/L) | 0-119 | 3 | | Metastatic cancer | 11 |
| | 120-134 | 2 | | Lymphoma | 13 |
| | 135-154 | 0 | | Hepatic failure | 16 |
| | 155 or more | 4 | | AIDS | 23 |
| Plasma albumin (g/L) | 0-19 | 11 | Neurological score | Use matrix | 0-48 |
| | 20-44 | 0 | | | |
| | 45 or more | 4 | | | |
| Acid base status | Use matrix | 0-12 | | | |

**STATISTICAL METHODS :**

All characteristics were summarized descriptively. For continuous variables, the summary statistics of N, mean, standard deviation (SD) were used. For categorical data, the number and percentage were used in the data summaries. Chi-square ($\chi 2$)/ Freeman-Halton Fisher exact test was employed to determine the significance of differences between groups for categorical data. The difference of the means of analysis variables was tested by unpaired t tes. If the p-value was $< 0.05$, then the results will be considered to be significant. Data were analyzed using SPSS software v.23.0..

**RESEARCH HYPOTHESIS:**.

To predict the outcome of patients with perforative peritonitis on the basis of APACHE 111 scoring system.

**SAMPLING:**

Study period from: October 2014 to June 2016.

All the patients admitted during this period, who will fulfill the inclusion criteria, will be included in this study.

Sample size is calculating using the formula,

$$n = \frac{Z_\alpha^2 p(1-p)}{D^2}$$

Where, n=sample size,

Zα=1.96,

p= Prevalence rate= 25%

q= 100-p,

E= allowable error=10%.

Minimum sample size is 75 patients.

For our study it is planned to conduct study on at least 100 patients, as our study is a prospective type.

Following statistical tests will be used to compare the results:

- Wilcoxon signed rank sum test or ‘t’ test for continuous variables.
- Chi-square test or the Fisher exact test.
- Multivariate analysis using logistic regression.
- Mean+/- Standard deviation.
- ROC curve

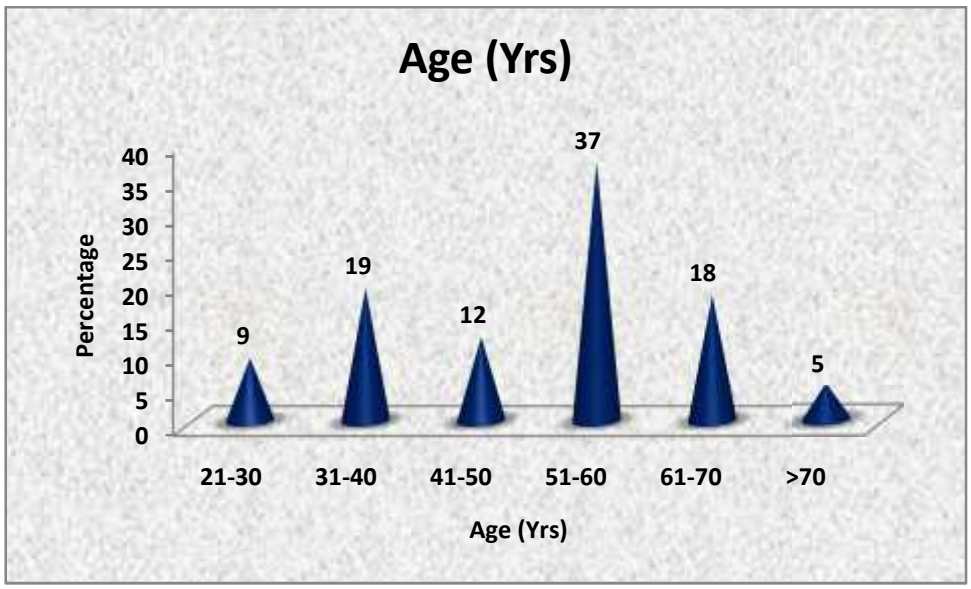
## RESULTS

### Age distribution

**Table9: Distribution of cases by Age**

| Age (Yrs.) | N | % |
|---|---|---|
| 21-30 | 9 | 9 |
| 31-40 | 19 | 19 |
| 41-50 | 12 | 12 |
| 51-60 | 37 | 37 |
| 61-70 | 18 | 18 |
| >70 | 5 | 5 |
| Total | 100 | 100 |

**Graph 1: Distribution of cases by Age**

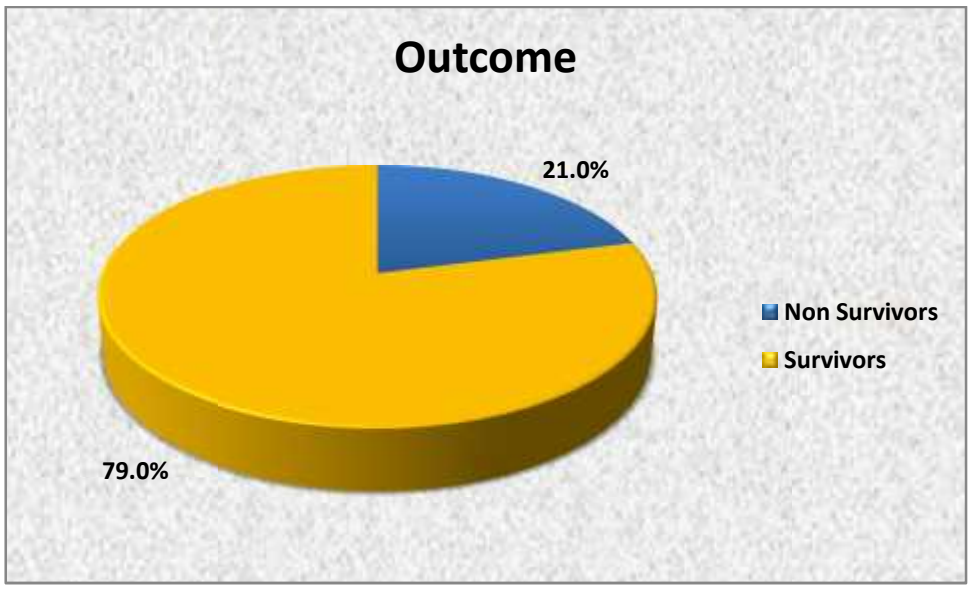



Age of the patients in the study ranged from 21years to 75years. Maximum number of patients 37 were in the age group (51-60), followed by (n=19) in age group 31 years to 40 years, (n=18) in age group 61 years to 70 years,(n=12_ in the age group 41 years to 50 years,(n=9) in the age group 21 years to 30 years, (n=5) in the age group >70 years. As depicted in graph no 1.

**Table10: Distribution of cases by Outcome**

| Outcome | N | % |
|---|---|---|
| Non Survivors | 21 | 21 |
| Survivors | 79 | 79 |
| Total | 100 | 100 |

**Graph 2: Distribution of cases by Outcome**

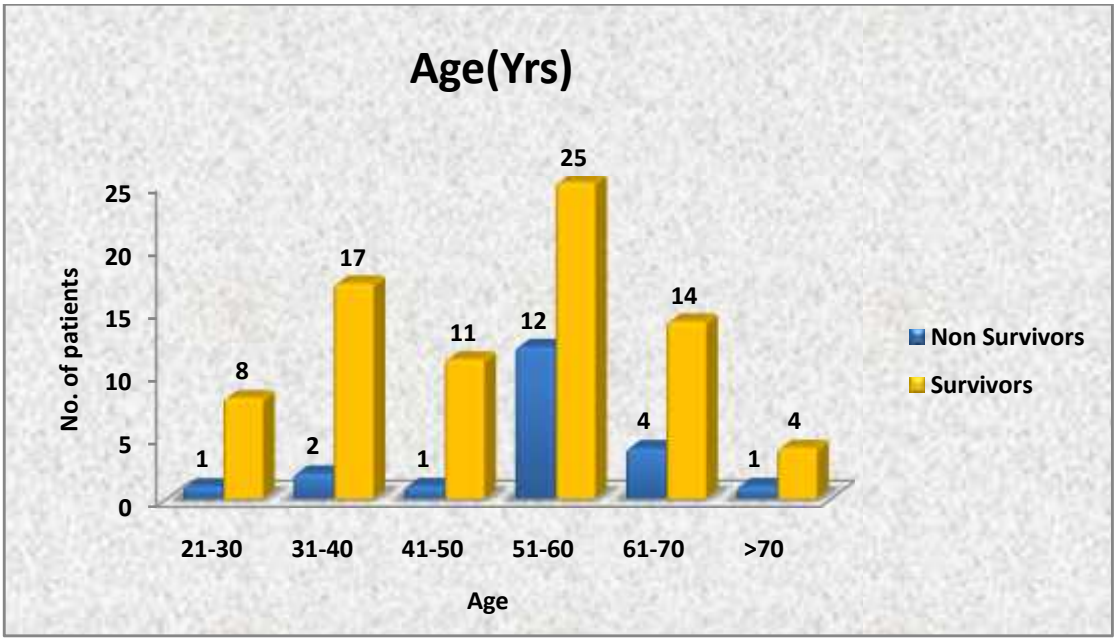



In present study it was observed that out of 100 patients with features of perforative peritonitis 79 patients were survivors and 21 patients were non-survivors.

**Table11: Distribution of Outcome by Age**

| Outcome | Non Survivors | | Survivors | | Total | p value |
|---|---|---|---|---|---|---|
| Age (Yrs.) | N | % | N | % | N | |
| 21-30 | 1 | 11.1% | 8 | 88.9% | 9 | |
| 31-40 | 2 | 10.5% | 17 | 89.5% | 19 | |
| 41-50 | 1 | 8.3% | 11 | 91.7% | 12 | |
| 51-60 | 12 | 32.4% | 25 | 67.6% | 37 | 0.372 |
| 61-70 | 4 | 22.2% | 14 | 77.8% | 18 | |
| >70 | 1 | 20.0% | 4 | 80.0% | 5 | |
| Total | 21 | 21.0% | 79 | 79.0% | 100 | |

**Graph 3: Distribution of Outcome by Age**

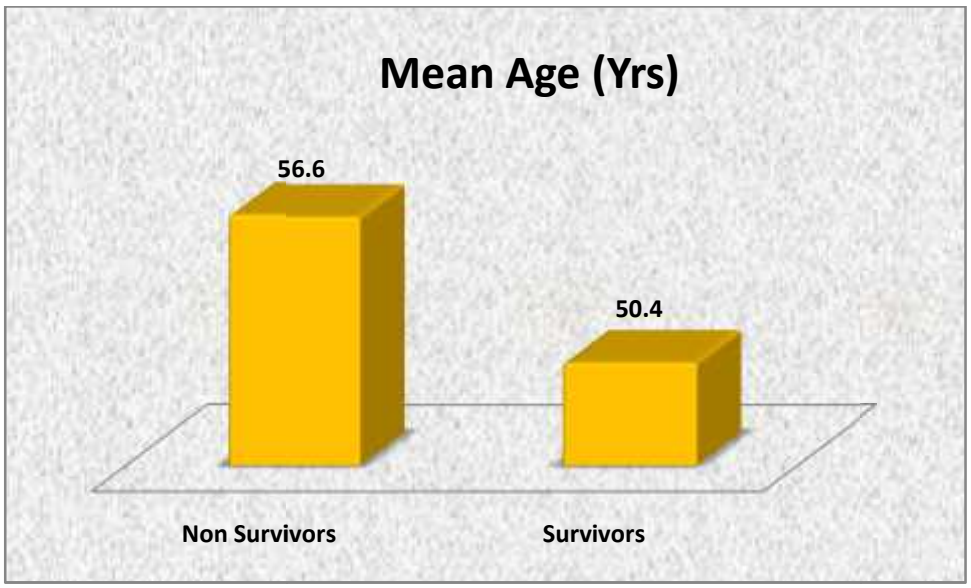



In the present study it was observed that highest no of survivors and non-survivors were seen in the age group of 51 years to 60 years, (n=12) non survivors and (n=25) survivors.

**Table12: Mean Age by Outcome**

| Parameters | Non Survivors | | Survivors | | p value |
|---|---|---|---|---|---|
| | Mean | SD | Mean | SD | |
| Age (Yrs.) | 56.6 | 13.1 | 50.4 | 16.0 | 0.104 |

**Graph 4: Mean Age by Outcome**

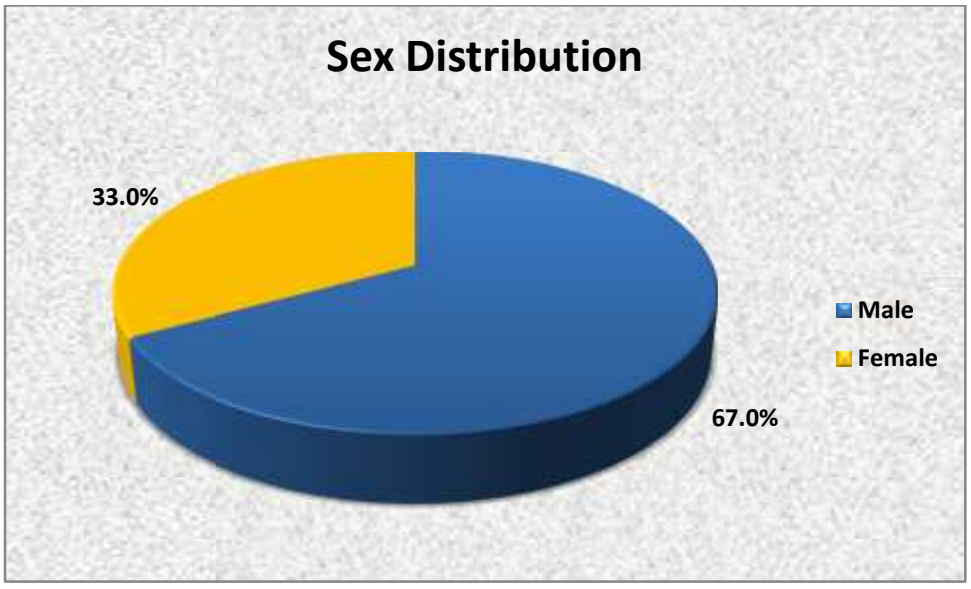



In the present study it was observed that mean age among survivors was 50.4(SD16.0) and mean age among non-survivors was 56.6(SD13.1)

**Table13: Distribution of cases by Sex**

| Sex | N | % |
|---|---|---|
| Male | 67 | 67 |
| Female | 33 | 33 |
| Total | 100 | 100 |

**Graph 5: Distribution of cases by Sex**

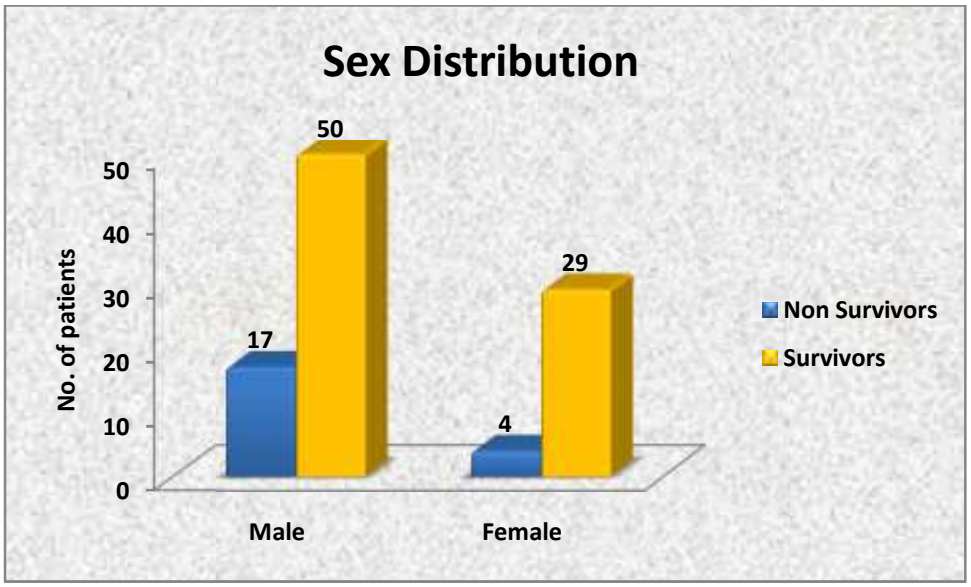



In the present study it was observed that out of 100 patients with perforative peritonitis 67% were male patients and 33.0% were female patients.

**Table14: Distribution of Outcome by Sex**

| Outcome | Non Survivors | | Survivors | | Total | p value |
|---|---|---|---|---|---|---|
| Sex | N | % | N | % | N | |
| Male | 17 | 25.4% | 50 | 74.6% | 67 | 0.191 |
| Female | 4 | 12.1% | 29 | 87.9% | 33 | |
| Total | 21 | 21.0% | 79 | 79.0% | 100 | |

**Graph 6: Distribution of Outcome by Sex**

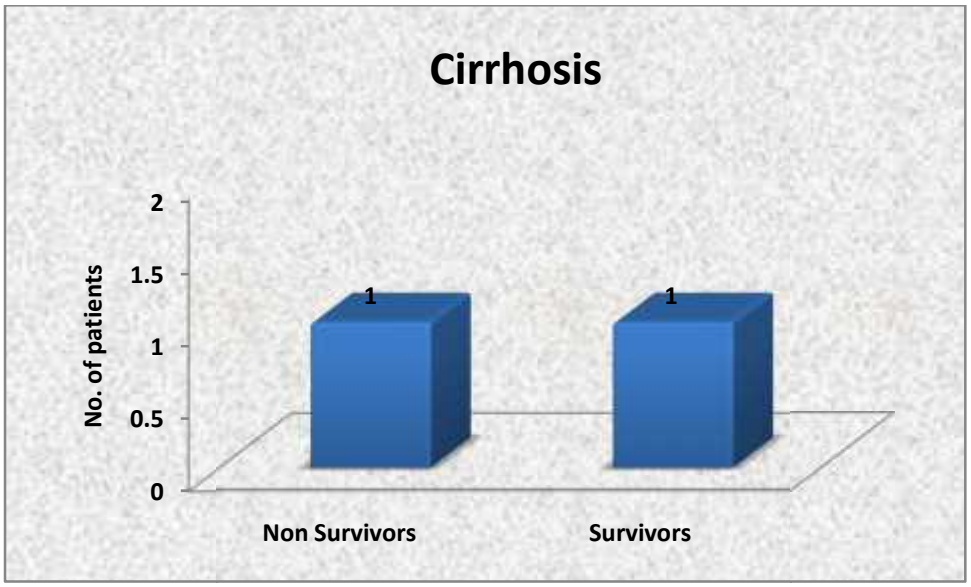



In the present study it was observed that out of 21 non-survivors 17 were male and 4were female patients. Among 79 survivors 50 were male patients and 29 were female patients.

**Table15: Distribution of Outcome by Chronic health evaluation**

| Outcome | Non Survivors | | Survivors | | Total | p value |
|---|---|---|---|---|---|---|
| **Chronic health evaluation** | **N** | **%** | **N** | **%** | **N** | |
| Cirrhosis | 1 | 50.0% | 1 | 50.0% | 2 | 0.378 |
| None | 20 | 20.4% | 78 | 79.6% | 98 | |
| Total | 21 | 21.0% | 79 | 79.0% | 100 | |

**Graph 7: Distribution of Outcome by Chronic health evaluation**

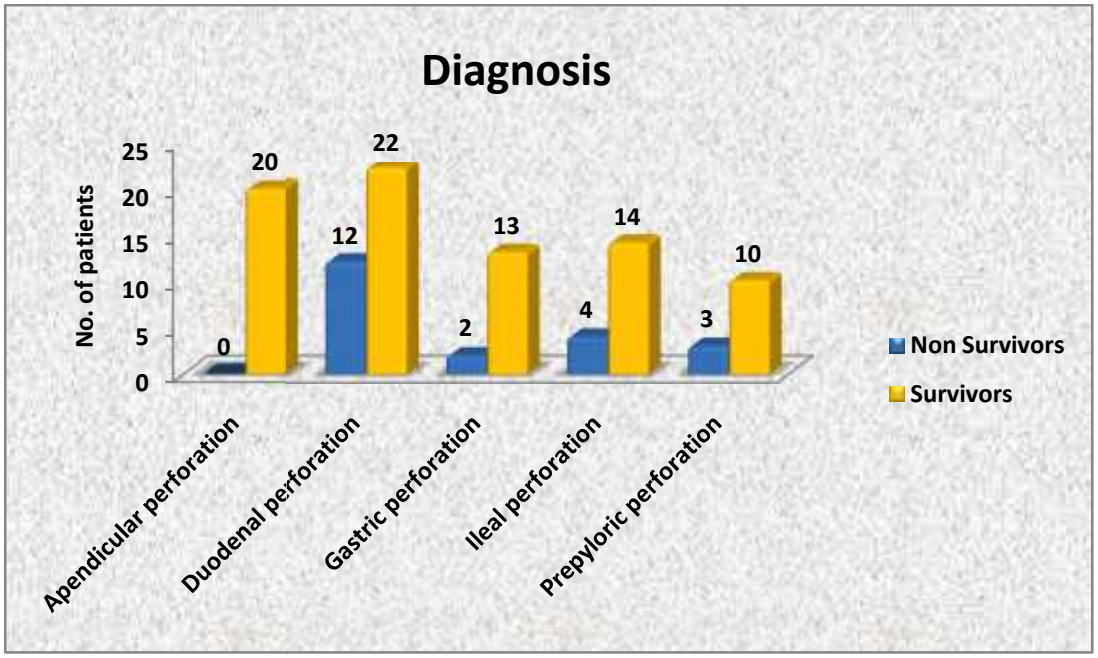



In the present study cirrhosis was observed among 1 patient in both non-survivor and survivor group.

**Table16: Distribution of Outcome by Diagnosis**

| Outcome | Non Survivors | | Survivors | | Total | p value |
|---|---|---|---|---|---|---|
| **Diagnosis** | **N** | **%** | **N** | **%** | **N** | |
| Appendicular perforation | 0 | 0.0% | 20 | 100.0% | 20 | |
| Duodenal perforation | 12 | 35.3% | 22 | 64.7% | 34 | |
| Gastric perforation | 2 | 13.3% | 13 | 86.7% | 15 | 0.020 |
| Ileal perforation | 4 | 22.2% | 14 | 77.8% | 18 | (Sig) |
| Prepyloric perforation | 3 | 23.1% | 10 | 76.9% | 13 | |
| Total | 21 | 21.0% | 79 | 79.0% | 100 | |

**Graph 8: Distribution of Outcome by Diagnosis**

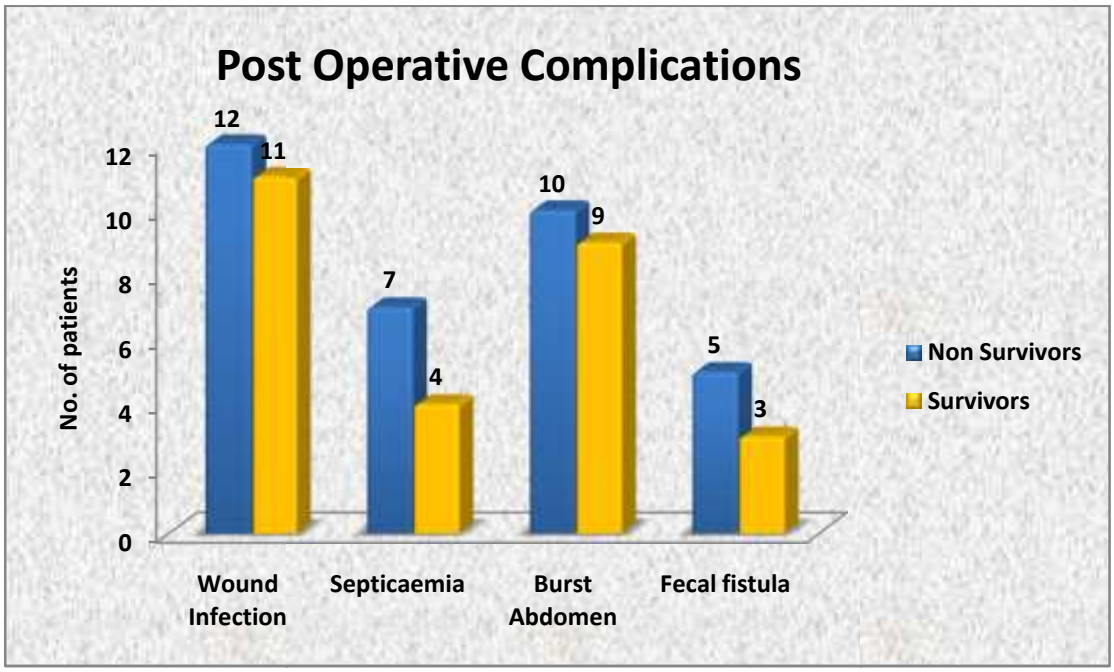



In the present study it was observed that highest no of mortality were observed among patients with duodenal perforation (n=12),ileal perforation(n=4),prepyloric perforation(n=3),gastric perforation(n=2) among non-survivor. Among survivors duodenal perforation was principal diagnosis followed by appendicular perforation.

**Table17: Distribution of Outcome by Post Operative Complications**

| Complications | Non Survivors | | Survivors | | Total |
|---|---|---|---|---|---|
| | N | % | N | % | N |
| Wound Infection | 12 | 52.2% | 11 | 47.8% | 23 |
| Septicemia | 7 | 63.6% | 4 | 36.4% | 11 |
| Burst Abdomen | 10 | 52.6% | 9 | 47.4% | 19 |
| Fecal fistula | 5 | 62.5% | 3 | 37.5% | 8 |

**Graph 9: Distribution of Outcome by Post Operative Complications**

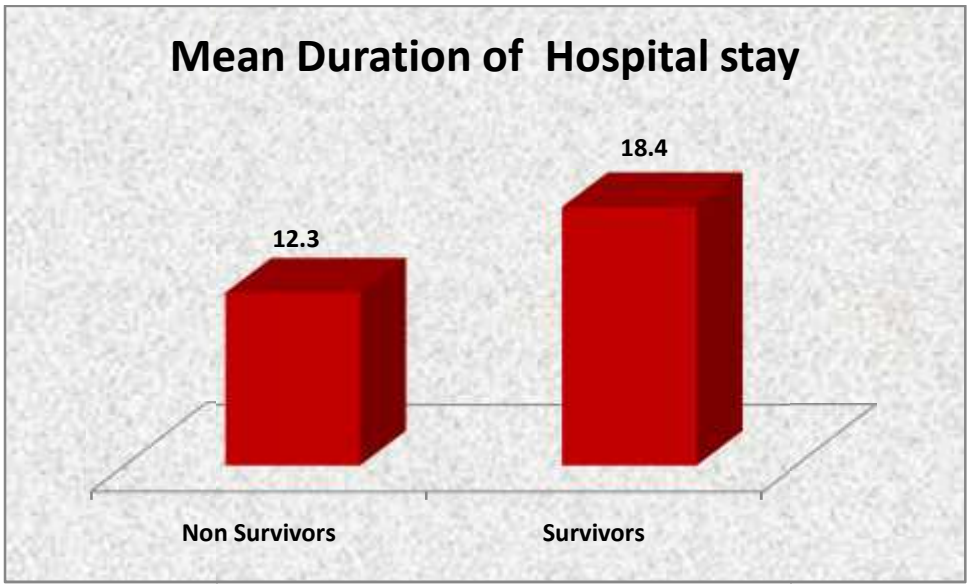



In the present study it was observed that most common post-operative complication among non-survivors and survivors was wound infection (n=12) and (n=11) followed by burst abdomen (n=10) and (n=9), septicemia (n=7) and (n=4), fecal fistula (n=5) and (n=3).

**Table18: Mean Duration of Hospital Stayby Outcome**

| | Non Survivors | Survivors | p value |
|---|---|---|---|
| **Mean Duration of Hospital stay** | 12.3±4.5 | 18.3±2.3 | <0.001 (Sig) |

**Graph 10: Mean Duration of Hospital Stayby Outcome**

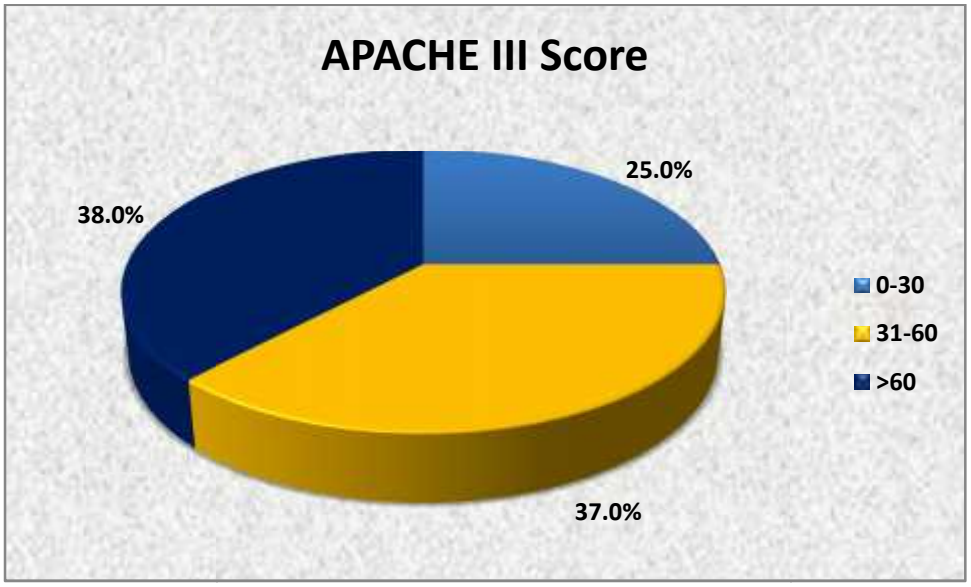



In the present study it was observed that mean duration of hospital stay was 18.4 days among survivors and 12.3 days among non-survivors.

**Table19: Distribution of cases by Total APACHE III Score**

| APACHE III Score | N | % |
|---|---|---|
| 0-30 | 25 | 25 |
| 31-60 | 37 | 37 |
| >60 | 38 | 38 |
| Total | 100 | 100 |

**Graph 11: Distribution of cases by Total APACHE III Score**

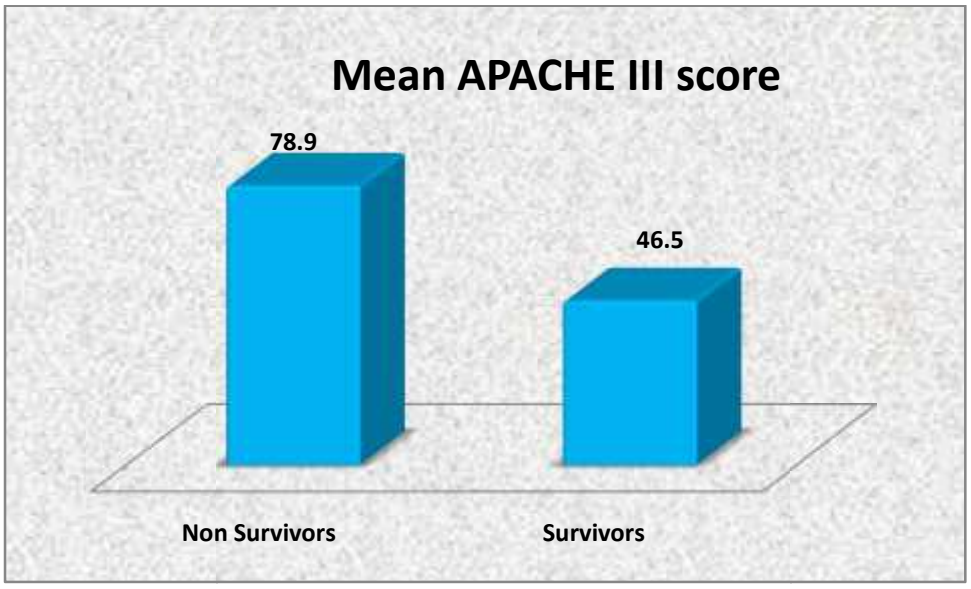



In the present study it was observed that out of 100 patients with perforative peritonitis 25 patients had APACHE 111 score in the range (0-30), 37 patients in the range (31-60) and 38 patients in the range (>60).

**Table20: Mean APACHE III Score by Outcome**

| Parameters | Non Survivors | | Survivors | | p value |
|---|---|---|---|---|---|
| | Mean | SD | Mean | SD | |
| Total APACHE III score | 78.9 | 20.2 | 46.5 | 20.3 | <0.001 (Sig) |

**Graph 12: Mean APACHE III Score by Outcome**

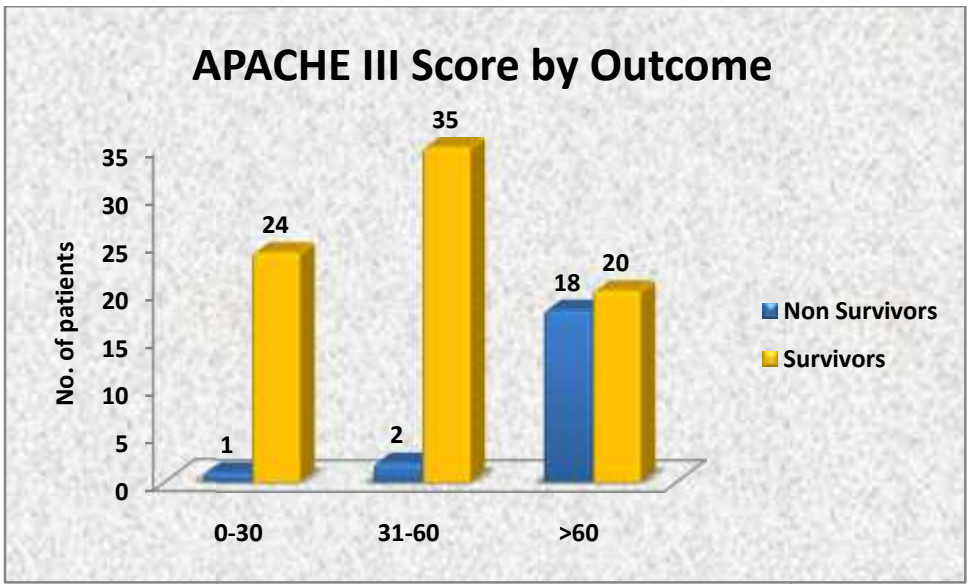



In the present study it was observed that mean APACHE 111 score among non-survivors was 78.9 and mean APACHE 111 score among survivors was 46.5.

**Table21: Distribution of cases by APACHE III Score and Outcome**

| Outcome | Non Survivors | | Survivors | | Total | p value |
|---|---|---|---|---|---|---|
| APACHE Score | N | % | N | % | N | |
| 0-30 | 1 | 4.0% | 24 | 96.0% | 25 | |
| 31-60 | 2 | 5.4% | 35 | 94.6% | 37 | 0.001 |
| >60 | 18 | 47.4% | 20 | 52.6% | 38 | (Sig) |
| Total | 21 | 21.0% | 79 | 79.0% | 100 | |

**Graph 13: Distribution of cases by APACHE III Score and Outcome**

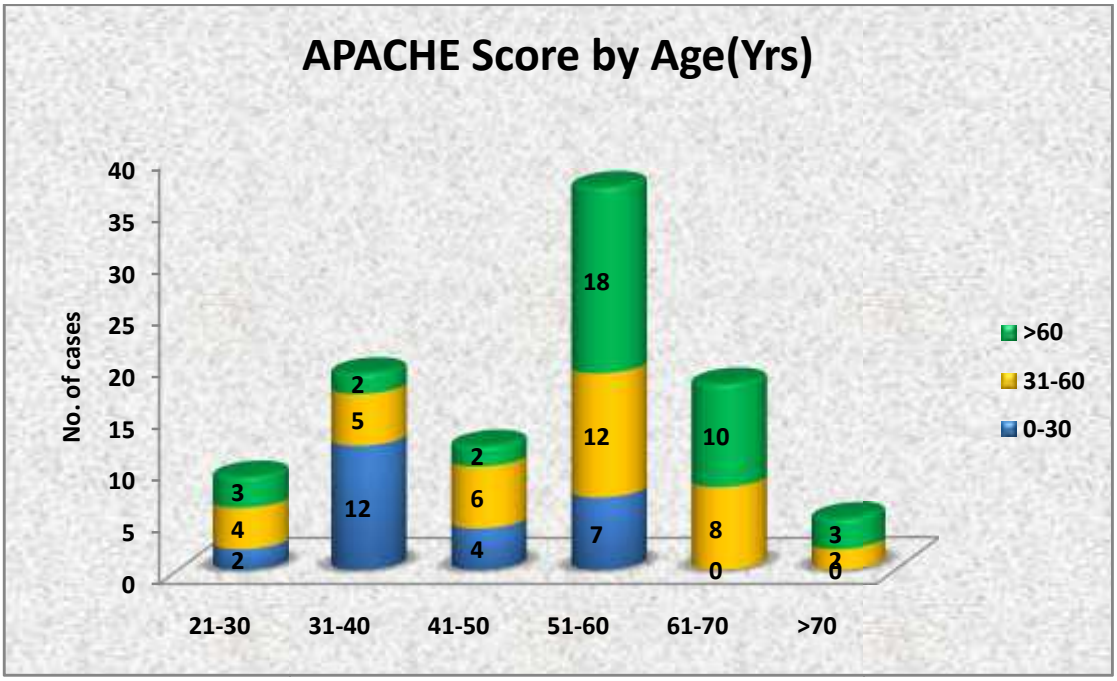



In the present study it was observed that 38 patients had score>60, 37 patients in the range (31-60) and 25 patients in the range (0-30). Mortality was observed among 18 patients with score> 60, 2 patients in the range (31-60) and 1 patient in the range (0-30).

**Table22: Distribution of cases by APACHE III Score and Age**

| APACHE Score | 0-30 | | 31-60 | | >60 | | Total | p value |
|---|---|---|---|---|---|---|---|---|
| Age(Yrs.) | N | % | N | % | N | % | N | |
| 21-30 | 2 | 22.2% | 4 | 44.4% | 3 | 33.3% | 9 | 0.002 (Sig) |
| 31-40 | 12 | 63.2% | 5 | 26.3% | 2 | 10.5% | 19 | |
| 41-50 | 4 | 33.3% | 6 | 50.0% | 2 | 16.7% | 12 | |
| 51-60 | 7 | 18.9% | 12 | 32.4% | 18 | 48.6% | 37 | |
| 61-70 | 0 | 0.0% | 8 | 44.4% | 10 | 55.6% | 18 | |
| >70 | 0 | 0.0% | 2 | 40.0% | 3 | 60.0% | 5 | |
| Total | 25 | 25.0% | 37 | 37.0% | 38 | 38.0% | 100 | |

**Graph 14: Distribution of cases by APACHE III Score and Age**

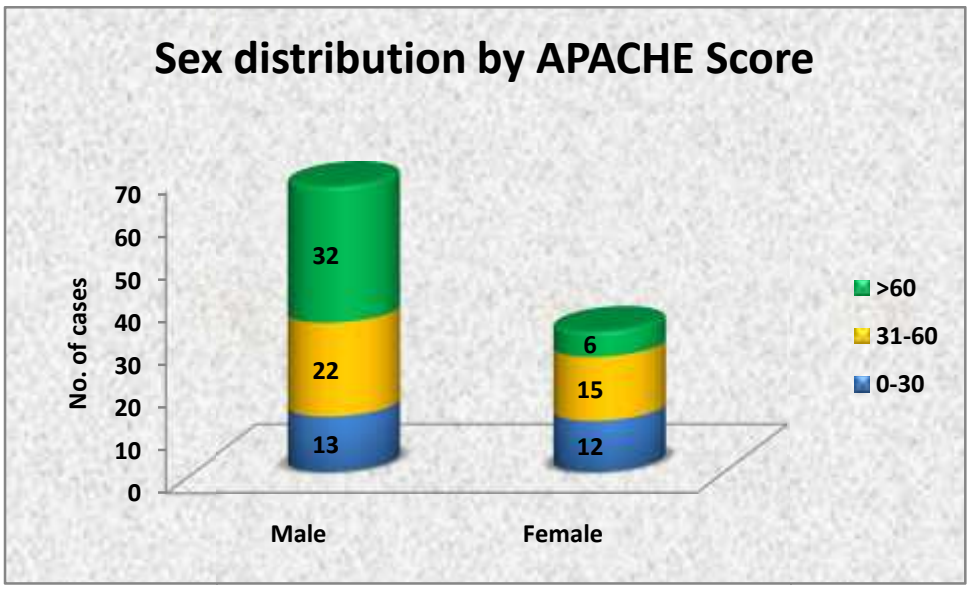



In the present study it was observed that among 38 patients with higher APACHE 111 score >60, 18 patients were in the age group (51-60 years), among 37 patients with score (31-60) 12 patients were in age group (51-60) and 25 patients with score less then 30 12 were in the age group (31-40 years).

**Table23: Distribution of cases by APACHE III Score and Sex**

| APACHE Score | 0-30 | | 31-60 | | >60 | | Total | p value |
|---|---|---|---|---|---|---|---|---|
| **Sex** | **N** | **%** | **N** | **%** | **N** | **%** | **N** | |
| Male | 13 | 19.4% | 22 | 32.8% | 32 | 47.8% | 67 | 0.014 (Sig) |
| Female | 12 | 36.4% | 15 | 45.5% | 6 | 18.2% | 33 | |
| Total | 25 | 25.0% | 37 | 37.0% | 38 | 38.0% | 100 | |

**Graph 15: Distribution of cases by APACHE III Score and Sex**

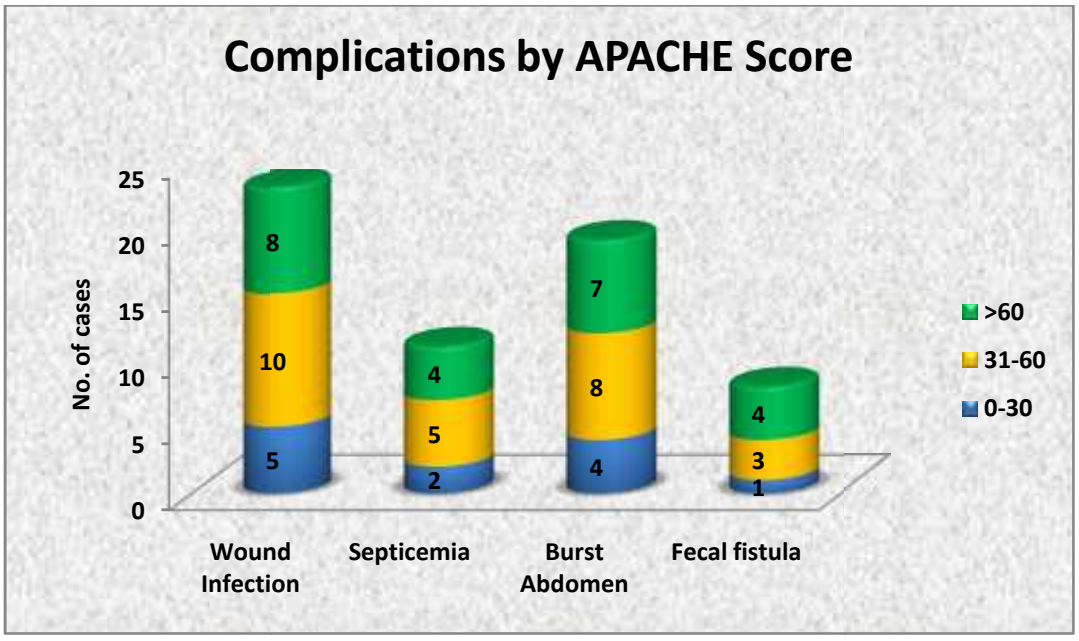



In the present study it was observed that among 38 patients with higher APACHE 111 score (>60) 32 were male and 6 were female patients, among 37 patients with APACHE 111 score (31-60) 22 were mal and 15 were female patients, among 25 patients with APACHE 111 score (<30) 13 were male patients and 12 were female patients.

**Table24: Distribution of cases by APACHE III Score and Complications**

| Complications | APACHE SCORE | | | | | |
|---|---|---|---|---|---|---|
| | 0-30 | | 31-60 | | >60 | |
| | N | % | N | % | N | % |
| Wound Infection | 5 | 21.7% | 10 | 43.5% | 8 | 34.8% |
| Septicemia | 2 | 18.2% | 5 | 45.5% | 4 | 36.4% |
| Burst Abdomen | 4 | 21.1% | 8 | 42.1% | 7 | 36.8% |
| Fecal fistula | 1 | 12.5% | 3 | 37.5% | 4 | 50.0% |

**Graph 16: Distribution of cases by APACHE III Score and Complications**

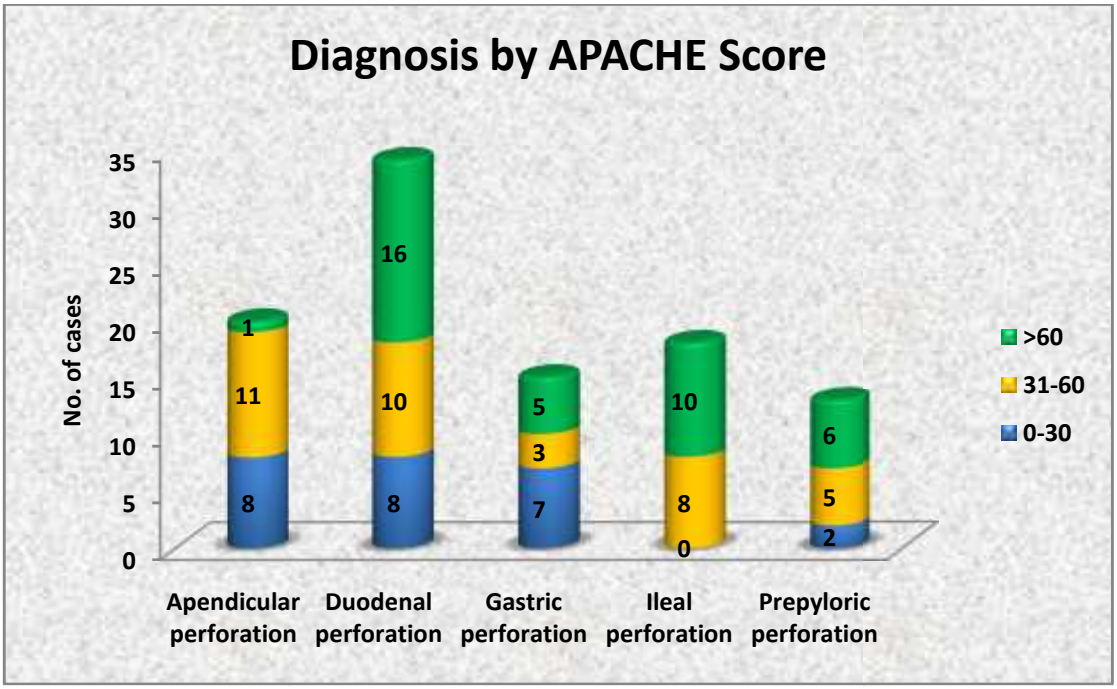



In the present study it was observed that wound infection was the most common complication among all three groups (n=23), followed by burst abdomen(n=19),septicemia (n=11),fecal fistula (n=8).

**Table25: Distribution of cases by APACHE III Score and Diagnosis**

| APACHE Score | 0-30 | | 31-60 | | >60 | | Total | p value |
|---|---|---|---|---|---|---|---|---|
| **Diagnosis** | **N** | **%** | **N** | **%** | **N** | **%** | **N** | |
| Appendicular perforation | 8 | 40.0% | 11 | 55.0% | 1 | 5.0% | 20 | 0.006 (Sig) |
| Duodenal perforation | 8 | 23.5% | 10 | 29.4% | 16 | 47.1% | 34 | |
| Gastric perforation | 7 | 46.7% | 3 | 20.0% | 5 | 33.3% | 15 | |
| Ileal perforation | 0 | 0.0% | 8 | 44.4% | 10 | 55.6% | 18 | |
| Prepyloric perforation | 2 | 15.4% | 5 | 38.5% | 6 | 46.2% | 13 | |
| Total | 25 | 25.0% | 37 | 37.0% | 38 | 38.0% | 100 | |

**Graph 17: Distribution of cases by APACHE III Score and Diagnosis**




In the present study it was observed that most common diagnosis among patients with higher APACHE 111 score was duodenal perforation (n=16). Most common diagnosis among patients with APACHE 111 score (31-60) was appendicular perforation (n=11), most common diagnosis among patients with APACHE 111 score<30 was both appendicular and duodenal perforation (n=8).

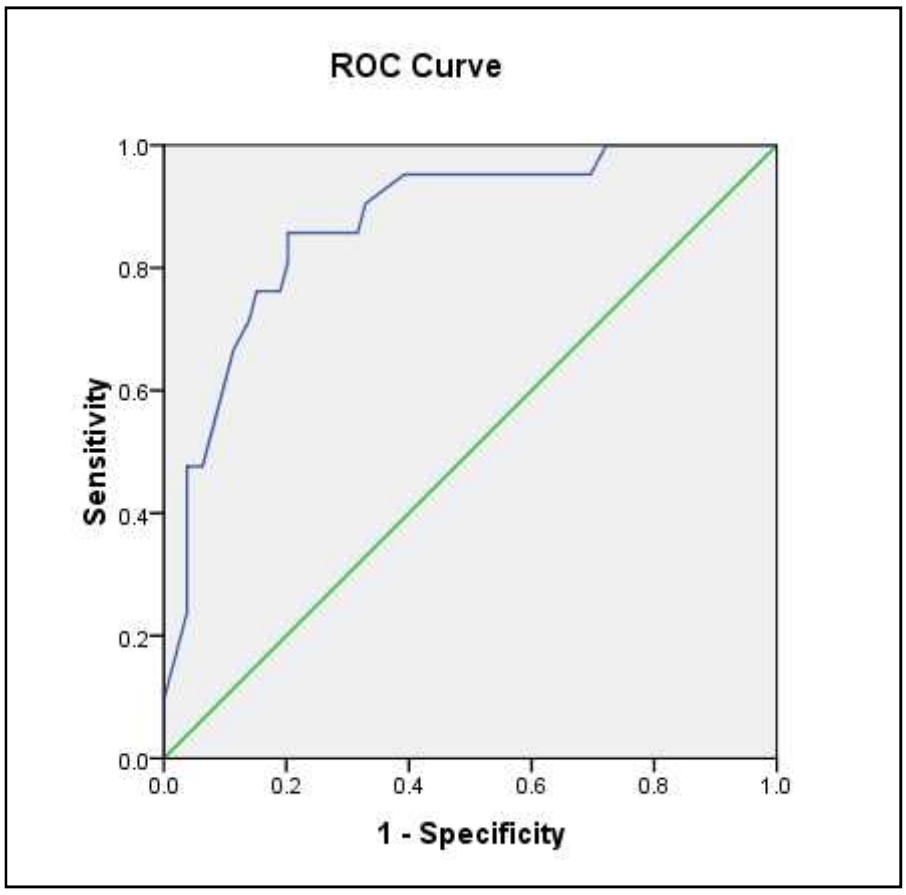
**Table26: Comparison of mean Parameters by Outcome**

| Parameters | Non Survivors | | Survivors | | p value |
|---|---|---|---|---|---|
| | Mean | SD | Mean | SD | |
| Temperature | 37.6 | 0.1 | 37.6 | 0.1 | 0.423 |
| Heart rate/min | 116.0 | 10.6 | 105.8 | 12.8 | 0.001 (Sig) |
| BP (mmhg) | 72.8 | 9.4 | 80.8 | 9.7 | 0.001 (Sig) |
| Plasma bilirubin (mg/dl) | 2.2 | 0.8 | 1.5 | 1.5 | 0.039 (Sig) |
| Urine volume(ml/24h | 742.9 | 396.6 | 1135.4 | 421.2 | <0.001 (Sig) |
| Serum creatinine(mg/dl) | 3.1 | 1.8 | 1.8 | 1.3 | <0.001 (Sig) |
| Respiratory rate/min | 28.7 | 5.5 | 26.6 | 4.3 | 0.068 |
| WBC count/cu mm | 9245.1 | 7676.9 | 10369.6 | 5129.6 | 0.427 |
| Hematocrit (%) | 39.3 | 8.3 | 39.0 | 8.5 | 0.873 |
| Plasma sodium (mmol/l) | 135.0 | 8.0 | 135.5 | 6.0 | 0.755 |
| Arterial po2/mmhg | 80.4 | 8.8 | 88.4 | 8.1 | <0.001 (Sig) |
| Plasma albumin (g/l) | 2.2 | 0.6 | 3.0 | 0.9 | <0.001 (Sig) |
| Ph. | 7.2 | 0.1 | 7.3 | 0.1 | <0.001 (Sig) |
| Glasgow coma scale | 10.0 | 2.7 | 13.7 | 2.3 | <0.001 (Sig) |
| Serum BUN mg/dl | 77.3 | 31.3 | 61.2 | 35.7 | 0.063 |
| Serum glucose (mg/dl) | 121.6 | 45.9 | 119.1 | 43.0 | 0.815 |

**Table27: Area Under curve in ROC Analysis for Total APACHE III score**

| Total APACHE III score | | | |
|---|---|---|---|
| Area Under curve | p value | 95% Confidence Interval | |
| 87.3% | <. 001 (Sig) | 0.791 | 0.955 |

**Graph 18: ROC Analysis for Total APACHE III score**




**Receiver operative characteristic curve**

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

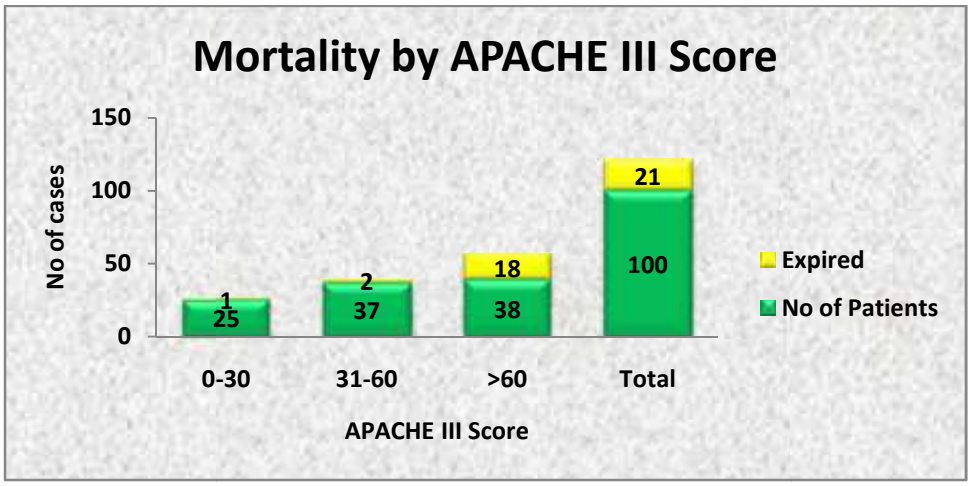
The ROC curves that related sensitivity to specificity for different cut-off points are shown

The area under curve was 87.3% for APACHE 111 score; the sensitivity of APACHE 111 was superior. And p value (<.001sig) indicating statistically significant.

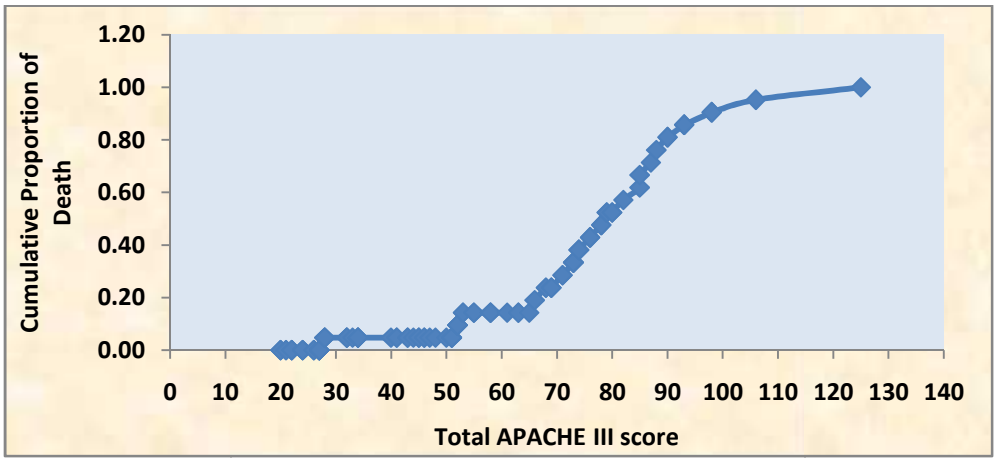
**Table28: Comparison of Observed and Predicted Mortality**

| APACHE III Score | No of Patients | Expired | Mortality | |
|---|---|---|---|---|
| | | | Observed | Predicted |
| 0-30 | 25 | 1 | 4.8% | 24.8% |
| 31-60 | 37 | 2 | 9.5% | 37.1% |
| >60 | 38 | 18 | 85.7% | 38.1% |
| Total | 100 | 21 | 100.0% | 100.0% |

**Graph 19: Proportion of Deaths by APACHE III Score**




**Graph 20: Cumulative Proportion of Deaths by APACHE III Score**




In the present study it was observed that among patients with higher APACHE 111 score> 60 observed mortality was 85.7% and predicted mortality was 38.1%, among patients with APACHE 111 score in the range (31-60) observed mortality was 9.5% and predicted mortality was 37.1%, among patients with APACHE 111 score < 30 observed mortality was 4.8% and predicted mortality was 24.8%.

## DISCUSSION

Prediction of outcome in patients with peritonitis is unpredictable due to certain unforeseen complications that occur during the course of the disease. 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 21% most of them were due to septicemia. The hospital mortality rate according to other studies ranged from 16% in Ajaz et al and reaching up to 21% per cent in case of C Ohmann et al, in these entire studies septicemia was main cause of death.

**COMPARISON OF PARAMETERS WITH OTHER STUDIES:**

**Demography: Age distribution:**

The prospective study involved 100 patients of both sexes with secondary peritonitis. Current study considered age range of 21-75 years. Mean age of the patients was 50.4(SD16). Maximum number of patients were in the age group51-60 (n=37), followed by (n=19) in 31-40 age group, (n=18) in 61-70 age group, (n=12) in 41-50 age group,(n=9) in 21-30 age group, (n=5) in age group>70 years. AshisAhujaetalalso stated predominant population from age group 21–40 years. C Ohmann et al study showed predominant population in 50-69years age group.

**Table 29: COMPARISON OF PREDOMINANT AGE GROUP IN PERITONITIS.**

| Study | Predominant age group |
|---|---|
| AshisAhuja et al[1] 2010-2011 | 21-40 years |
| C Ohmann et al[49] 2012-2014 | 50-69years |
| Our study | 51-60 years |

**Mean Age group with highest mortality**

Highest mortality in our study was observed among mean age group of 56.6years. As compared to 46.6 years in study conducted by G S Shrestaet al[50]

In our study it was observed that mortality rate increases with increase in age. Indicating Age is a significant factor contributing to survival.

**Table 30: MEAN AGE GROUP WITH HIGHEST MORTALITY**

| Studies | Mean age group with highest mortality |
|---|---|
| G S Shresta[50] 2010 | 46.6 years |
| Our study | 56.6 years |

**Sex distribution:**

Current study showed the male preponderance in peritonitis. Male preponderance was also found in Dr. S. K. Katiyar, Dr. S. K. Gahlotet al[51] study. In our study mortality rate was 25.4% among male sex as compared to 12.4% among female sex, indicating male sex is a risk factor.

**ETIOLOGICAL SPECTRUM OF PERFORATION:**

Site of peroration show a wide variability in different studies, 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[52]

In our study most common diagnosis was duodenal perforation, followed by appendicular perforation, which was similar to study conducted byRaj ender sing jhobta[54]

**Table 31: SITE OF PERFORATION IN DIFFERENT STUDY GROUP:**

| | Study | SITE OF PERFORATION | |
|---|---|---|---|
| | | Gastro duodenal | Appendicular |
| 1 | Raj ender sing jhobta et al[54] 2006 | 57% | 11% |
| 2 | NithinAgarwal et al[53] 2006 | 23% | 43% |
| 3 | Our study | 64% | 20% |

**Site specific mortality:**

Overall mortality rate in peritonitis due to hollow viscus perforation in our study was 21%.The individual mortality according to etiology showed highest with small intestine perforation (40.6%) as seen in NithinAgarwalstudy,Rajender Singh Jhobtaet al in their study concluded that most common cause of

Perforation was perforated duodenal ulcer (57%) followed by appendicitis (11%)Our study showed maximum mortality with duodenal perforation.

**Table 32: COMPARING SITE SPECIFIC MORTALITY RATE**

| Study | Site specific mortality rate | |
|---|---|---|
| | Gastro duodenal | Appendicular |
| Raj ender sing jhobta et al[54] 2006 | 57% | 11% |
| NithinAgarwal et al[53] 2006 | 23% | 43% |
| Our study | 64% | 20% |

**APACHE III Score:**

All the patients were assigned APACHE III score. APACHE III score in our study was from 0-299-reference range. Patients were divided into 3 score groups in the range of 0-30, 31-60 and >60 score. Mortality was observed among 18 patients with score>60, 2 patients with score in the range 31-60 and 1 patient with score<30 similar results were seen in the study conducted by Knauset al[55]

Where theyfound an increase in the mortality risk as the scores increased from < 30 to > 60.

**Table 33: APACHE III Score in various studies**

| Various studies | Scores | Mortality |
|---|---|---|
| Our Study | >60 | 47.4% |
| Knaus et al[55] 2002 | >60 | 41.6% |
| G S Shresta[50] 2010 | >60 | 45.6% |

**Table 34: POSTOPERATIVE COMPLICATIONS**

| Post operative complications | Wound infection | Burst abdomen | Septicemia | Fecal fistula |
|---|---|---|---|---|
| Raj ender sing jhobta et al[54] 2006 | 25% | 15.2% | 11% | 2.7% |
| Ashish ahuja[2] 2010-2011 | 56% | 12% | 7% | 3% |
| Our study | 23% | 19% | 11% | 8% |

Most common postoperative complication in our study among patients with higher APACHEIII score was wound infection followed by burst abdomen, septicemia and fecal fistula. Similar results were seen in the study by Raj ender sing jhobtaet al[54]

Where most common post operative complication among patients with higher APACHE III score was wound infection followed by burst abdomen. Hence higher APACHE III score is associated with worst outcome and trouble some postoperative complication.

**Table 35: MEAN APACHE 111 SCORE**

| Mean APACHE III score | Non survivors | Survivors |
|---|---|---|
| G S Shresta[50] 2010 | 94.95 | 50.14 |
| Bohnen et al[56] 2009 | 22.4 | 8 |
| Our study | 78.9 | 46.5 |

Mean APACHEIII score among non survivors was 78.9(SD20.2), among survivors was 46.5(SD20.6) similar results were seen in the study by G S Shresta[50]

Where mean APACHE III score among non-survivors was 94.95 and among survivors was 50.14.Comparatively, in study conducted by Bohnen et al., Adesunkanmi et al., Agarwal S et al[56]., the mean APACHE II score among survivors was 8 (low risk group) and among non–survivors was 22.4 (high risk group). Thus conclusive of the fact that mortality is directly linked with higher scores.

**Table 36: MEAN DURATION OF HOSPITAL STAY**

Mean duration of hospital stay was 12.3 days among non-survivors and 18.4 among survivors similar results were seen in study by Dr. S. K. Katiyar[57] and Bohnen et al[56]

Where mean duration of hospital stay was 13 days among non-survivors and 17.8 days for survivors indicating that higher APACHE III score is associated with reduced duration of hospital stay due to mortality.

| Mean duration of hospital stay | Non survivors | Survivors |
|---|---|---|
| Dr. S. K. Katiyar[51] | 13 | 17.8 |
| Bohnen et al[56] | 13 | 18 |
| Our study | 12.3 | 18.4 |

**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 87.3% for APACHE III in our study and was consistent with Knauset al[55]

90% and Zimmerman et al[57] 89%implying that it has an excellent discriminative ability. Our analysis resulted in APACHE 111 score being accurate.

**Table 37: COMPARISON OF AREA UNDER ROC CURVE IN VARIOUS STUDIES**

| | Study | Area under ROC curve in APACHE III |
|---|---|---|
| 1 | Our study | 0.955 |
| 2 | Knaus et al[55] | 0.900 |
| 3 | Zimmerman et al[57] | 0.890 |

**Table38: Comparison of Observed and Predicted Mortality**

Observed and predicted mortality for patients with APACHE III score (0-30) was 4.8% and 24.8%, for patients with score (31-60) observed and predicted mortalitywas 9.5% and 37.1%, observed and predicted mortality for with score > 60 was 85.7% and 38.1%, in study by Dr. S. K. Katiyar[51]

Observed and predicted mortality with score (0-30) was 2.8% and 7.5%, for score with (31-60) observed and predicted mortality was 8% and 25.2%, for score >60 observed and predicted mortality was 41.6% and 42.6%. These results were slightly different from our study results probably owing to larger sample size (n=100) in our study as compared to (n=72) in their study. In study by Markgrof R et

Showed that hospital mortality rate was higher than predicted for patients with higher APACHE III score (>60) was consistent with results of our study in which observed mortality was significantly higher than predicted mortality for patients with score >60, 85.7% and 37.5%. Indicating that score above 60 is an important prognostic factor.

**Table38: COMPARISON OF OBSERVED AND PREDICTED MORTALITY**

**Our study**

| APACHE III Score | No of Patients | Expired | Mortality | |
|---|---|---|---|---|
| | | | Observed | Predicted |
| 0-30 | 25 | 1 | 4.8% | 24.8% |
| 31-60 | 37 | 2 | 9.5% | 37.1% |
| >60 | 38 | 18 | 85.7% | 38.1% |
| Total | 100 | 21 | 100.0% | 100.0% |

**Dr. S. K. Katiyar[51]**

| APACHE III Score | No of Patients | Expired | Mortality | |
|---|---|---|---|---|
| | | | Observed | Predicted |
| 0-30 | 35 | 1 | 2.8% | 7.5% |
| 31-60 | 25 | 2 | 8% | 25.2% |
| >60 | 12 | 5 | 41.6% | 42.6% |

## SUMMARY

In our study we analyzed 100 patients with perforative peritonitis confirmed on emergency laparotomy. Mortality rate as cited in various studies ranged from 10% to 60%, our study had 21% mortality rate. Highest mortality is in the age group of > 51-60(30.83%). There were 37 patients in this group out of which 12 patients died. Lowest mortality (1) is seen in age group of 21-30years. In our study mortality increases with age, consistent with other studies. Out of 67 male patients (50) 74.6% patients survived and 12(17.9%) patients died. 33 female patients were included in the study among them 29 (87.8) survived and 4(12.2%) died.

Thus in our study mortality was observed more in males. The perforation of duodenum was most common diagnosis (29.4%) inn our study. Site of peroration show a wide variability in different studies. All the patients were subjected to emergency exploratory laparotomy. 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 APACHE III scoring system. Each patient was assigned APACHE III score, based on the APACHE III score patients were divided into 3 groups with scores of <60,31-60 and >60.

Number of patients scoring less than 30 was 25 out of 100(25%) in study group. 37 patients scored in the range of 31-60, 35 patients survived with mortality observed among 2 patients (5.4%), there were 38 patients who scored more then 60, with mortality observed among 18(47%) and 20 patients (52.6) survived.

Thus in our study mortality increased with increasing score.

Mean duration of hospital stay in our study was 12.3 among non-survivors and 18.3 days among survivors. These results are similar to study by Dr. S. K. Katiyar[57] and Bohnenet al[56]

Where mean duration of hospital stay was 13 days among non-survivors and 17.8 days for survivors indicating that higher APACHE III score is associated with reduced duration of hospital stay due to mortality.

Most common postoperative complication in our study among patients with higher APACHE III score was wound infection (23%) followed by burst abdomen (18%), septicemia (11%) and fecal fistula (8%). Similar results were seen in the study by Raj ender sing jhobtaet al[54]

Where most common post operative complication among patients with higher APACHE III score was wound infection followed by burst abdomen. Hence higher APACHE III score is associated with worst outcome and trouble some postoperative complication.

APACHE III score were accurate in predicting the outcome. Accuracy i.e. discriminative ability of the scoring system is measured by area under receiver operative curve. The area below the curve wasthe area below the curve was 87.3% for APACHE III in our study and was consistent with Knauset al[55]

90% and Zimmerman et al[57] 89%implying that it has an excellent discriminative ability. Our analysis resulted in APACHE 111 score being accurate.

In APACHE III score <30, observed mortality was 4.8% and predicted mortality was 24.8% for patients with score (31-60) observed and predicted mortality was 9.5% and 37.1%, observed and predicted mortality for with score > 60 was 85.7% and 38.1%, these results were similar to study by Markgrof R et[58]

Which Showed that hospital mortality rate was higher than predicted for patients with higher APACHE III score (>60), Indicating that score above 60 is an important prognostic factor.

APACHE III score were reliable in predicting mortality in patients with score <30 and 31-60. In patients with higher APACHE III score > 60, predicted mortality did not correlate with observed hospital mortality in our study.

As per analysis APACVHE III score was reliable in predicting mortality for patients with score <60.

## CONCLUSION

Perforative peritonitis is most common in elderly males. In hospital mortality rate for perforative peritonitis remains high in spite of advances in investigation, improved treatment modality, better inpatient care and advanced hospital resources.

A scoring system is efficient if it is accurate and sharp in predicting prognosis and also reliable and which can be reproduced if needed to stratify the patients to risk category. This will help us to divert the resources of hospital to appropriate patient help in decisions like transfer of patients to intensive care unit, the choice of more effective but expensive antibiotics and treatment modality. By comparing expected against observed outcome the score can be used to monitor quality of patient care.
Patients with lower APCHE III scores have more favorable prognosis than patients

With higherAPCHE III score. In patients with higher APACHE III score > 60, predicted mortality did not correlate with observed hospital mortality in our study.

Thus it was concluded from this study that APACHE III score was reliable in predicting mortality for patients with score < 60.And APACHE III score, as measured beforethe treatment of perforation peritonitis correlates significantly with the outcome of disease With respect to both morbidity and mortality.

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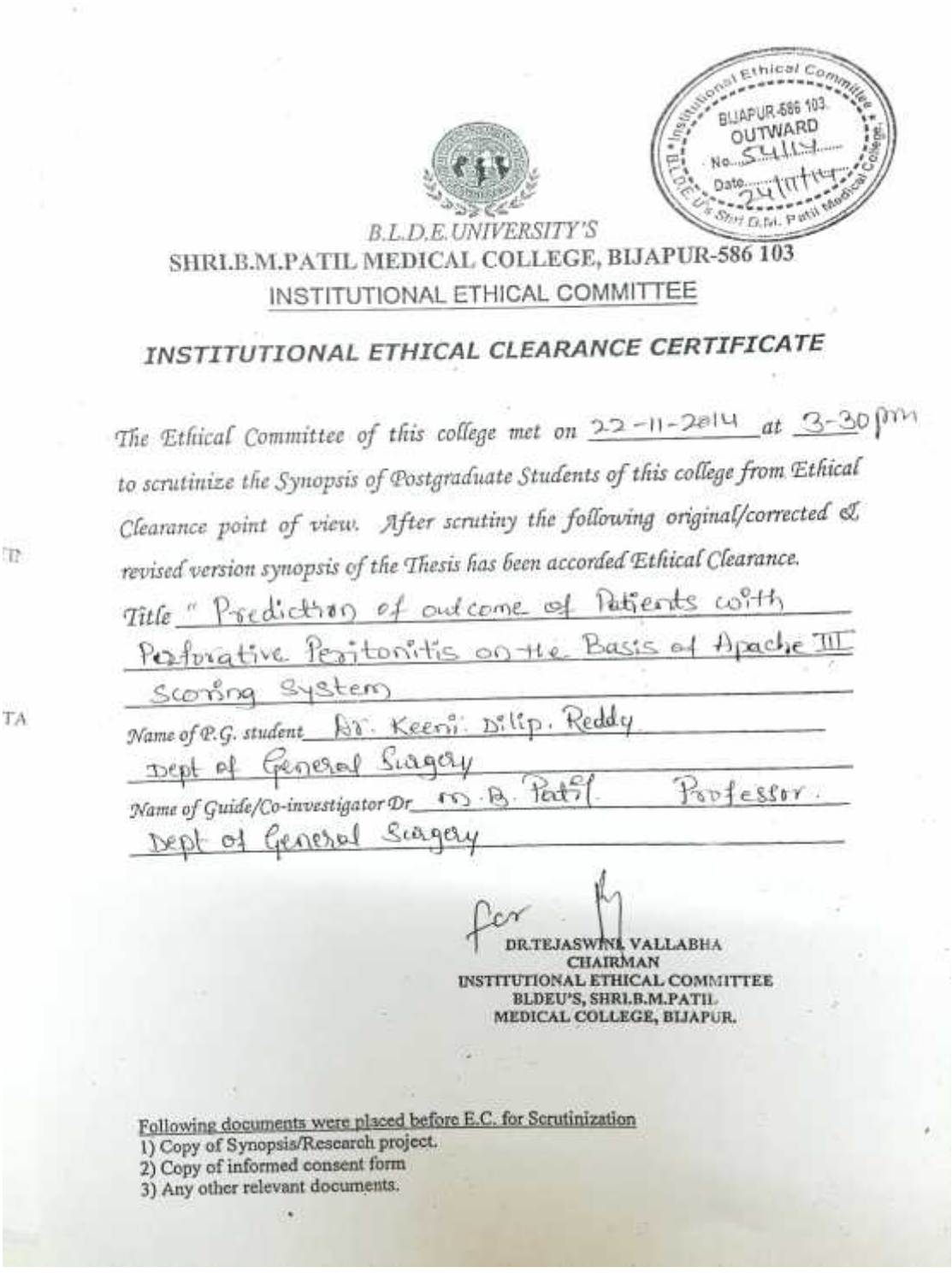
## ANNEXURES-I

## ETHICAL CLEARENCE CERTIFICATE



B.L.D.E. UNIVERSITY'S
SHRI.B.M.PATIL MEDICAL COLLEGE, BIJAPUR-586 103
INSTITUTIONAL ETHICAL COMMITTEE

**INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE**

The Ethical Committee of this college met on 22-11-2014 at 3-30 pm to scrutinize the Synopsis of Postgraduate Students of this college from Ethical Clearance point of view. After scrutiny the following original/corrected & revised version synopsis of the Thesis has been accorded Ethical Clearance.

Title " Prediction of outcome of Patients with Perforative Peritonitis on the Basis of Apache III Scoring System

Name of P.G. student Dr. Keeni. Dilip. Reddy
Dept of General Surgery

Name of Guide/Co-investigator Dr M.B. Patil Professor.
Dept of General Surgery

for
DR.TEJASWINI VALLABHA
CHAIRMAN
INSTITUTIONAL ETHICAL COMMITTEE
BLDEU'S, SHRI.B.M.PATIL
MEDICAL COLLEGE, BIJAPUR.

Following documents were placed before E.C. for Scrutinization
1) Copy of Synopsis/Research project.
2) Copy of informed consent form
3) Any other relevant documents.

## ANNEXURES-II

## SAMPLE INFORMED CONSENT FROM

## BLDEU'S SHRI B. M. PATIL MEDICAL COLLEGE HOSPITAL AND RESEARCH CENTER, BIJAPUR- 586103

**TITLE OF THE PROJECT – Prediction of outcome of patients with perforative peritonitis on the basis of APACH III scoring system.**

PRINCIPAL INVESTIGATOR - DR. KEENI DILIP REDDY

GUIDE - DR. M.B PATIL$_{MS}$

PROFESSOR OF SURGERY

**PURPOSE OF RESEARCH:**

I have been informed that this is a prospective study to evaluate the prognostic scoring system in perforative peritonitis, APACHE III scoring system.

**PROCEDURE:**

I am aware that in addition to routine care received I will be asked series of questions by the investigator. I have been asked to undergo the necessary investigations and treatment, which will help the investigator in this study.

**RISK AND DISCOMFORTS:**

I understand that I may experience some pain and discomfort during the examination or during my treatment. This is mainly the result of my condition and the procedure of this study is not expected to exaggerate these feelings that are associated with the usual course of treatment.

**BENEFITS:**

I understand that my participation in this study will help in analyzing whether APACHE III scoring systems is a better predictor of mortality in peritonitis.

**CONFIDENTIALITY:**

I understand that the medical information produced by this study will become a part of Hospital records and will be subject to the confidentiality and privacy regulation. Information of a sensitive personal nature will not he a part of the medical records, but will be stored in the investigator's research file and identified only by a code number. The code-key connecting name to numbers will be kept in a separate location.

If the data are used for publication in the medical literature or for teaching purpose, no name will be used and other identifiers such as photographs and audio or videotapes will be used only with my special written permission. I understand that I may see the photographs and videotapes and hear the audiotapes before giving this permission.

**REQUEST FOR MORE INFORMATION:**

I understand that I may ask more questions about the study at anytime Dr.Keeni Dilip reddy is available to answer my questions or concerns. I understand that I will be informed of any significant new findings discovered during the course of the study, which might influence my continued participation.

If during the study, or later. I wish to discuss my participation in or concerns regarding this study with a person not directly involved, I am aware that the social worker of the hospital is available to talk with me.

**REFUSAL FOR WITHDRAWAL OF PARTICIPATION:**

I understand that my participation is voluntary and that I may refuse to participate or may withdraw consent and discontinue participation in the study at any time without prejudice to my present or future care at this hospital. I also understand that Dr.Keeni Dilip reddymayterminate my participation in the study after she has explained the reasons for doing so and has helped arrange for my continued care by my own physician or physical therapist, if this is appropriate.

**INJURY STATEMENT:**

I understand that in the unlikely event of injury to me resulting directly from my participation in this study, if such injury were reported promptly, the appropriate treatment would be available to me, but no further compensation would be provided. I understand that by my agreement to participate in this study I am not waiving any of my legal rights.

I have explained to _____ the purpose of the research, the procedures required and the possible risks and benefits to the best of my ability in patients own language.

Dr.Keeni Dilip reddy Date
(Investigator)

**STUDY SUBJECT CONSENT STATEMENT:**

I confirm that Dr.Keeni Dilip reddy has explained to me the purpose of research, the study procedures that I will undergo. and the possible risks and discomforts as well as benefits that I may experience in my own language. I have read and I understand this consent form. Therefore, I agree to give consent to participate as a subject in this research project.

Participant / Guardian Date

Witness to signature Date

**ANNEXURES-III**

**PROFORMA**

SL NO

NAME

AGE IP NO

SEX UNIT

RELIGION DOA

OCCUPATION DOO

ADDRESS DOD

SOCIO-ECONOMIC STATUS

**Complaints:**

**HISTORY OF PRESENT ILLNESS**

**A.HISTORY OF PAIN**:

1.MODE OF ONSET

2. SITE OF PAIN

3.HOW LONG IS THE HISTORY OF PRESENTING COMPLAINT OF PAIN

4. DOES PAIN RADIATES

5. CHARACTER OF PAIN

6. RELIEF OF PAIN

7. NUMBER OF HOURS SINCE ACUTE PAIN STARTED.

B.**VOMITING**

1.PROJECTILE/ NON-PROJECTILE

2. NATURE OF VOMITUS

3. NO. OF TIMES

4. HAEMATEMESIS

C. **FEVE**R:

D.**DISTENSION OF ABDOMEN:**

E. **CONSTIPATION:**

**PAST HISTORY:**

**PERSONAL HISTORY:** SMOKER/ALCOHOLIC

**GENERAL PHYSICAL EXAMINATION**

BUILT: WELL/MODERATE/POOR

NOURISHMENT: WELL/MODERATE/POOR

PALLOR

ICTERUS

FEBRILE

PEDAL EDEMA

GENERAL LYMPHADENOPATHY

NUTRITIONAL STATUS:

a. GENERAL APPEARANCE: NORAMAL/THIN

b. ANTHROPOMETRY: HT

WT

**VITAL DATA**:

TEMPERATURE:

PULSE

RESPIRATORY RATE

BLOOD PRESSURE:

**SYSTEMIC EXAMINATION:**

**PER ABDOMEN:**

**INSPECTION:**

CONTOUR OF ABDOMEN

MOVEMENTS WITH RESPIRATION

UMBILICUS

VISIBLE PERISTALSIS

VISIBLE PULSATION

SKIN OVER ABDOMEN

HERNIAL ORIFICES

**PALPATION**

LOCAL RAISE OF TEMPERATURE

HYPERAESTHESIA

TENDERNESS

RIGIDITY/GAURDING

LUMP

PALPATION OF HERNIAL SITES

ABDOMIN GIRTH

**PERCUSSION**

SHIFTING DULLNESS

FLUID THRILL

OBLITERATION OF LIVER DULLNESS

**AUSCULTATION**

BOWEL SOUNDS

**PER RECTAL**:

**RESPIRATORY SYSTEM**

**CARDIOVASCULAR SYSTEM**

**CENTRAL NERVOUS SYSTEM**

**CLINICAL DIAGNOSIS:**

**LABORATORY TESTS**

HB%

TOTAL COUNT

DIFFERENTIAL COUNT

N/L/E/B/M:

PT

APTT

INR

URINE ROUTINE:

RBS

FBS

PPBS

B.UREA

S.CREATININE

TOTAL PROTEIN

S.ALBUMIN

SERUM ELECTROLYTES

Na

K

Cl

CA

PERITONIAL ASPIRATION

PERITONIAL FLUID ANALYSIS AND CULTURE SENSITIVITY

BLOOD CULTURE

BLOOD GROUPING

HIV

HBsAg

CHEST X RAY:

ERECT ABDOMEN X-RAY:

ULTRASONOGRAPHY OF ABDOMEN AND PELVIS:

CT SCAN OF ABDOMEN:

ARTERIAL BLOOD GAS ANALYSIS:

OTHERS: PHYSIOLOGICAL PARAMETERS OF APACHE-111 SCORE

1} PULSE RATE

2} RESPIRATORY RATE

3} MEAN ARTERIAL PRESSURE

4} TEMPERATURE

5} URINE OUTPUT {24HRS}

6} HAEMATOCRIT {%}

7} WHITE BLOOD CELL COUNT

8} SERUM SODIUM {mmol/l}

9} SERUM CREATININE{mg/dl}

10} SERUM ALBUMIN {g/dl}

11} SERUM BILIRUBIN {mg/dl}

12} Blood urea nitrogen {mg/dl}}

13} Blood sugar {mg/dl}

14} ARTERIAL $P^H$

15} OXYGENATION {pa o2 in mm of hg with fio2 < 0.05}

16] GLASGOW COMA SCALE

OPERATIVE PROCEDURE (DATE AND TIME):

**INTRA-OPERATIVE DIAGNOSIS:**

DURATION OF PROCEDURE:

POST OPERATIVE INVESTIGATIONS:

LENGTH OF STAYIN HOSPITAL AFTER PROCEDURE:

**POST OPERATIVE COMPLICATIONS**

1. BLEEDING.
2. POST OPERATIVE SURGICAL SITE INFECTIONS.
3. SEPTIC COMPLICATIONS. a) WOUND INFECTION.
   b) INTRA-ABDOMINAL INFECTION.
4. URINAY COMPLICATIONS.
5. CARDIAC.
6. RESPIRATORY COMPLICATIONS.
7. GLYCEMIC CONTROL.

These values were scored in accordance to the APACHE-III chart scoring for abnormally high or low range. Zero score represents a normal value. These parameters represent acute physiology score.

**MASTER CHART**

| sl no | age | sex | diagnosis | temperature | heart rate/min | mean BP(mmhg) | plasma bilirubin(mg/dl) | urine volume(ml/24h | serumcreatinine(mg/dl) | respiratory rate/min | WBC count/cumm | hematocrit(%) | plasma sodium(mmol/l) | arterial po2/mmhg | chronic health evaluation | plasma albumin(g/l) | Ph | Glasgow coma scale | Serum BUNmg/dl | Serum glucose(mg/dl) | total APACHE III score | Outcome |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | 60 | male | duodenal perforation | 37.6 | 130bpm | 75 | 1.8 | 300 | 1.8 | 28 | 7600 | 38 | 138 | 90 | none | 1.7 | 7.2 | 6/15 | 92 | 91 | 125 | mortality |
| 2 | 60 | male | duodenal perforation | 37.8 | 110bpm | 65 | 2 | 800 | 3 | 28 | 3330 | 32 | 140 | 78 | none | 1.8 | 7.2 | 8/15 | 12 | 90 | 88 | mortality |
| 3 | 68 | male | ileal perforation | 37.6 | 106bpm | 75 | 2.4 | 700 | 1.6 | 32 | 3500 | 40 | 130 | 76 | none | 3.3 | 7.1 | 6/15 | 49 | 131 | 82 | mortality |
| 4 | 35 | male | prepyloric perforation | 37.4 | 110bpm | 65 | 2.6 | 600 | 6.8 | 29 | 35790 | 24 | 129 | 72 | none | 1.8 | 7.1 | 6/15 | 80 | 90 | 87 | mortality |
| 5 | 60 | male | duodenal perforation | 37.6 | 110bpm | 75 | 2.5 | 900 | 1.6 | 26 | 4270 | 49 | 139 | 84 | none | 3.7 | 7.2 | 8/15 | 80 | 99 | 69 | survived |
| 6 | 28 | female | ileal perforation | 37.5 | 110bpm | 75 | 2.6 | 600 | 9.1 | 32 | 12800 | 35 | 116 | 74 | none | 2.1 | 7.1 | 9/15 | 255 | 178 | 65 | survived |
| 7 | 95 | male | duodenal perforation | 37.5 | 130bpm | 65 | 1.1 | 900 | 1.9 | 26 | 9620 | 42 | 142 | 88 | none | 1.8 | 7.3 | 9/15 | 70 | 137 | 73 | survived |
| 8 | 58 | male | prepyloric perforation | 37.6 | 120bpm | 70 | 2 | 700 | 1.8 | 30 | 16600 | 38 | 141 | 89 | none | 1.8 | 7.3 | 11/15 | 90 | 146 | 66 | mortality |
| 9 | 45 | male | duodenal perforation | 37.3 | 140bpm | 55 | 1.3 | 400 | 8 | 32 | 7180 | 35 | 159 | 89 | none | 1.8 | 7.4 | 13/15 | 53 | 90 | 85 | mortality |
| 10 | 60 | female | gastric perforation | 37.6 | 130bpm | 70 | 1.2 | 600 | 2.6 | 30 | 16170 | 36 | 145 | 66 | none | 1.8 | 7.2 | 9/15 | 66 | 140 | 85 | mortality |
| 11 | 65 | male | ileal perforation | 37.5 | 100bpm | 78 | 1.8 | 2000 | 2.4 | 12 | 6000 | 54 | 136 | 85 | none | 2 | 7.2 | 13/15 | 65 | 84 | 68 | mortality |
| 12 | 55 | female | duodenal perforation | 37.6 | 110bpm | 75 | 4.3 | 900 | 0.9 | 28 | 5450 | 33 | 128 | 86 | none | 1.8 | 7.3 | 11/15 | 75 | 78 | 52 | mortality |
| 13 | 60 | male | duodenal perforation | 37.7 | 106bpm | 80 | 2 | 700 | 3.9 | 28 | 7430 | 30 | 128 | 88 | none | 1.8 | 7.5 | 13/15 | 60 | 110 | 53 | mortality |
| 14 | 83 | female | duodenal perforation | 37.6 | 116bpm | 72 | 3.3 | 750 | 1.9 | 38 | 7400 | 55 | 128 | 90 | none | 3.3 | 7.4 | 13/15 | 55 | 198 | 76 | mortality |
| 15 | 25 | female | duodenal perforation | 37.4 | 126bpm | 75 | 3.6 | 600 | 2.3 | 26 | 2000 | 29 | 132 | 80 | none | 2.2 | 7.1 | 9/15 | 75 | 50 | 98 | mortality |
| 16 | 65 | male | prepyloric perforation | 37.5 | 112bpm | 71 | 2.2 | 550 | 3.2 | 34 | 11400 | 44 | 130 | 78 | none | 2.5 | 7.2 | 9/15 | 58 | 205 | 71 | mortality |
| 17 | 70 | male | duodenal perforation | 37.6 | 122bpm | 76 | 2.8 | 600 | 1.8 | 34 | 12600 | 44 | 130 | 74 | none | 3.2 | 7.3 | 11/15 | 65 | 220 | 90 | mortality |
| 18 | 60 | male | ileal perforation | 37.5 | 108bpm | 76 | 1.4 | 1200 | 1.8 | 36 | 18600 | 55 | 138 | 76 | none | 2.6 | 7.1 | 13/15 | 66 | 180 | 73 | mortality |
| 19 | 60 | male | gastric perforation | 37.7 | 110bpm | 100 | 2.5 | 400 | 5.7 | 24 | 2290 | 35 | 131 | 90 | cirrhosis | 1.9 | 7.3 | 9/15 | 172 | 83 | 79 | mortality |

| | | | | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 20 | 52 | male | duodenal perforation | 37.6 | 122bpm | 75 | 2.2 | 600 | 3.7 | 25 | 6678 | 38 | 142 | 75 | none | 1.8 | 7.2 | 11/15 | 95 | 110 | 78 | mortality |
| 21 | 68 | male | ileal perforation | 37.6 | 132bpm | 65 | 2.1 | 700 | 3.5 | 32 | 6500 | 40 | 133 | 75 | none | 2 | 7.3 | 9/15 | 75 | 90 | 69 | survived |
| 22 | 55 | male | prepyloric perforation | 37.6 | 122bpm | 70 | 1.8 | 600 | 2.6 | 25 | 8500 | 42 | 145 | 85 | none | 1.6 | 7.2 | 11/15 | 90 | 110 | 80 | survived |
| 23 | 60 | male | ileal perforation | 37.7 | 110bpm | 75 | 2.2 | 800 | 3 | 22 | 3500 | 40 | 132 | 70 | none | 2.6 | 7.1 | 9/15 | 90 | 100 | 74 | mortality |
| 24 | 60 | male | duodenal perforation | 37.4 | 122bpm | 65 | 2 | 400 | 4 | 32 | 6600 | 42 | 122 | 65 | none | 1.6 | 7.1 | 6/15 | 120 | 100 | 93 | mortality |
| 25 | 33 | male | duodenal perforation | 37.8 | 126bpm | 55 | 2.4 | 500 | 3.5 | 28 | 5500 | 38 | 140 | 75 | none | 1.8 | 7.2 | 9/15 | 100 | 130 | 106 | mortality |
| 26 | 66 | female | ileal perforation | 37.6 | 130bpm | 80 | 1.1 | 1500 | 1.1 | 36 | 12400 | 48 | 128 | 86 | none | 4.4 | 7.3 | 15/15 | 42 | 140 | 46 | survived |
| 27 | 40 | female | duodenal perforation | 37.4 | 94bpm | 83 | 1 | 1400 | 0.9 | 32 | 7600 | 46 | 133 | 90 | none | 4.8 | 7.3 | 15/15 | 45 | 170 | 33 | survived |
| 28 | 66 | female | gastric perforation | 37.7 | 126bpm | 80 | 1.1 | 1700 | 0.9 | 32 | 12400 | 48 | 132 | 92 | none | 4.4 | 7.3 | 15/15 | 44 | 160 | 43 | survived |
| 29 | 30 | female | appendicular perforation | 37.7 | 108bpm | 100 | 1.1 | 1800 | 1 | 32 | 13200 | 49 | 131 | 92 | none | 4.6 | 7.3 | 15/15 | 48 | 150 | 32 | survived |
| 30 | 40 | female | duodenal perforation | 37.5 | 98bpm | 80 | 1.1 | 1600 | 0.9 | 28 | 14600 | 47 | 128 | 86 | none | 4.1 | 7.3 | 15/15 | 48 | 210 | 26 | survived |
| 31 | 62 | male | ileal perforation | 37.7 | 106bpm | 90 | 0.9 | 1100 | 1.4 | 26 | 12600 | 39 | 146 | 92 | none | 3.2 | 7.4 | 15/15 | 53 | 115 | 34 | survived |
| 32 | 60 | male | ileal perforation | 37.4 | 98bpm | 70 | 0.9 | 1300 | 2.4 | 26 | 15900 | 40 | 142 | 92 | none | 2.8 | 7.4 | 15/15 | 55 | 103 | 43 | survived |
| 33 | 55 | male | appendicular perforation | 37.7 | 110bpm | 80 | 0.9 | 900 | 1.4 | 26 | 9030 | 48 | 130 | 91 | none | 2.2 | 7.4 | 15/15 | 60 | 90 | 40 | survived |
| 34 | 32 | male | appendicular perforation | 37.6 | 90bpm | 88 | 0.9 | 1100 | 0.9 | 28 | 11600 | 38 | 141 | 92 | none | 3.8 | 7.4 | 15/15 | 45 | 90 | 22 | survived |
| 35 | 40 | female | duodenal perforation | 37.6 | 110bpm | 80 | 1 | 1300 | 0.6 | 26 | 14380 | 28 | 131 | 95 | none | 3 | 7.4 | 15/15 | 19 | 71 | 20 | survived |
| 36 | 53 | female | gastric perforation | 37.7 | 110bpm | 80 | 1 | 900 | 1.1 | 24 | 9800 | 36 | 133 | 91 | none | 4.2 | 7.4 | 15/15 | 29 | 116 | 24 | survived |
| 37 | 42 | male | ileal perforation | 37.6 | 116bpm | 88 | 0.9 | 1700 | 1.8 | 28 | 13860 | 49 | 133 | 88 | none | 4.3 | 7.4 | 15/15 | 58 | 205 | 41 | survived |
| 38 | 42 | female | appendicular perforation | 37.6 | 80bpm | 80 | 0.9 | 1450 | 1.6 | 26 | 13300 | 49 | 134 | 90 | none | 4.8 | 7.4 | 15/15 | 44 | 170 | 32 | survived |
| 39 | 66 | male | appendicular perforation | 37.8 | 88bpm | 125 | 1.2 | 2500 | 1 | 24 | 14220 | 48 | 138 | 73 | none | 4 | 7.3 | 15/15 | 42 | 110 | 34 | survived |
| 40 | 65 | male | prepyloric perforation | 37.6 | 82bpm | 80 | 0.4 | 2000 | 0.7 | 24 | 14250 | 38 | 136 | 73 | none | 3 | 7.4 | 15/15 | 23 | 148 | 44 | survived |
| 41 | 65 | male | prepyloric perforation | 37.5 | 108bpm | 90 | 0.8 | 1700 | 2.8 | 36 | 7400 | 48 | 138 | 92 | none | 3.8 | 7.4 | 15/15 | 48 | 110 | 46 | survived |
| 42 | 45 | male | ileal perforation | 37.7 | 110bpm | 65 | 1.6 | 1000 | 1.5 | 26 | 4460 | 54 | 133 | 92 | none | 4.2 | 7.4 | 15/15 | 51 | 128 | 48 | survived |
| 43 | 55 | female | appendicular perforation | 37.6 | 92bpm | 105 | 0.8 | 1400 | 1.1 | 22 | 6300 | 48 | 141 | 96 | none | 3.9 | 7.4 | 15/15 | 23 | 126 | 21 | survived |
| 44 | 31 | female | appendicular perforation | 37.6 | 90bpm | 80 | 1 | 1400 | 2.1 | 24 | 8500 | 38 | 148 | 94 | none | 3.6 | 7.4 | 15/15 | 21 | 102 | 21 | survived |
| 45 | 55 | female | prepyloric perforation | 37.6 | 98bpm | 88 | 2 | 900 | 1 | 22 | 6900 | 39 | 136 | 90 | none | 3.5 | 7.4 | 15/15 | 20 | 112 | 22 | survived |

| | | | | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 46 | 55 | female | prepyloric perforation | 37.7 | 98bpm | 85 | 2 | 1000 | 0.6 | 26 | 5800 | 33 | 134 | 94 | none | 1.6 | 7.4 | 15/15 | 14 | 90 | 34 | survived |
| 47 | 50 | female | gastric perforation | 37.5 | 110bpm | 80 | 1.4 | 900 | 1 | 28 | 19860 | 37 | 132 | 96 | none | 3.5 | 7.4 | 15/15 | 67 | 93 | 34 | survived |
| 48 | 56 | male | ileal perforation | 37.4 | 100bpm | 80 | 1 | 900 | 1.5 | 26 | 10620 | 49 | 141 | 92 | none | 2.8 | 7.4 | 15/15 | 50 | 90 | 32 | survived |
| 49 | 60 | female | duodenal perforation | 37.8 | 100bpm | 75 | 0.7 | 1400 | 1.4 | 28 | 2300 | 19 | 133 | 84 | none | 2.6 | 7.5 | 15/15 | 49 | 92 | 47 | survived |
| 50 | 45 | female | appendicular perforation | 37.4 | 90bpm | 80 | 0.8 | 1200 | 0.9 | 26 | 12690 | 20 | 141 | 96 | none | 3.6 | 7.4 | 15/15 | 60 | 80 | 27 | survived |
| 51 | 69 | male | duodenal perforation | 37.6 | 90bpm | 90 | 0.5 | 1100 | 0.9 | 18 | 2890 | 43 | 141 | 85 | none | 2.4 | 7.3 | 15/15 | 74 | 55 | 45 | survived |
| 52 | 60 | male | gastric perforation | 37.4 | 102bpm | 85 | 1 | 1500 | 1.7 | 22 | 4770 | 46 | 141 | 60 | none | 2 | 7.3 | 15/15 | 106 | 84 | 63 | survived |
| 53 | 70 | male | gastric perforation | 37.5 | 86bpm | 83 | 0.8 | 550 | 3.1 | 23 | 17630 | 37 | 130 | 94 | none | 2.9 | 7.2 | 15/15 | 102 | 88 | 63 | survived |
| 54 | 80 | male | prepyloric perforation | 37.8 | 110bpm | 80 | 1 | 1600 | 3.3 | 30 | 18600 | 48 | 133 | 90 | none | 4.2 | 7.4 | 15/15 | 55 | 210 | 58 | survived |
| 55 | 42 | male | appendicular perforation | 37.6 | 120bpm | 80 | 0.6 | 1950 | 0.8 | 22 | 15870 | 41 | 144 | 90 | none | 2.5 | 7.4 | 13/15 | 81 | 110 | 55 | survived |
| 56 | 55 | female | gastric perforation | 37.3 | 104bpm | 80 | 6.5 | 1100 | 2.2 | 28 | 22340 | 28 | 138 | 92 | none | 2 | 7.4 | 15/15 | 88 | 110 | 51 | survived |
| 57 | 60 | female | duodenal perforation | 37.6 | 100bpm | 75 | 0.5 | 1000 | 0.9 | 26 | 18000 | 38 | 131 | 90 | none | 1.7 | 7.3 | 15/15 | 24 | 110 | 58 | survived |
| 58 | 22 | male | appendicular perforation | 37.8 | 86bpm | 90 | 12 | 1100 | 1.6 | 22 | 8350 | 21 | 140 | 96 | none | 1.9 | 7.5 | 15/15 | 74 | 110 | 52 | survived |
| 59 | 45 | male | appendicular perforation | 37.4 | 106bpm | 75 | 1 | 900 | 3.1 | 26 | 4640 | 38 | 126 | 96 | none | 2 | 7.3 | 15/15 | 125 | 238 | 61 | survived |
| 60 | 80 | female | ileal perforation | 37.8 | 90bpm | 75 | 0.8 | 900 | 1 | 28 | 6600 | 39 | 139 | 92 | none | 3 | 7.5 | 15/15 | 65 | 125 | 52 | survived |
| 61 | 65 | female | prepyloric perforation | 37.4 | 95bpm | 80 | 0.9 | 1400 | 2.4 | 27 | 9220 | 28 | 134 | 94 | none | 2.1 | 7.4 | 15/15 | 80 | 110 | 51 | survived |
| 62 | 60 | male | duodenal perforation | 37.7 | 126bpm | 75 | 0.6 | 600 | 2 | 24 | 12110 | 22 | 139 | 96 | none | 2.7 | 7.4 | 15/15 | 118 | 60 | 50 | survived |
| 63 | 50 | male | ileal perforation | 37.8 | 110bpm | 75 | 1 | 800 | 1.7 | 26 | 2400 | 46 | 139 | 92 | none | 2.5 | 7.4 | 15/15 | 48 | 138 | 52 | survived |
| 64 | 32 | female | appendicular perforation | 37.5 | 120bpm | 55 | 0.8 | 700 | 0.8 | 28 | 23340 | 34 | 121 | 82 | none | 2.5 | 7.5 | 13/15 | 60 | 110 | 58 | survived |
| 65 | 55 | male | duodenal perforation | 37.5 | 100bpm | 80 | 1 | 1500 | 0.8 | 26 | 8530 | 47 | 137 | 96 | none | 2.5 | 7.4 | 15/15 | 86 | 128 | 28 | mortality |
| 66 | 22 | male | appendicular perforation | 37.2 | 100bpm | 80 | 1.1 | 1000 | 2.1 | 26 | 6930 | 51 | 139 | 98 | none | 2.5 | 7.2 | 13/15 | 47 | 88 | 52 | against medical advice |
| 67 | 33 | female | appendicular perforation | 37.8 | 110bpm | 85 | 0.8 | 900 | 0.6 | 30 | 9550 | 27 | 135 | 97 | none | 3.8 | 7.4 | 15/15 | 40 | 130 | 27 | against medical advice |
| 68 | 56 | male | gastric perforation | 37.6 | 90bpm | 90 | 2 | 1400 | 1.3 | 22 | 11000 | 38 | 143 | 93 | none | 3.6 | 7.4 | 15/15 | 60 | 110 | 26 | survived |
| 69 | 42 | male | prepyloric perforation | 37.7 | 110bpm | 85 | 0.8 | 1300 | 1.6 | 26 | 14600 | 38 | 141 | 97 | none | 2.5 | 7.4 | 15/15 | 30 | 90 | 27 | survived |
| 70 | 35 | female | appendicular perforation | 37.5 | 90bpm | 85 | 2 | 1100 | 0.6 | 24 | 1500 | 27 | 138 | 94 | none | 2 | 7.4 | 15/15 | 25 | 90 | 28 | survived |
| 71 | 35 | male | appendicular perforation | 37.8 | 106bpm | 85 | 0.8 | 1400 | 1.6 | 26 | 19100 | 39 | 137 | 96 | none | 3.9 | 7.4 | 15/15 | 55 | 193 | 27 | survived |

| | | | | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 72 | 39 | male | duodenal perforation | 37.8 | 116bpm | 88 | 0.9 | 1700 | 1.8 | 28 | 13860 | 49 | 133 | 88 | none | 4.3 | 7.4 | 15/15 | 58 | 205 | 41 | survived |
| 73 | 22 | male | appendicular perforation | 37.6 | 90bpm | 90 | 0.5 | 1100 | 0.9 | 18 | 2890 | 43 | 141 | 85 | none | 2.4 | 7.3 | 15/15 | 74 | 55 | 45 | survived |
| 74 | 31 | male | appendicular perforation | 37.6 | 120bpm | 80 | 0.6 | 1950 | 0.8 | 22 | 15870 | 41 | 144 | 90 | none | 2.5 | 7.4 | 13/15 | 81 | 110 | 55 | survived |
| 75 | 35 | male | appendicular perforation | 37.4 | 95bpm | 80 | 0.9 | 1400 | 2.4 | 27 | 9220 | 28 | 134 | 94 | none | 2.1 | 7.4 | 15/15 | 80 | 110 | 51 | survived |
| 76 | 50 | male | gastric perforation | 37.6 | 90bpm | 88 | 0.9 | 1100 | 0.9 | 28 | 11600 | 38 | 141 | 92 | none | 3.8 | 7.4 | 15/15 | 45 | 90 | 22 | survived |
| 77 | 32 | female | duodenal perforation | 37.6 | 110bpm | 80 | 1 | 1300 | 0.6 | 26 | 14380 | 28 | 131 | 95 | none | 3 | 7.4 | 15/15 | 19 | 71 | 20 | survived |
| 78 | 55 | male | gastric perforation | 37.7 | 110bpm | 80 | 1 | 900 | 1.1 | 24 | 9800 | 36 | 133 | 91 | none | 4.2 | 7.4 | 15/15 | 29 | 116 | 24 | survived |
| 79 | 22 | male | duodenal perforation | 37.8 | 110bpm | 85 | 0.8 | 900 | 0.6 | 30 | 9550 | 27 | 135 | 97 | none | 3.8 | 7.4 | 15/15 | 40 | 130 | 27 | survived |
| 80 | 33 | female | gastric perforation | 37.6 | 90bpm | 90 | 2 | 1400 | 1.3 | 22 | 11000 | 38 | 143 | 93 | none | 3.6 | 7.4 | 15/15 | 60 | 110 | 26 | survived |
| 81 | 56 | male | duodenal perforation | 37.7 | 110bpm | 85 | 0.8 | 1300 | 1.6 | 26 | 14600 | 38 | 141 | 97 | none | 2.5 | 7.4 | 15/15 | 30 | 90 | 27 | survived |
| 82 | 42 | male | gastric perforation | 37.5 | 90bpm | 85 | 2 | 1100 | 0.6 | 24 | 1500 | 27 | 138 | 94 | none | 2 | 7.4 | 15/15 | 25 | 90 | 28 | survived |
| 83 | 35 | female | duodenal perforation | 37.8 | 106bpm | 85 | 0.8 | 1400 | 1.6 | 26 | 19100 | 39 | 137 | 96 | none | 3.9 | 7.4 | 15/15 | 55 | 193 | 27 | survived |
| 84 | 35 | male | gastric perforation | 37.6 | 90bpm | 88 | 0.9 | 1100 | 0.9 | 28 | 11600 | 38 | 141 | 92 | none | 3.8 | 7.4 | 15/15 | 45 | 90 | 22 | survived |
| 85 | 39 | male | duodenal perforation | 37.6 | 110bpm | 80 | 1 | 1300 | 0.6 | 26 | 14380 | 28 | 131 | 95 | none | 3 | 7.4 | 15/15 | 19 | 71 | 20 | survived |
| 86 | 22 | male | appendicular perforation | 37.7 | 110bpm | 80 | 1 | 900 | 1.1 | 24 | 9800 | 36 | 133 | 91 | none | 4.2 | 7.4 | 15/15 | 29 | 116 | 24 | survived |
| 87 | 65 | male | ileal perforation | 37.5 | 100bpm | 78 | 1.8 | 2000 | 2.4 | 12 | 6000 | 54 | 136 | 85 | none | 2 | 7.2 | 13/15 | 65 | 84 | 68 | survived |
| 88 | 55 | female | duodenal perforation | 37.6 | 110bpm | 75 | 4.3 | 900 | 0.9 | 28 | 5450 | 33 | 128 | 86 | none | 1.8 | 7.3 | 11/15 | 75 | 78 | 52 | survived |
| 89 | 60 | male | duodenal perforation | 37.7 | 106bpm | 80 | 2 | 700 | 3.9 | 28 | 7430 | 30 | 128 | 88 | none | 1.8 | 7.5 | 13/15 | 60 | 110 | 53 | survived |
| 90 | 83 | female | duodenal perforation | 37.6 | 116bpm | 72 | 3.3 | 750 | 1.9 | 38 | 7400 | 55 | 128 | 90 | none | 3.3 | 7.4 | 13/15 | 55 | 198 | 76 | survived |
| 91 | 25 | female | duodenal perforation | 37.4 | 126bpm | 75 | 3.6 | 600 | 2.3 | 26 | 2000 | 29 | 132 | 80 | none | 2.2 | 7.1 | 9/15 | 75 | 50 | 98 | survived |
| 92 | 65 | male | prepyloric perforation | 37.5 | 112bpm | 71 | 2.2 | 550 | 3.2 | 34 | 11400 | 44 | 130 | 78 | none | 2.5 | 7.2 | 9/15 | 58 | 205 | 71 | survived |
| 93 | 70 | male | duodenal perforation | 37.6 | 122bpm | 76 | 2.8 | 600 | 1.8 | 34 | 12600 | 44 | 130 | 74 | none | 3.2 | 7.3 | 11/15 | 65 | 220 | 90 | survived |
| 94 | 60 | male | ileal perforation | 37.5 | 108bpm | 76 | 1.4 | 1200 | 1.8 | 36 | 18600 | 55 | 138 | 76 | none | 2.6 | 7.1 | 13/15 | 66 | 180 | 73 | survived |
| 95 | 60 | male | gastric perforation | 37.7 | 110bpm | 100 | 2.5 | 400 | 5.7 | 24 | 2290 | 35 | 131 | 90 | cirrhosis | 1.9 | 7.3 | 9/15 | 172 | 83 | 79 | survived |
| 96 | 52 | male | duodenal perforation | 37.6 | 122bpm | 75 | 2.2 | 600 | 3.7 | 25 | 6678 | 38 | 142 | 75 | none | 1.8 | 7.2 | 11/15 | 95 | 110 | 78 | survived |
| 97 | 68 | male | ileal perforation | 37.6 | 132bpm | 65 | 2.1 | 700 | 3.5 | 32 | 6500 | 40 | 133 | 75 | none | 2 | 7.3 | 9/15 | 75 | 90 | 69 | survived |
| 98 | 55 | male | prepyloric perforation | 37.6 | 122bpm | 70 | 1.8 | 600 | 2.6 | 25 | 8500 | 42 | 145 | 85 | none | 1.6 | 7.2 | 11/15 | 90 | 110 | 80 | survived |

| | | | | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 99 | 60 | male | ileal perforation | 37.7 | 110bpm | 75 | 2.2 | 800 | 3 | 22 | 3500 | 40 | 132 | 70 | none | 2.6 | 7.1 | 9/15 | 90 | 100 | 74 | survived |
| 100 | 60 | male | duodenal perforation | 37.4 | 122bpm | 65 | 2 | 400 | 4 | 32 | 6600 | 42 | 122 | 65 | none | 1.6 | 7.1 | 6/15 | 120 | 100 | 93 | survived |