

Evaluation of prognosis in patients' with perforation peritonitis using Mannheim's peritonitis index

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Abstract- Peritonitis is defined as inflammation of the serous membrane that lines the abdominal cavity and the organs contained therein. Most cases of peritonitis are caused by an invasion of the peritoneal cavity by bacteria, so that when the term peritonitis is used without specification bacterial peritonitis is implied. Peritonitis is one of the most common infections, and an important problem that a surgeon has to face.

Index Terms- Perforation peritonitis, Mannheims' peritonitis index, prognosis

I. INTRODUCTION

Despite the surgical treatment, sophisticated intensive care units, last generation antibiotics and a better understanding of pathophysiology, the mortality rate of perforation peritonitis are still high. The outcome of an abdominal infection depends on the complex interaction of many different factors and the success obtained with the early institution of specific therapeutic procedures. It also depends upon the exact recognition of the seriousness of the diseases and an accurate assessment and classification of the patients risks. Early prognostic evaluation of peritonitis is desirable to select high risk patients for more aggressive therapeutic procedure such as radical debridement, lavage system, open management and planned relaparotomy. An accurate risk index classification is the only way to settle a standard of comparison between group of patients and different treatment methods which would allow prospective adequate comparative studies.

There is no single, easily available laboratory test that predicts severity or prognosis in patients with peritonitis. Despite controversial discussion, there is agreement that prospective controlled clinical trials are necessary in the field of intra abdominal infections. Randomised controlled clinical trials are the preferred methods for comparing clinical efficacy of treatment strategies. They remain a vital bridge between advances in basic science on one hand and improvement in health care on the other. Therefore all measures should be undertaken to perform clinical trials with a high quality in this field. Many of the problems such as terminology and definitions, assessment of severity of disease, case selection, and exclusion of patients, confounding factors, end point, and generalised ability of a study result can be dealt adequately with the help of

scoring systems. Scoring systems have been advocated as prognostic predictors, they reduce all the clinical problems including lots of variables to a simple number¹.

Reproducible scoring system that allow a surgeon to determine the severity of intra-abdominal infections are essential to rectify the effectiveness of different treatment regimens, to scientifically compare surgical intensive care units, to select a more aggressive surgical approach for high risk patients and to able to inform patient's relatives with greater objectivity. The results of treatment for peritonitis are especially difficult to evaluate because these patients may correspond to various aetiologies, treatments differ and there is a lack of universally valid criteria and definitions. Identifying both prognostic factors and severity scales that provide objective description of the patient condition at specific points such as the preoperative and postoperative period is useful to improve our understanding of the problem involved¹.

Intra-abdominal infections and secondary peritonitis are a frequently encountered surgical emergency in tropical countries. The spectrum of perforation peritonitis in India continues to be different from its western counterparts. In India, the most commonly affected population is the young men in the prime of their life as compared to the west where the mean age for the occurrence of perforation peritonitis is usually 45-60 yrs. In majority of cases in tropical countries like India the presentation to the hospital is late with established generalized peritonitis with faecal or purulent contamination and varying degree of septicaemia. In India perforations of the proximal gastrointestinal tract were more common as compared to the distal ones.

Despite advances, mortality from many forms of intra-abdominal infection remains unacceptably high. Substantial differences between conventional & more recently developed therapy has been found in randomized prospective studies. It has become apparent that approaches for managing patient profoundly ill from intra-abdominal infection require further critical review at that new methods for analyzing the results of various therapeutic intervention must be found.

With this background an international congress on intra abdominal infection was organized in Hamburg in 1987, supported by the Surgical Infection Society (SIS) & the Paul Ehrlich society. Surgeons from all continents came to review the current status of definition as well as statistical techniques &

severity of illness scoring system, to allow for more sophisticated analysis of results.

The following classification of peritonitis is considered as a standard -

1. Primary Peritonitis

- A. Spontaneous peritonitis of childhood
- B. Spontaneous peritonitis of adults
- C. Peritonitis in patients with CAPD (continuous ambulatory peritoneal dialysis)
- D. Tubercular peritonitis

2. Secondary Peritonitis (Acute Suppurative)

A. Perforation peritonitis (spontaneous acute)

- 1. GIT perforation
- 2. Bowel wall necrosis
- 3. Pelvipertonitis
- 4. Peritonitis after translocation of bacteria

B. Postoperative Peritonitis

- 1. Leak of an anastomosis
- 2. Leak of suture line
- 3. Stump insufficiency.

Despite controversial discussion there is agreement that multicentre prospective controlled clinical trials are necessary in the field of intra-abdominal infection. Randomized controlled clinical trials are the preferred scientific methods for comparing the clinical efficacy of treatment strategy. They remain the vital bridge between advances in basic science on the one hand and improvement in health care on the other. Therefore all measure should be undertaken to perform clinical trials with a high quality in this field. Many of these problem areas such as terminology and definitions, assessment of severity of disease, case selection,

exclusion of patients, confounding factors, end points and generalizability of study results can be dealt with adequately with scoring systems. Clinical study in intra abdominal infection can be improved considerably by incorporation of scoring systems. Scoring systems assessing severity of disease can help to support comparison between results of different studies or centres².

Various scoring systems have been used to indicate prognosis of patients with peritonitis. These scores can be broadly divided into two groups:

A) Disease independent scores for evaluation of serious patients;

- APACHE II score
- simplified acute physiology score (SAPS II)
- sepsis severity score
- multiple organ dysfunction score

B) Peritonitis specific score;

- Mannheim Peritonitis Score (MPI)
- Peritonitis index altona II
- left colonic perforation score.

II. MANNHEIM PERITONITIS INDEX

It was developed by **Wacha and Linder**³ in 1983.

It was developed based on the retrospective analysis of data from 1253 patients with peritonitis in which 20 possible risk factors were considered. Of these 20 factors, only 8 were proved to be of prognostic relevance and were entered into **MANNHEIM PERITONITIS INDEX**. These factors were classified according to their predictive power.

MANNHEIM PERITONITIS INDEX SCORE

Study Variable	Adverse factor	Points	Favorable factor	Points
1.Age	>50 yrs	5	< 50 years	0
2.Sex	Female	5		0
3.Organ Failure	Present	7		0
4.Malignancy	Present	4		0
5.Evolution time	>24 hrs	4		0
6.Origin of sepsis	Non-colonic	4		0
7 Extension of peritonitis	Generalized	6		0
8.Character of exudate	Purulent Fecal	6 12	Clear	0

Maximal possible score is 47 and minimal possible score is zero. Patients were divided in three categories according to **MPI** score:

- 1. Score less than equal to 21
- 2. Score between 21 to 29
- 3. Score equal to greater than 29.

The MPI appears to be more practical than other scoring system, such as the APACHE II, which is time consuming and may be impossible to apply in the setting of intra-abdominal

sepsis. Also in a multicentre study of 2003 patients, the MPI had an acceptable specificity and sensitivity.

Much has been said and published about peritonitis but a consolidated analytical study of peritonitis and peritonitis grading scale is not found.

The secondary peritonitis being a common problem with a high mortality and morbidity rate made us interested in conducting the study. Gastric and duodenal perforations have been included in the present study.

No claim for originality can be advanced because similar works has been published, but if it can add a trifling bit to existing knowledge on this subject and its management, labour will be rewarded.

The present thesis is only a part of the project, in an attempt to fill up the gap mentioned earlier. The scope is indefinite and the greatest gain in research can come from its scope being indefinite. At the same time to the every inch that was there, there may be acres of waste.

In the end, we can only say that in the golden age of surgery, mortality from peritonitis is still a challenge to surgeon resplendent with brilliant achievements. Much has been learnt about the diagnosis and treatment of this catastrophe, but there is often more to learn. Still we should not be stressing the "What's new", until we have mastered the "What's old".

III. AIMS AND OBJECTIVES

1. To study the spectrum of perforation peritonitis.
2. To study the prognostic factors in perforation peritonitis.
3. To evaluate the outcome of patients with perforation peritonitis using **MANNHEIM PERITONITIS INDEX**.
4. To evaluate the **MANNHEIM PERITONITIS INDEX** to allow identification of high risk patients.
5. To confirm the predictive value of the **MANNHEIM PERITONITIS INDEX**.
6. To identify the patients using **MANNHEIM PERITONITIS INDEX** for intensive post-operative or ICU care.
7. Role of **MANNHEIM PERITONITIS INDEX** in decision making regarding surgical intervention in peritonitis.

IV. MATERIALS AND METHODS

STUDY DESIGN

"Evaluation of prognosis in patient's with peritonitis using Mannheim's peritonitis index" for approximate time period from 2009 to 2011.

SELECTION OF CASES

From cases attending our institute in which diagnosis of peritonitis is established by operative findings or surgical interventions during management. Therefore nonrandomized sampling technique was used.

INCLUSION CRITERIA

Cases of peritonitis secondary to hollow viscous perforation attending our institute in the study period were included in the study.

EXCLUSION CRITERIA

- Patients absconded or discharged against medical advice (DAMA) during hospital admission.
- All patients with primary peritonitis (Spontaneous bacterial peritonitis)

- All patients with tertiary peritonitis - Patients with peritonitis due to anastomotic dehiscence or leak
- Patients with acute appendicitis (without perforation)
- Diagnosis was made by a combination of history, clinical examination and on the basis of the reports of the radiological examinations after which the patients is posted for emergency laparotomy.
- Once the diagnosis of peritonitis was confirmed by the operative findings of the patients, the patients were accepted for the study.

The following parameters were recorded meticulously for the calculation of the Mannheim Peritonitis Index :

1. Age
2. Sex
3. Organ Failure
 - ❖ The criteria which were used for the presence of organ failure are as follows Published by *Deitch*⁴ (1992):
 - **Renal failure:** serum creatinine >177mmol / L (> 2 mg/dl) or serum urea >16.7 mmol/L (>46.78 mg/dl) {conversion factor is 88.40 and 0.3570 respectively} or oliguria < 20 ml/ hour.
 - **Shock:** Hypotension is defined as a systolic BP of <90 mmHg or a reduction of >40 mmHg from baseline, in the absence of other causes for the fall in blood pressure.
 - **Intestinal obstruction** (only if profound): paralysis >24 hours or complete mechanical ileus.
 - **Respiratory failure:** pO₂ <50 mmHg or pCO₂ >50 mmHg.

4. Malignancy

Patients with known malignancy or with features of malignancy on gross examination e.g. malignant gastric perforations, perforation of a colonic growth suspicious of malignancy, perforation of proximal bowel due to distal obstruction by malignant growth on gross examination were included in the study.

5. Evolution time

Patients were divided into two groups (<24 hour / >24 hour) on the basis of history and timing of surgery.

6. Origin of sepsis (colonic / noncolonic)

This parameter is recorded on the basis of findings of laparotomy.

7. Extension of peritonitis (Diffuse/ localized)

8. Character of exudates or peritoneal fluid

- a) Clear
- b) Cloudy/purulent
- c) Faecal

Bilious collections in cases of recent perforation without superadded infection were grouped as clear. Serohaemorrhagic collection of recent origin is taken as clear in traumatic peritonitis.

The individual score of each parameter is added to calculate Mannheim peritonitis index score of each case. Patients were divided into three categories according to the score:

1. Score less than 21.
2. Score between 21 to 29.
3. Score more than 29.

V. REVIEW OF LITERATURE

HISTORICAL REVIEW^{5,6}

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 Hertzler⁵ (1919), "Infections of the peritoneum" by Steinberg(1944), and reviews by Hedberg and Welch and Hauet al⁶. 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.

The benign gastro-duodenal perforation is more common in the males than the females, the ratio being 10:1(Bailey & Love). It is more common in winter than summer (Turner 1951). It is much less common in children than the adults and the clinical features are less dramatic than that in adults (Bell, 1953). Peptic ulcer complications are rare in pregnant women (Sandweiss et al, 1943). Commonly these perforations occur in the afternoon between 3 pm and 6 pm (Illingworth et al 1944, Jamieeson 1955).

ANATOMY OF PERITONEUM AND PERITONEAL CAVITY^{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.

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.

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.

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 parietal 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.

The peritoneal cavity is subdivided into interconnected compartments or spaces by 11 ligaments and mesenteries. The peritoneal ligament and mesenteries includes the coronary, gastrohepatic, hepatoduodenal, falciform, gastrocolic, duodenocolic, gastrosplenic, splenorenal, and phrenocolic ligaments and the transverse mesocolon and small bowel mesentery.

These structures partition the abdomen into nine potential spaces viz.

1. Right subphrenic
2. Left subphrenic
3. Subhepatic
4. Supramesenteric
5. Inframesenteric
6. Right paracolic gutter
7. Left paracolic gutter
8. Pelvis
9. Lesser sac

This compartmentalization directs the circulation of fluid in the peritoneal cavity and thus may be useful in predicting the route of spread of infection and malignant diseases. For example; Perforation of the duodenum from peptic ulcer disease may result in the movement of the fluid (and the development of abscesses) in the subhepatic space, the right paracolic gutter and the pelvis.

FUNCTIONS OF PERITONIUUM

The peritoneal membrane provides lubrication for the loops of intestine by secreting a highly viscous fluid.

The mesothelial cells are also able to secrete lytic enzymes, prostaglandins, interferons and lymphokines some of which probably discourages infection.

PERITONEAL PHYSIOLOGY¹⁰

The peritoneum is a bidirectional, semi permeable membrane that controls the amount of fluid within the peritoneal cavity, promotes the sequestration and removal of bacteria from the peritoneal cavity, promotes the sequestration and removal of bacteria from the peritoneal cavity, and facilitates the migration of inflammatory cells from the microvasculature into the peritoneal cavity. Normally, the peritoneal cavity contains less than 100 ml of sterile serous fluid. Microvilli on the apical surface of the peritoneal mesothelium markedly increase the surface area and promote the rapid absorption of fluid from the peritoneal cavity into the lymphatics and the portal and systemic circulation. The amount of fluid within the peritoneal cavity may increase too many litres in various diseases such as cirrhosis, nephritic syndrome, and peritoneal carcinomatosis.

The circulation of fluid within the peritoneal cavity is driven in part by the movement of the diaphragm. Intercellular pores in the peritoneum covering the inferior surface of the diaphragm (termed stomata) communicate with lymphatic pools within the diaphragm. Lymph flows from these diaphragmatic lymphatic channels through sub pleural lymphatic to the regional lymph nodes and ultimately the thoracic duct. Relaxation of the diaphragm during exhalation opens the stomata, and the negative intra thoracic pressure draws fluid and particles, including bacteria, into the stomata. Contraction of the diaphragm during inhalation propels the lymph through the mediastinal lymphatic channels into the thoracic duct.

It is postulated that this so-called diaphragmatic pump drives the movement of peritoneal fluid in a cephalic direction towards the diaphragm and into the thoracic lymphatic vessels. This circulatory pattern of peritoneal fluid towards the diaphragm and into central lymphatic channel is consistent with the rapid appearance of sepsis in patients with generalized intra-abdominal infections as well as the perihepatitis of Fitz-Hugh-Curtis syndrome in patients with acute salpingitis.

The peritoneum and peritoneal cavity respond to infection in the following ways:

The bacteria are rapidly removed from the peritoneal cavity through the diaphragmatic stomata.

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 immunoglobulins into the peritoneal space. Protein within the peritoneal fluid opsonises 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.

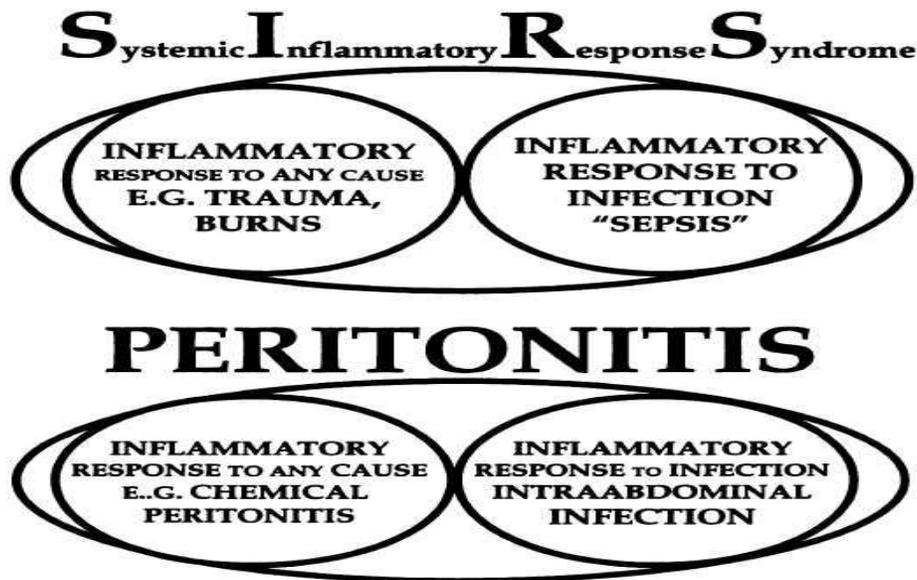
PATHOPHYSIOLOGY OF PERITONITIS^{7,10-13} CLASSIFICATION AND STRATIFICATION¹⁴

Definition

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. Intra-abdominal infection denotes peritonitis caused by bacteria (e.g., a local inflammatory process initiated by bacteria and their toxins).

It may be regarded as the localized equivalent of systemic sepsis. Intra-abdominal abscess is an intra-abdominal infection that has been confined within the abdominal cavity. Because the vast majority of clinically significant peritonitis is caused by bacteria, both terms are used interchangeably. Intra-abdominal infection is defined as an inflammatory response of the peritoneum to micro-organisms and their toxins, which results in purulent exudates in the abdominal cavity.

Conditions without such peritoneal inflammatory response, in which contamination has occurred but infection is not established (e.g., early traumatic bowel perforation), or in which the infectious process remains contained within a diseased, but resectable organ (e.g. gallbladder or appendix), represent "simple" form of peritonitis, easily cured by an operation and not requiring prolonged additional antibiotic therapy.



Relation between SIRS & sepsis vs. Peritonitis & intraabdominal infection

Peritonitis results from any local trigger of inflammation. Usually infection is the trigger, although infection may not necessarily be present at the localized peritoneal space surrounding an infected but resectable intraabdominal organ, such as the appendix or gallbladder. In contrast, there may be contamination of the peritoneum from a defect in the intestinal wall, before establishment of infection or onset of an inflammatory response, e. g. immediately following penetrating abdominal trauma. Peritonitis has been categorized as primary, secondary, or more recently tertiary.

Primary peritonitis is an infection of the peritoneal cavity not directly related to other intra abdominal abnormalities. The vast majority of cases are due to bacterial infection; it is commonly known as spontaneous bacterial peritonitis (SBP). Usually it occurs in the presence of ascites. In the preantibiotic era, primary peritonitis accounted for ~ 10% of all paediatric abdominal emergencies; it now accounts for < 1-2%. The decline has been attributed to widespread use of antibiotic for minor upper respiratory tract illness.

The infrequency of primary peritonitis in form of ascites other than due to liver disease emphasizes the importance of intrahepatic shunting in the pathogenesis of the disease.

Secondary intraabdominal infection is usually due to spillage of gastrointestinal or genitourinary micro-organisms into the peritoneal space as a result of loss of integrity of the mucosal barrier. Examples include appendicitis, diverticulitis, cholecystitis, penetrating wound of bowel, and perforation of a gastric or duodenal ulcer. Secondary infection is relatively common; taking the form of either generalized or localized abscesses. Abscesses may be restricted to the immediate peritoneal space around a disease intra abdominal organ, such as pericholecystic, periappendiceal, or peridiverticular abscesses, or to certain peritoneal recesses, such as interloopal, subdiaphragmatic, subhepatic, lesser sac or pelvic abscesses.

Tertiary peritonitis is conceived as a later stage in the disease, when clinical Peritonitis and systemic signs of sepsis (e.

g., fever, tachycardia, tachypnoea, hypotension, elevated cardiac index, low systemic vascular resistance, leucopenia or leukocytosis, and multiorgan failure) persists after treatment for secondary peritonitis and either organisms or low virulence pathogens, such as *enterococci* or *fungi*, are isolated from peritoneal exudates. This organism may gain access to the peritoneal cavity by contamination during operative Interventions, by selection from the initial polymicrobial peritoneal inoculum by antibiotic therapy, or by translocation of bowel flora. Translocation may be promoted by intestinal ischemia, endotoxemia, malnutrition, or proliferation of resistant bowel flora by antibiotic pressure.

Undoubtedly, many of the systemic as well as abdominal manifestation of the peritonitis are mediated by cytokines, such as TNF, IL-1, IL-6, IFN-gamma, and others.

Cytokines appear in the systemic circulation of patients with peritonitis and to a much greater extent in the peritoneal exudates. These cytokines are produced by macrophages and other host cells in response to bacteria or bacterial products, such as endotoxin, or by tissues traumatized during operative procedures. Another potential source is direct translocation of cytokines through the intestinal barrier.

With gastrointestinal perforation as the precipitating event, the number and type of micro-organism isolated from the peritoneal cavity depend on level of perforation. The stomach in fasting state contain sparse microflora of a few relatively more acid-resistant species, e. g., lactobacilli or candida species. Similarly, the duodenum and the proximal bowel contain a sparse microflora in the fasting state, whereas the colon contains a high microbial density, i.e., about 10^{12} per gram are obligate anaerobes, mainly of *bacteroides fragilis* group.

Gastric perforations are associated with either sterile chemical peritonitis or peritonitis due to above mentioned pathogens, depending on the underlying gastric condition. Similarly, the normal sparse flora of small bowel may be altered by gastric disease or small bowel ileus.

Peritonitis is thought to pass through three phases:-

PHASE 1- Involves rapid removal of contaminants from the peritoneal cavity into systemic circulation. The contaminated peritoneal fluid passes through stomata in the diaphragmatic peritoneum and is absorbed into lymphatic lacunae. The lymph flows into the main lymphatic duct through the substernal nodes. The resultant septicaemia predominantly involves Gram negative facultative anaerobes and is associated with high morbidity.

PHASE 2- Involves synergistic interaction between aerobes and anaerobes as they encounter host complement and phagocytes. The complement is activated by classical pathway, with the alternate and lectin pathway in support.

Phospholipids surfactant produced by the parietal mesothelial cells work synergistically with complement to increase opsonisation and phagocytosis. Peritoneal mesothelial cells are also potent secretors of proinflammatory mediators, therefore peritoneal mesothelial cells play a major role in the cell signaling pathway leading to recruitment of phagocytes to the peritoneal cavity and the upregulation of mast cells and fibroblast in the submesothelium.

PHASE 3- It is an attempt by host defences to localize infection mainly via production of fibrinous exudates that traps microbes within its matrix and promotes local phagocytic effectors mechanism. It also serves to promote development of abscesses.

Factors favouring localization of generalization of peritonitis –

Localization

- Fibrinous Exudates
- Anatomical compartmentalization of peritoneum
- Greater omentum (Adheres to inflamed peritoneum)

Generalization

- Sudden visceral perforation
- Violent peritonitis
- Virulent infecting organisms
- Injudicious handling
- Immunocompromized state.

MICROBIOLOGY OF PERITONITIS^{15, 16}

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). After a perforation of colon more than 400 different species invades the bacteria invades the peritoneal cavity. Only a few are involved in ensuing infection. 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.

MOLECULAR BASIS OF PERITONITIS

Topley N et al¹² in their study on macrophages and mesothelial cells in bacterial peritonitis examined the mechanism underlying cellular host defence in the peritoneal cavity. This study has established that the resident cell of the peritoneal cavity, the peritoneal macrophages (PM phi) and the mesothelial cells (HPMC) contribute to the initiation, amplification and resolution of peritoneal inflammation. Ex vivo measurements of intraperitoneal inflammatory mediators during peritonitis has elucidated the time course for the generation of proinflammatory, chemotactic and anti-inflammatory cytokines and have identified that their secretion occurs largely within the peritoneum .This study provide evidence that both PM phi and HPMC derived mediators are directly involved in controlling inflammation. It has been widely accepted that resident PM phi forms the first line of defence against peritoneal infection, a more contemporary view would suggest that the direct or indirect (via secreted proinflammatory cytokines) interactions between PM phi and HPMC cytokine synthesis .Once the inflammatory response is initiated, recent evidence suggest that mesothelial cells upon activation by PM phi derived interleukin I,TNF alpha & beta are capable of amplifying inflammation and generating signals (via the creation of a gradient of chemotactic cytokines, IL-8, MCP-1, RANTES) for the recruitment of leukocytes into the peritoneum. This process is also facilitate via the cytokine driven up-regulation of adhesion molecule expression (ICAM –I & VCAM-I) on HPMC. Much less is understood about the mechanism by which inflammation molecules (IL-6&IL1) by receptors by PM phi and HPMC may be important in this process.

The existence of a peritoneal cytokine network controlling inflammation is now well established within this the interaction of PM phi and HPMC appear to play a pivotal role in host response to peritoneal infection.

Wolfgang Sendt et al¹³ compare the degree of the inflammatory response of human peritoneum with the severity of peritonitis.

They concluded that the pattern of peritoneal inflammatory reaction is relatively uniform and does not correlate with the clinical grading of severity.

CLINICAL MANIFESTATIONS^{7, 8, 10}

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 catecholamines 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 and anxious; 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.

INVESTIGATIONS^{7, 8, 10}

Peritonitis is mainly a clinical diagnosis and urgent laparotomy should not be delayed for unnecessary investigations. Blood tests are discussed below:

Full blood count will demonstrate leukocytosis.

Urea and electrolytes will confirm dehydration and acute renal failure; results are used to guide replacement of fluid and electrolytes.

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).

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

Grouping and cross matching – laparotomy may be indicated and therefore cross matched blood will be required.

IMAGING :

Erect radiograph of the chest will show pneumoperitoneum in about 70–80% of visceral perforations. A left lateral decubitus radiograph of the abdomen is an alternative in those who are unable to sit up. A supine radiograph of the abdomen is less informative, but has a 'ground glass' appearance in cases of diffuse peritonitis.

Ultrasound may play a role in confirming or excluding specific diagnoses (e.g. subphrenic abscess).

Computerized Tomography (CT) is far more accurate in negative prediction than ultrasound, and has largely replaced blind, diagnostic laparotomy in the search for occult sepsis. The diagnostic accuracy of both ultrasound and CT has also been affirmed in clinically equivocal cases of acute appendicitis.

DIFFERENTIAL DIAGNOSIS:

Basal pneumonia, myocardial infarction, gastroenteritis, hepatitis and urinary tract infection may be misdiagnosed as peritonitis.

Other causes of severe abdominal pain (e.g. intestinal obstruction, ureteric or biliary colic) tend to cause restlessness.

MANAGEMENT:

Conservative-

Medical treatment is indicated if the:

Infection has localized (e.g. appendix mass)

- Cause of peritonitis does not require surgery (e.g. acute pancreatitis)
- Patient is not fit for general anaesthesia (e.g. elderly, moribund patient with severe co morbidity)
- Medical facilities are unable to support safe surgical management

The principal elements of medical treatment are fluid hydration (i.v.) and broad spectrum antibiotics. Supportive care should include early enteral feeding (in preference to total parenteral nutrition) for patients with complex abdominal sepsis in the ICU.

Immediate-

High-flow oxygen is vital for all shocked patients. Hypoxia can be monitored by pulse oximetry or measurement of arterial blood gases.

Fluid resuscitation is initially with crystalloids (i.v.), the volume being dependent on the degree of shock and dehydration. Electrolyte (especially potassium) replacement may be required. The patient should be catheterized in order to monitor the hourly output of urine. Monitoring of central venous pressure and the use of inotropes may be appropriate in severe sepsis or in patients with comorbidity.

Analgesia – opiate analgesia (i.v.) and an appropriate antiemetic will be required.

Antibiotics should be broad-spectrum, cover aerobes and anaerobes, and given intravenously. A third-generation cephalosporin and metronidazole is a common primary strategy. For patients who acquire peritonitis in hospital (e.g. anastomotic leak) or who require intensive care, second-line therapy with meropenem or a combination of piperacillin and tazobactam is advised. Antifungal therapy should also be considered to cover possible *Candida* species. Early and appropriate use of

antibiotics is a key to reducing mortality in patients with septic shock associated with peritonitis.

Nasogastric tube and aspiration alleviates vomiting and abdominal distension and reduces the risk of aspiration pneumonia.

Definitive-Surgery:

The prerequisite for the surgical treatment of peritonitis and for abdominal surgery in general was the foundation of experimental physiology and medicine by *Francois Magendy* and *Claude Bernard*, the development of cellular pathology by *Virchow*, the advent of the germ theory connected with the names of *Pasteur* and *Koch*, the introduction of antiseptics and asepsis by *Lister* and *Semmelweis*, the introduction of the systemic physical examination and the correlation between clinical and pathological findings by the Paris clinical school, and the introduction of general anaesthesia by *Wells* and *Morton*. With this background the knowledge of pathophysiology and bacteriology of peritonitis as well as the surgical treatment of the

disease developed rapidly around the turn of the century. The principles of the latter were summarized by *Kirschner* in 1926. The most important are mandatory surgical exploration, secure elimination of the focus of infection, and an effective peritoneal toilet.

Advances in the treatment of peritonitis during the last five decades were due to the advent of antibiotics and intensive care medicine, the better understanding of the synergism of bacteria in the peritoneal cavity, the systemic inflammatory response due to intraperitoneal infections, and the development of scoring systems and their application to patients with peritonitis.

Laparotomy is usually performed through an upper or lowermidline incision (depending on the suspected site of pathology). The objectives are to:

- ❖ establish the cause of peritonitis
- ❖ control the origin of sepsis by removal of the inflamed or ischaemic organ (or closure of the perforated viscous)
- ❖ perform effective peritoneal toilet/lavage.

MANAGEMENT PRINCIPLES OF PERITONITIS¹⁴

I. Supportive measures

- A. To combat hypovolemia and shock and maintain adequate tissue oxygenation
- B. To treat bacteria, not eliminated by surgery, with antibiotics
- C. To support failing organ systems
- D. To provide adequate nutrition

II. Operative treatment

Principle 1 (Repair)

Control the source of infection

Principle 2 (Purge)

Evacuate bacterial inoculum, pus, and adjuvants (peritoneal "toilet")

Principle 3 (Decompress)

Treat abdominal compartment syndrome

Principle 4 (Control)

Prevent or treat persistent and recurrent infection or verify both repair and purge

Control of the primary source of sepsis is essential. However, there is a trend towards undertaking primary anastomosis in patients with peritonitis (providing that they are haemodynamically stable and have no other significant risk factors).

There is little evidence of clinical benefit for peritoneal irrigation, possibly because of resistance of microbial peritoneal colonies to lavage, or concomitant damage inflicted to mesothelial cells. Rather than robust irrigation of the peritoneal cavity, removal of debris, faecal or purulent material may suffice.

Mass closure of the abdomen is undertaken using interrupted or continuous monofilament sutures. Antibiotics are continued for five days postoperatively in cases of generalized or complex peritonitis.

Re-laparotomy has an important role in the treatment of patients with severe secondary peritonitis who, after primary laparotomy, have ongoing or worsening features of sepsis. Reoperations may be performed 'on demand', or in a more aggressive 'planned' strategy at regular intervals. Planned relaparotomy often involves leaving the abdominal wall open with a sheet of synthetic mesh *in situ* to prevent evisceration. Modifications are 'primary open management', and semi-open approaches such as 'staged abdominal repair'. However, recent studies have concluded that in-hospital and long-term survival rates are higher in those patients managed by on-demand relaparotomy than in those with disease of comparable severity treated by planned relaparotomy.

Combining clinical data with frequent CT imaging is the key to timely and appropriate selection of patients requiring on demand

relaparotomy. However, it should always be remembered that many septic patients do not require relaparotomy but may simply require extended periods of mechanical ventilation, antimicrobials and organ support.

Obtaining effective control of sepsis at the first operation is vitally important because each subsequent operation is met with an increasing risk of morbidity and mortality.

Laparoscopy:

The theoretical risks of malignant hypercapnia and septic shock secondary to absorption of carbon dioxide and endo toxin through an inflamed peritoneum have not been proven. Instead, laparoscopy has proved effective in the management of acute appendicitis and perforated duodenal ulcer. It can be used in cases of colonic perforation, but the conversion rate to laparotomy is higher. Shock or major ileus are contraindications to laparoscopy.

Drains tend to be effective if used to drain a localized space, but are generally quickly 'walled off' and fail to drain the entire peritoneal cavity. There is a lack of evidence to support the prophylactic use of drain tubes after laparotomy.

VARIOUS FACTORS AFFECTING PROGNOSIS IN PATIENTS WITH PERITONITIS AND EVALUATION OF MANNHEIM PERITONITIS INDEX:

J. L. Dawson¹⁷ et al (1963) performed a study in which analysis of the record of 665 consecutive cases of diffuse peritonitis was undertaken to investigate factors affecting the mortality rate. The factors which were studied are the causal lesion, age, sex, the length of history and the method of treatment. About one third of cases were due to appendicitis and another third due to perforated duodenal ulcer.

There was a preponderance of male patients and it was largely accounted for by the large number of male patients with perforated duodenal ulcer.

The sex ratio in other condition is almost equal. Apart from patients with appendicitis the majority were aged above 40 years. In appendicitis there is little rise in mortality rate with age; this differs considerably from all the other groups.

The overall mortality rate ignoring sex, age, and the causal lesion, was 19.5%. The mortality rises with the age and below the age of 40 years the mortality was negligible. The difference in mortality between two sexes was not striking. In appendicitis there was little rise in mortality with increase in age. In perforated duodenal ulcer the mortality rises steeply after the age of 60 years. The mortality in perforated gastric ulcer was much higher than in perforated duodenal ulcer.

A study was conducted between 1935 to 1985 by Cecilie Svanes¹⁸. One thousand one hundred and twenty-eight patients treated for perforated gastro-duodenal ulcer during the year 1935 - 1985 were studied at the Haukeland University Hospital. The majority of patients (97.7 %) were treated surgically. The total post perforation lethality was 7.4%, the post surgical death rate was 6.6%, and the death rate among conservatively treated patients was 42.3%. Lethality was significantly influenced by year of hospital admission and increased markedly with age of the patients. For all age groups, the lethality decreased markedly with time. Treatment delay was associated with a moderate but significant increase in lethality. In patients with gastric ulcer the

lethality was 3.6 times higher than in those with duodenal ulcer. The death rate was similar in the duodenal and pyloric ulcer groups. Death rate decreased with time in both stomach ulcer, duodenal, and pyloric ulcer patients. There was no sex difference and no difference between patients treated with simple suture or gastric resection.

In 1987 Linder MM, Wacha H³, Feldmann U, Wesch G et al conducted a study based on experience with 1243 patients suffering from purulent peritonitis. 255 patients were included in the study and they were studied prospectively. For intra abdominal infection an index named the MANNHEIM PERITONITIS INDEX was established that allows for the first time to predict lethal outcomes of the disease in the individual patients.

Thereafter in 1988 Fugger R¹⁹ Validated the Mannheim peritonitis index. 113 patients suffering from purulent peritonitis entered this retrospective study for evaluation of the prognostic value of the Mannheim peritonitis index. There was no lethality below an index $x = 21$, between $x = 21$ and $x = 29$ % and lethality increased to 100% in patients with an index x greater than or equal to 30. Statistical validation showed that prognosis was correct in 93%. Between $x = 21$ and $x = 29$ prognosis of the MPI was correct in at least 65%. This study concluded that the Mannheim peritonitis index is a prognostic index for peritonitis with high accuracy in individual prognosis and that it is also very easily documented.

In 1994 the reliability of the Mannheim peritonitis index was assessed and its predictive power for different populations examined in a study of 2003 patients from seven centers in three European countries by Billing A, Frohlich D and Schildberg FW²⁰. The prevalence of risk factors varied considerably between the groups. For a threshold index score of 26, the sensitivity was 86 (range 54-98) percent, specificity 74 (range 58-97) percent and accuracy 83% (range 70-94) percent in predicting death. For patients with a score less than 21 the mean mortality rate was 2.3 (range 0-11) percent, for score 21-29 22.5 (range 10.6-50) percent, for score 21-29 22.5 (range 10.6-50) percent and for score greater than 29 59.1 (range 41-87) percent.

This study concludes that the mean index score and the mean mortality rate correlated in different groups, reflecting a homogenous standard of therapy for peritonitis and that the Mannheim peritonitis index provides an easy and reliable means for risk evaluation and classification for patients with peritoneal inflammations.

Demmel N²¹ et al studied 438 patients with intra abdominal infections prospectively. 300 of them were managed by closed treatment with drainage, 138 by open treatment with planned relaparotomy. 63 patients (14.4%) died, 34 of them due to sepsis, 29 due to another cause. In this study stepwise logistic regression analysis showed that only preoperative shock, concomitant disease and sepsis to be significant and independent factors leading to death. MPI had a strong correlation to mortality, statistical validation showed a sensitivity of 88% and a specificity of 78 at a critical score of 26 points. This study concludes that MPI is an early documented prognostic index for peritonitis with high accuracy in individual prognosis.

Liverania A, Correnti SF²² et al (1998) studied 235 patients operated on for acute peritonitis in a retrospective manner. The aim of this study was to estimate the prognostic reliability of

Mannheim peritonitis index. In this study the overall mortality was 8.1% for Mannheim peritonitis index score of less than 26 the mortality was 2% while the mortality was 40.5% for scores greater than 26. The study concluded that Mannheim peritonitis index is a threshold over which the therapeutic approach has to be more aggressive. Planned multiple laparotomies or the open abdomen technique could be the best option to explore and clean up the peritoneal cavity from septic debris.

Rogy M. Fugger R¹⁹ et al (1990) studied seventy patients suffering from purulent peritonitis. Two score were compared, the Mannheim peritonitis index and the APACHE II Score. The study concludes that the Mannheim peritonitis index is a prognostic index for peritonitis with high accuracy in individual prognosis. The simultaneous use of both the scores leads to a negligible improvement of prognostic accuracy. The sensitivity and specificity of MPI is more than that for the APACHE II Score.

Pacelli F²³ et al (1996) retrospectively reviewed 604 patients who consecutively underwent emergency operation for intra – abdominal infections. The study showed that the APACHE II Score, the Mannheim peritonitis index hypoalbuminaemia, hypercholesterolemia and preoperative organ impairment are independent predictors of death. They also concluded that the Mannheim peritonitis index has the advantage of being easier to calculate.

James M Watter²⁴ et al (1996) conducted a study to evaluate the influence of age on the evolutions and severity of peritonitis. They studied 122 patients with acute appendicitis and 100 patients with acute colonic diverticulitis requiring operation or per cutaneous drainage. They concluded that the biological before of peritonitis differ in the elderly, who are more likely to present with an advanced or severe process than young patients. They further stated that if these retrospective clinical observations reflect a generalisable, age related change in the biological features of peritonitis, then an understanding of the basis for such a change may allow improvement in the care of elderly surgical patients with abdominal infections.

Bosscha K²⁵ Reijnders K et al (1997) evaluated the various scoring systems such as the APACHE II Score, simplified acute physiology score, sepsis severity score, multiple organ failure score, Mannheim peritonitis index and the Ranson and Imrie score in about 50 patients. Additionally scoring systems were combined to obtain a combined score for the prediction of peritonitis related mortality. In this study it was found that only the APACHE II Score and the Mannheim peritonitis index provides the best scoring system fitting clinical goals.

Christian Ohmann² et al (1997) studied 355 patients of peritonitis confirmed at laparotomy. The objective of the study was to develop and to evaluate a new score to aid management of peritonitis. Computation of four different prognostic systems APACHE II; APACHE II and successful operation; APACHE II successful operation and Gori's score on first post operative day and the multivariate analysis was done. It was found that multivariate analysis is superior. From the analysis a new prognostic model (prognostic peritonitis model: PPM) was derived. The author finally concludes that clinical trials are required for the validation of this new score.

TM Cook²⁶ et al (1998) studied 107 patients age over 80 years undergoing urgent or emergency laparotomy. The factors

that predict hospital survival were studied. Age and ASA status were significant predictors of survival ($P<0.05$) and of anaesthetists prediction of mortality both before and after operation. They also found that women had a greater probability of dying. The estimated probability of death was increased in patients who were admitted to the ICU or who had invasive hemodynamic monitoring.

Sokmen S²⁷ (2001) studied three hundred and twenty five patients retrospectively. Among them 258 patients were discharged in a well condition with 67 patients died. In the patients who died the Mannheim peritonitis index score was always equal or more than 26. When all descriptive factors were revealed to be statistically significant between the exitus and the discharged group. They concluded that the Mannheim peritonitis index is effective scoring system in terms of predicting final outcomes in patients with peritonitis and intra abdominal sepsis.

Murat Kologlu²⁸ et al (2001) Studied 473 patients. 75 of these patients were operated for postoperative peritonitis and their data was compared with the remaining 398 patients with secondary bacterial peritonitis due to other causes. All patients were scored according to the Mannheim peritonitis index and the peritonitis index of ALTONA II score. Using multiple logistic regression, Mannheim peritonitis index and the peritonitis index of ALTONA II were combined in an equation and this new variable was called combined peritonitis score (CPS): $CPS = 9 + (0.8 \times MPI) + (-1.2 \times PIA II)$. All patients were scored according to Mannheim peritonitis index and the peritonitis index of ALTONA II and CPS. Receiver – operator characteristic (ROC) curves and sharpness of scores were compared. Also mean scores in both groups, proportions of correct predictions of outcome according to scores and correlation of scores with mortality were compared. Overall mortality was 17.8% in OTHER group and 33.3% in POSTOP group ($P=0.018$). Higher MPI scores, lower PIA II scores and higher CPS scores were associated with higher mortality in both groups ($P<0.0001$). mean MPI values were higher, mean PIA II values were lower and mean CPS value were higher in POSTOP group ($P<0.001$) the areas under ROC curves of CPS were bigger than MPI and PIA II in both groups. Sharpness of CPS was higher in both groups compared to MPI and PIA II ($P<0.05$).

Proportion of correct predictions of outcome was highest in CPS among the three scores ($P=0.0074$). CPS had the best correlation with observed mortality. They concluded that the Mannheim peritonitis index and the PI A II are scoring systems which are specially designed to predict outcomes in peritonitis and they depend mainly on preoperative and intraoperative objective criteria. They are practical and useful for clinical practice and clinical studies. They further concluded that instead of developing new scores modifying the existing scores would be more useful when this two indices were combined in the form of combined peritonitis score (CPS) all the parameters improved.

MM Corriea²⁹ et al (2001) studied 89 patients with cancer who undergo an emergency operation for secondary peritonitis. Frequencies of MPI component were calculated and the total score was obtained. The ages of the patient ranged from 0 to 89 with a mean of 58.4 years. 65 patients were men (73.3%) and 24 women (26.7%), only 8 patients were pre-operative and all other were post – operative. Most of the underlying causes were gastrointestinal. The overall mortality was 61.8%. The

preoperative duration of peritonitis was longer than 24 hours in 65.5%. Purulent exudates were observed in 63.3% and generalized diffuse peritonitis occurred in 62.2% of the patients. In 55.6% the origin of peritonitis was non – colonic and organ failure was observed in 48.9 % of cases. Comparison of the MPI variables in two groups showed that only organ failure, age older than 50 years and diffuse generalized peritonitis reached statistical significance. Preoperative peritonitis duration > 24 hours was slightly more frequent among patients who died than among survivors, but the difference was not significant. The mortality increased proportionally according to the MPI score. The authors recommended that *Mannheim peritonitis index* is accurate to be used for oncological patients with peritonitis but the MPI cut off should be adjusted for each hospital.

Scapellato (2004) studied 255 cases of secondary acute peritonitis. The Mannheim peritonitis index score was calculated for each patient to predict the peritonitis related in hospital mortality. The patients were divided into 3 groups according to MPI score of less than 21, between 21 to 29 and greater than 29. There was no mortality in the first group and there was significantly less mortality in the second group as compared to the third group. The study suggests that intervention time may be considered the main determinant of mortality in patients with peritonitis. This observation is especially relevant since intervention time is a modifiable risk factor while other factors are not modifiable.

Thomas Koperna³⁰ et al (2004) studied Eighty five emergency surgical patients admitted for the surgical ICU. The APACHE II Score was calculated before surgery, after admission to the ICU and on the postoperative days 3, 7 and 10. The study concludes that though longitudinal APACHE II Scoring reveals continuous improvement of the score in the surviving patients but it has no therapeutic relevance in the individual patients.

Rodolfo L³¹ et al (2002) studied 176 patients with acute perforation peritonitis. Patients were divided into groups according to the following categories (MPI points) : a) <21, 21-29, >29 and b) ≤ 26 and > 26. The mortality was about 6%. Each factor of the Mannheim peritonitis index was studied separately. Mean age of the patient was 34.6 years. The mean age of survivors was 32.7 years. Among the non-survivors the mean age was 63 years. Though age is a significant factor to predict mortality further studies may be undertaken to establish critical values related to age and intervals into which this variable should be subdivided. In this study patient with generalized peritonitis corresponds to 34% of patients.

Among survivors localized peritonitis was found more frequent than generalized peritonitis (68% vs. 32%) which in non survivors this relationship was inverted (27% vs. 73%) the extension of the peritonitis process was thus related with mortality. Considering survival related with the character of the peritoneal fluid they found that clear fluid had mortality of 5.8%, purulent fluid had mortality of 6.3% and faecal fluid had mortality of 25%, approximately 48% of patients were females and 52% were males with mortality rate of 7% and 6% respectively. It was not possible to calculate significance for the variables of presentation greater than 24 hours and organ failure because all deaths presented this adverse factor. Non colonic origin was also not found to be a significant factor in this study related to adverse outcomes as the mortality was 6% in pts with

non colonic origin of peritonitis and it was 15.38% in patients with colonic origin of sepsis. It was found that the anatomical origin of bacterial contamination and the microbiologic findings are not the main predictors of patient outcome. It was concluded the Mannheim peritonitis index is a useful method of determine outcome in patients of peritonitis. All the MPI adverse factors except the colonic origin of sepsis behaved as expected and the following were especially useful presence of organic failure, time elapsed > 24 hours, presence of malignancy, age greater than 50 years and the generalized extension of peritonitis.

Juan J³² et al (2003) studied 710 patients aged 70 years or older who underwent emergency surgery for intra-abdominal disorders. Patients were divided into two group one between 70 to 79 years and other greater than 80 years of age. In the analysis patient's age, sex, preoperative risk, the time interval between onset of symptoms and admission to hospital and between admission to surgery, type of surgery, operative findings, mortality, morbidity and the length of stay in hospital were studied.

The analysis reveals that most of the factors analysed increased mortality such as the ASA grading IV-V increased time lapse, intestinal infarction, malignant disease with distant metastasis and only palliative surgery. Independent mortality related factors were found to be preoperative risk (ASA grade), the time elapsed, disorders that only permit palliative surgery (gastrointestinal by pass, colostomy) or no surgical intervention (non therapeutic laparotomy) such as the advanced mesenteric vascular disease or advanced malignancy. In contrast to other studies increased age of the patient was not associated with higher morbidity or mortality.

Shuhei Komastu³³ et al (2003) studied 26 consecutive patients who underwent emergency operation for colorectal perforation. Several clinical factors were analyzed as possible predictive factors and APACHE II, SOFA, MPI and MOF Scores were calculated. The 26 patients comprise 14 men and 12 women. The mean age was 69 years. The overall mortality was 26.9% in this study with respect to the pre and perioperative clinical factors, Hinchey's stage III –IV, low preoperative market of pH, base excess and an MPI score of more than 30 were significant poor prognostic factors. Mortality was more in patients with diffuse peritonitis than in those with localized peritonitis. Concerning the post operative clinical factors low postoperative white blood cell count, Pao₂/Fio₂ ratio and renal output was associated with poor prognosis. The time from onset to operation did not significantly influence in hospital mortality in this study. The study also concludes that patients with an APACHE II score of 19, a SOFA score of 8 an MOF score of 7 and MPI score of 30 or more represents the highest risk group and early classification with this prognostic scoring systems to predict outcomes in desirable to select patients for appropriate antiseptic treatment.

Muluri K³⁴ et al (2004) retrospectively studied 66 patients with secondary peritonitis caused by gastrointestinal tract perforation. The overall hospital mortality rate was 30% significant risk factors in the univariate analysis includes advanced age, pre existing illness, chronic medications, hospital transfer, non-traumatic course of perforation, high Mannheim peritonitis index score and a high c-reactive protein level in the early post operative phase.

Kusumoto Yashiko³⁵ et al (2004) studied 108 patients operated on for intra abdominal infections (excluding the patients with appendicitis) the objective of the study was to evaluate the reliability of the Mannheim peritonitis index in predicting the outcome of patients with peritonitis. In this study the mortality was 5.3% in men and 15.2% in women with deaths occurring only in patients older than 50 years of age. The study demonstrates that the MPI score of 26 or less has mortality of 3.8% while score of more than 26 is associated with significant mortality thus establishing the score of 26 as an out off. The study concludes that the Mannheim peritonitis index is a simple and useful prognostic index for assessing the severity of peritonitis. The reliability of index may be improved by including complications associated with multiple organ failure and severe chronic health problems.

Abraal Maqbool Qureshi³⁶ et al (2005) studied on hundred and Twenty Six patients of secondary peritonitis who presented to the surgery unit II of the Rawalpindi general hospital. MPI score was calculated for each patient on a predesigned proforma and the patient was followed up till discharge or death. Death was the main outcome measure against which the MPI score was analyses under 2 categories i) <26 or ≥ 26 ii) <21 , $21-29$, > 29 . For MPI score ≥ 26 the mortality was 28.1% while it was 4.3% for scores less than 26, for a score less than the mortality was 1.8% for scores between 21 to 29 it was 21.9% and for score of 30 or more it was 28%.

Odds ratio calculated were significant for age > 50 year, malignancy, organ failure, preoperative duration > 24 hrs and cloudy, purulent exudates. The study concludes that increasing MPI score is strongly associated with the outcome.

Jyrki Tapani Makela³⁷ et al (2004) studied 172 patients with diverticular perforation. The clinical variables were evaluated as prognostic indicator of postoperative complications, mortality and the time of hospitalizations. The overall complication rate was 33% in patients under 70 years the Mannheim peritonitis index and the American society of anaesthesiologist (ASA) scores were independent prognostic factors. The Hinchey's classes and malnutrition also correlates with mortality. Only the Mannheim also correlates with mortality. Only the Mannheim peritonitis index was shown as a predictor of mortality. The study concludes that mortality is related to age but age alone is not an independent predictor of mortality. The MPI score is useful in the prediction of death in patients with diverticular perforation.

Ali Yaghoobi Notash³⁸ et al (2005) studied 80 consecutive patients with secondary peritonitis. The aim of the study was to predict the outcome of patients with peritonitis using the Mannheim peritonitis index and the multiple organ failure score. The MPI and the revised and original MOF scores were calculated at admission or during management. Patients were followed up until death or discharge non survivors had higher mean original and revised MOF scores (6.8 and 4.8) whereas survivors had mean MOF score of 0.3. Survivors had mean MPI of 19.39 but that of non survivors was 33.07. The risk of in hospital mortality was higher in patients aged above 60 years and time interval from presentation until surgery of greater than 24 hours.

The MPI has been shown to be an appropriate objective prognostic factor in patients with peritonitis to predict the

outcome. The study finally says that a combination of MPI and MOF can give better results.

Rajendra Singh Jhobta³⁹ et al reviewed say consecutive cases of perforation peritonitis over a period of five years in terms of clinical presentation, operative findings and the post – operative course at GMCH Chandigarh. The objective of the study was to highlight the spectrum of perforation peritonitis encountered therein. The most common cause of perforation in this series was perforated duodenal ulcer (289 cases) followed by appendicitis (59 cases), GI perforation due to blunt trauma abdomen (45 cases), typhoid fever (41 cases) and tuberculosis (20 cases). Despite delay in seeking medical therapy (53%) the overall mortality was 10% but the morbidity was unusually high at 50%. The study concludes that in contrast to the western literature where lower GI tract perforations predominate, upper GI tract perforation constitutes majority of cases in India. The author also sounds warning due to the increase in the number of GI tract perforations due to blunt abdominal trauma as a result of high speed motor vehicular accident and warrants early recognition and prompt treatment.

Atsushi Floriuchi⁴⁰ et al (2007) studied 26 patients (9 men, 17 women) with mean age of 72.7 ± 11.6 year who underwent emergency surgery for colorectal perforation. Several clinical factors were measured preoperatively and 19 hours postoperatively. APACHE II Score, the Mannheim peritonitis index and the peritonitis index Altona was calculated preoperatively. APACHE II Score was significantly less in survivors than in non survivors. Non survivors tend to display a high Mannheim peritonitis index score and a low PIA score, but no significant difference was identified.

Thomas E Riz⁴¹ and Tom bates performed a search of the literature of ram 1977 – 2007 using midline this search was performed to identify those predictive risk scores relevant to sick elderly patients in whom emergency surgery may be life saving. The study concludes that risk scores may be help but in sick elderly patients needing emergency abdominal surgery an experience clinical opinion is still essential.

Jebrey Vermeulen⁴² et al (2007) studied 200 consecutive patients with acute perforated diverticulitis. Mortality and morbidity was compared in relation to type of surgery. ASA classification, age, gender, Mannheim peritonitis index, Hinchey score, surgeon's experience and type of operation. The object of this study was to compare patients of perforated diverticulitis treated either with primary anastomosis or Hartmann's procedure. Total mortality was 27%, 47 patients died after Hartmann's procedure compare to 7 after primary anastomosis. Age, Mannheim peritonitis index and the ASA score were significantly related to mortality. The study concludes that whether to do Hartmann's procedure or a primary anastomosis should be done taking into account the patients concomitant disease, response of preoperative resuscitation and the availability of a surgeon experienced in colorectal surgery.

M Hyninen⁴³ et al (2007) studied 163 consecutive patients with secondary peritonitis. 58 patients among this were treated in an ICU. The hospital mortality was 19% and one year mortality was 23%, on admission 44 patients (27%) were in septic shock and 10 patients (6%) presented with severe sepsis. Mortality varied with the site of perforation and highest mortality was noted in colonic and biliary perforation. In 5 patients malignancy

was the etiological factor and all of them survived for 1 year. Analysis revealed that APACHE II Score, previous functional status and the sepsis category were independent predictors of mortality. SOFA score (sequential organ failure assessment) was assessed in patients admitted to ICU and it was shown to have the best predictive power for mortality. The study highlights the importance of early recognition, prevention & treatment of organ dysfunction in our attempt to improve the short and long term outcome of patients with peritonitis.

CG Nwigwe⁴⁴ et al (2007) studied 67 consecutive patients with generalized peritonitis. The clinical data and the Mannheim peritonitis index were collected manually for each patient after the surgery. The Mannheim peritonitis index score was related to mortality and the long stay in the hospital which donates morbidity. The mean MPI score for survivors was 30.6, for MPI > 30 mortality was 92.3%. In this study the cut off point for Mannheim peritonitis index of 25 was associated with the highest degree of accuracy.

Cristian P. Schneider⁴⁵ et al evaluated a cohort of 319 consecutive patients with secondary peritonitis requiring intensive care treatment. Four month mortality was calculated and it was related to various variables such as the sex, emergency admissions, main source of infection, adequacy of empirical antibiotic therapy, presence of malignancy, palliative or curative surgery, need for ventilation and admission, presence of pneumonia/ liver cirrhosis, age, APACHE II Score on admission day, duration of artificial ventilation, duration of catecholamine therapy, need for renal replacement therapy, no. of transfused red cell units and need for surgical revisions. Four month survival after ICU admission was 31.7%, 1 year survival was 82.7%. Inability to control the source of infection was the most important determinant of mortality.

Other factors associated with worse 4 monthly survival were high disease severity at ICU admission and during the ICU stay, specific co morbidities such as the extended malignancies and presence of liver cirrhosis, source of infection being distal oesophagus and stomach and inadequate empirical antibiotic therapy.

Chao-Wen-Hsu⁴⁶ studied spectrum of malignant small bowel perforations data on 19 patients (6 women and 13 men), with malignancy perforation through small bowel tissue was

retrospectively reviewed. The median patient age was 57 year (range, 41-48 years). The histopathology included lymphoma (seven patients), leiomyosarcoma (two patients), metastatic carcinomas with unknown primary tumour (seven patients), gastrointestinal stromal tumour (one patient), adenocarcinoma (one patient), metastatic carcinomas with unknown primary tumour (four patients) and metastatic adenocarcinoma from the lung (one patient). Resection of a segment of perforated bowel with primary anastomosis was performed in 16 patients, wedge resection of perforated lesion with plication in two patients, and loop ileostomy in one patient. Postoperative deaths occurred in 10(52.6%) patients, owing to sepsis and organ functional failure, seven patients died from the primary malignancy at a median follow up of 6.5 month (range, 5 months to 1 year 9 months) after surgery. Moreover, two patients with small bowel lymphoma were alive with disease at 4 years 8 months and 7 years 1 month after surgery. In conclusion, perforation through small bowel malignant tumours had a high postoperative mortality rate. High index of suspicion of the disease with early surgical treatment may improve treatment outcomes.

A Mishra⁴⁷ et al of G. I. Surgery Unit, Department of Surgery, NSCB Government Medical College, Jabalpur, Madhya Pradesh Constructed and Assessed a simplified scoring system for peptic perforation, which can be easily used in developing countries. One hundred and forty consecutive patients with perforated pre-pyloric or duodenal ulcer undergoing Graham patch omentopexy closure were studied prospectively. Each factor was given a score based on its severity in accordance with the APACHE –II scoring system to construct the simplified prognostic (Jabalpur) Scoring system, and multiple regression analysis was used to identify risk factors, this system was prospectively validated in the next 50 consecutive patients and compared to existing systems. Factors associated with mortality were age, presence of co-morbid illness, perforation- to- operation interval, preoperative shock, heart rate, and serum creatinine. The mean score in survivors (4.9) was less than that in those who died (12.5 p<0.0001). This scoring system compared favourably with other scoring systems. The Jabalpur scoring system is effective for prognostication in case of peptic perforation. It is simple and user-friendly as it uses only six routinely documented clinical risk factors.

VI. OBSERVATION AND ANALYSIS

Table 1a: Number of patients in each age group

Age group (years)	Frequency	Percent
<15	14	7.0
15-30	28	14.0
31-45	55	27.5
46-60	57	28.5
61 and above	46	23.0

The mean age of the study group was 43.74 years and the age group of 46-60 contains maximum (28.5%) patients followed by 31 – 45 years. Oldest patients was 84 years and youngest was of 9 days.

Table 1b: Showing age distribution of the patients (< 50 years & > 50 years)

Age groups (years)	Frequency	Percent
<50	119	59.5
50 or more	81	40.5

Table 2: Showing sex distribution of the patients

Sex	Frequency	Percent
Male	136	68.0
Female	64	32.0

Table 3: Showing anatomical site of perforation in study patients

Site of perforation	Frequency (n = 200)	Percent
DUODENAL PERFORATION	67	33.5
COLONIC PERFORATION	25	12.5
ILEAL PERFFORATION	51	25.5
APPENDICULAR PERFORATION	16	8.0
GASTRIC PERFORATION	13	6.5
JEJUNAL PERFORATION	7	3.5
GALL BLADDER PERFORATION	7	3.5
MECKEL'S DIVERTICULUM PERFORATION	3	1.5
URINARY BLADDER PERFORATION	3	1.5
CBD PERFORATION	2	1.0
ILEAL AND UTERINE PERFORATION	1	.5
UTERINE PERFORATION	3	1.5
HYDATID CYST PERFORATION	1	.5
RECTAL PERFORATION	1	.5

As the table shows maximum number of patients had duodenal perforation 67

(33.5 %) followed by Ileal perforation 51 (25.5 %). Colonic, appendicular and gastric perforations were 25 (12.5 %), 16(8 %) and 13(6.5%) respectively. Rectal, hydatid cyst of liver and combined ileal - uterine perforations were among the least common perforations i.e.1 (0.5 %).

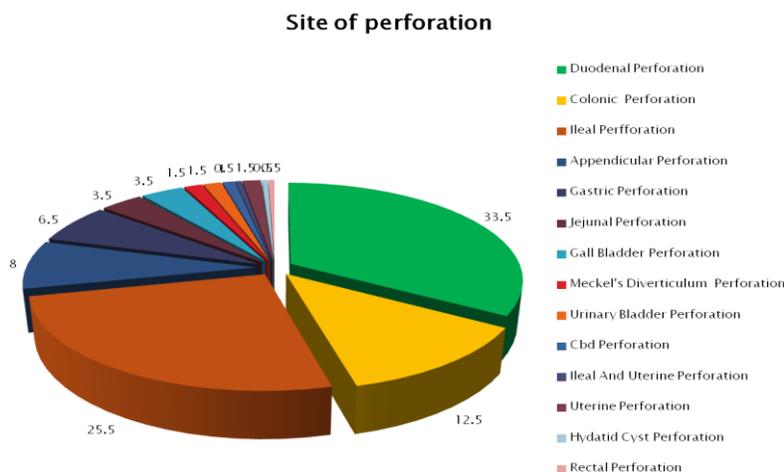


Figure 1: Showing anatomical site of perforation in study patients

Table 4: Showing various clinical features in patients with peritonitis

Symptoms		Frequency	Percent
Abdominal pain	Absent	6	3.0
	Present	194	97.0
Distension	Absent	94	47.0
	Present	106	53.0
Not passed Flatus	Absent	118	59.0
	Present	82	41.0
Not passed Stools	Absent	119	59.5
	Present	81	40.5
Fever	Absent	84	42.0
	Present	116	58.0
Vomiting	Absent	77	38.5
	Present	123	61.5

The commonest symptom was abdominal pain seen in 194 (97 %) patients followed by vomiting & abdominal distension seen in 123 (61.5 %) & 106 (53%) respectively.

Table 5: Showing various intraoperative procedures done

Intraoperative Procedures	Frequency	Percent
EL & OMENTAL PATCH REPAIR	63	31.5
LAPAROSCOPIC CLOSURE OF PERFORATION	1	.5
EL & PERITONEAL LAVAGE WITH DRAIN TUBE INSERTION	7	3.5
TRANSEVERSE COLOSTOMY	4	2.0
TUBE DUODENOSTOMY WITH FJ	1	.5
EL WITH RA (RESECTION & ANASTOMOSIS)	27	13.5
EL WITH PRIMARY CLOSURE	57	28.5
EL WITH RA WITH ILEOSTOMY	3	1.5
EL WITH HEMICOLECTOMY	4	2.0
EL WITH APPENDECTOMY	13	6.5
EL WITH ILEOSTOMY	2	1.0
DISTAL GASTRECTOMY WITH GJ	2	1.0
HARTMANN'S PROCEDURE	3	1.5
HEMICOLECTOMY WITH ILEOSTOMY	3	1.5
EL WITH PRIMARY CLOSURE WITH TRANSEVERSE COLOSTOMY	3	1.5
EL WITH CHOLECYSTECTOMY	7	3.5

Most common procedure performed was exploratory laparotomy with omental patch repair in 63 (31.5 %) patients followed by primary closure and resection and anastomosis in 57(28.5%) and 27 (13.5%) patients respectively 1(0.5%) patient underwent laparoscopic closure of duodenal perforation 1(0.5%) patient underwent tube duodenostomy with feeding jejunostomy.

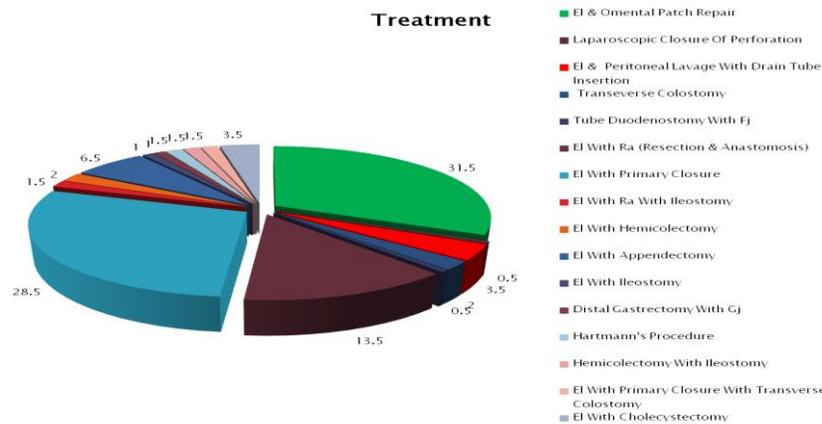


Figure 2: Showing various intraoperative procedures done.

Table 6: Showing distribution of organ failure in patients with peritonitis

Organ failure	Frequency	Percent
Absent	155	77.5
Present	45	22.5

In present study 45 i.e.22.5 % patients showed presence of organ failure

Table 7: Showing preoperative duration wise distribution of patients

	Preoperative duration	Frequency	Percent
1.	<24 hours	32	16.0
2.	24 hours and more	168	84.0

Table 8: Showing presence of malignancy in patients with peritonitis

Malignancy	Frequency	Percent
Absent	186	93.0
Present	14	7.0

Table 9: Table showing origin of sepsis (colonic /noncolonic) in our study

Origin of sepsis	Frequency	Percent
Non-colonic	175	87.5
Colonic	25	12.5

In our study 25 i.e.12.5 % patients origin of sepsis was colonic while in 175 i.e. 87.5 % patients origin of sepsis was noncolonic.

Table 10: Showing type of peritonitis in study population

Type of peritonitis	Frequency	Percent
Localised	26	13.0
Diffuse	174	87.0
Total	200	100.0

In our study 174 i.e. 87 % patients had Diffuse peritonitis while 26 i.e.13 % had localised peritonitis

Table 11: Showing character of exudates in study population

Exudate	Frequency	Percent
Clear	40	20.0
Purulent	124	62.0
Fecal	36	18.0

In our study 124 (62%) patients had purulent exudates while clear & fecal exudates were present in 40 (20%) & 36 (18%) patients respectively.

Table 12: Showing MPI score wise distribution of patients

MPI	Frequency	Percent
<21	87	43.5
21-29	70	35.0
>29	43	21.5

In 87 (43.5 %) patients total MPI score was < 21 while 70 (35%) patients total score was 21-29 & it was > 29 in 43 (21.5%) patients

Table13: Showing outcome of the patients in our study

Outcome	Frequency	Percent
Discharged	168	84.0
Death	32	16.0

Table 14: Showing mortality in each age group

Age Group (years)		Mortality in age group	Total mortality
<15	N	0	0
	%	.0%	.0%
15-30	N	1	1
	%	3.6%	3.1%
31-45	N	3	3
	%	5.5%	9.4%
46-60	N	14	14
	%	24.6%	43.8%
61 and above	N	14	14
	%	30.4%	43.8%

The highest mortality was in the age group 61 years & above followed by 46 - 60 years. The lowest mortality was in the age group < 15 years followed by 15-30 years.

Table 15: Showing correlation of Age > 50 yrs with incidence of mortality

Age group	Outcome		Mortality according to age	X ²	Df	p-value
	Discharged	Death				

			group			
<50 (n)	112	7	7	22.12	1	<0.001
%	94.1%	5.9%	21.9%			
50 or more (n)	56	25	25			
%	69.1%	30.9%	78.1%			

$X^2 - 22.12$ $d(f) - 1$ $p - < 0.001$

In correlation between Age > 50 yrs with incidence of mortality, our study showed statistically significant result with $p < 0.001$

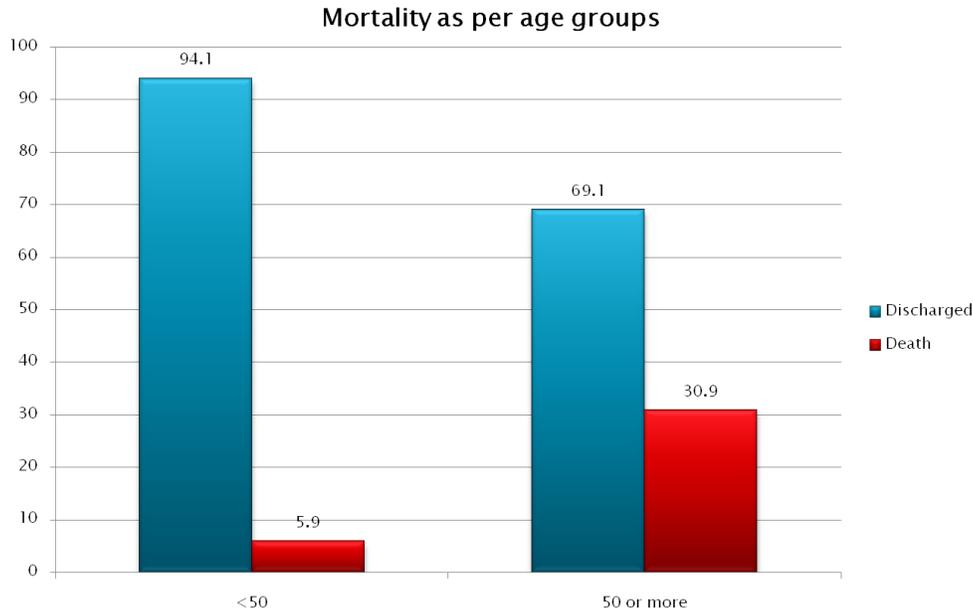


Figure 3: Showing correlation of Age > 50 yrs with incidence of mortality

Table 16: Showing correlation of sex with incidence of mortality

Sex	Outcome		Mortality according to gender	X^2	Df	p-value
	Discharged	Death				
Male (n)	112	24	24	0.858	1	0.354
%	82.4%	17.6%	75%			
Female (n)	56	8	8			
%	87.5%	12.5%	25%			

$X^2 - 0.858$ $d(f) - 1$ $p - 0.354$

In correlation of sex with incidence of mortality, p value in our study was 0.354 which is statistically not significant & shows contrast results with MPI.

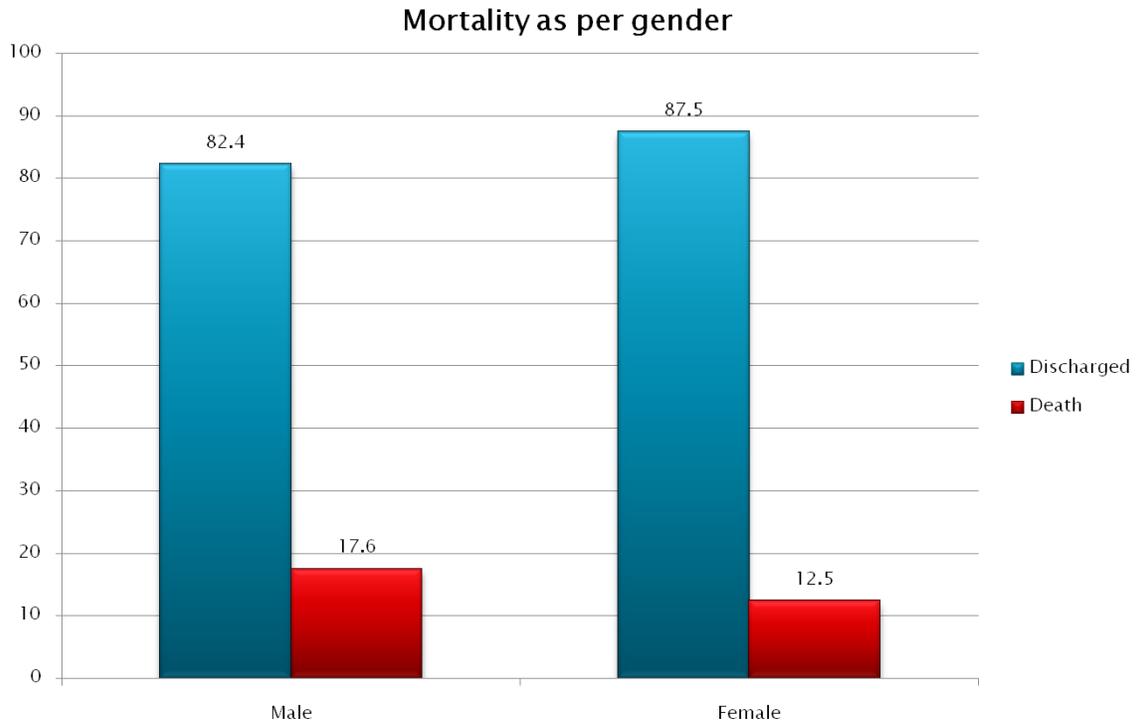


Figure 4: Showing correlation of sex with incidence of mortality

Table 17: Showing correlation of organ failure with incidence of mortality

Organ failure	Outcome		Mortality according to Organ failure	X ²	Df	p-value
	Discharged	Death				
Absent (n)	153	2	2	110.91	1	<0.001
%	98.7%	1.3%	6.3%			
Present (n)	15	30	30			
%	33.3%	66.7%	93.8%			

X²- 110.91 d(f)- 1 **p<0.001**

In correlation of organ failure with incidence of mortality p value in our study was **< 0.001** which is statistically significant.

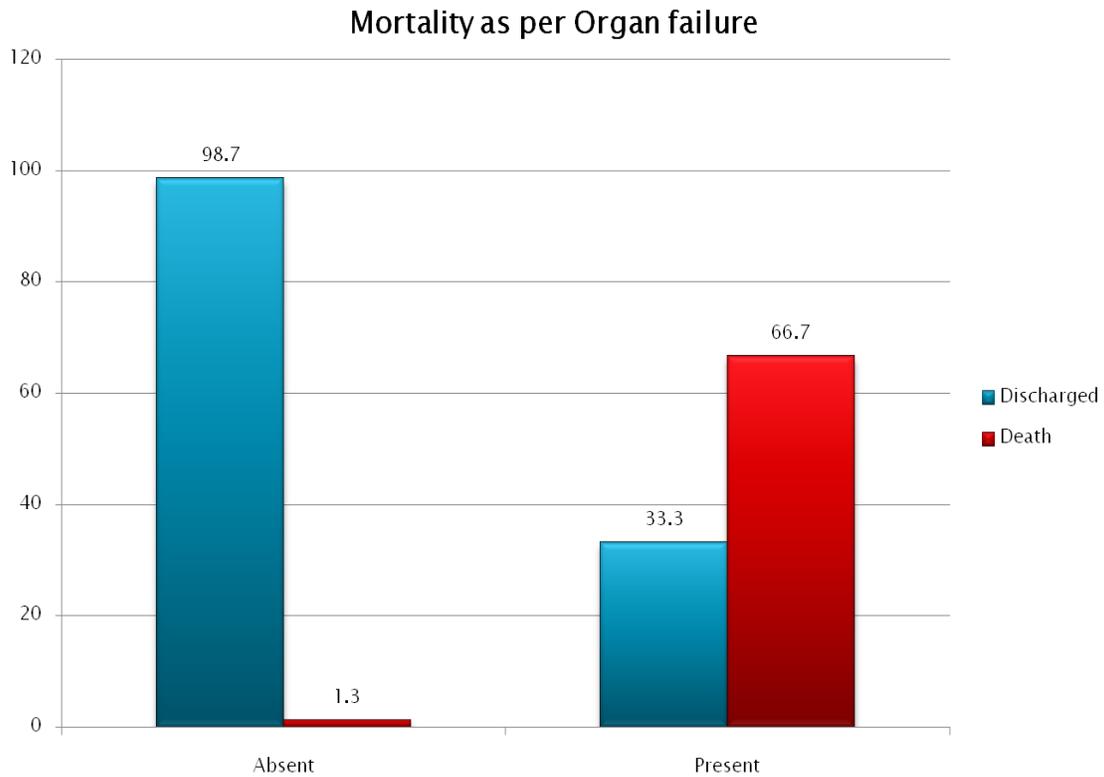


Figure 5: Showing correlation of organ failure with incidence of mortality

Table 18: Showing correlation of preoperative duration with incidence of mortality

Pre-op duration	Outcome		Mortality according to Pre-op duration	X ²	Df	p-value
	Discharged	Death				
<24 hours (n)	32	0	0	7.256	1	0.007
%	100.0%	.0%	0			
24 hours and more (n)	136	32	32			
%	81.0%	19.0%	100%			

X²- 7.256 d (f) -1 p - <0.007

In correlation of preoperative duration with incidence of mortality, our study showed statistically significant result with **p <0.007**.

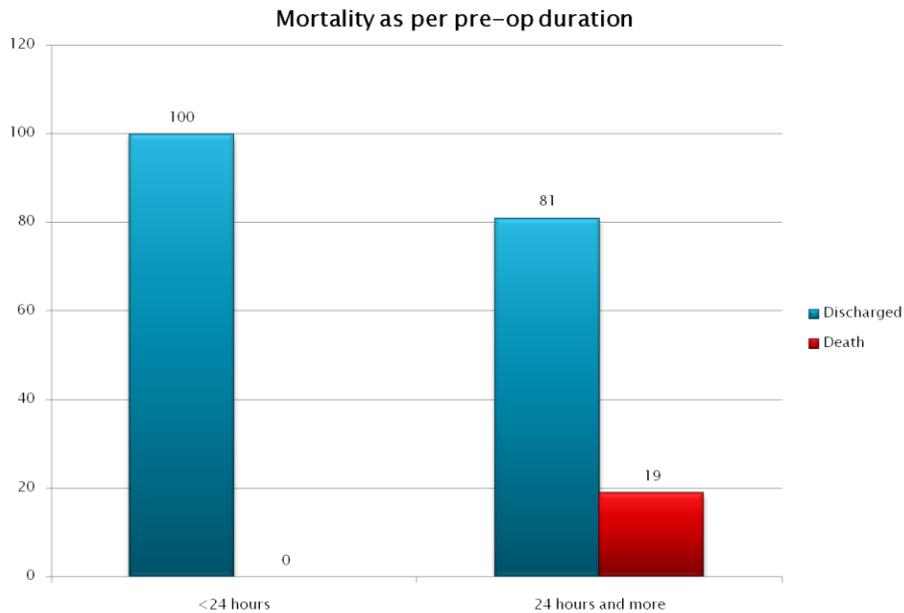


Figure 6: Showing correlation of preoperative duration with incidence of mortality

Table 19: Showing correlation between presence of malignancy with incidence of mortality

Malignancy	Outcome		Mortality according to Malignancy	X ²	Df	p-value
	Discharged	Death				
Absent (n)	163	23	23	26.12	1	<0.001
%	87.6%	12.4%	71.9%			
Present (n)	5	9	9			
%	35.7%	64.3%	28.1%			

X² – 26.12 d(f) – 1 p - <0.001

In correlation between presence of malignancy with incidence of mortality, our study showed statistically significant result with p <0.001

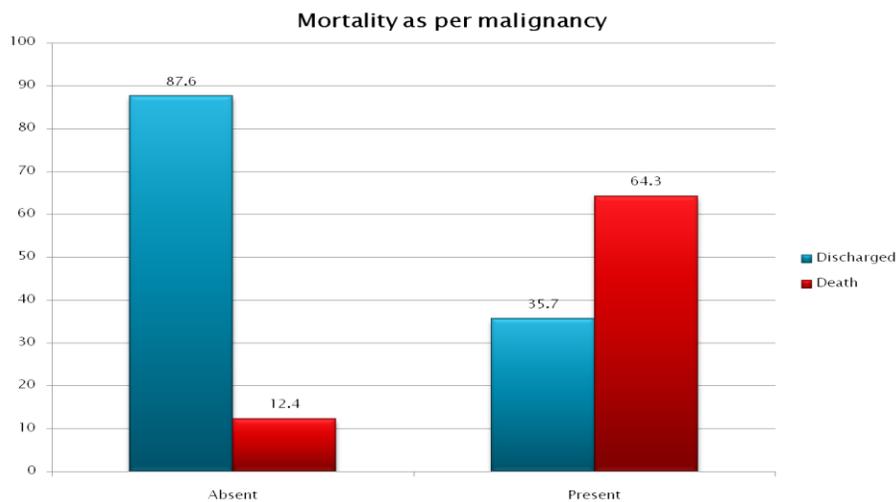


Figure 7: Showing correlation between presence of malignancy with incidence of mortality

Table 20: Showing correlation between type of peritonitis with incidence of mortality

Peritonitis	Outcome		Mortality according to Peritonitis	X ²	Df	p-value
	Discharged	Death				
Localised (n)	26	0	0	5.692	1	0.017
%	100.0%	.0%	0			
Diffuse (n)	142	32	32			
%	81.6%	18.4%	100			

$X^2 - 5.692$ $d(f) - 1$ $p - <0.017$

In correlation between type of peritonitis with incidence of mortality, our study showed statistically significant result with $p < 0.017$

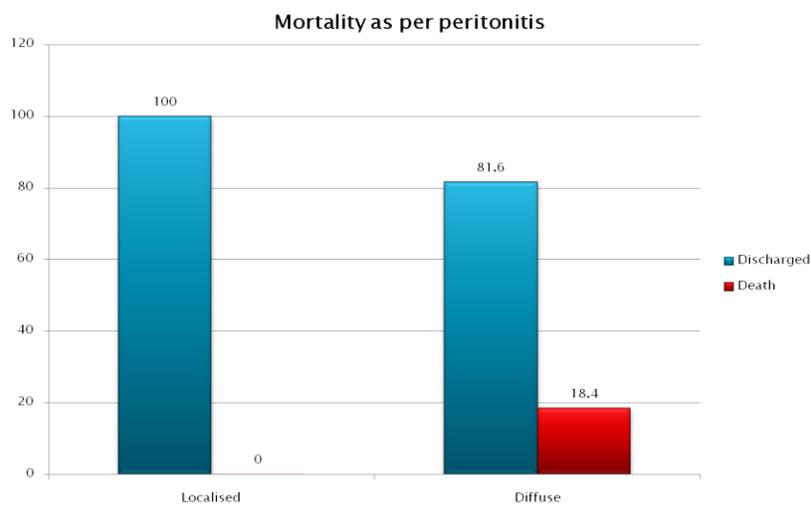


Figure 8: Showing correlation between type of peritonitis with incidence of mortality

Table 21: Showing correlation between origin of sepsis (colonic / noncolonic) with incidence of mortality

Diagnosis	Outcome		Mortality according to diagnosis	X ²	Df	p-value
	Discharged	Death				
Non Colonic(n)	150	25	25	3.061	1	0.145
%	85.7%	14.3%	78.1%			
Colonic (n)	18	7	7			
%	72.0%	28.0%	21.9%			

$X^2 - 3.061$ $d(f) - 1$ $p - <0.145$

In correlation between origin of sepsis (colonic / noncolonic) with incidence of mortality p value in our study was 0.145, which is statistically not significant & shows contrast results with MPI.

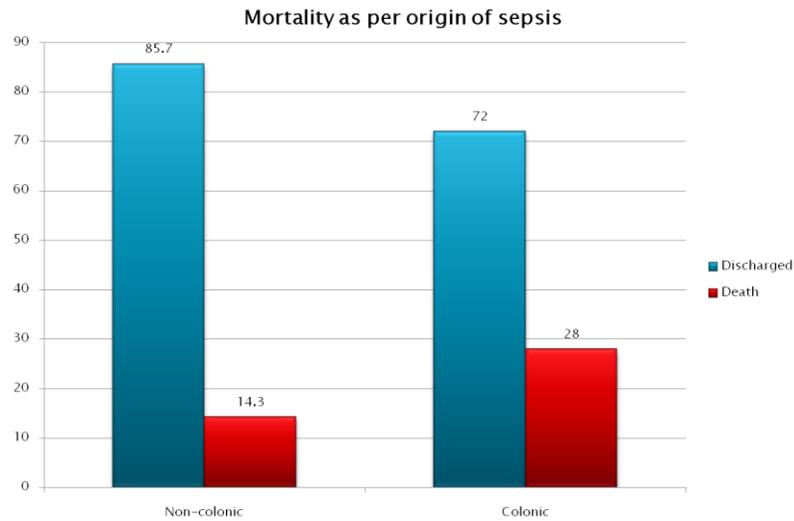


Figure 9: Showing correlation between origin of sepsis (colonic / noncolonic) with incidence of mortality

Table 22: Showing correlation between character of exudate with incidence of mortality

Exudate	Outcome		Mortality according to exudates	X ²	Df	p-value
	Discharged	Death				
Clear (n)	38	2	2	18.52	2	<0.001
%	95.0%	5.0%	6.3%			
Purulent (n)	108	16	16			
%	87.1%	12.9%	50%			
Fecal (n)	22	14	14			
%	61.1%	38.9%	43.8%			

X²- 18.52 d (f) – 2 p- < 0.001

In correlation between character of exudate with incidence of mortality, our study showed statistically significant result with **p < 0.001**

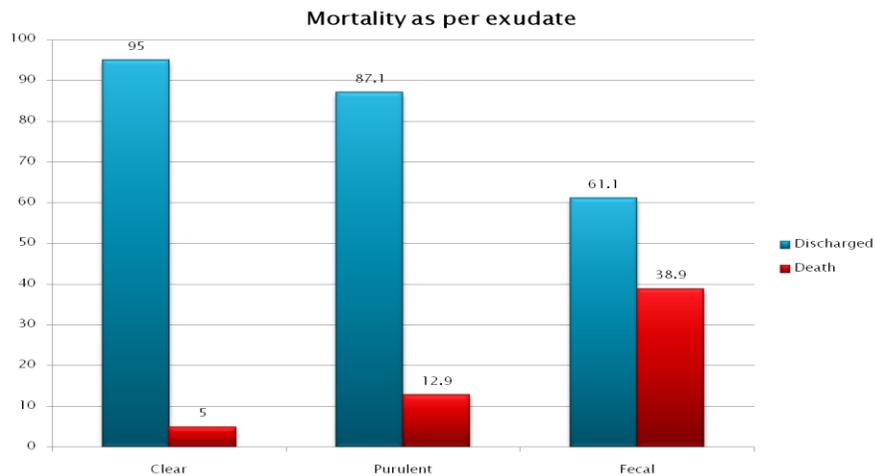


Figure 10: Showing correlation between character of exudate with incidence of mortality

Table 23: Showing duration of hospital stay of the patients

Hospital stay	Frequency	Percent
<10 days	43	21.5
10-20 days	125	62.5
21-30 days	24	12.0
>30 days	8	4.0

In our study most of the patients i.e. 125 (62.5 %) stay in the hospital for 10 - 20 days while 8 (4 %) patients stay in the hospital for > 30 days.

Table 24: Showing correlation of MPI score with incidence of mortality

MPI	Outcome		Mortality according to MPI	X ²	Df	p-value
	Discharged	Death				
<21 (n)	87	0	0	108.38	2	<0.001
%	100.0%	.0%	0			
21-29 (n)	67	3	3			
%	95.7%	4.3%	9.4%			
>29 (n)	14	29	29			
%	32.6%	67.4%	90.6%			

X²- 108.38 d(f)- 2 p-<0.001

In our study mortality rate among patients with MPI score > 29 was 67.4% and with MPI < 21 was 0, which is statistically significant with p <0.001

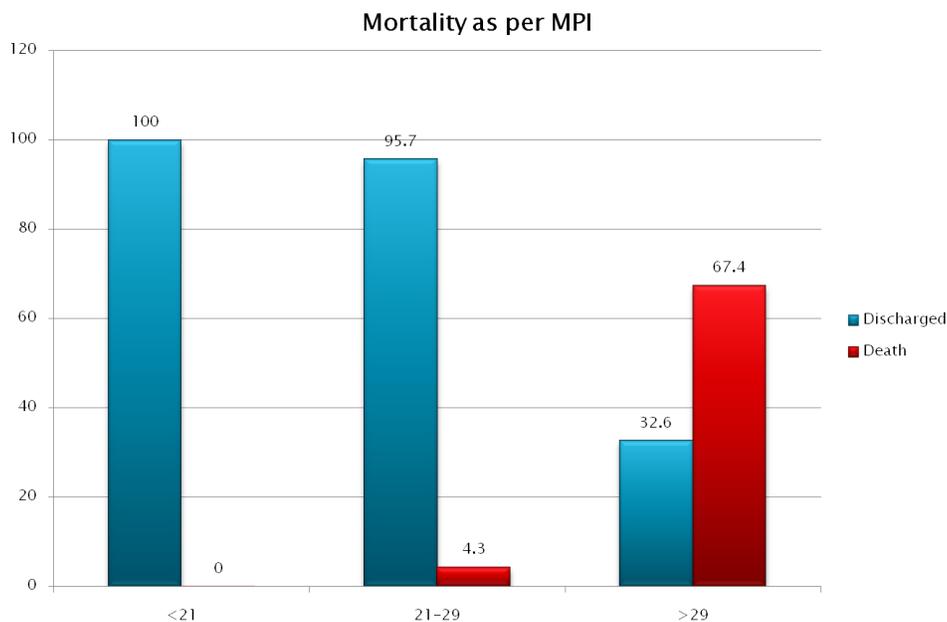


Figure 11: Showing correlation of MPI score with incidence of mortality

Table 25: Showing distribution of MPI variables and outcome of patients

Variables		Outcome		p-value
		Discharged	Death	
50 or more years (n)		56	25	<0.001
%		69.1%	30.9%	
Female (n)		56	8	0.354
%		87.5%	12.5%	
Organ failure (n)		15	30	<0.001
%		33.3%	66.7%	
24 hours or more duration (n)		136	32	0.007
%		81.0%	19.0%	
Malignancy (n)		5	9	<0.001
%		35.7%	64.3%	
Diffuse peritonitis (n)		142	32	0.017
%		81.6%	18.4%	
Exudate	Clear (n)	38	2	<0.001
	%	95.0%	5.0%	
	Purulent (n)	108	16	
	%	87.1%	12.9%	
	Fecal (n)	22	14	
	%	61.1%	38.9%	
Noncolonic (n)		150	25	0.08
%		85.7%	14.3%	

In our study correlation between noncolonic origin of sepsis and female sex with outcome did not showed statistically significant results while other factors showed statistically significant results.

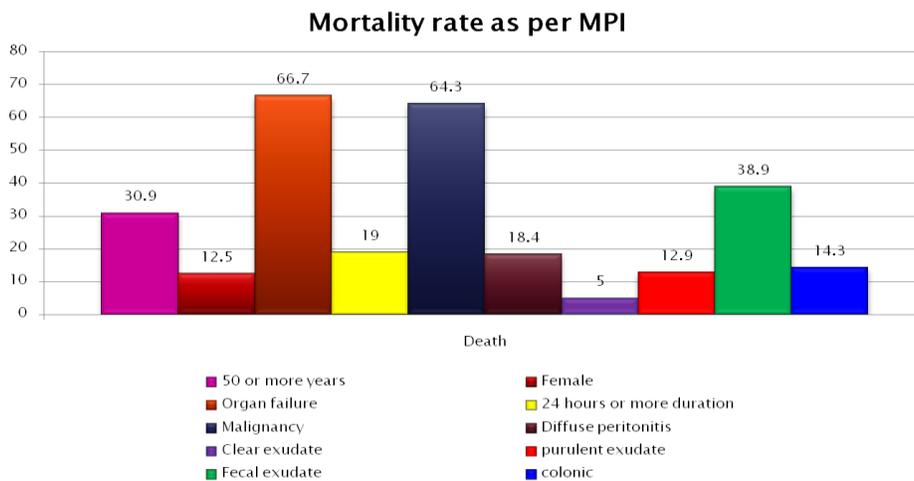


Figure 12: Showing distribution of MPI variables and outcome of patients

Table 26: Showing microbiological profile in patients with peritonitis

S. no.	Type of microorganism	Number of patients
1.	Sterile	20

2.	E.Coli	15
3.	Klebsiella	08
4.	Pseudomonas	06
5.	Enterococcus	01

Random sample of 50 patients were drawn and it showed above mentioned results.

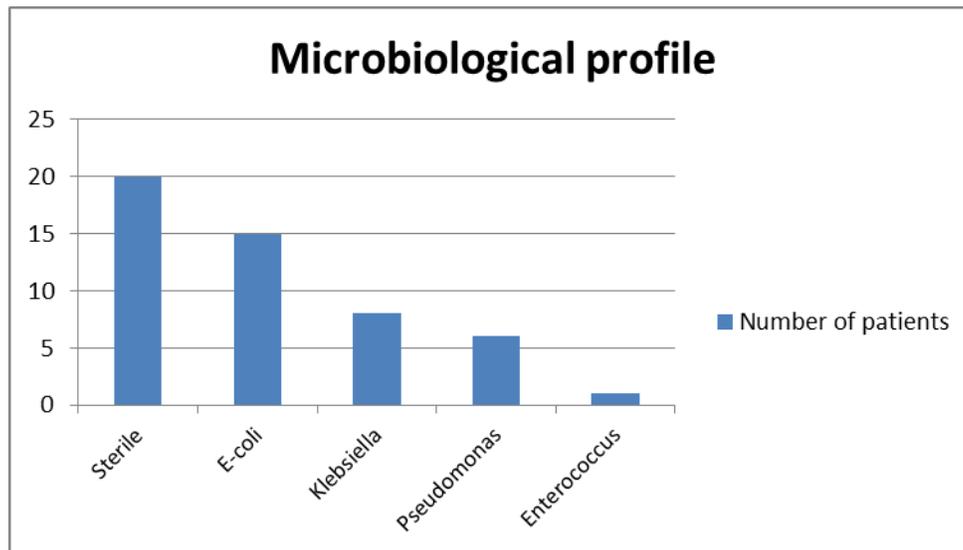


Figure 13: Showing microbiological profile in patients with peritonitis

VII. DISCUSSION

SPECTRUM OF PERFORATION PERITONITIS

AGE (Table 1a & 1 b)

- Total of 200 patients were studied.
- The age range is from 9 days to 84 years. The mean age of the study population was 43.7 years.
- The highest numbers of patients were found in the age group of 46-60 years and they constitute about 28.5% of the study population.

In a study by Rajendra Singh Jhobta³⁹ et al (2006) the mean age was 36.8 years and the age range was 3 years to 90 years. In a study by Aijaz A Memon⁴⁸ (2008) in which the spectrum of acute abdomen was studied the age range was from 13 years to 87 years.

AUTHOR	YEAR	AGE (yrs)
Ohmann C et al	1997	56
Roduez et al	1999	39.8
Corriea et al	2001	58.9
Rodofo L et al	2002	34.6
Present Study	2011	43.7

- } The number of patients in the age group <50 years were 119 i.e. 59.5% and 81 patients of the study population i.e. 40.5% were in the age group >50 years.

- } The increased prevalence of the perforation in the age group of 31- 60 years in our study can be attributed to the fact that gastro duodenal perforations due to peptic ulcer disease is a major cause of perforation peritonitis in our study and the increased prevalence of the etiological risk factors such as smoking, alcoholism and NSAID abuse in this age group.
- } After increasing steeply at the beginning of the twentieth century perforation incidence during the last decade has declined in young and has risen among elderly. These changes can be attributed to the cohort phenomenon: ulcer perforation risk is particularly common in the cohorts born after the turn of 20 century and is less common in previous and succeeding cohorts³⁰.
- } It is also attributed to the increased numbers of traumatic perforations in the younger age group leading to parallel increase in the overall prevalence of perforation peritonitis in this age group. Also appendicular perforation is more common in the age group of 20-30 years but no age is exempted. Majority of the ileal perforations are seen in the age group of 10-30 years, typhoid being the main etiological factor.

Sex (Table 2)

- } In our study the incidence of male sex was 68 % while that of female sex was 32 %.
- } In a study by Rajender Singh Jhobta³⁹ (2006) regarding the spectrum of perforation peritonitis in India 84% patient’s were male.

AUTHOR	YEAR	FEMALE	MALE
Tripathi et al.	1993	45.5 %	54.5 %
Yilmazlar et al.	1999	37 %	63 %
Corriea et al.	2001	26.7 %	73.3 %
Rudolfo L et al.	2002	48 %	52 %
Present study	2011	32 %	68 %

In a study by Rajender Singh Jhobta³⁹ regarding the spectrum of perforation peritonitis in India , 422 of the 504 patients studied were males i.e.84%.

In a study by Aijaz A Meman⁴⁸ (2008) et al about the spectrum of disease in patients with acute abdomen, 70.30 % was males and 29.69% were females.

In a study by Rudolfo L³¹ (2004) out of the 174 patients, 84 were females (48%) and 90 were males (52%).

- The increased prevalence of male sex in our study is mainly due to increased number of male patients in the category of duodenal perforation

SITE OF PERFORATION (Table 3)

In our study duodenal perforations account for 33.5%, illeal perforation for 25.5%, colonic perforation for 12.5%, appendicular perforations for 8% ,gastric perforation for 6.5 %, jejunal and gall bladder perforations for 3.5 % each, meckel’s diverticulum and urinary bladder perforations for 1.5 % each & CBD perforation in 1 % of patients.

Rectal and hydatid cyst of liver perforations were among the least common perforations, constituting 0.5 % in each group.

In a study by Rajender Singh Jhobta et al³⁹ (2006) the result was as below:

duodenum 57%, gastric 8%, jejunal 3%, ileal 15%, appendicular 12%, colonic 4% and oesophageal 0.5 %.

SITE	Tripathi et al (1993)	Desa L.A et al (1983)	Kachroo et al.(1984)	Bohner et al.(1999)	Present study
Duodenal	15 %	32.29 %	18.7 %	22.7 %	33.5 %
Ileal	24.5 %	27.3 %	15 %	-	25.5 %
Appendicular	10 %	18.1 %	41.1 %	15.9 %	8 %
Others	50.5%	22.31%	25.2%	61.4%	36%

In a study by Rodolfo L³¹ et al appendicular perforations constitute 48.28% while gastric pathology and small bowel pathology constitutes 2.87% each and colonic pathology 2.30%. The increased number of duodenal perforations in our study is due to increased prevalence of the acid peptic disease. Also the increase number of jejunal perforations all of which were secondary to trauma reveals the hazard of trauma.

The perforations of the proximal gastro intestinal tract were six times as common as the perforations of the distal gastrointestinal tract as has been noted by earlier studies from India. This is in sharp contrast to studies from the developed countries which reveal that distal gastrointestinal tract perforations are more common³¹.

Clinical features (Table 4)

- } In our study pain in abdomen was the most common symptom and 97 % of patients had pain abdomen at presentation while 41% of patients have difficulty in passing flatus or motion.
- } Distension of abdomen was present in 53 % of patients, 61.5% patients had episodes of vomiting, 58 % patients had fever at presentation.
- } In a study by Shantanu Kumar Sahu et al⁴⁹ the commonest presenting symptom was abdominal pain (100%), followed by distension of abdomen (82%), constipation, vomiting and fever.
- } In a study by Rajender Singh Jhobta et al³⁹ pain was present in 98% of patients, followed by vomiting (59%), abdominal distension (44%), constipation (58%), fever (35%), and diarrhoea (7%).
- } Perforation peritonitis is a clinical condition with a wide spectrum of presentation and high index of suspicion is always warranted.
- } Not every patients of perforation peritonitis will present with signs of pain, distension of abdomen, guarding and rigidity of the anterior abdominal wall. A thorough examination from head to toe is mandatory in every patient.
- } Diagnosis of perforation peritonitis is always clinical and immediate resuscitative measures should be initiated.
- } Radiological investigations are only for the confirmation of diagnosis. Unnecessary investigations unless the diagnosis is in doubt should be avoided, and after initial adequate resuscitation exploratory laparotomy should be done in an emergency basis. Delay in treatment can lead to the development of sepsis and multiorgan failure with concomitant increase in morbidity and mortality of patients

INTRA OPERATIVE PROCEDURE DONE (Table 5)

- } EXPLORATORY LAPAROTOMY and OMENTAL PATCH REPAIR was done in patients with duodenal perforation.
- } 1 patient underwent laproscopic closure.
- } 27 (13.5 %) patients underwent resection and anastomosis, while in 3 (1.5%) patients ileostomy was done along with resection and anastomosis.
- } 3 (1.5 %) patients underwent hemicolectomy.
- } 3 (1.5 %) patients underwent transeverse colostomy.
- } 3 (1.5 %) patients underwent Hartmann’s procedure.

In our study no patients was managed by a definitive procedure for acid peptic disease. All the patients with gastroduodenal perforation due to acid peptic disease were prescribed proton pump inhibitor at the time of discharge.

Primary closure of the gastroduodenal perforation with edge biopsy with omental patch was done in all of the cases of gastroduodenal perforations of peptic origin.

In the study by Rajender Singh Jhobta³⁹ 304 patients i.e. 60% were managed by simple closure of perforations, 46 patients i.e. 9% were managed by resection and anastomosis. Resection

without anastomosis was done in 64 patients, ileostomy /colostomy with mucous fistula / Hartmann’s procedure was done in these patients. Definitive procedure in the form of Billroth I and II gastrectomy with truncal vagotomy and drainage procedure was done in 33% patients and appendicectomy in 57 patients.

- } The classic, pedicled omental patch i.e. performed for the plugging of this perforation was first described by Cellan Jones in 1929, although it is commonly and wrongly attributed to Graham, who described the use of a free graft of the omentum to repair the perforation in 1937. However large perforations may be encountered in which there exists the threat of postoperative leakage following closure by the simple method. Here other surgical options such as partial gastrectomy, jejunal serosal patch, jejunal pedicled graft or even gastric disconnection may be deemed necessary for the secure closure.

DISTRIBUTION OF ORGAN FAILURE (Table 6)

- } In our study 45 patients i.e.22.5% of the study population shows evidence of organ failure at presentation.

Distribution of organ failure in different studies are –

- 48.5 % in MM Correia et al²⁹
- 11.5 % in Rodolfo L et al³¹
- 20 % in Murut Kologlu et al²⁸

- } In peritonitis a systemic inflammatory response induced by the peritoneal infection may progress to septic shock and multiorgan failure. The high rate organ failure in our study denotes a delay in presentation of most cases.

PREOPERATIVE DURATION (Table 7)

- } In our study 32 patients i.e. 16 % presented within 24 hours while 168 patients i.e. 84 % presented after 24 hours of onset of the disease
- } In other studies the distribution of preoperative duration is as below-

Study	<24 hrs.	>24 hrs.
Rodolfo L ³¹	49.42%	54.48%
MM Correia ²⁹	34.5%	65.5%

In our institute the cause of delayed presentation i.e. a preoperative duration of peritonitis more than 24 hours was mainly related to the

- } Illiteracy among the study population
- } Lack of proper referral services
- } In some patients the delay was due to diagnostic dilemma which demands early use of more sophisticated investigations like CT scan, which is not available at the peripheral hospitals

PRESENCE OF MALIGNANCY (Table 8)

- } In our study 14 patient's (7 %) had malignancy. 9 were cases of colonic malignancy with perforation and 3 were of carcinoma stomach with perforation and 2 had a malignancy as an associated finding.
- } In a study by Rodolf L³¹ 2 patients had malignancy.
- } In a study by M.M. Correia²⁹ 89 patients with cancer were studied. Among them 8 were preoperative and all other were postoperative. Chronic use of NSAIDs in patients of malignancies exposes them to an increased risk of perforation.

ORIGIN OF SEPSIS (Table 9)

- } In our study 25 patients i.e. 12.5 % had colonic origin of sepsis while in the rest 175 patients the origin of sepsis was non colonic.
- } In the study by Rudolf L³¹ 12.64% of patient's had colonic origin of sepsis.
- } In the study by Rajendra Singh Jobhta³⁹ 3.76% of patient's had colonic origin of sepsis.
- } The various causes of the perforation of the colon are trauma, diverticulum perforation, perforated malignancy and mesenteric ischaemia.
- } Colonic perforation presents with faecal exudates and a severe form of peritonitis.

TYPE OF PERITONITIS (Table 10)

- } In our study patients 174 i.e. 87% presented with a diffuse form of peritonitis while the remaining 26 i.e. 13 % presented with localized peritonitis.
- } In other study the distribution of type of peritonitis was as below

<u>STUDY</u>	<u>DIFFUSE</u>
<u>LOCALISED</u>	
Rajender Jhobta ³⁹ 17 %	83 %
Rodolf L ³¹ 65.51	34.49 %
Ohmann ¹ 34.64 %	65.36 %

- } Diffuse peritonitis is associated with a severe inflammatory reaction and development of sepsis and multiorgan failure.

Localization of peritonitis is body's defense mechanism and will lead to formation of abscess.

NATURE OF EXUDATES (Table 11)

- } In our study 40 patients i.e. 20 % had clear exudates, 124 patients i.e. 62 % had purulent exudates and 36 patients i.e. 18 % had faecal exudates.
- } In a study by Rodolf L³¹ 69.5% has clear exudates and 21.8% had purulent exudates.
- } In a study by Rajender Singh Jhobta³⁹ 15% had clear exudates, 71% had purulent and 13% had faecal exudates.

- } Purulent and faecal exudates are associated with delayed presentation and presence of varying degree of septicaemia.

DISTRIBUTION OF PATIENTS AS MPI CUT OFF POINTS (Table 12)

- } 87 (43.5%) patients had MPI score of less than 21.
- } 70 (35%) patients had MPI score between 21 to 29
- } 43 (21.5%) patients had MPI score greater than 29
- } Of the present prognostic scoring system the Mannheim Peritonitis Index is one of the easiest to apply and the determination of risk is easily available during the initial operation. Retrospective data collection is possible and valid, as only standard information available from the operation report of the patients record is required.
- } In the original study by Wacha and Linder³ the cut off point of 26 MPI point was used. But in our study many patients had attended higher values in the range of 40 (due to presence of malignancy and faecal contamination) so a lower cut off value of 21 MPI point was used so that the sensitivity and the specificity of the study could be increased.

OUTCOME (Table 13)

- } Among the 200 patients studied by us 32 patients died thus placing the mortality at 16%.
- } *Atsushi Hourichi*⁴⁰ in their study of perforation peritonitis had a mortality of 23.1%.
- } *Koperna T*³⁰ et al in their study of secondary bacterial peritonitis had a average total mortality rate of 18.5%.
- } The mortality rate in various studies on perforation peritonitis ranges between 20 to 30%.
- } Thus inspite of improvement in the medical management, availability of new broad spectrum antibiotics and vast development in the field of intensive care with easy availability of intensive care and life support measure the mortality from perforation peritonitis remains high.
- } Development of organ failure and sepsis are important determinants of mortality
- } Therefore research and development should be directed in the understanding of pathogenesis and evolution of these factors so that new and more effective treatment strategies could be evolved.
- } Delay in the presentation for appropriate treatment should be addressed by means of strengthening the referral services and improving the means of transportation.

CORRELATION BETWEEN AGE AND MORTALITY (Table 14 & 15)

- } In our study a total of 119 patients were less than 50 years of age. Out of 119 patients of age less than 50 years 7 (5.9 %) patients died while out of 81 patients with age more than 50 years 25 (30.9 %) patients died
- } In a study by Rodolfo L Braco³¹ the mean age of the survivors was 32.7 years (SD ± 16.64), among non-survivors mean age was 63 years (SD ± 18.94).

Pacelli F et al²³ confirms age as a decisive factor related with mortality. They showed that patients with age of less than 70 years had a mortality rate of 17.2% compared to mortality rate of 37.7% in patients with age more than 70 years.

Ali Yaghoobi Notash et al³⁸ confirms that the risk of in hospital death was higher in patients aged above 60 years.

Kusumoto Yoshiko et al³⁵ in their study of patients operated on for intraabdominal infection found that there was no mortality in less than 50 years age group, while mortality occurring only in patients older than 50 years.

- } Cecilie Svanes¹⁸ et al in their study found that among 581 patients with age < 49, 18 patients died i. e. a mortality of 3.09% , while in patients with age >49 years the mortality was 11.94%.
- } Death and other outcomes of acute surgical illness are uniformly worse in the elderly than in young patients and the adverse impact of age on outcome from abdominal sepsis in particular is well recognized. The higher death rate among the elderly undoubtedly reflects an increased prevalence of pre existing cardiovascular and other diseases as well as a predictable decline in many physiological functions.
- } As patients get older coincident disease are more common. Even if there is no evidence of disease there may be a decrease in the physiological reserve such as the decrease in the glomerular filtration rate despite a normal creatinine. The initial disease that requires surgery may be complicated by tissue hypo perfusion and acidosis from vomiting and loss of fluid into the gastrointestinal tract or bleeding in the elderly population.
- } In our study we confirm that patients over 50 years undergoing emergency surgery for laparotomy have a higher risk of mortality. Mortality after surgery undoubtedly increases with age but this could be because of increased prevalence of comorbid medical conditions in the elderly.

CORRELATION BETWEEN SEX AND MORTALITY (Table 16)

- } In our study total of 136 patients belong to the male sex among which 24 died resulting in a mortality of 17.6 % . Similarly, female sex had a mortality of 12.5 % . & thus female sex has not qualified to be included in the variables of adverse outcome.
- } T M Cook et al²⁹ found out in their study that female sex is one of the parameter associated with death with an odds ratio of 0.21.
- } Yoshiko Kusumoto³⁵ et al found out in their study of 108 patients operated for intra abdominal infections the mortality was 5.3% in men and 15.2% in women
- } In a study by MM Correia²⁹ the factor of female sex has not reached statistical significance between the groups, but it showed a good performance (accuracy of 69.7%) when all MPI components were considered together.

CORRELATION BETWEEN ORGAN FAILURE AND MORTALITY (Table 17)

- } In our study a total of 45 patients showed evidence of organ failure. 30 patients died among this 45 patients thus resulting in a mortality rate of 66.7 %.
- } 2 patients out of 155 patients who showed no evidence of organ failure died resulting in a mortality of 1.3 %.
- } In the study by Rodolfo L et al³¹ 11(6.32 %) patient's died and all of them presented with the variable of organ failure.
- } Daniel A et al²¹ in their study found that the crude relative risk of death in patients with systemic sepsis was 13 times greater than those without. Severe sepsis was present in 424 patients (62%) among the 628 decedents. The author concludes that severe sepsis complicates the course of 11% of all patients with peritonitis.
- } M Hynninen⁴³ et al showed that the degree of organ dysfunction as measured by the SOFA (Sequential Organ Failure Assessment) score was the best predictor for hospital mortality in patients suffering from secondary peritonitis.
- } A systemic inflammatory response induced by the peritoneal infection may further progress to septic shock and multi organ failure.

Organ failure is not an all or none phenomenon, rather it is a continuation of alterations in organ function from normal function, through varying degrees of dysfunction, to organ failure. The description of organ dysfunction needs to be based on simple, easily repeatable variables specific to the organ in question and readily available. Organ dysfunction is not static and it will alter over time.

- } These result mentioned above highlight the importance of early recognition, prevention, and treatment of organ dysfunction in our attempt to improve the short and long term outcome in patients with peritonitis.

CORRELATION BETWEEN PREOPERATIVE DURATION OF PERITONITIS AND MORTALITY (Table 18)

- } In our study out of the 32 patients with a preoperative duration of peritonitis of less than 24 hrs no patient died. Out of the 168 patients who have preoperative duration of peritonitis of more than 24 hrs, 32 patients died thus placing the mortality rate of 19 %.

Ali Yaghoobi Notash³⁸ found mortality of 11.4% in patients presenting within 24 hours of the onset of symptoms while the mortality was 25% in patients presenting late.

In the study by Rodolfo L³¹ all the patients who died were having a preoperative duration of greater than 24 hours.

In the natural history of perforation peritonitis there is a gradual evolution from sepsis to resuscitation, empirical broad spectrum antibiotics and surgical intervention for the clearance of septic debris and control of the source of infection are key in the management of perforation peritonitis.

- } Scapellato S et al suggests that intervention time may be considered the main determinant of mortality in patients with peritonitis, since intervention time is a modifiable prognostic factor while many other factors are not. Therefore in cases of perforation peritonitis after the initial resuscitation of the patient's immediate laparotomy should be done as a surgical emergency.

CORRELATION BETWEEN MALIGNANCY AND OUTCOME (Table 19)

In our study 14 patients had malignancy. 9 out of the 14 patients expired thus placing the mortality rate in presence of malignancy to a whopping 64.3 %.

- } MM Correia et al²⁹ found that in presence of malignancy the mortality rate under the score of 21 was of 33.3% and for score equal to or greater than 21 the mortality rate was 70.6%.
- } Chao- Wen Hsu⁴⁶ in their study of colorectal perforations found out that although the overall mortality was 36.9% the highest disease specific mortality was due to malignancy (61.5%).
- } Peritonitis in oncological patients is generally caused by a ruptured viscous. The classic clinical manifestations are fever, abdominal pain, nausea, vomiting, diffuse abdominal tenderness, rebound tenderness and paralytic ileus. The diagnosis may be delayed by recent postoperative status, immunodepression, concomitant use of antibiotics and advancing age.
- } Peritonitis in oncologic patients presents high mortality rates, essentially related to the severity of the underlying disease.
- } These patients are less prone to survive serious infections.
- } Many disturbances of the immune system have been identified in oncologic patients, such as destruction of the anatomic barriers and derangement in the phagocytic activities and humoral and cellular responses. A consumption of opsonins may occur in the course of severe infection leading to failure of the immune system.

CORRELATION BETWEEN TYPE OF PERITONITIS AND MORTALITY (Table 20)

- } In our study 174 patients had diffuse peritonitis and 26 patients had localized peritonitis.
- } There was no mortality in patients with localized peritonitis while in patients with diffuse peritonitis there were 32 deaths with a mortality of 18.4 %.
- } In the study by Pacelli F²³ generalized peritonitis corresponded to 30.66% of the study group.
- } In the study by Rodolfo L³¹ generalized peritonitis corresponded to 34%.
- } Wahl N and associates⁵⁰ have rated diffuse peritonitis with mortality of 47% as one of the most unfavourable factor. According to them 10 - 15% patients in this group may need a relaparotomy for persistent and recurring infection.

- } As expected the extension of the peritoneal inflammation process was related to increased mortality.

CORRELATION BETWEEN ORIGIN OF SEPSIS (COLONIC / NONCOLONIC) AND MORTALITY (Table 21)

- } In our study 25 patients had colonic origin of sepsis out of which 7 patients died resulting in a mortality of 28% while in non colonic origin of sepsis the mortality rate in our study was 14.3%.
- } Thus according to our study colonic origin of sepsis is associated with poorer prognosis (increased mortality) than the non colonic origin of sepsis. This is in contrast with the MPI as introduced by Wacha and Linder³ where colonic origin of sepsis was considered as a favourable factor.
- } John Bohnen⁵¹ et al in their study of 176 patients found mortality of 10% in appendicitis and duodenal perforation, 50% in peritonitis of intraperitoneal origin other than appendix and the duodenum and 60% in postoperative peritonitis. Thus in this study the significance of the septic focus was high -lighted and it showed that colonic perforation is a higher risk while appendicular and duodenal perforations had a good recovery rate.
- } Chao -Wen Hsu et al⁴⁶ in their study of 141 patients with colorectal perforations found a mortality of 36.9%.

CORRELATION BETWEEN CHARACTER OF EXUDATE AND MORTALITY (Table 22)

- } In our study among the 40 patients with clear exudates 2 (5%) patient's died.
- } 16 (12.9 %) patients died among the 124 patients with purulent exudates
- } 14 (38.9 %) patients died among 36 patients with faecal exudates.
- } Thus the mortality in patients with clear exudates was 5 % purulent exudate was 12.9 % while in faecal exudate the mortality was 38.9 %.
- } In the study of Rodolfo L³¹ clear fluid had a mortality of 5.8% (7/121), purulent fluid had a mortality of 6.3% and faecal fluid had a mortality of 25%.
- } In a study by Chao-Wen-Hsu⁴⁶ in fecal peritonitis the mortality was 57.10% while in purulent peritonitis it was 30.25%.
- } In a study by Christian Ohmann et al² out of 166 patients with clear or purulent exudates 24 (14.45 %) died while out of 188 patients with turbid or feculent exudates 35 (18.61 %) died.
- } In 1983 Killingback⁵² reported a mortality rate of over 70% in case of faecal peritonitis complicating diverticular disease. The mortality rate from purulent peritonitis in the same study was much less than that, between 10 to 30% depending upon coexisting factors such as age, cardio respiratory disease steroid therapy and timing of surgical intervention.
- } The nature of exudates and its mortality has got direct relationship with the amount of micro organism that it contains.

- } Clear exudates are generally sterile to start with so evolution of sepsis is slow.
- } Purulent exudates and fecal exudates had a significant number of microorganisms many of which are gram negative anaerobes and they result in endotoxaemia and septic shock.

- } Rodolfo L et al³¹ in their study found out that 26 MPI point was a useful reference.
- } Patients with >26 points had mortality rate >40% whereas patients having a score <26 did not reach a 3% mortality.
- } A Billing et al²⁰ in their study of 2003 patients of perforation peritonitis found out a mortality rate of 2.3% in MPI score < 21, in MPI score between 21 and 29 the mortality was 22.5% & it was 51.1% for MPI score greater than 29.
- } Abrar Maqbool Qureshi et al³⁶ in their study found out that for MPI score of less than 21 the mortality was 1.9%, for scores in between 21 - 29 it was 21.9% & for scores 30 or more it was 21.8%.
- } In our study there was no death in patients with MPI score less than 21, in MPI score between 21 to 29 the mortality was 4.3%, while in patients with MPI score greater than 29 the mortality was 67.4%.

MORBIDITY OF PATIENT'S WITH PERITONITIS (Table 23)

- } The duration of hospital stay is good measure of morbidity of patients due to peritonitis.
- } In our study most of the patients i.e. 125 (62.5 %) stay in the hospital for 10-20 days. Very few of patient i.e. 8 (4 %) stay in the hospital for > 30 days.
- } Presence of secondary infections, malnutrition, delayed presentation contribute for longer period of hospital stay and associated increased morbidity in our study population

STATISTICAL VALIDATION OF MANNHEIM PERITONITIS INDEX (Table 24 ,25)

STATISTICAL VALIDATION OF MANNHEIMS PERITONITIS INDEX

AUTHOR	YEAR	SENSITIVITY	SPECIFICITY	PPV	NPV
Billing A.et al. 1 st Series	1994	70	67	-	-
Billing A.et al.3 rd Series	1994	85	61	-	-
Billing A.et al. 4 th Series	1994	69	97	-	-
Billing A.et al. 6 th Series	1994	98	76	-	-
Billing A.et al. 7 th Series	1994	76	58	-	-
Lombordoand et al.	1998	87	88	93	94
Watch et al.	1987	88	90	87	90
Von-Laarhosen et al.	1988	24	35	-	-
Altaca et al.	1992	90	94	-	-
Demmel et al.	1994	89	92	-	-
Corriea M.et al.	2001	87.3	41.2	-	-
Present Study	2011	90.62	91.7	67.44	98.12

- } When considering each risk factor constructing a contingency table in which presence and absence of adverse factor and result (death or survival) are considered the p value allow us to weight In descending order of significance , each of risk factors as follows:
 - a) Presence of organ failure b) Malignancy c) Age > 50 yrs d) Type of exudate e) Duration >24hrs; f) Diffuse / localised peritonitis
 - b) Non colonic origin of sepsis and female sex is also considered as a adverse prognostic factor by Linder and Waccha³ contrary to our study.
 - c) In our study the mortality rate is 28 % for colonic origin of peritonitis and 14.3 % for noncolonic origin of peritonitis. These are statistically not significant with p value of 0.145. Our findings are in agreement with other studies namely John Bohnen⁵¹ et al, Chao –Wen Hsu et al⁴⁶.
 - d) In our study the mortality rate is 12.5 % for female sex and 17.6 % for male sex & was statistically not significant with p value of 0.354, which highlights the fact that female sex is not an adverse prognostic factor,

this is not in agreement with the founders of the MANNHEIM PERITONITIS INDEX.

Other studies like Pacelli et al²³ have shown that factors related to host overshadow type and source of infection in evaluation of patients with intra abdominal infection. This is consistent with result of our study.

MICROBIOLOGICAL PROFILE OF ORGANISMS ISOLATED FROM PERITONIAL FLUID IN PATIENT'S WITH PERITONITS (Table 26)

In our study a random sample of 50 patients were drawn and culture reports of peritoneal/drained fluid were analysed, as in all cases microbiological reports were not available in the records. Most common culture result was sterile in 20 (40 %) cases. E.coli was the next common bacteria present in 15 (30%) cases, followed by Klebsiella in 8 (16%) cases, were as pseudomonas and enterococcus was isolated in 6 (12 %) and 1 (2%) cases respectively.

In the study by P. Panhofer, M.Riedl¹⁶ et al(2007), 43 patients with a positive microbiology were investigated, in which 33 patients (76.7%) had a gram positive microbiology. In the study by A.Prakash, D.Sharma⁽³⁾ et al (2006) found that 42 (50 %) out of total 84 patients had positive peritoneal fluid cultures.

In the study by Berger d et al⁵¹ (1998) found that culture swab taken during operations performed after 24 hours of onset of symptoms shows no growth, which is consistent with our results.

As per our study we were able to come to the conclusion that all the organisms that were cultured showed a trend to be sensitive to Aminoglycosides (Amikacin) and Cephalosporins (Cefeperazone with Sulbactam).

VIII. CONCLUSIONS

- } Mannheim Peritonitis index is a useful method to determine study group outcome in patients with peritonitis.
- } All the MPI variables of adverse outcome namely, presence of organ failure; time elapsed > 24hrs; presence of malignancy; age>50 years, generalized extension of peritonitis and type of exudate behaved as expected, except the noncolonic origin of sepsis in peritonitis and female sex.
- } **In our study we found that :**
 - Colonic origin of sepsis was associated with worse outcome probably due to presence of faecal exudates which was more commonly associated with colonic origin of sepsis.
 - Female sex was associated with better outcome as compared to male sex.
 - **Our study differs from MPI in these 2 variables of adverse outcome.**
- } Mortality can be further reduced by early arrival of the patients to hospital and early intervention.
- } Reproducible scoring systems that allow a surgeon to determine the severity of the intra abdominal infections are essential to:
 - 1) Ratify the effectiveness of different treatment regimen.
 - 2) Indicate individual risk to select patient's who may require a more aggressive surgical approach.
 - 3) Inform patient relatives with greater objectivity.
- } In the past 30years, many prognostic scoring system have been developed for critical patients. Presently one of the most accepted score is APACHE II score which integrates various physiological variables during the first 24 hours within the ICU. They are however both complex and time consuming.
- } The MPI is one of the most simple scoring system in use that allows the surgeon to easily determine the outcome risk during initial surgery.
- } Early evaluation of severity of illness using MPI allows us to estimate the probability of patient's survival.
- } The MPI cutoff points should be adjusted for each hospital on individual basis as in our study it was divided into 3 groups, <21, 21-29, >29.

- } Death rate in patients with MPI score < 21 was 0%, 21-29 was 4.3% and >29 was 67.4%
- } The simplicity of MPI makes ideal for hospitals with serious shortages of staff and resources.

Based on our study results we conclude that:

- } MPI is accurate to be used with patients with peritonitis and should be considered reliable and simple reference for estimating their risk of death.
- } **As our study differs in two adverse outcome variables, female sex & noncolonic origin of sepsis, we advocate need for further studies on Mannheim Peritonitis index to include colonic origin of sepsis and to remove female sex as variables of adverse outcome in Mannheim Peritonitis index.**

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