EVALUATION OF OUTCOME IN PATIENTS WITH PERFORATION PERITONITIS USING MANNHEIM'S PERITONITIS INDEX

A RETROSPECTIVE STUDY

Dissertation submitted

in partial fulfillment of the requirements for the degree of

MS. DEGREE- BRANCH I

GENERAL SURGERY

STANLEY MEDICAL COLLEGE

THE TAMILNADU DR.M.G.R. MEDICAL UNIVERSITY

CHENNAI, TAMILNADU

April 2017

CERTIFICATE

This is to certify that the dissertation titled "EVALUATION OF OUTCOME IN PATIENTS WITH PERFORATION PERITONITIS USING MANNHEIM'S PERITONITIS INDEX" is the bonafide work done by Dr. G.P.Kumaran, Post graduate student in the Department of General Surgery, Government Stanley Medical College and Hospital, Chennai under my guidance and supervision, in partial fulfilment of the requirements of the The Tamilnadu Dr. M.G.R. Medical University, Chennai for the M.S. Degree Branch I General Surgery Examination to be held in April 2017.

Prof.Dr.A.K.RAJENDRAN M.S.,D.Ortho,
Professor of Surgery
Dept. of General Surgery
Stanley Medical College
Chennai - 600001

Prof.Dr. D. NAGARAJAN M.S.
Head of the Department
Dept. of General Surgery
Stanley Medical College
Chennai - 600001

PROF.Dr.ISSAC CHRISTIAN MOSES M.D.,
The Dean
Govt. Stanley Medical College
Chennai - 600001

DECLARATION

I, Dr. G.P.Kumaran solemnly declare that this dissertation titled "EVALUATION OF OUTCOME IN PATIENTS WITH PERFORATION PERITONITIS USING MANNHEIM'S PERITONITIS INDEX " is a bonafide work done by me in the Department of General Surgery, Government Stanley Medical College and Hospital, Chennai under the guidance and supervision of my unit chief Prof. Dr. A.K.RAJENDRAN, Professor of Surgery and my head of the department Prof. Dr.D.NAGARAJAN. This dissertation is submitted to The Tamilnadu Dr. M.G.R. Medical University, Chennai towards partial fulfilment of the university regulations for the award of M.S. Degree (Branch-I) in General Surgery.

Place: Chennai

Date :September 2016 **Dr. G.P.Kumaran**

ACKNOWLEDGEMENT

It gives me immense pleasure to thank everyone who has helped me during the course of my study and during preparation of dissertation.

It is my proud privilege to express my sincere thanks to Dean **PROF.Dr.ISSAC CHRISTIAN MOSES M.D.,** Stanley Medical College for permitting to utilize the clinical materials of this hospital.

I take this opportunity to express my deep sense of gratitude, although I have no words to express the greatness of my teacher **Prof. Dr. A.K.RAJENDRAN ,M.S., D.Ortho.,** Professor, Department of General Surgery, Stanley Medical College for permitting me to use the clinical materials and for his valuable advice and encouragement in conducting the study. I consider myself fortunate and privileged to work under his affectionate guidance, excellent supervision and sustained support.

I express my sincere and heartfelt gratitude to **Prof. Dr. D.NAGARAJAN,M.S.,** Head of the Department, Department of General Surgery for his constant advice and guidance throughout this study. He has been the pillar of discipline, courage and immense kindness and who was instrumental in guiding me throughout the course of this dissertation.

I express my deepest sense of thankfulness to my assistant professors **Dr. C.ARUN BABU, Dr.D.S.KUMARESAN, DR.VIJAYALAKSHMI** for their valuable inputs and constant encouragement without which this dissertation could not have been completed.

I express my sincere gratitude to my mentor **Prof. G.V. MANOHARAN** for their constant support, able guidance and valuable help.

I am thankful to my fellow postgraduates for their valuable support. I also thank my senior postgraduates for their constant encouragement and support. I am also thankful to my junior postgraduate Dr.M.GNANACHEZHIYAN for his help in completing the study.

It is my earnest duty to thank my dear parents without whom accomplishing this task would have been impossible.

I am extremely thankful to my PATIENTS who consented and participated to make this study possible.

CONTENTS

ABBREVIATIONS

ICU- Intensive Care Unit

MPI-Mannheim Peritonitis Index

GIT- Gastrointestinal tract

CLD-Chronic Liver disease

SBP-Spontaneous Bacterial Peritonitis

SIRS- Systemic Inflammatory Response Syndrome

TNF- Tumour Necrosis Factor

IL-Interleukin

Ifn-Interferon

GOO-Gastric Outlet Obstruction

BP-Blood Pressure

PR-Per Rectal Examination

RFT- Renal Function Test

AKI- Acute Kidney Injury

INTRODUCTION

While there has been improvements in the surgical treatment, more sophistications in ICU and development of newer antibiotics, the mortality rate of peritonitis due to perforation is still high.

There are many factors which influence the outcome of any abdominal infection and the importance in early commencement of corresponding therapeutic procedures couldn't be stressed enough. The seriousness of the diseases should be recognised and an accurate assessment of the patients risks should be done.

Any marker which help in early evaluation of prognosis in a peritonitis patient will help in selecting high risk patients for more aggressive therapeutic procedures such as radical debridement, planned relapararotomy etc.

If there exists an accurate risk index classification ,it would be a way to set a standard of comparison between groups of patients and different therapeutic methods. Such an index would also allow prospective comparative studies between such groups of patients.

It is difficult and rather impossible to point out the severity or prognosis in Perforation peritonitis with a single laboratory investigation. Scoring systems help in combining various clinical problems into one single score, reducing the number of variables. It is for this reason that they have been advocated as prognostic predictors.

Reproducible scoring system that allow a surgeon to determine the severity of perforation peritonitis are essential to

1. ratify the effectiveness of different treatment regimens ,
2. to scientifically compare surgical intensive care units ,
3. to select a more aggressive surgical approach for high risk patients and
4. to able to inform patient's relatives with greater objectivity.

The assessment and comparison of treatment outcomes to various modalities also pose a difficulty in the patients with perforation peritonitis in that these patients may correspond to various etiologies which might require different treatment methods 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 involved.

Intra-abdominal infections and secondary peritonitis are a frequently encountered surgical emergency in tropical countries. The spectrum of perforation peritonitis in India remains to be different from western countries. In India, the most commonly affected population is the young men as compared to the west where the mean age for the occurrence of perforation peritonitis is usually 45-60 yrs.

It is also important to note that the patients with perforation peritonitis present to the hospital late in countries like ours and they have established generalized peritonitis at the primary presentation itself and they have some degree of septicemia. In India perforations of the proximal gastrointestinal tract were more common as compared to the distal ones. While there has been many advances, mortality from perforation peritonitis remains high.

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. Pelvic peritonitis
4. Peritonitis after translocation of bacteria

B. Postoperative Peritonitis

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

Randomized controlled clinical trials are the best methods for comparing efficacy of various treatment strategies for any specific disease. They remain a bridge between advances in basic sciences and improvement in health care setup. Therefore It couldn't be stressed enough the importance of performing clinical trials with high quality, in this field. 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 centers

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.

MANNHEIM PERITONITIS INDEX

It was developed by **Wacha and Linder** 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.

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.

While the scoring systems such as the APACHE II are time consuming and cumbersome, MPI is more practical, reproducible and easy to use.MPI has shown an acceptable specificity and sensitivity in prior studies

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.

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”.

Risk Factor	Weightage, if any
Age >50 years	5
Female gender	5
Organ failure*	7
Malignancy	4
Pre-operative duration of peritonitis > 24 hours	4
Origin of sepsis not colonic	4
Diffuse generalised peritonitis	6
Exudates	
Clear	0
Cloudy, purulent	6
Faecal	12

*Definitions of organ failure: Kidney: creatinine >177 μmol/L, urea >167 μmol/L, oliguria <20 ml/h; Lung: pO2 <50 mmHg, pCO2 >50 mmHg; Shock: hypodynamic or hyperdynamic; Intestinal obstruction (only if profound): Paralysis >24 h or complete mechanical ileus.

REVIEW OF LITERATURE- HISTORICAL REVIEW

Peritonitis was recognized as a universal fatal condition from the earliest of times. An historical perspective of the slow unraveling of the pathology, microbiology, and evolution of the treatment is best appreciated in “The peritoneum” by Hertzler (1919), “Infections of the peritoneum” by Steinberg(1944),and reviews by Hedberg and Welch and Hauet al. 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 gastro duodenal 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

The peritoneum is a glistening , slippery transparent serous membrane lining the abdominal cavity and invests the viscera.

The peritoneum is of two continuous layers the:

THE PARIETAL PERITONEUM, which lines the inner surface of the abdominal wall, and

THE VISCERAL PERITONEUM, which invests viscera such as hollow viscera and solid organs.

Both layers of peritoneum consist of mesothelium, a layer of simple squamous epithelial cells.

The vasculature and somatic supply of the parietal peritoneum is same as is the region of the abdominal wall it lines. As it has the same somatic supply as the skin , the peritoneum lining the inner surface of the abdominal wall is sensitive to various sensations such as pressure, pain, and temperature. Pain from the parietal peritoneum is generally well localized, but for the part covering the central tendon of diaphragm, as it is innervated by phrenic nerve, irritation here is often referred to the C3-C4 dermatomes over the shoulder.

Likewise, The visceral peritoneum shares the vascular supply of the organs it covers and they share the visceral nerve supply. The visceral peritoneum is insensitive to sensations such as touch, heat and cold; it is stimulated primarily by stretching and

chemical irritation. The pain produced is poorly localized, being referred to dermatomes of spinal ganglia providing sensory fibers, 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. This cavity is a potential space between the partial and visceral layers of peritoneum. While It contains no organs, it contains a thin film of peritoneal fluid. The composition of peritoneal fluid is mainly water, electrolytes, and substances derived from interstitial fluid. 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 the lubrication function, the peritoneal fluid contains leukocytes and antibodies that resist infection.

Lymphatic vessels, particularly on the inferior surfaces of the diaphragm, absorb the peritoneal fluid. While the peritoneal cavity is closed completely in men, there is a communicating pathway in women to the exterior of the body via 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, spenorenal, and phrenocolic ligaments and the transverse mesocolon and small bowel mesentry.

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 PERITONIUM

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 PHYSIOLOGY

The peritoneum is a bidirectional, semi permeable membrane that has control on the amount of the peritoneal fluid. It has an active role in the sequestration and removal of pathogens from the peritoneal cavity. It also facilitates the migration of immune cells from the vasculature into the peritoneal cavity. Normal volume of the peritoneal fluid is less than 100 ml and it is sterile and serous. The apical surface of the peritoneal mesothelium contain microvilli which markedly increase the surface area and thereby make it possible, the rapid absorption of peritoneal fluid from the cavity into the lymphatics and the portal and systemic circulation. Peritoneal fluid may increase in amount even to an extent of around few liters in conditions such as cirrhosis, nephrotic syndrome, and peritoneal carcinomatosis.

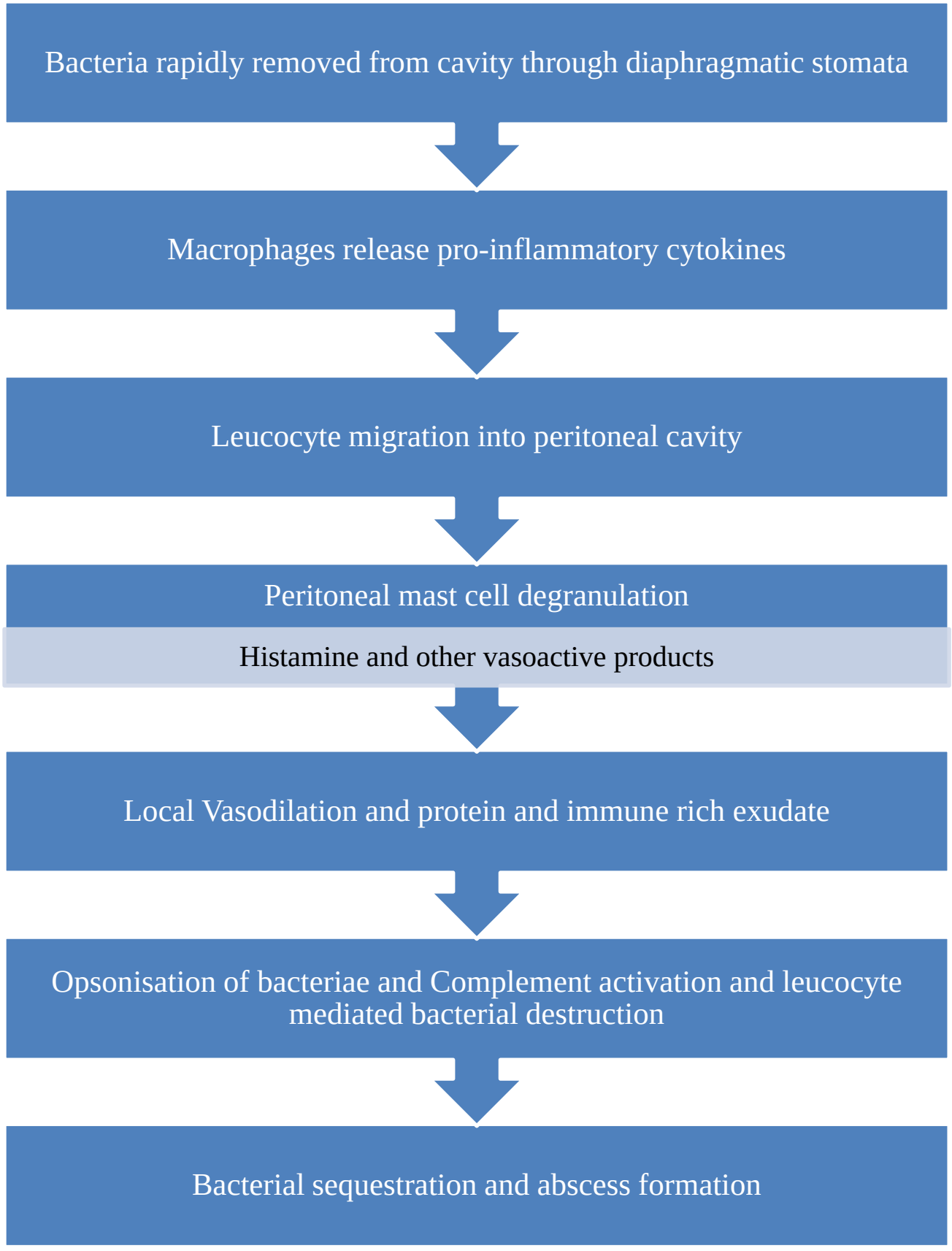
Movements of diaphragm is an important factor which drives the circulation of peritoneal fluid within the peritoneal cavity.There are numerous minute openings in the peritoneum covering the under surface of the diaphragm. They are termed as stomata. They form connection channels with lymphatic channels of the diaphragm.

The pathway of lymph from these channels goes through lymphatics in the sub pleural space to the regional lymph nodes. From these lymph nodes, they reach the Thoracic duct. During expiration, relaxation of the diaphragm opens the pores, and the fluid and particles flow through these pores facilitated by the negative intra thoracic pressure. . During inspiration, contraction of the diaphragm opens the pores, and the lymph flow through the mediastinal lymphatic channels into the thoracic duct.

It is postulated that this so-called This diaphragmatic pump drives the upward movement of peritoneal fluid in a direction towards the under surface of the diaphragm and into the thoracic lymphatic vessels. This circulatory pattern from the peritoneal fluid into the central lymphatic channels forms one of the reasons for rapid onset of systemic sepsis in patients with intra abdominal infections. This is also the reason for development of peri-hepatitis of Fitz – Hugh – Curtis syndrome in patients with acute salpingitis.

Response of Peritoneum to Infection:

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


Bacteria rapidly removed from cavity through diaphragmatic stomata
Macrophages release pro-inflammatory cytokines
Leucocyte migration into peritoneal cavity
Peritoneal mast cell degranulation
Histamine and other vasoactive products
Local Vasodilation and protein and immune rich exudate
Opsonisation of bacteriae and Complement activation and leucocyte mediated bacterial destruction
Bacterial sequestration and abscess formation

PATHOPHYSIOLOGY OF PERITONITIS

CLASSIFICATION AND STRATIFICATION

Definition

Peritonitis and intra-abdominal infection are not synonymous. Peritonitis denotes inflammation of the peritoneum from any cause. Any trigger of systemic inflammatory response (SIRS) can cause an inflammation of the peritoneum also. Any infection that is intra-abdominal such as one caused by bacteria lead to inflammation of peritoneum ie. Peritonitis.

It forms a part of the local equivalent of the inflammation abdominal cavity in a setting of systemic sepsis. When such an intra-abdominal infection gets contained and walled-off, it may form an abscess, in this case an intra-abdominal abscess. As most if not all of the peritonitis is due to infection by bacteria, the terms “Bacterial peritonitis” and “Peritonitis” are often used inter changeably.

There are conditions in which even though contamination has occurred, the infective process has not settled well (e.g., in an early setting of trauma with bowel perforation) and there are conditions in which the infective process gets localized to an organ which is resectable as in Cholecystitis and Appendicitis. These represent a “simple” form of peritonitis, easily cured by a surgery to remove the organ concerned.

Relation between SIRS & sepsis vs. Peritonitis &intraabdominal infection

Any local inflammatory trigger in abdominal cavity can cause Peritonitis. Commonest of such an inflammatory trigger is usually infection. Such an infection may not necessarily be present at the localized peritonitis as in Appendicitis or cholecystitis etc. In contrast, the infectious trigger may be by contamination of the peritoneum by intraluminal contents as in perforation/defect in the abdominal wall. In case of an acute traumatic intestinal perforation, the contamination may occur which takes some time in progression to infection/ inflammation. Peritonitis has been categorized as primary, secondary, or more recently tertiary.

Primary peritonitis or Spontaneous bacterial peritonitis (SBP) is a bacterial infection of the peritoneal cavity usually it occurs in the presence of ascites as in conditions like Chronic liver disease(CLD). The incidence of SBP has been reducing as a result of widespread use of antibiotics. The infrequent occurrence of SBP in form of ascites other than due to CLD shows the role of intrahepatic shunting in the pathogenesis of SBP.

Secondary peritonitis is usually due to contamination of the cavity by microbes form the GI tract or genitourinary tract. This occurs as a result of loss of mucosal integrity and its barrier action. Examples include traumatic penetration of bowel wall, and gastric or duodenal ulcer perforation. This form of peritonitis can occur either in a localized form or diffuse form. Such infections form abscesses may be limited to

the surrounding peritoneum around a diseased organ, such as around the gall bladder, appendix, or a perforated diverticulum. They may also limit themselves to one of the few recesses in the peritoneum, such as subdiaphragmatic, subhepatic, lesser sac or pelvic abscesses.

Tertiary peritonitis is a condition in which the patient continues to have signs of peritonitis and sepsis even after treatment of secondary peritonitis. This is usually supported by evidence of retrieval of low virulence pathogens from the peritoneal exudate such as fungi, Enterococci etc. This infection may result either from contamination during surgical procedures undertaken for the treatment of secondary peritonitis, or by selection by antibiotic pressure, from the initial organisms causing secondary peritonitis.

Undoubtedly, many manifestations of the peritonitis are mediated by inflammatory mediators such as such as TNF, IL-1, IL-6, IFN-gamma, and others.

The concentration of the cytokines is much higher in the peritoneal exudates than in the systemic circulation of peritonitis patients. Bacteria or bacterial endotoxins serve as the trigger for the production of these cytokines by immune cells of the host. Tissue trauma during the surgeries and direct outflow form the intestinal barriers also serve additional sources.

When gastrointestinal perforation is the causative event for secondary peritonitis , the number and type of micro-organism isolated from the peritoneal cavity depend on level of perforation. In fasting state, stomach contain few of acid resistant organisms such as lactobacilli or candida species. Similarly, the duodenum has less microbial load in a fasting state, whereas the large intestine contains a high microbial density, i.e., about 1012 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. Similarly, the normal limited flora of small intestine may be altered by gastric disease or small bowel ileus.

Peritonitis is thought to pass through three phases:-

PHASE 1-

Rapid translocation of bacteria mainly Gram negative facultative anaerobes from the peritoneal cavity into lymphatic lacunae through the stomata in the diaphragm. From these lacunae, through the substernal nodes and thoracic duct, these organisms gain entry into the systemic circulation. The ensuing gram negative septicemia is associated with high morbidity.

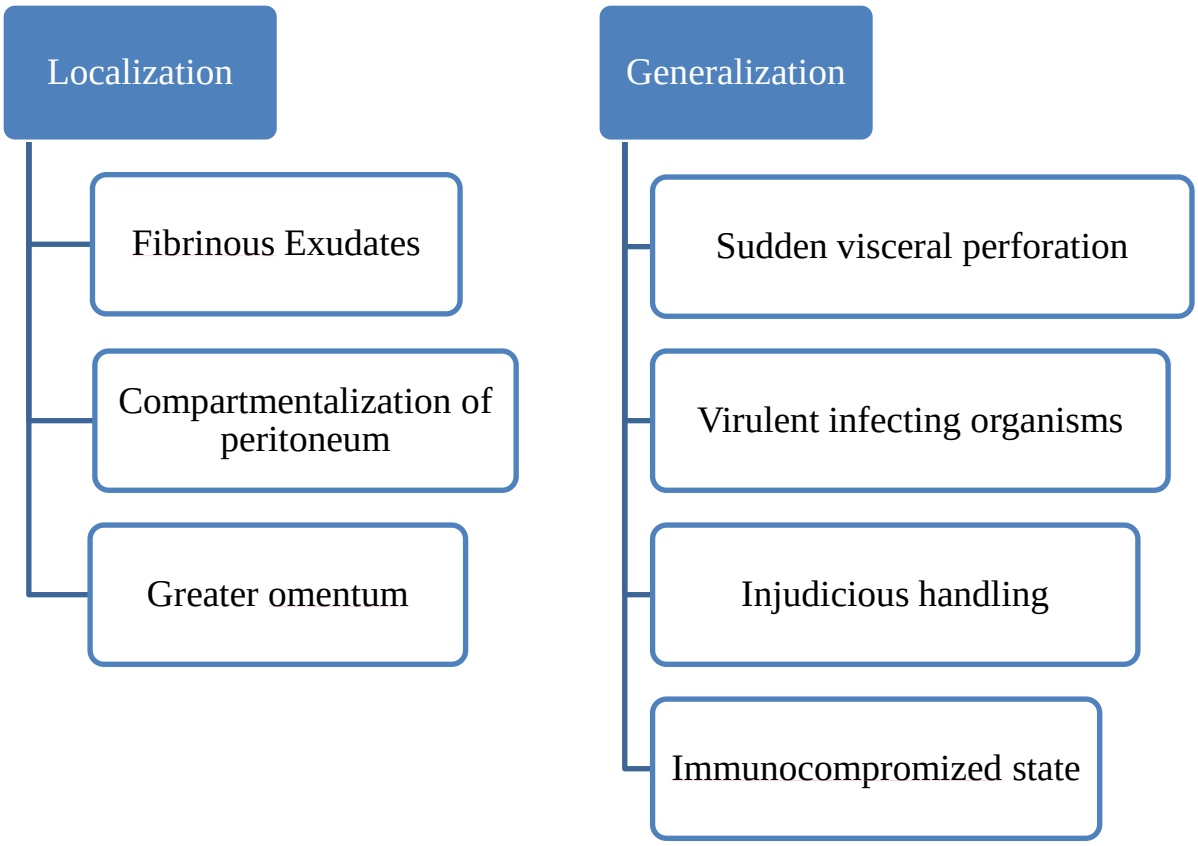
PHASE 2-

Involves activation of complement cascade mainly the classical pathway and also the alternative pathway. Mesothelial cells in the peritoneum play an important role in the opsonisation and phagocytosis of the microbes by producing a phospholipids surfactant which work synergistically with complement. They also secrete pro inflammatory cytokines thereby playing a major role in the signaling pathway leading to phagocyte recruitment and the up regulation of mast cells and fibroblast.

PHASE 3-

The host defenses attempt to localize the infection. They do so mainly by exudation of fibrin rich exudates. They trap the bacteria and other micro organisms within its matrix and paves way for local phagocytes to act. They also act in organizing an abscess.

Factors favouring localization or generalization of peritonitis:



MICROBIOLOGY OF PERITONITIS

Primary Spontaneous bacterial peritonitis (SBP) is usually aerobic infection and is mono microbial. Whereas secondary peritonitis resulting from conditions such as a perforation of hollow visceral organs usually is polymicrobial and may contain obligate anaerobes or a mixed flora. Such a polymicrobial infection may also result from conditions such as post anastomotic leaks.

In fasting state, Stomach and duodenum are usually sterile albeit for a few coliforms which are acid resistant. Having said that, diseases of the stomach such as CA, GOO and acid reducing drugs may pave way for colonization of the otherwise sterile stomach. The microbial load goes on increasing as we go down the GI tract with large intestines having the highest load and concentration – 1 gram of stool up to 1012 obligate anaerobes and 108 facultative anaerobes (formally aerobes) and there is a change in trend from aerobic to anaerobic organisms.In the event of a colonic perforation, more than 400 different bacterial species contaminate the peritoneum of which only a few are involved in ensuing peritonitis.

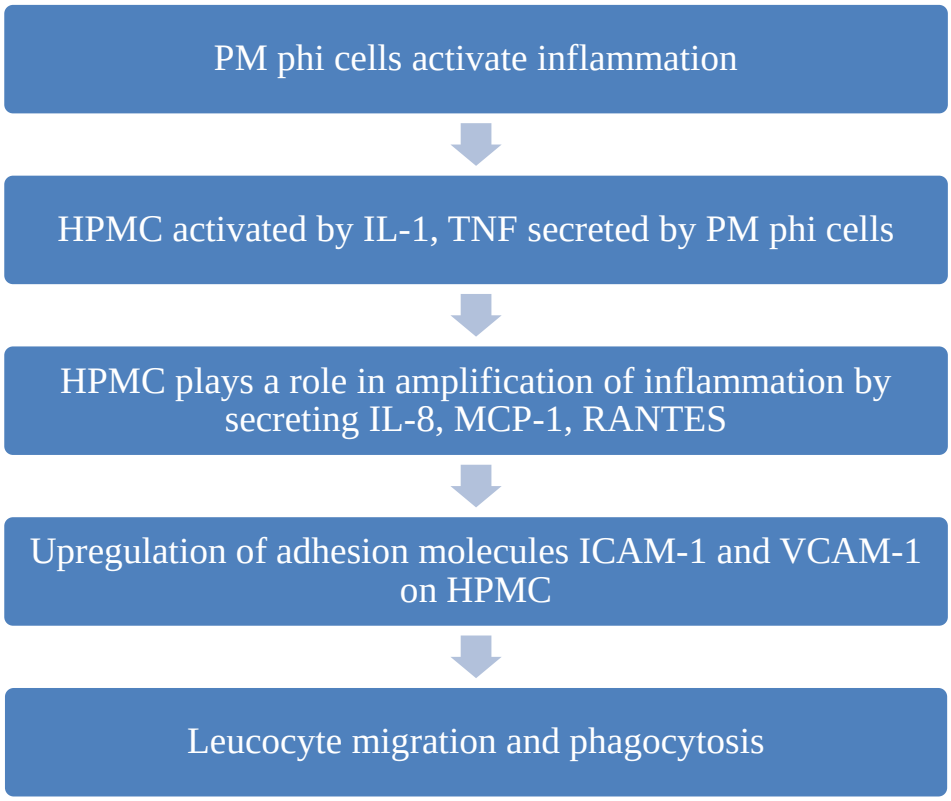
This normal ecology may be varied in patients in postoperative state, and during administration of systemic and luminal antibiotics. They may have organisms even of low virulence colonizing the foregut e.g., fungi, coagulase negative staphylococci and gram negative bacteria. These are the organisms that may be found in tertiary peritonitis.

MOLECULAR BASIS OF PERITONITIS

Topley N et al in their study on macrophages and mesothelial cells in bacterial peritonitis examined the mechanism underlying cellular host defence in the peritoneal cavity.

There are two important cells that reside in peritoneum, the peritoneal macrophages (PM phi) and the mesothelial cells (HPMC) which carry on 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 .

Mediators form Both PM phi and HPMC play a direct role in inflammatory control. The PM phi cells form the initial line of defense.



Wolfgang Sendt et al 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

The clinical manifestations of peritonitis are mainly a result of fluid shifts and metabolic disturbance. There is initial tachycardia and tachypnoea as a result of intestinal, diaphragmatic, and pain reflexes. Subsequent alterations in cardiac output and respiratory pattern are due to aldosterone excess, catecholamine secretion and ADH secretion.

Protein break down and hepatic glycogenolysis marks the beginning of a highly catabolic state. Paralytic ileus develops, and there ensues fluid and electrolyte loss and exudation of protein rich secreta. As the disease progresses , there occurs abdominal distension which causes elevation of diaphragm and lung changes such as a passive collapse and/or pulmonary infection. MODS and death will follow if generalized peritonitis persists.

DIAGNOSIS / PRESENTATION :

Abdominal pain is the commonest symptom, may be confined or generalized; The pain is usually continuous and is of a sharp pricking character. In case of a viscus perforation, there is initial acute, severe pain in the perforation are which then becomes generalized as peritoneal contamination proceeds. Referral of pain occurs to the shoulder of the same side in case of involvement of diaphragmatic peritoneum.

Loss of appetite , weakness, tiredness , nausea and vomiting are other common associated features. As a result of ileus, there is accompanying constipation but there is diarrhea in case of pelvic abscess.

EXAMINATION:

General:

A peritonitis patient is pale, irritable and anxious; signs of dehydration such as sunken eyes , dry tongue may be there. In addition, the patient might be febrile and there might be tachycardia, tachypnea and other signs of sepsis such as fever, tachycardia, tachypnoea, hypotension, elevated cardiac index, low systemic vascular resistance or features of multiorgan failure

Abdomen:

The patient usually lies on his back and relatively motionless as any movement may increase the irritation of the peritoneum and increase pain. Respiratory movements are shallow and the movement of abdominal quadrants with respiration

reduce or cease in case of generalized peritonitis. The patients keep their knees flexed to reduce stretch in the abdomen thereby reducing pain. “Board-like rigidity” occurs as a result of spasm of the abdominal muscles .

Abdominal palpation shows diffuse guarding, rigidity and the abdomen is exquisitely tender. Palpation should be gentle. It will show tenderness, guarding and rebound tenderness; The site of pathology may be deduced by the site of maximal tenderness. While initially there will be voluntary guarding, it becomes involuntary rigidity as the inflammatory process progresses.

PR examination may elicit Pouch of Douglas tenderness in case of pelvic peritonitis/ abscess.

Auscultation will show reduced/absent bowel sounds depending on the severity of the ileus

INVESTIGATIONS

Peritonitis is mainly a clinical diagnosis and we shouldn’t postpone emergency surgical intervention just for the want of investigations.

Blood tests are discussed below:

1.Complete blood count will show neutrophilic leucocytosis.

2.RFT elevation may show the degree of dehydration and presence of AKI.

3.Elecrolyte values guide us in fluid resuscitation along with RFT values.

4.Liverfunction tests- moderate elevation occur in sepsis as it is one of the early marker of sepsis.

5.Serum amylase – while very high values of amylase such as above 1000 may point to acute pancreatitis, moderate elevation can also occur in perforation peritonitis.

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

7. Grouping and cross matching of blood

IMAGING :

Erect radiograph of the chest and abdomen

will show pneumo peritoneum as air under the diaphragm in about 70–80% of hollow viscus perforations.

An alternative is left lateral decubitus Xray of abdomen for those who couldn't stand erect.

A supine radiograph of the abdomen may show a 'ground glass' appearance in cases of diffuse peritonitis.

Ultrasound is useful in ruling out conditions such as subphrenic abscess etc.

Computerized Tomography (CT) is very accurate in negative prediction than ultrasound.CT has helped in reducing number of negative diagnostic laparatomies in search of sepsis, thereby reducing morbidity.

DIFFERENTIAL DIAGNOSIS:

1. Basal pneumonia
2. Myocardial infarction
3. Gastroenteritis
4. Hepatitis and
5. urinary tract infection
6. Ureteric/biliary colic may be misdiagnosed as peritonitis.

MANAGEMENT:

Conservative-

Conservative management is indicated in cases

such as appendicular mass where the infection has localized and

an unfit patient for General anesthesia (moribund state with severe co-morbidities)

The supportive treatment of the conservative management include fluid resuscitation (i.v.) and broad spectrum antibiotics.

In case of a patient with perforation peritonitis, there is no role for expectant management and surgery is almost always essential and necessary.

Immediate-

1. *High flow Nasal O_2*with ABG/spO_2 monitoring.
2. *Ryle`s tube and aspiration* reduces vomiting and abdominal distension
3. *Fluid resuscitation* - initially with crystalloids (i.v.), depending on the degree of dehydration and shock along with potassium replacement. Catheterization to monitor urine output and to maintain I/O chart. Administration of inotropic support based on CVP (central venous pressure) monitoring in patients with severe sepsis.
4. *Analgesia – with* opioids
5. *Antibiotics* –iv broad spectrum with both aerobic and anaerobic coverage. A common combination is a Cephalosporin with metronidazole is used. Mortality and morbidity can be reduced by Early and appropriate use of antibiotics.

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 antisepsis 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 intra peritoneal infections, and the development of scoring systems and their application to patients with peritonitis.

*Laparotomy (upper/ lower midline)*is usually performed and the objectives are to:

- find out the reason for 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 PERITONITIS

One of the principal aim in management of peritonitis is bringing under Control, the primary source of sepsis. Usefulness of peritoneal irrigation has not shown any positive evidence, possibly because of the micro organisms in peritoneum being resistant to lavage, or more so because of the risk of damage inflicted to peritoneal mesothelium. Removal of debris, faecal or purulent exudates must be the primary aim.

Mass closure of the abdomen is undertaken using interrupted or continuous monofilament sutures. It is advisable to continue broad spectrum antibiotics for atleast a period of five to seven days postoperatively in cases of generalized peritonitis.

Secondary peritonitis Patients with continuing or worsening sepsis even after primary surgery will require a re-laparotomy for clearance of sepsis. Relaparatomies may be performed as and when the condition warrants, or in a more aggressive manner wherein the relaparatomy is planned to be done at a regular interval during the initial procedure itself. If a relaparatomy is planned for, then instead of closing the abdomen, it is left open with a sheet of mesh insitu to prevent evisceration. Modifications are “primary open management”, and “semi-open approaches such as staged abdominal repair”. However, evidences from recent studies have shown that survival benefits are

better in the patients managed by on-demand relaparotomy than in those treated by planned relaparotomy.

Continued clinical monitoring with CT imaging as and when required, is the key to time the re laparotomy and to select the patients who would require an “on demand relaparotomy”. Even for many of the septic patients performing poorly in the post operative period may not require a re laparotomy but rather require ICU monitoring and care and prolonged antibiotics and organ support.

It couldn`t be stressed enough the importance of removing the source of sepsis during the primary procedure itself and proper peritoneal lavage/toileting plays an important role in that. Besides, each and every repeat procedure done on the patient increases chances of his/her morbidity and mortality manifold.

Laparoscopy:

Laparoscopy in an inflamed peritoneum have a theoretical possibility of systemic absorption of carbon di oxide and the endotoxins into the blood stream and hence the risk of hypercapnia and septic shock. But these have not yet been proven. Instead, both acute appendicitis and peptic ulcer perforation has been managed effectively with a laparoscopic setting. While there is a choice to use laparoscopy incases of colonic perforation, it should be borne in mind that the conversion rate to laparotomy is high. Contraindications to laparoscopy are Shock and major ileus.

A localized space or collection could be drained well by usage of intra abdominal drains but they couldn’t be trusted to drain the entire peritoneal cavity as they get quickly walled off. There is a lack of evidence to support the prophylactic use of drain tubes after laparotomy.

‘

AIMS AND OBJECTIVES

To evaluate the efficacy of MANNHEIM PERITONITIS INDEX in predicting mortality in patients with perforation peritonitis..

MATERIALS AND METHODS

PLACE OF STUDY:

DEPARTMENT OF GENERAL SURGERY, STANLEY MEDICAL COLLEGE AND HOSPITAL

DURATION:

OCTOBER 2015 TO SEPTEMBER 2016[12 months]

STUDY DESIGN:

OBSERVATIONAL STUDY – RETROSPECTIVE

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.

SAMPLE SIZE

A total of 50 patients of perforation peritonitis who were admitted in surgery department over a period of 1 year are included in the study. All the patients who

were operated for perforation peritonitis and whose OT records were complete were included in the study.

INCLUSION CRITERIA:

Patients with clinical suspicion and investigatory support for the diagnosis of peritonitis due to hollow viscous perforation who are later confirmed by intra op findings.

EXCLUSION CRITERIA:

1. patients with hollow viscous perforation due to trauma
2. patients with associated injuries to other organs
3. patients with associated vascular, neurogenic injuries
4. Patients with any other significant illness which is likely to affect the outcome more than the disease in study.
5. Patients absconded or discharged against medical advice (AMA) during hospital admission.
6. All patients with primary peritonitis (Spontaneous bacterial peritonitis)
7. All patients with tertiary peritonitis - Patients with peritonitis due to anastomotic dehiscence or leak
8. HIV Patients with CD Count < 200/mcL

Methodology:

An informed consent to be obtained. After obtaining a detailed history, complete general physical examination and systemic examination ,the patients will be subjected to relevant investigations.

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 included 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 (1992):

o 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.

o 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.

o Intestinal obstruction (only if profound):paralysis >24 hours or complete mechanical ileus.

o 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 / non colonic)

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. Sero haemorraghic 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 to29.
3. Score more than 29.

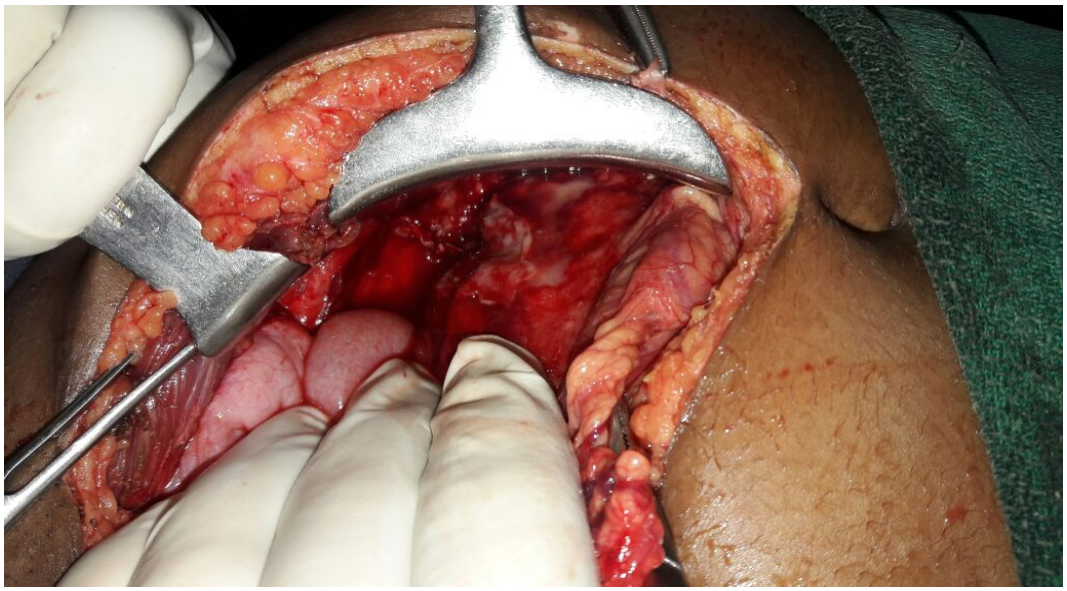
Ethical consideration :

study was approved by the institutional review board prior to commencement of data collection. Data were collected by approved data collection form.

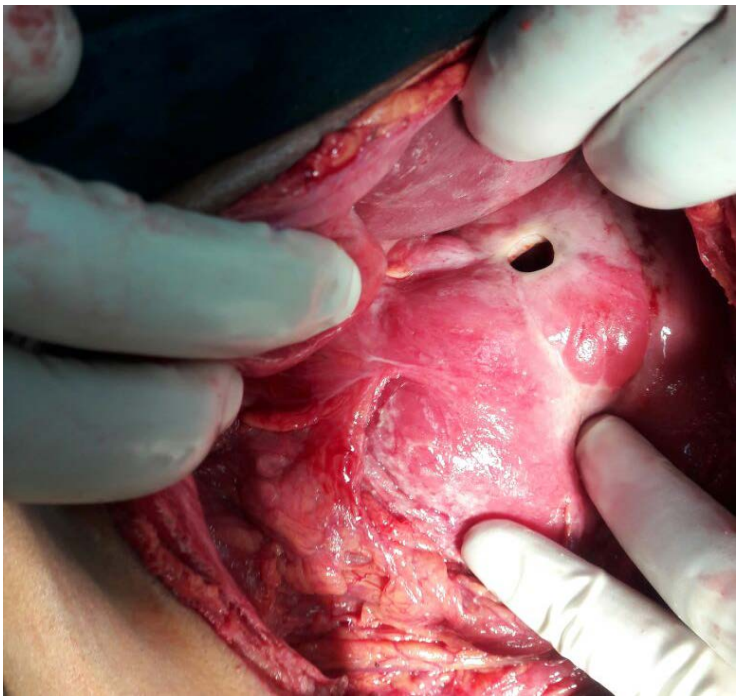
Data analysis :

Data analysis was done both manually and by using computer. Calculated data were arranged in systemic manner, presented in various tables and figures and statistical analysis was made to evaluate the objectives of this study with the help of Statistical Package for Social Science (SPSS).

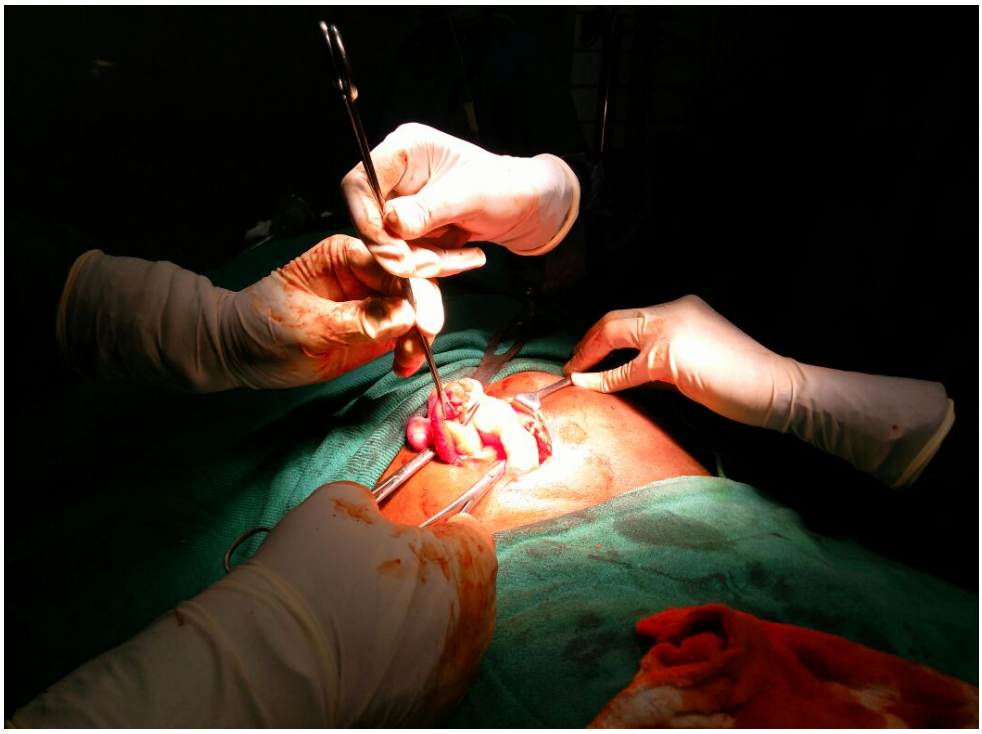
CLINICAL PHOTOS



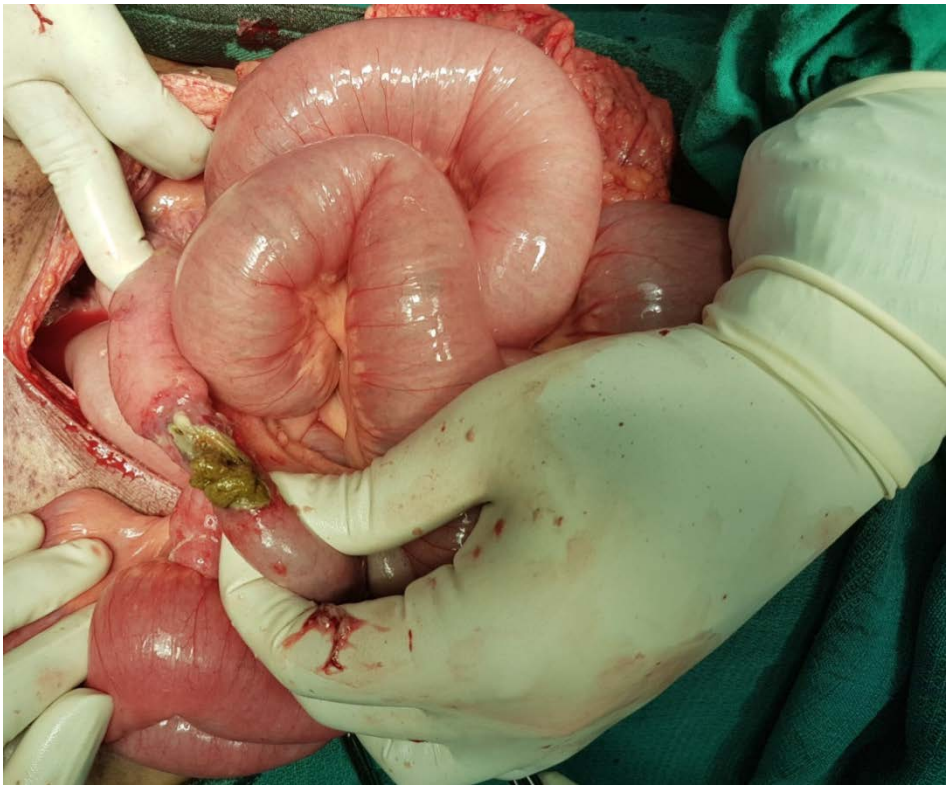
1.Photo showing midline laparotomy with Appendicectomy



2. Photo showing Duodenal Perforation



3. Photo showing open appendicectomy



4.Photo showing Ileal perforation

RESULTS

Table Showing age distribution of the patients

Age group	Frequency	Percent
<15	2	4%
16-30	13	26%
31-45	17	34%
46-60	12	24%
>60	6	12%

The mean age of the study group was 41.4 years and the age group of 31-45 contains maximum (34%) patients followed by 16-30 years. Oldest patients was 80 years and youngest was of 13 years.

Table Showing age distribution of the patients as per MPI

Age	Frequecny	Percent
<50	35	70%
>50	15	30%

Table showing sex distribution of the patients

Sex	Frequency	Percent
Male	45	90%
Female	5	10%

Table Showing anatomical site of perforation in study patients

Etiology	Males	Females	Total
Duodenal Perforation	28	1	29
Gastric Perforation	5	1	6
Small Bowel Perforation	4	1	5
Appendix	8	2	10
Total	45	5	50

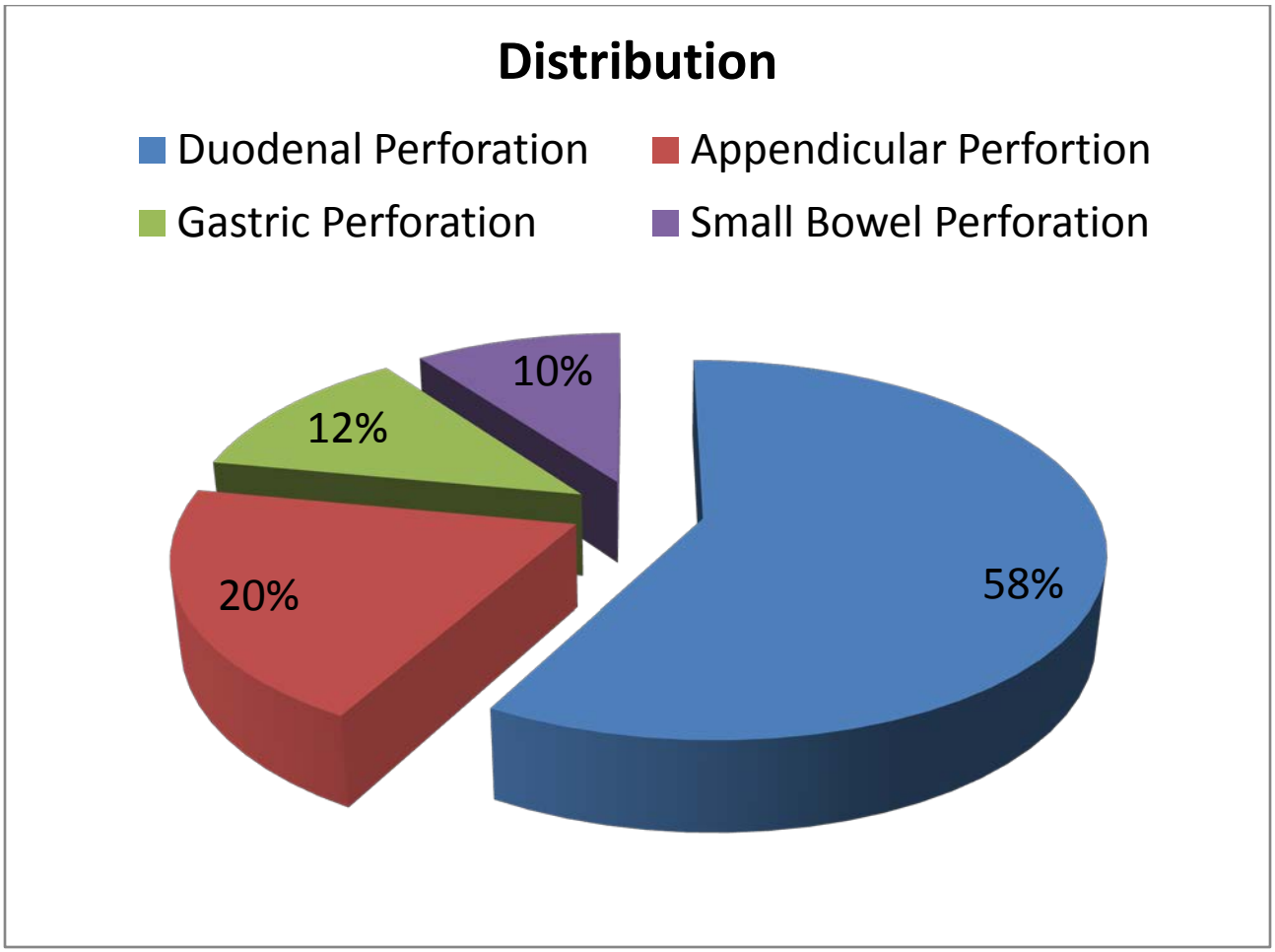
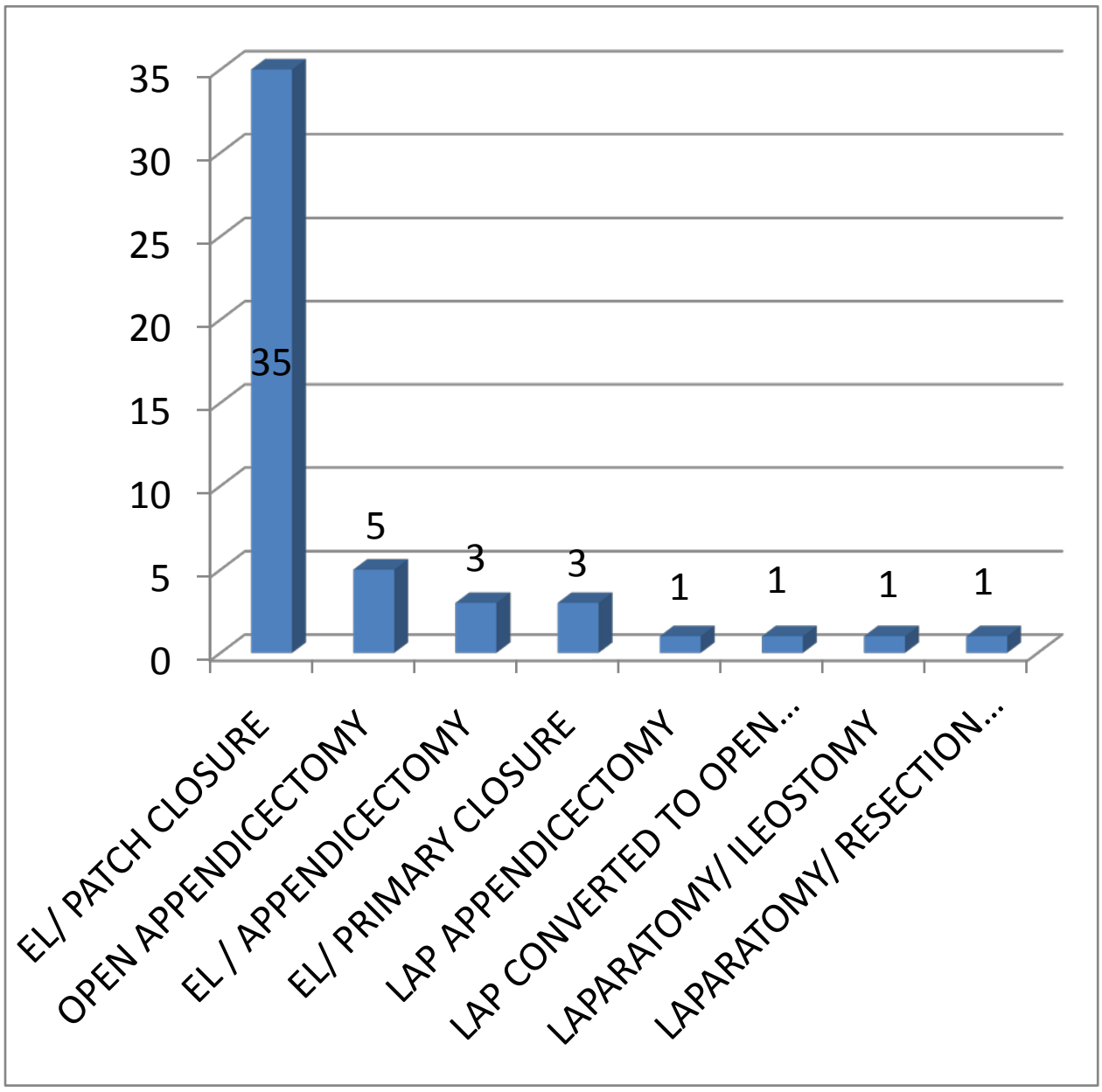


Figure Showing anatomical site of perforation in study patients

As the table shows maximum number of patients had duodenal perforation 29(58 %) followed by Appendicular perforation (20 %), gastric perforations were (12%), and ileal perforation (10%)

Procedures performed

Most common procedure performed was exploratory laparotomy with omental patch repair in 35 (70 %) patients followed by open appendicectomy in 5 (10%) and Laparatomy and Appendicectomy in 3 (6%) , Emergencylaparotomy and primary closure in 3(6%)of patients.



LaparascopicAppendicectomy, Lap converted to Openappendicectomy, Laparatomy with ileostomy, Laparatomy with resection anastomosis was done in 1 patient each. EL in the chart denote Emergency Laparatomy

Various clinical features in patients with peritonitis

Symptoms		Frequency	Percent
Abdominal pain	Absent	1	2%
	Present	49	98%
Distension	Absent	23	46%
	Present	27	54%
Not passed Flatus	Absent	30	60%
	Present	20	40%
Not passed Stools	Absent	31	62%
	Present	19	38%
Fever	Absent	21	42%
	Present	29	58%
Vomiting	Absent	19	38%
	Present	31	62%

The commonest symptom was

1.Abdominal pain (98 %)

Followed by

2.Vomiting (62%)

Table showing distribution of organ failure in patients with peritonitis

Organ Failure	Frequency	Percent
Absent	30	60%
Present	20	40%

In our study, 20 patients (40%) showed organ failure features

Table showing preoperative duration wise distribution of patients

PreOperative duration of peritonitis	Frequency (Percent)	Percent
<24 hours	24	48%
>24 hours	26	52%

In our study, 24 patients (48%) presented within 24 hrs of onset of peritonitis symptoms and 26 (52%) presented beyond 24 hours

Table showing presence of malignancy in patients with peritonitis

Malignancy	Frequency	Percent
Absent	48	96%
Present	2	4%

4% of patients in our study had malignancy. Both of them were gastric carcinoma patients.

Table showing origin of sepsis (colonic /non colonic) in our study

Origin of sepsis	Frequency (Percent)	Percent
Colonic	10	20%
Non Colonic	40	80%

In our study 10 i.e.20 % patients origin of sepsis was colonic while in 40 i.e. 80 % patients origin of sepsis was non colonic

Table showing type of peritonitis in study population

Distribution of Peritonitis	Frequency (Percent)	Percent
Localized	15	30%
Generalized	35	70%

In our study 35 i.e. 70 % patients had Diffuse peritonitis while 15 i.e.30% had localised peritonitis

Table showing character of exudates in study population

Exudates	Frequency	Percent
Clear	7	14%
Purulent	41	82%
Fecal	2	4%

In our study 41 (82%) patients had purulent exudates while clear & fecal exudates were present in 7 (14%) & 2 (4%) patients respectively

Table showing MPI score wise distribution of patients

MPI	Frequency(Percent)	Percent
<21	25	50%
21-29	21	42%
>29	4	8%

In 25 (50 %) patients total MPI score was < 21 while 21 (42%) patients total score was 21-29 & it was > 29 in 4 (8%) patients

Table showing outcome of the patients in our study

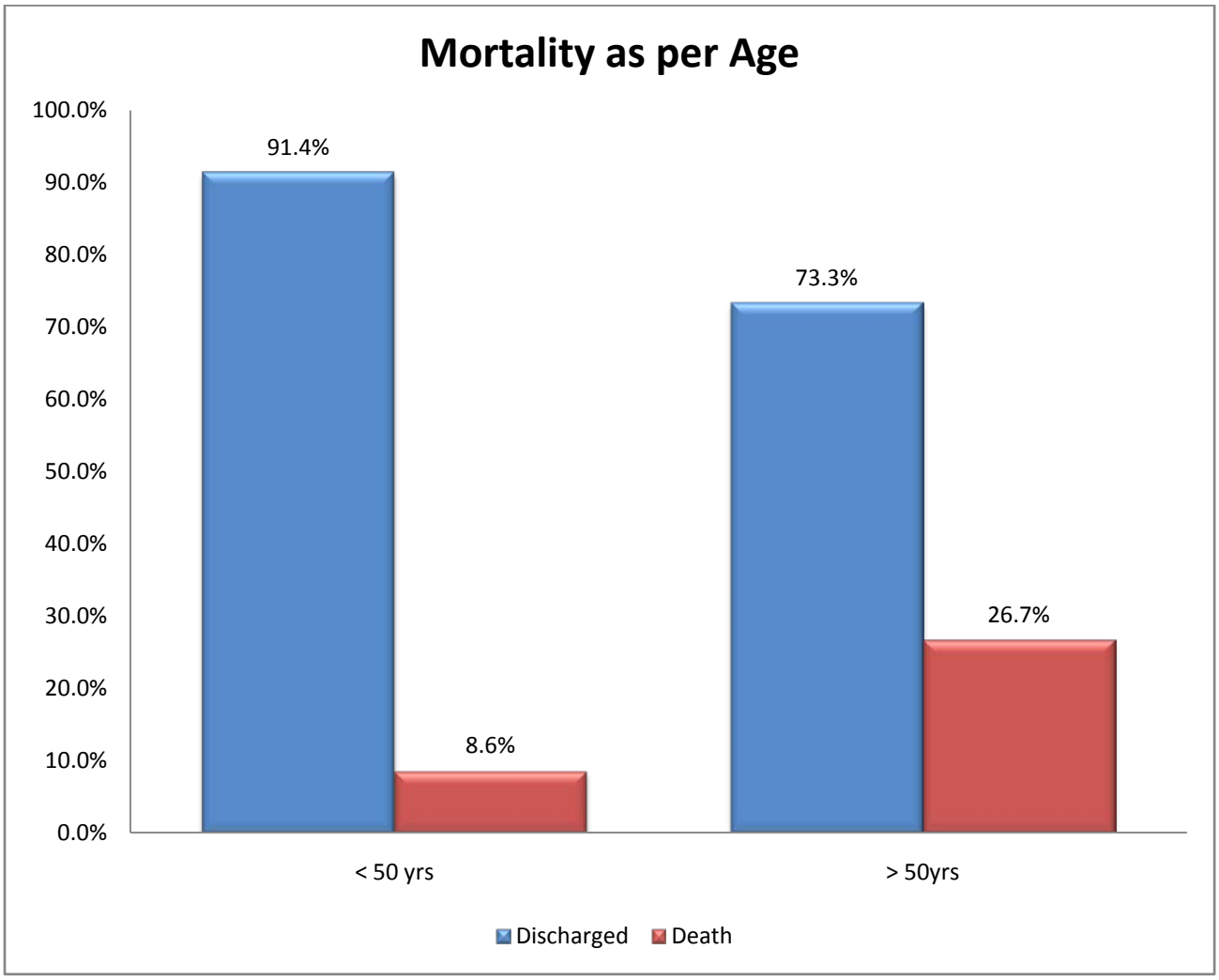
Outcome	Frequency	Percentage
Discharged	**43**	**86%**
Death	**7**	**14%**
Total	**50**	**100%**

Table showing mortality in each age group

			OUTCOME	
			Mortality in group	Total Mortality
Agegroup	Upto 30 yrs	Count	1	1
		%	7.1%	0.02%
	31 - 45 yrs	Count	2	2
		%	12.5%	4.0%
	46 - 60 yrs	Count	2	2
		%	14.3%	4.0%
	> 60 yrs	Count	2	2
		%	33.3%	4.0%
Total		Count	7	7
		%	14.0%	14.0%

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

Showing correlation of Age > 50 yrs with incidence of mortality

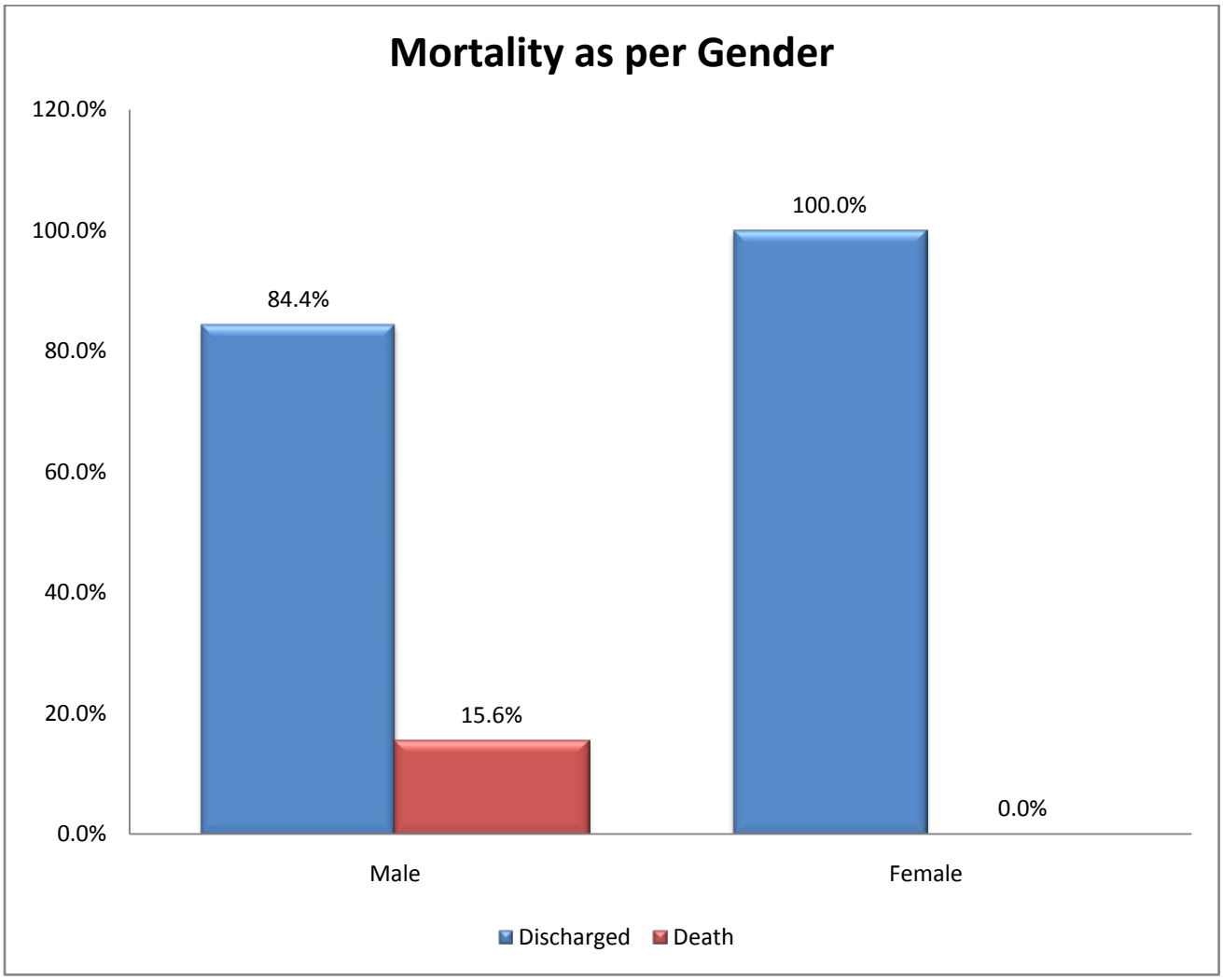


Age	Frequecny	Mortality	Discharged
<50	35 (70%)	3(8.6%)	32(91.4%)
>50	15 (30%)	4(26.7%)	11(73.3%)

Chi-Square Tests					
	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	2.856[a]	1	.091		
Continuity Correction[b]	1.550	1	.213		
Likelihood Ratio	2.623	1	.105		
Fisher's Exact Test				.176	.109
Linear-by-Linear Association	2.798	1	.094		
N of Valid Cases	50				

In correlation between Age > 50 yrs with incidence of mortality, our study didn't show statistically significant result with p >0.05 ie**. 0.176**

Showing correlation of sex with incidence of mortality



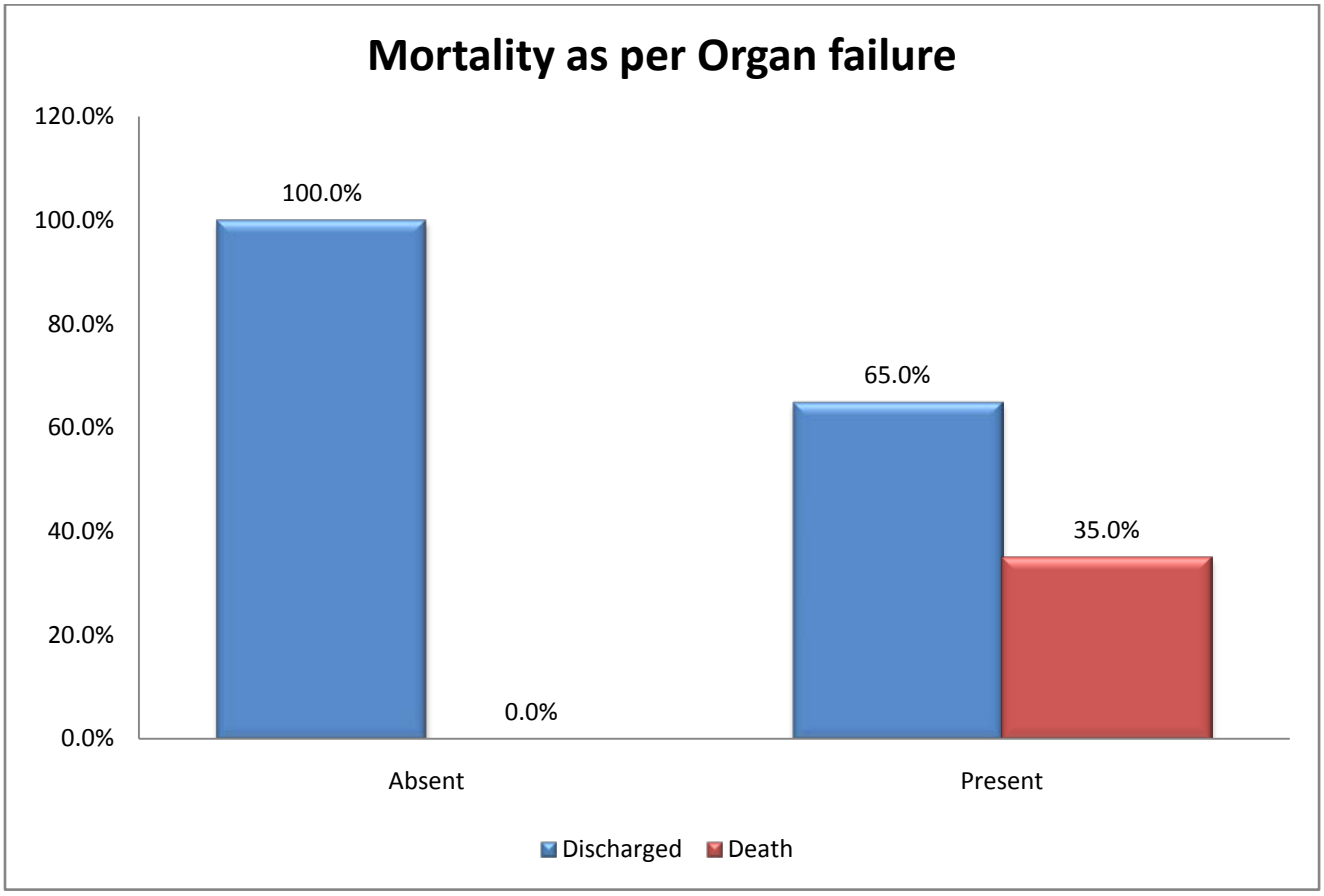
Sex	Frequency	Mortality	Discharged
Male	45 (90%)	7(15.6%)	38(84.4%)
Female	5 (10%)	0	5(100%)

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	.904[a]	1	.342		
Continuity Correction[b]	.074	1	.786		
Likelihood Ratio	1.596	1	.206		
Fisher's Exact Test				1.000	.454
Linear-by-Linear Association	.886	1	.346		
N of Valid Cases	50				

In correlation of sex with incidence of mortality, p value in our study was 1.000 which is statistically not significant &shows contrast results with MPI.

Showing correlation of organ failure with incidence of mortality

Organ Failure	Frequency (Percent)	Mortality	Discharged
Absent	30 (60%)	0	30(100%)
Present	20 (40%)	7(35%)	13(65%)

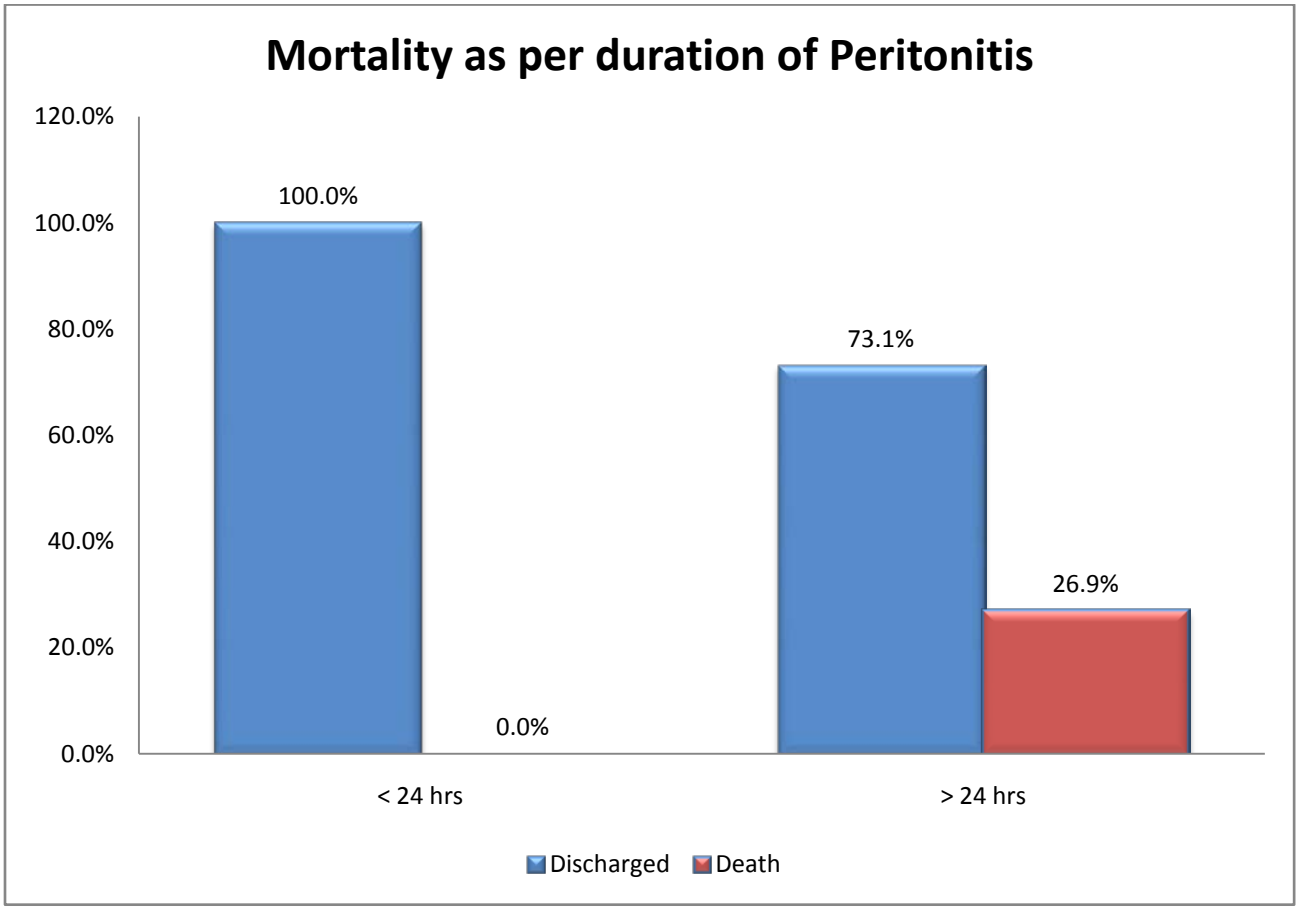


	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	12.209[a]	1	.000		
Continuity Correction[b]	9.475	1	.002		
Likelihood Ratio	14.598	1	.000		
Fisher's Exact Test				.001	.001
Linear-by-Linear Association	11.965	1	.001		
N of Valid Cases	50				

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

Showing correlation of preoperative duration with incidence of mortality

PreOperative duration of peritonitis	Frequency (Percent)	Mortality	Discharged
<24 hours	24 (48%)	0	24(100%)
>24 hours	26(52%)	7(26.9%)	19(73.1%)

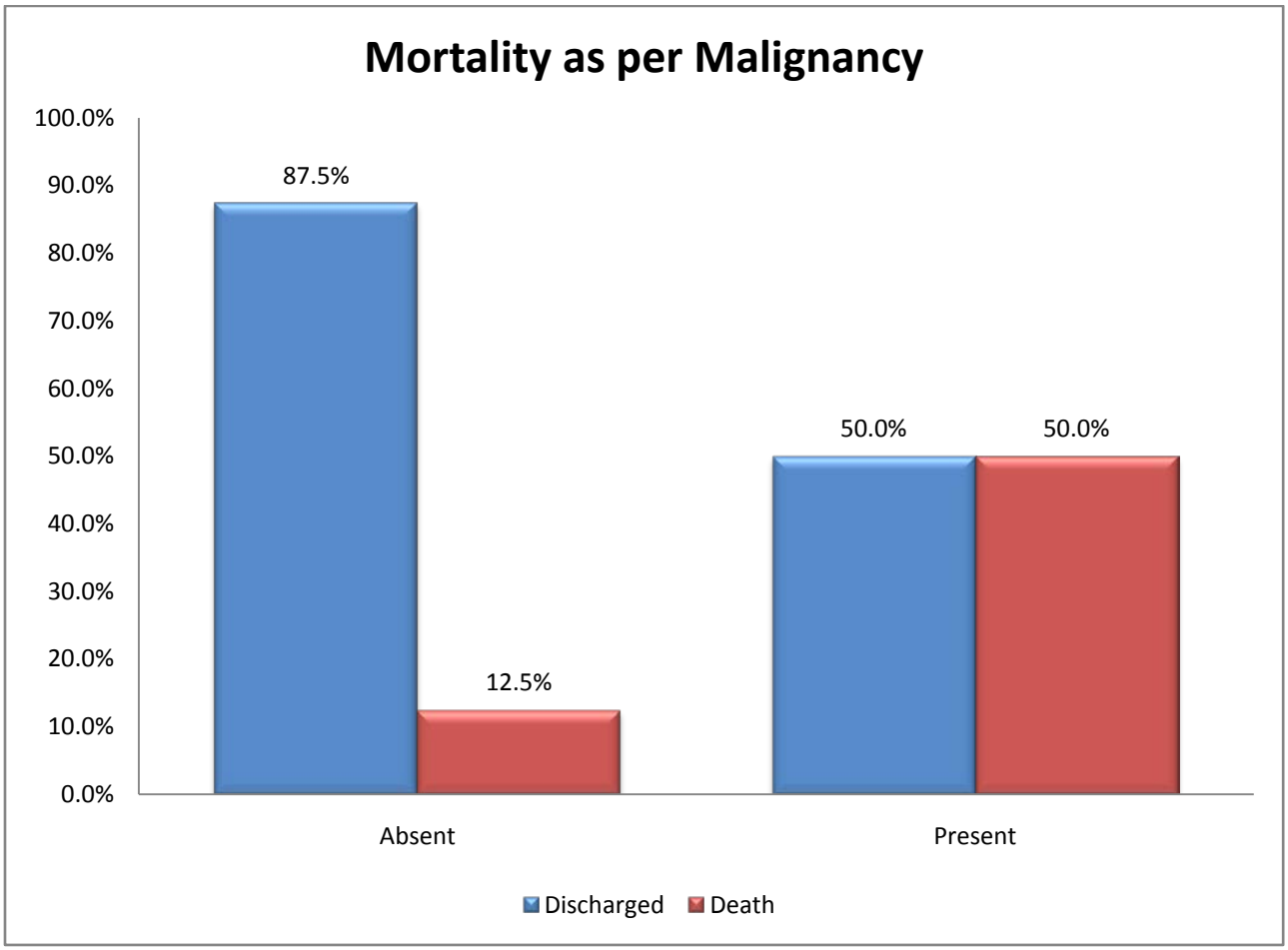


	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	7.513[a]	1	.006		
Continuity Correction[b]	5.444	1	.020		
Likelihood Ratio	10.207	1	.001		
Fisher's Exact Test				.010	.007
Linear-by-Linear Association	7.363	1	.007		
N of Valid Cases	50				

In correlation of preoperative duration with incidence of mortality, our study showed statistically significant result with p <0.010 showing correlation between pre operative duration with incidence of mortality

Showing correlation between presence of malignancy with incidence of mortality

Malignancy	Frequency (Percent)	Mortality	Discharged
Absent	48 (96%)	6(12.5%)	42(87.5%)
Present	2(4%)	1(50%)	1(50%)

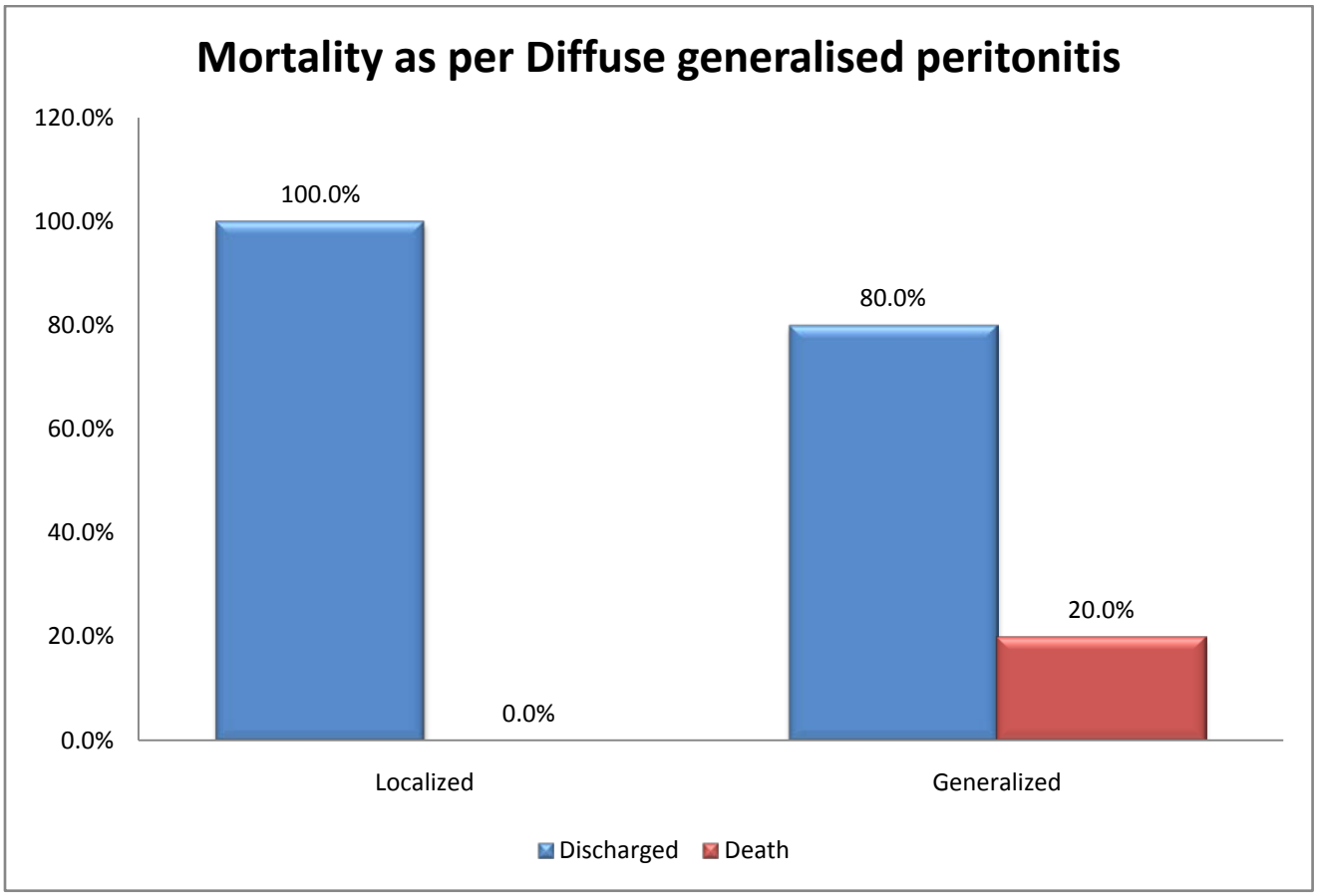


	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	2.243[a]	1	.134		
Continuity Correction[b]	.209	1	.647		
Likelihood Ratio	1.554	1	.213		
Fisher's Exact Test				.263	.263
Linear-by-Linear Association	2.198	1	.138		
N of Valid Cases	50				

In correlation of malignancy with incidence of mortality, p value in our study was 0.263 which is statistically not significant &shows contrast results with MPI.

Showing correlation between type of peritonitis with incidence of mortality

Distribution of Peritonitis	Frequency (Percent)	Mortality	Discharged
Localised	15 (30%)	0	15(100%)
Generalised	35(70%)	7(20%)	28(80%)

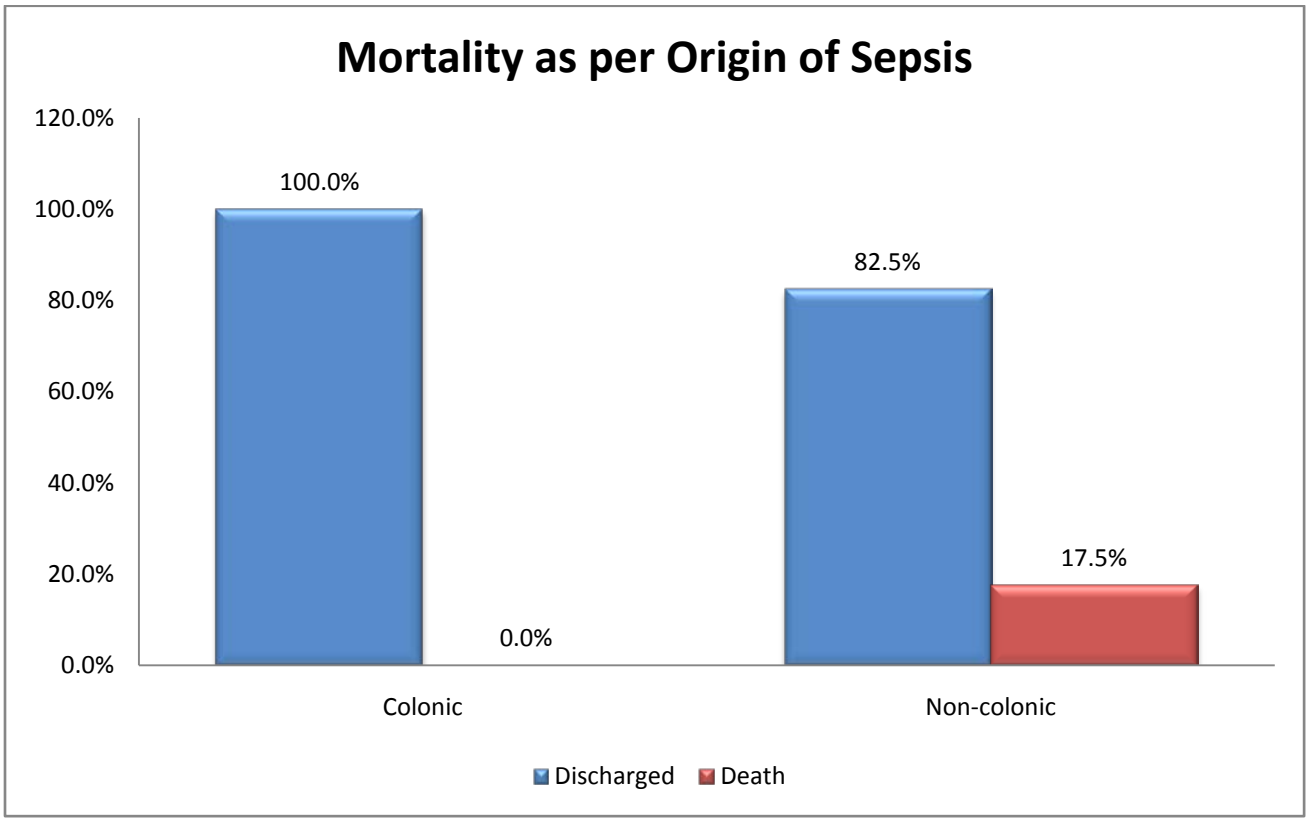


	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	3.488[a]	1	.062		
Continuity Correction[b]	2.025	1	.155		
Likelihood Ratio	5.468	1	.019		
Fisher's Exact Test				.087	.067
Linear-by-Linear Association	3.419	1	.064		
N of Valid Cases	50				

In correlation of type of peritonitis with incidence of mortality, p value in our study was 0.062 which is statistically not significant.

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

Origin of sepsis	Frequency (Percent)	Mortality	Discharged
Colonic	10 (20%)	0	10(100%)
Non Colonic	40 (80%)	7(17.5%)	33(82.5%)

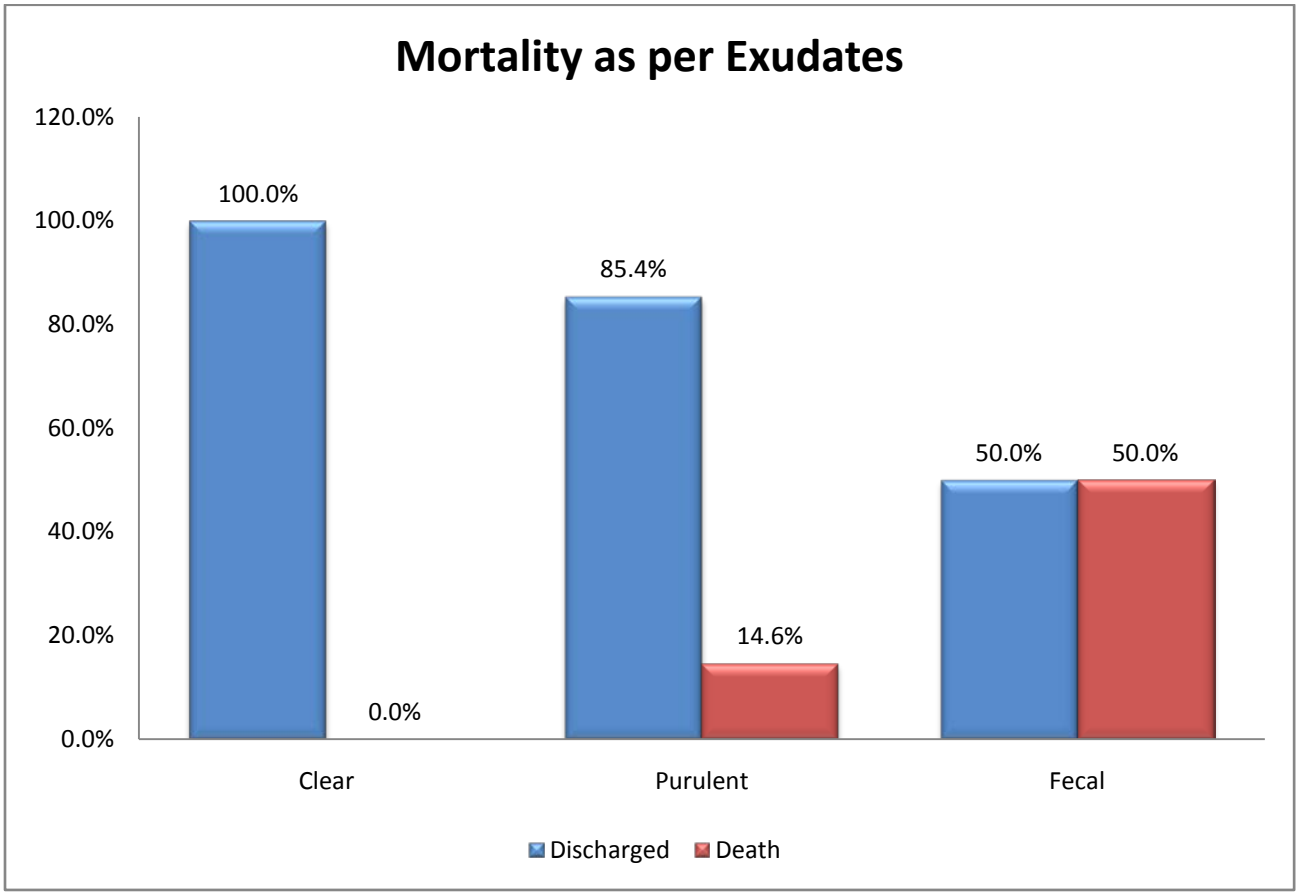


	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	2.035[a]	1	.154		
Continuity Correction[b]	.841	1	.359		
Likelihood Ratio	3.398	1	.065		
Fisher's Exact Test				.319	.187
Linear-by-Linear Association	1.994	1	.158		
N of Valid Cases	50				

In correlation of origin of sepsis with incidence of mortality, p value in our study was 0.154 which is statistically not significant.

Showing correlation between character of exudate with incidence of mortality

Exudates	Frequency (Percent)	Mortality	Discharged
Clear	7 (14%)	0	7(100%)
Purulent	41 (82%)	6(14.6%)	35(85.4%)
Fecal	2 (4%)	1(50%)	1(50%)

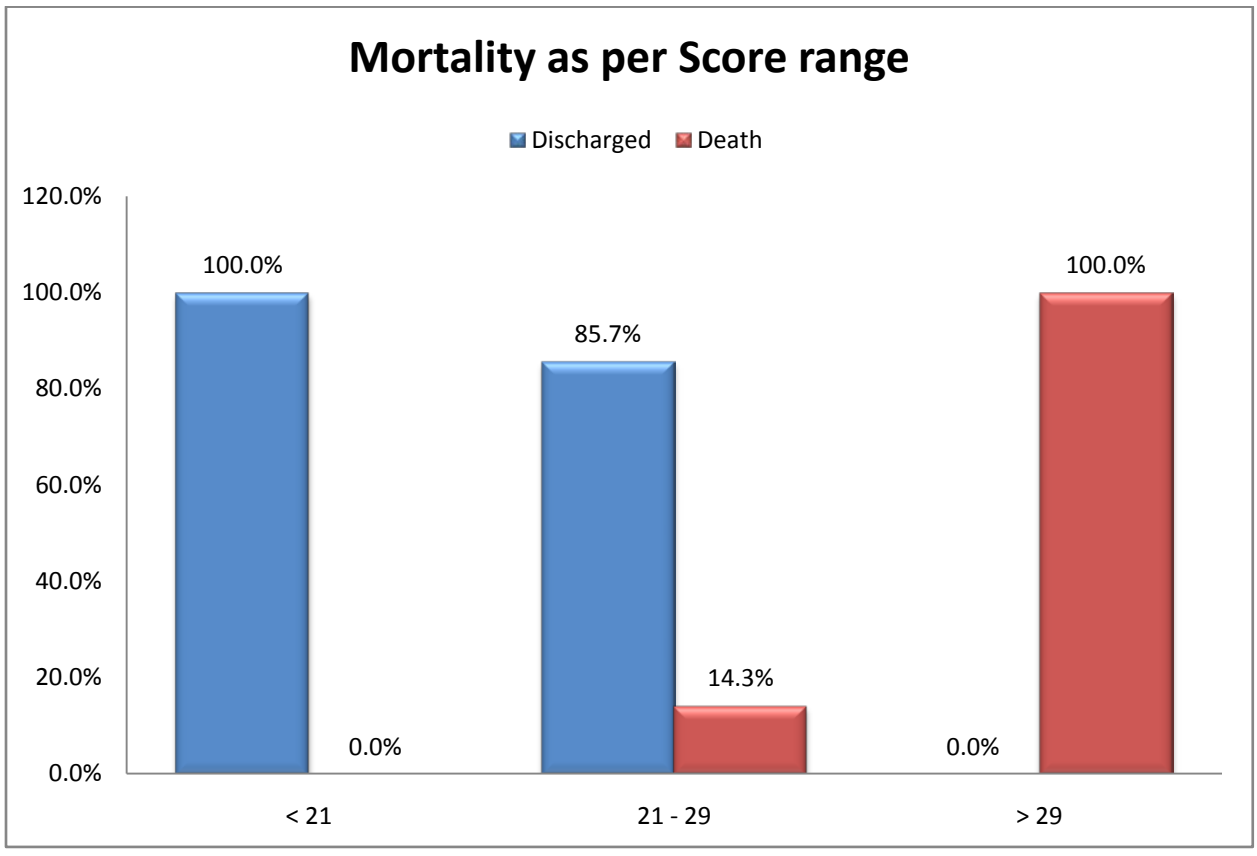


	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	3.306[a]	2	.191
Likelihood Ratio	3.586	2	.166
Linear-by-Linear Association	2.767	1	.096
N of Valid Cases	50		

In correlation of type of exudate with incidence of mortality, p value in our study was 0.191 which is statistically not significant .

Showing correlation of MPI score with incidence of mortality

MPI	Frequency	Mortality
<21	25	0
21-29	21	3 (14.28%)
>29	4	4 (100%)

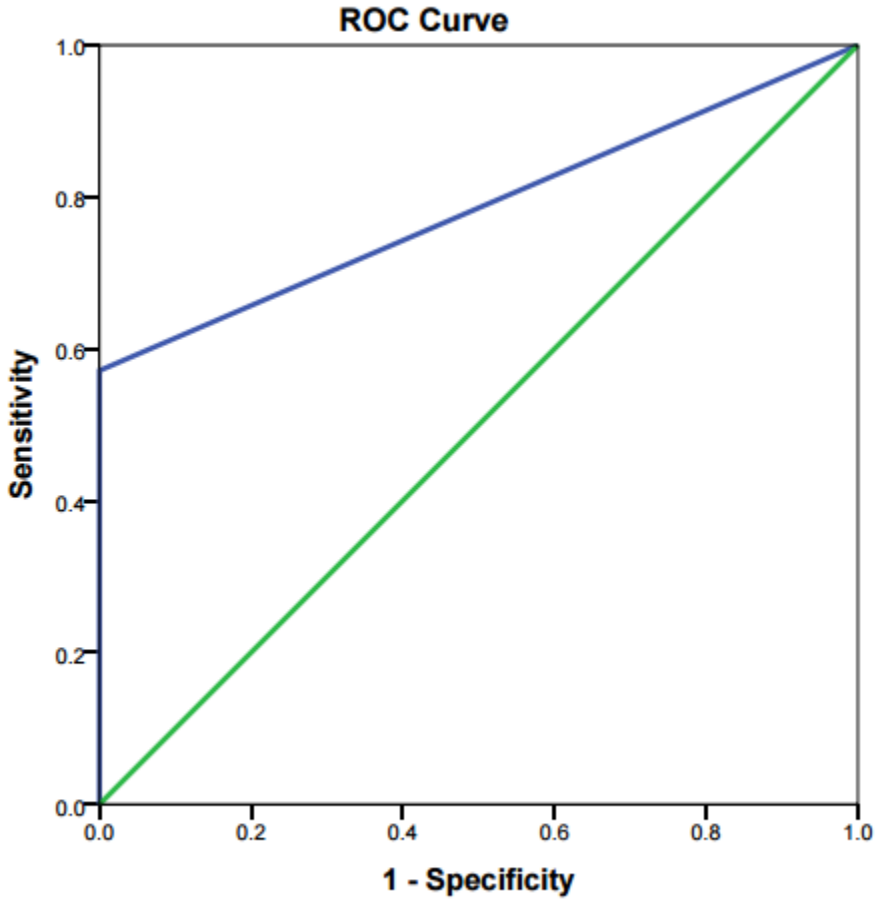


	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	26.708[a]	1	.0005		
Continuity Correction[b]	19.508	1	.0005		
Likelihood Ratio	18.316	1	.0005		
Fisher's Exact Test				.0005	.0005
Linear-by-Linear Association	26.174	1	.0005		
N of Valid Cases	50				

In our study mortality rate among patients with MPI score > 29 was 100% and with MPI < 21 was 0, which is statistically significant with p <0.0005

MPI Score range		Outcome		Total
		Death	Discharged	
<21	Count	0	25	25
	%	0.0%	100.0%	100.0%
21-29	Count	3	18	21
	%	14.3%	85.7%	100.0%
>29	Count	4	0	4
	%	100.0%	0.0%	100.0%
Total	Count	7	43	50
	%	14.0%	86.0%	100.0%

ROC Curve



Diagonal segments are produced by ties.

Sensitivity	57.1
Specificity	100.0
PPV	100.0
NPV	93.5
Accuracy %	94.0

Case Processing Summary

OUTCOME	Valid N (listwise)
Positive[a]	7
Negative	43

Area Under the Curve

Test Result Variable(s): Score

Area	Std. Error[a]	Asymptotic Sig.[b]	Asymptotic 95% Confidence Interval	
			Lower Bound	Upper Bound
.786	.120	.016	.551	1.000

Variant		Outcome		P Value
		Discharged	Death	
Age >50 years(n)		11	4	.091
		73.33%	26.67%	
Female(n)		5	0	.342
		100%	0%	
Organ Failure(n)		13	7	<0.0005
		65%	35%	
Malignancy(n)		1	1	.134
		50%	50%	
Duration > 24 hrs(n)		19	7	.006
		73.07%	26.93%	
Origin of Sepsis Non Colonic(n)		33	7	.154
		82.5%	17.5%	
Generalized Peritonitis (n)		28	7	.062
		80%	20%	
Exudate	Clear	7(100%)	0	.191
	Purulent	35(85.36%)	6(14.64%)	
	Fecal	1(50%)	1(50%)	

Showing distribution of MPI variables and outcome of patients

Among the MPI variables of adverse outcome ,Organ Failure and time >24 hrs showed statistical significance in predicting mortality.

Even though other parameters such as age ,malignancy , generalised peritonitis showed suggestion with mortality, they failed to show statistical significance in our study

DISCUSSION

SPECTRUM OF PERFORATION PERITONITIS

AGE

Total of 50 patients were studied. The mean age of the study group was 41.4 years and The age group of 31-45 contains maximum (34%) patients followed by 16-30 years. Oldest patients was 80 years and youngest was of 13 years..

In a study by Rajendra Singh Jhobta 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 Memon (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 2016 41.4

The number of patients in the age group <50 years were 35 i.e. 70% and 15 patients of the study population i.e. 30% 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.

SEX

In our study the incidence of male sex was 90 % while that of female sex was 10 %.

In a study by Rajender Singh Jhobta (2006) regarding the spectrum of perforation peritonitis in India 84% patients 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	2016	10%	90%

In a study by Aijaz A Meman (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 L (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

In our study , maximum number of patients had duodenal perforation 29(58 %) followed by Appendicular perforation (20 %), gastric perforations were (12%), and ileal perforation (10%).

In a study by Rajender Singh Jhobta et al (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)
Duodenal	15 %	32.29 %	18.7 %	22.7 %
Ileal	24.5 %	27.3 %	15 %	-
Appendicular	10 %	18.1 %	41.1 %	15.9 %
Others	50.5%	22.31%	25.2%	61.4%

Present Study duodenal- 58% , Ileal(10%), Appendicular (20%),others- gastric (12%).In a study by Rodolfo L 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.

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 common.

CLINICAL FEATURES

In our study pain in abdomen was the most common symptom and 98 % of patients had pain abdomen at presentation, while 38% of patients have difficulty in passing flatus or motion. Distension of abdomen was present in 54 % of patients, 62% patients had episodes of vomiting, 58 % patients had fever at presentation.

In a study by Shantanu Kumar Sahuet al the commonest presenting symptom was abdominal pain (100%), followed by distension of abdomen (82%), constipation, vomiting and fever.

In a study by Rajender Singh Jhobtaet al pain was present in 98% of patients, followed by vomiting (59%), abdominal distension (44%), constipation (58%), fever (35%), and diarrhoea (7%).

DISTRIBUTION OF ORGAN FAILURE

In our study 20 patients i.e.40% of the study population shows evidence of organ failure at presentation.

Distribution of organ failure in different studies are –

48.5 % in MM Correiaet al

11.5 % in Rodolf L et al

20 % in Murut Kologluet al

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

PREOPERATIVE DURATION

In our study 24patients i.e. 48 % presented within 24 hours while 26 patients i.e. 52 % 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 L31	54.48%	49.42%
MM Correia29	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 and lack of awareness among the study population

PRESENCE OF MALIGNANCY

In our study 2 patient's (4 %) had malignancy. Both of them were carcinoma stomach with perforation.

In a study by Rodolf L patients had malignancy.

In a study by M.M. Correia 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

In our study 10 patients i.e. 20 % had colonic origin of sepsis while in the rest 40 patients the origin of sepsis was non colonic.

In the study by Rudolf L 12.64% of patient’ s had colonic origin of sepsis.

In the study by Rajendra Singh Jobhta 3.76% of patient’ s had colonic origin of sepsis..

Colonic perforation may present with faecal exudates and a severe form of peritonitis.

TYPE OF PERITONITIS

In other study the distribution of type of peritonitis was as below

STUDY	DIFFUSE	LOCALISED
Rajender Jhobta	83 %	17 %
Rodolf L	49 %	65.51
Ohmann	65.36 %	34.64 %

In our study patients 35 i.e. 70% presented with a diffuse form of peritonitis while the remaining 15 i.e. 30 % presented with localized peritonitis.

Diffuse peritonitis is associated with a severe inflammatory reaction and development of sepsis and multi organ failure.Localization of peritonitis is body’s defense mechanism and will lead to formation of abscess.

NATURE OF EXUDATES

In our study 7 patients i.e. 14 % had clear exudates, 41 patients i.e. 82 % had purulent exudates and 2 patients i.e. 4 % had faecal exudates.

In a study by Rodolf L 69.5% has clear exudates and 21.8% had purulent exudates.

In a study by Rajender Singh Jhobta 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

25 (50%) patients had MPI score of less than 21.

21 (42%) patients had MPI score between 21 to 29

4 (8%) patients had MPI score greater than 29

In the original study by Wacha and Linder 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

Among the 50 patients studied by us 7 patients died thus placing the mortality at 14%.

Atsushi Hourichi in their study of perforation peritonitis had a mortality of 23.1%.

Koperna T 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 35 patients were less than 50 years of age. Out of 35 patients of age less than 50 years 3 (8.6%) patients died while out of 15 patients with age more than 50 years 4 (26.7 %) patients died

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..

CORRELATION BETWEEN SEX AND MORTALITY

In our study total of 45 patients belong to the male sex among which 7 died resulting in a mortality of 15.6 %. But female sex had no mortality. & thus female sex has not qualified to be included in the variables of adverse outcome.

CORRELATION BETWEEN ORGAN FAILURE AND MORTALITY

In our study a total of 20 patients showed evidence of organ failure. 7 patients died among this 20 patients thus resulting in a mortality rate of 35 %.none of the patients who showed no evidence of organ failure died.

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. 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 PREOPRATIVE DURATION OF PERITONITIS AND MORTALITY

In our study out of the 24 patients with a preoperative duration of peritonitis of less than 24 hrs no patient died. Out of the 26 patients who have preoperative duration of peritonitis of more than 24 hrs, 7 patients died thus placing the mortality rate of 26.9 %.

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

In our study 2 patients had malignancy. 1 out of the 2 patients expired thus placing the mortality rate in presence of malignancy to a whopping 50 %.

Peritonitis in oncological patients is generally caused by a ruptured viscous. The diagnosis may be delayed by recent postoperative status, immuno depression, 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.

CORRELATION BETWEEN TYPE OF PERITONITIS AND MORTALITY

In our study 35 patients had diffuse peritonitis and 15 patients had localized peritonitis.There was no mortality in patients with localized peritonitis while in patients with diffuse peritonitis there were 7 deaths with a mortality of 20 %.

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

In our study 10 patients had colonic origin of sepsis out of which no patients died while in non colonic origin of sepsis the mortality rate in our study was 17.5%.

John Bohnen 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 al in their study of 141 patients with colorectal perforations found a mortality of 36.9%.

CORELATION BETWEEN CHARACTER OF EXUDATE AND MORTALITY

In our study among the 7 patients with clear exudates none of the patients died.6 (14.6 %) patients died among the 41 patients with purulent exudates.1 (50 %) patients died among 2 patients with faecal exudates.

Thus the mortality in patients with clear exudates was 0 % purulent exudate was 14.6 % while in faecal exudate the mortality was 50 %.

In the study of Rodolfo L 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 al 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.

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.

STATISTICAL VALIDATION OF MANNHEIM PERITONITIS INDEX

Rodolfo L et al 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 al 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.

AbrarMaqbool Qureshi et al 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 the there was no death in patients with MPI score less than 21 ,in MPI score between 21to 29 the mortality was 14.28%.,while in patients with MPI score greater than 29 the mortality was 100%.

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) female sex is also considered as a adverse prognostic factor by Linder and Waccha contrary to our study.

c) In our study the mortality rate is 0 % for female sex and 15.6 % for male sex & was statistically not significant with p value of 0.342, 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 al 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

CONCLUSION

Mannheim Peritonitis index is a useful method to determine study group outcome in patients with peritonitis.

Among 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 ,**Organ Failure** and **time >24 hrs** showed statistical significance in predicting mortality.

Even though other parameters such as age ,malignancy , generalised peritonitis showed correlation with mortality, they failed to show statistical significance in our study.

In our study we found that :.

1.Female sex was associated with better outcome as compared to male sex.

2.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:

- ❖ Ratify the effectiveness of different treatment regimen.
- ❖ Indicate individual risk to select patient's who may require a more aggressive surgical approach.
- ❖ 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 14.28% and >29 was 100%

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.”

BIBLIOGRAPHY

1. C. Ohmann, prognostic scores and Design of clinical studies, Infection 26 (1998) No. 5.
2. Christian Ohmann, Qin Yang, Toni Hau, Prognostic Modelling in Peritonitis. Eur J. Surg 1997; 163 : 53-60.
3. Linder MM, Wacha H. The Mannheim peritonitis index. An instrument for the intraoperative prognosis of peritonitis. Chirurg,1987, Feb ; 58 (2) 84-92.
4. Deitch EA, multiple organ failure: pathophysiology & potential future therapy.AnnSurg 1992, 216: 117-34.
5. Hertzler A.E. The peritoneum. St. Louis: Mosby CV Co; 1919:12.
6. James W. Dobbie, Surgical peritonitis: Its Relevance to The pathogenesis of peritonitis in CAPD, peritioneal Dialysis, International Journal of Oral and Maxillofacial Surgery, 31, 206-209Vol.8: pg 241-248: 1998 .
7. F Charles Brunicardi, Schwartz Principles of surgery. 8th Edition.
8. Gerard M Doherty, Current surgical diagnosis and treatment. 12th Edition.
9. Keith L Moore, Arthur F. Dalley, Clinically oriented anatomy. 5th Edition.
10. Sabiston text book of surgery. The biological basis of modern surgical practice. 18th Edition.
11. Caroline C. Johnson, James Baldessarre. Peritonitis: Update on pathophysiology, clinical manifestations, and management. Clinical infectious Disease 1997-24: 1035-47.
12. Topely N. Mackenzie RK, Williams JD. Macrophages and Mesothelial cells in bacterial peritonitis. Immunobiology, 1996, Oct.:195 (4-5):563-73.
13. Wolfgang Sendit, Rainer Amberg. Secondary peritonitis: Severity of disease and Activation of peritoneal cells. Eur J Surg 2001; 167; 426-432.

14. D.H. Witmann, Management of secondary peritonitis, Annals of Surgery 1996, 224;10-18
15. A.Prakash, D. Sharma, et al, Effect of Candida infection on outcome in patients with perforation peritonitis; Indian Journal of Gastroenterology may-Jun: 27: 107-109, 2008.
16. P. Panhofer, M.Riedl, Clinical outcome and microbial flora in patients with secondary and tertiary peritonitis, EurSurg (2007) 39/4: 259-264.
17. J.L. Dawson. A study of some factors affecting the mortality rate in peritonitis, Gut 1963. 4(4): 368–372.
18. CecilieSvanes M.D. A multifactorial analysis of factors related to lethality after treatment of perforated gastroducodenal ulcer 1935-1985 Ann surgery 1988, 0182.
19. Fugger R, Rogy M et al Validation study of the Mannheim peritonitis index. Chirurg. 1988; 59:598-601.
20. A Billing, D. Frohlich. Predication of outcome using the Mannheim peritonitisindex in 2003 patients. British Journal of Surgery 1994, 81, 209-213.
21. Demmel N, Magg A. The value of clinical parameters for determining the prognosis of peritonitis validation of the mannheium peritonitis index. Langenbecks Arch Chirurgy, 1994, 379(3):152-158.
22. Liverani A, Correnti SF. The value of 2 distinct prognostic scores in patients with peritonitis. The MPI versus the APACHE II score ,Chirurg. 1990; 61 (4) : 297-300.
23. Arch Surgery, Prognosis in intra abdominal infection; Multivariate analysis of 604 patients. 1996, 131 (6): 641-5.
24. James M. Watters, The influence of age on the severity of peritonitis. Canadian Journal of surgery 1996; 39; 142-146.

25. Bosscha K. Br, Prognostic scoring systems to predict outcome in peritonitis and intra- abdominal sepsis.British J. Surg. 1997 Nov ; 84 (11): 1532-34.
26. T.M. Cook, C.J.E. Day Hospital mortality after urgent and emergency laparotomy in patients aged 65 yr and over. Risk and prediction of risk using multiple logistic regression analysis. British Journal of Anaesthesia 1998; 80:776-781.
27. Sokmen S; Effectiveness of the Mannheim Peritonitis index in patients with peritonitis. Turkish Journal of trauma & emergency surgery. 2001 ; 792) :100-3.
28. Murat Kologlu, Validation of MPI and PIA II in two different groups of patients with secondary peritonitis. HepatoGastroenterology 2001;48: 147-151.
29. M.M. Correia, Prediction of Death using the Mannheim Peritonitis Index in Oncologic patients. RevistaBrasileira de Cancerologia, 2001 47 (1) : 63-68.
30. Thomas Koperna et al, Relaparotomy in peritonitis: Prognosis and treatment of patients with persisting intraabdominal infections. World Journal of Surgery. Vol. 24 Number 1, Jan. 2000 page 32-37.
31. Rodolfo L. Bracho-RiquelmeMC,Men C, Mannheim Peritonitis Index Validation Study at the Hospital General de Durango (Mexico), Cir Circuj 2002;70:217-225.
32. Juan J., Mortality associated with emergency abdominal surgery in the elderly. Can. J. surgery, Vol. 46, No. 2 April 2003.
33. Shuhei Komatsu, Prognostic factors and scoring system for survival in colonic perforation. Hepato-Gastroenterology 2005; 52: 761-764.
34. Mulari K. Severe secondary peritonitis following gastrointestinal tract perforation. Scand J. Surg. 2004: 93(3) : 204-8.
35. Yoshiko Kusumoto, Study of Mannheim peritonitis index to predict outcome of patients with peritonitis. Jpn. Journal Gastroenterological Surg 37; 7-1.

36. AbrarMaqbool Qureshi, Predictive power of Mannheim peritonitis index. Journal of College of Physicians and Surgeons of Pakistan 2005 Nov; 15 (11) 693-6.
37. JyrkiTapaniMakela, Prognostic factors of perforated sigmoid diverticultitis in the elderly. Digestive Surg. 2005; 22 : 100-106.
38. Ali YoghoobiNotash et al. Evaluation of Mannheim peritonitis index and multiple organ failure score in patients with peritonitis. Indian Journal of Gastroenterology 2005; Vol. 25, Issue 5 Pg 197200.
39. Rajender Singh Jhobta, Ashok Kumar Attri, Spectrum of performation peritonitis in India-review of 504 consecutive cases. World Journal of Emergency Surgery 2006, 1:26.
40. Atsushi Horiuchi, Evaluation of prognostic factors and scoring system in colonic perforation. World J of gastroenterology 2007. June 21 13 ; (23) 3228-3231.
41. Thomas E Rix, Per-operative risk scores for the prediction of outcome in elderly people who require emergency surgery. World Journal of Emergency Surgery 2007, 2: 16.
42. JefreyVermeulen, Outcome after emergency surgery for acute perforated diverticulitis in 200 cases. Digestive Surg. 2007; 24:361-366.
43. M. Hynninen, Organ dysfunction and long term outcome in secondary peritonitis. Langenbecks Arch Surg (2008) 393:81-86.
44. C G Nwigwe, Validation of Mannheim peritonitis index (A Nigerian study) Ebonyl Medical Journal Vol. 6 (1) 2007 pg: 3-8.
45. Christian P. Schneider, Prognostic factors in critically III patients suffering from secondary peritonitis: A retrospective, observational, survival time analysis. World J Surg. (2009) 33: 34–43.

46. Chao-Wen Hsu, Colorectal perforation: Spectrum of the disease and its mortality. J Soc Colon Rectal Surgeon (Taiwan) September 2007, 81.
47. A Mishra, D. Sharma et al, A Simplified scoring system for peptic ulcer perforation in developing countries. Indian Journal of Gastroenterology 2003; 22: 49-53.
48. Aijaz A Memon, Spectrum of disease in patients with non-traumatic acute abdomen. World J of Emergency Surgery 2006, 25: 2537-2545.
49. Shantanu Kumar Sahu, Amit Gupta. Outcome of secondary peritonitis Based on Apache II score. The Internet Journal of Surgery ISSN : 1528-8242.
50. Wahl N,Minkus A .- Prognostically relevant factors in intraabdominal infection, Langenbecks Arch Chir,1992,377: 237.
51. John Bohnen, Micheline Boulanger- Prognosis in generalized peritonitis, Arch Surg. 1983; 118(3):285-290.
52. Killingback M. Diverticular disease, In : Allan RN,KeighleyMRB,Eds. Inflammatory bowel diseases ,Edinburgh: Churchill Livingstone, 1983: 504 -11.

APPENDIX - I : ETHICAL COMMITTEE CLEAREANCE

INSTITUTIONAL ETHICAL COMMITTEE,
STANLEY MEDICAL COLLEGE, CHENNAI-1

Title of the Work : Evaluation of outcomes in patients with perforation Peritonitis using Mannheim's peritonitis index.

Principal Investigator : Dr. G. P. Kumaran

Designation : PG MS (General Surgery)

Department : Department of General Surgery,
Government Stanley Medical College,
Chennai-01.

The request for an approval from the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 14.06.2016 at the Council Hall, Stanley Medical College, Chennai-1 at 2PM

The members of the Committee, the secretary and the Chairman are pleased to approve the proposed work mentioned above, submitted by the principal investigator.

The Principal investigator and their team are directed to adhere to the guidelines given below:

1. You should inform the IEC in case of changes in study procedure, site investigator investigation or guide or any other changes.
2. You should not deviate from the area of the work for which you applied for ethical clearance.
3. You should inform the IEC immediately, in case of any adverse events or serious adverse reaction.
4. You should abide to the rules and regulation of the institution(s).
5. You should complete the work within the specified period and if any extension of time is required, you should apply for permission again and do the work.
6. You should submit the summary of the work to the ethical committee on completion of the work.

19/9/16

MEMBER SECRETARY,
IEC, SMC, CHENNAI

MEMBER SECRETARY
ETHICAL COMMITTEE,
STANLEY MEDICAL COLLEGE
CHENNAI-600 001.

APPENDIX-II

PROFORMA

EVALUATION OF OUTCOME IN PATIENTS' WITH PERFORATION PERITONITIS USING MANNHEIM'S PERITONITIS INDEX

Investigator: Dr.G.P.KUMARAN, PG 3rd year – MS (General Surgery)

Guide: Prof. Dr.A.K.RAJENDRAN, Chief, Unit S4

NAME : SL. NO:

AGE /SEX:

ADDRESS WITH CONTACT NUMBER:

IP NO:

DATE OF ADMISSION:

DATE OF SURGERY:

HISTORY OF PRESENTING ILLNESS:

PAST HISTORY:

Whether a known case of DM/Hypertension/Asthma/TB/epilepsy/cardiac illness

H/O SIMILAR EPISODES IN THE PAST, IF ANY:

CLINICAL EXAMINATION:

GENERAL EXAMINATION: TEMP: P.R: B.P: R.R

SYSTEMIC EXAMINATION:

CVS

RS

PER ABDOMEN:

CLINICAL DIAGNOSIS:

INVESTIGATIONS:

HEMATOLOGY

HB PCV RBC TC DC PLT ESR

RBS

B.UREA

S.CREAT

LFT

S.Na+

S.K+

S.Cl-

S.HCO3-

PaO2

PaCO2

CHEST X RAY :

ABD X RAY:

USG ABD:

CT/CECT:

LAPARATOMY FINDINGS:

PATIENT CLINICAL COURSE:

MANNHEIM PERITONITIS INDEX:

Patient Information Module

You are being invited to be a subject in this study.

Before you participate in this study, I am giving you the following details about this trial, which includes the aims, methodology, intervention, possible side effects, if any and outcomes:

All consenting patients who are admitted with perforative peritonitis will be included in this study. A detailed clinical history will be taken following a standardized proforma. A detailed clinical examination will be made and relevant basic investigations will be done at the time of admission. Effectiveness of Mannheim`s peritonitis index scoring system will be evaluated. The results arising from this study will be analyzed and used for academic purposes. You will begiven clear instructions at every step and you are free to ask/ clarify any doubts. Your identity will remain confidential. You are free to withdraw from this trial at any point of time, without any prior notice &/ or without any medical or legal implications.

I request you to volunteer for this study.

Thanking You,

Investigator's Sign

(Dr.G.P.KUMARAN)

Patient's Sign

(Name:)

Informed Consent

Name: Age/ Sex: IP:

I herewith declare that I have been explained in a language fully understood by me regarding the purpose of this study, methodology, proposed intervention, plausible side effects, if any and sequelae.

I have been given an opportunity to discuss my doubts and I have received the appropriate explanation.

I understand that my participation in this study is completely voluntary and that I am free to withdraw from this study at anytime without any prior notice &/ or without having my medical or legal rights affected.

I permit the author and the research team full access to all my records at any point, even if I have withdrawn from the study. However my identity will not be revealed to any third party or publication.

I herewith permit the author and the research team to use the results and conclusions arising from this study for any academic purpose, including but not limited to dissertation/ thesis or publication or presentation in any level.

Therefore, in my full conscience, I give consent to be included in the study and to undergo any investigation or any intervention therein.

Patient's Sign Investigator's Sign

(Dr.G.P.KUMARAN)

ஆராய்ச்சிஒப்புதல்படிவம்

ஆராய்ச்சியின் தலைப்பு:ஸ்டான்லி பொது மருத்துவமனையில் துளையினாலான வயிற்றறை உறையழற்சி (**PERFORATION PERITONITIS**) அறுவை சிகிச்சை செய்து கொள்பவர்களின் விளைவை **MANNHEIM PERITONITIS INDEX** மதிப்பெண்களின் மூலம் மதிப்பாய்வு

பங்குகொள்வரின் பெயர் :

ஆராய்ச்சிசெய்பவரின் பெயர் :குமரன்.ஜி.பி.

இடம் :அரசு பொது மருத்துவமனை, சென்னை – 600001

_____ எனும் நான், எனக்கு கொடுத்துள்ள தகவல் தாளை படித்து புரிந்து கொண்டேன். நான் பதினெட்டு வயதை கடந்துள்ளதால், என்னுடைய சுயநினைவுடனும், முழுசுகந்திரதுடனும், இந்த ஆராய்ச்சியில் என்னை சேர்த்துக் கொள்ள சம்மதிக்கிறேன்.

நான், எனக்கு கொடுத்துள்ள தகவல் தாளை படித்து புரிந்துகொண்டேன்.

எனக்கு இந்த ஆராய்ச்சியின் ஒப்புதல் படிவம் விளக்கபட்டுள்ளது.

எனக்கு இந்த ஆராய்ச்சியின் நோக்கமும், விவரங்களும் விளக்கப்பட்டது.

எனக்கு என்னுடைய உரிமைகள் பற்றி விளக்கப்பட்டது.

இந்த ஆராய்ச்சியில் இருந்து நான் என்நேரமும் பின்வாங்கலாம் என்றும், அதனால் எந்த பின்விளைவும் ஏற்படாது என்று புரிந்துகொண்டேன்.

என்னை பற்றி எந்ததகவலும், அடையாளங்களும் வெளியிடப்படமாட்டது என்பதையும் புரிந்து கொண்டேன்.

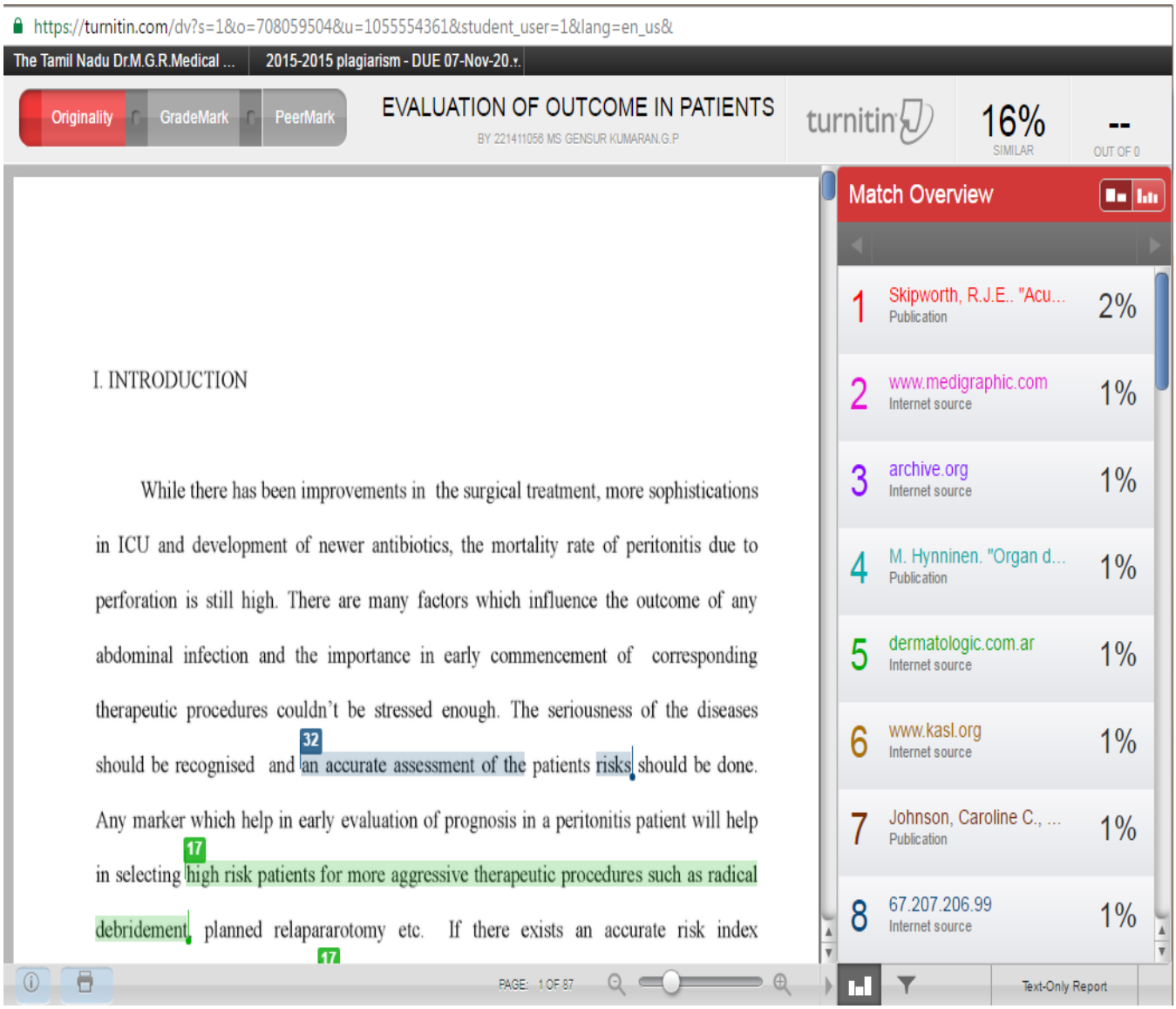
என்னுடைய முழுசுகந்திரதுடனும் இந்த ஆராய்ச்சியில் சேர்த்து கொள்ள சம்மதிக்கிறேன்.

பங்கேற்பாளர் கையொப்பம் ஆராய்ச்சியாளர் கையொப்பம்

நாள் :

இடம் : (குமரன்.ஜி.பி.)

APPENDIX III - - PLAGIARISM



APPENDIX IV — MASTER CHART

NAME	AGE	SEX	IP NO	DIAGNOSIS	SURGICAL PROCEDURE	AGE	GENDER	ORGAN FA	MALIGNA	DURATION	ORIGIN OI	DIFFUSE G	EXUDATES	TOTAL SC	POST OPEI
MR.VENKATESAN	39	M	1560688	CA STOMACH WITH PERFORATION	EL/ PATCH CLOSURE	0	0	7	4	0	4	6	6	27	
MR.GUNA	45	M	1561190	DUODENAL PERFORATION	EL/ PATCH CLOSURE	0	0	7	0	0	4	6	6	23	
MR.RAMESH	37	M	1562574	DUODENAL PERFORATION	EL/ PATCH CLOSURE	0	0	7	0	4	4	6	6	27	
MR.KALAIYARASAN	19	M	1562833	ILEAL PERFORATION	EL/ PRIMARY CLOSURE	0	0	0	0	4	4	6	6	20	
MR.GANESAN	47	M	1564478	DUODENAL PERFORATION	EL/ PATCH CLOSURE	0	0	7	0	4	4	6	6	27	
MR.MANIKANDAN	26	M	1566062	APPENDICULAR PERFORATION	EL / APPENDICECTOMY	0	0	0	0	4	0	6	0	10	
MRS.JANSI RANI	35	F	1566483	ILEAL PERFORATION	EL/ PRIMARY CLOSURE	0	5	7	0	0	4	0	6	22	
MR.BENNY	80	M	1566857	DUODENAL PERFORATION	EL/ PATCH CLOSURE	5	0	0	0	0	4	6	6	21	
MR.HARI	25	M	1567078	DUODENAL PERFORATION	EL/ PATCH CLOSURE	0	0	0	0	4	4	6	6	20	
MR.MUNUSAMY	33	M	1567563	DUODENAL PERFORATION	EL/ PATCH CLOSURE	0	0	0	0	0	4	0	6	10	
MR.ARUNACHALAM	41	M	1570456	DUODENAL PERFORATION	EL/ PATCH CLOSURE	0	0	0	0	4	4	0	6	14	
MR.RAVI	46	M	1570698	DUODENAL PERFORATION	EL/ PATCH CLOSURE	0	0	7	0	4	4	6	6	27	
MRS.THENMOZHI	21	F	1571076	APPENDICULAR PERFORATION	OPEN APPENDICECTOMY	0	5	0	0	0	0	0	0	5	
MR.BIJIMON	15	M	1590764	APPENDICULAR PERFORATION	EL / APPENDICECTOMY	0	0	0	0	4	0	6	6	16	
MR.KUMARAN	28	M	1590808	DUODENAL PERFORATION	EL/ PATCH CLOSURE	0	0	0	0	0	4	6	6	16	
MR.SOLAIYAPPAN	65	M	1591222	CA STOMACH WITH PERFORATION	EL/ PATCH CLOSURE	5	0	7	4	4	4	6	6	36	RE-DO LAF
MR.SIVALINGAM	70	M	1591455	DUODENAL PERFORATION	EL/ PATCH CLOSURE	5	0	0	0	4	4	0	6	19	
MR.DHAYALAN	40	M	1591460	DUODENAL PERFORATION	EL/ PATCH CLOSURE	0	0	7	0	4	4	6	6	27	
MR.JEEVA	22	M	1591628	DUODENAL PERFORATION	EL/ PATCH CLOSURE	0	0	0	0	0	4	6	6	16	
MRS.NIRMALA	77	F	1592503	GASTRIC PERFORATION	EL/ PATCH CLOSURE	0	5	0	0	0	4	6	6	21	
MR.CHANDRASEKAR	57	M	1592509	DUODENAL PERFORATION	EL/ PATCH CLOSURE	5	0	7	0	4	4	6	6	32	EXPIRED P
MR.RAMKUMAR	35	M	1592242	DUODENAL PERFORATION	EL/ PATCH CLOSURE	0	0	7	0	4	4	6	6	27	EXPIRED P
MR.SELVAVINAYAGAM	75	M	1593093	APPENDICULAR PERFORATION	EL / APPENDICECTOMY	5	0	0	0	0	0	6	6	17	
MR.NAGARATHINAM	50	M	1593305	GASTRIC PERFORATION	EL/ PATCH CLOSURE	5	0	0	0	4	4	0	6	19	

MR.MOHAMMED YUSU	13	M	1593388	APPENDICULAR PERFORATION	OPEN APPENDICECTOMY	0	0	0	0	4	0	0	0	4	
MR.SHAHUL	18	M	1593673	APPENDICULAR PERFORATION	OPEN APPENDICECTOMY	0	0	0	0	0	0	6	0	6	
MR.SHANKAR	35	M	1594053	APPENDICULAR PERFORATION	LAP CONVERTED TO OPEN APPENDICEC	0	0	0	0	0	0	0	6	6	
MR.BALAMURUGAN	35	M	1594306	DUODENAL PERFORATION	EL/ PATCH CLOSURE	0	0	7	0	0	4	6	6	23	
MR.KONDAIYAH	40	M	1600038	DUODENAL PERFORATION	EL/ PATCH CLOSURE	0	0	0	0	0	4	6	6	16	
MR.RAJ	55	M	1603024	DUODENAL PERFORATION	EL/ PATCH CLOSURE	5	0	7	0	0	4	6	6	28	
MR.MOHAMMED ALI	47	M	1603265	DUODENAL PERFORATION	EL/ PATCH CLOSURE	0	0	7	0	0	4	6	6	23	
MR.PRABHU	35	M	1603822	ILEAL PERFORATION	LAPARATOMY/ RESECTION ANASTAMO	0	0	7	0	0	4	0	6	17	
MR.MUNUSAMY	76	M	1604706	ILEAL PERFORATION	EL/ PRIMARY CLOSURE	5	0	7	0	4	4	6	12	38	EXPIRED P
MR.KAMARAJ	51	M	1605819	APPENDICULAR PERFORATION	OPEN APPENDICECTOMY	5	0	0	0	0	0	0	0	5	
MR.KUMAR	50	M	1606030	DUODENAL PERFORATION	EL/ PATCH CLOSURE	5	0	7	0	4	4	6	6	32	EXPIRED P
MR.MUNUSAMY	55	M	1606302	DUODENAL PERFORATION	EL/ PATCH CLOSURE	5	0	0	0	4	4	6	6	25	RE-DO LAP
MR.SHANKAR	42	M	1606564	DUODENAL PERFORATION	EL/ PATCH CLOSURE	0	0	0	0	4	4	0	6	14	
MR.SAMPATH	25	M	1608417	DUODENAL PERFORATION	EL/ PATCH CLOSURE	0	0	7	0	4	4	6	6	27	EXPIRED P
MR.MOHAN	25	M	1608838	DUODENAL PERFORATION	EL/ PATCH CLOSURE	0	0	0	0	4	4	6	6	20	
MR.RAMALINGAM	50	M	1609040	DUODENAL PERFORATION	EL/ PATCH CLOSURE	5	0	0	0	4	4	6	6	25	
MR.JANAKIRAMAN	35	M	1609010	DUODENAL PERFORATION	EL/ PATCH CLOSURE	0	0	7	0	4	4	6	6	27	EXPIRED P
MRS.KALAVATHY	58	F	1609779	APPENDICULAR PERFORATION	LAP APPENDICECTOMY	5	5	0	0	0	0	0	0	10	
MR.VIMAL DAVID	19	M	1610464	APPENDICULAR PERFORATION	OPEN APPENDICECTOMY	0	0	0	0	0	0	0	0	0	
MR.THIRUMALAI	29	M	1610826	GASTRIC PERFORATION	EL/ PATCH CLOSURE	0	0	7	0	0	4	6	6	23	
MR.THULUKANAM	57	M	1611224	DUODENAL PERFORATION	EL/ PATCH CLOSURE	5	0	0	0	0	4	6	6	21	
MR.RAJESHKUMAR	37	M	1612800	ILEAL PERFORATION	LAPARATOMY/ ILEOSTOMY	0	0	0	0	4	4	6	12	26	
MR.MURUGESAN	32	M	1615030	DUODENAL PERFORATION	EL/ PATCH CLOSURE	0	0	0	0	0	4	0	6	10	
MRS.BABY	46	F	1617206	DUODENAL PERFORATION	EL/ PATCH CLOSURE	0	5	7	0	0	4	6	6	28	
MR.RAMACHANDRAN	50	M	1619819	GASTRIC PERFORATION	EL/ PATCH CLOSURE	5	0	0	0	4	4	0	6	19	
MR.KUMAR	28	M	1622818	DUODENAL PERFORATION	EL/ PATCH CLOSURE	0	0	0	0	4	4	6	6	20	

KEY

EL/ PATCH CLOSURE - EMERGENCY LAPARATOMY WITH PATCH CLOSURE
LAPARATOMY / ILEOSTOMY - LAPARATOMY WITH ILEOSTOMY
EL/PRIMARY CLOSURE - EMERGENCY LAPARATOMY WITH PRIMARY CLOSURE
LAP CONVERTED TO OPEN APPENDICECTOMY- LAPROSCOPIC APPENDICECTOMY CONVERTED TO OPEN APPENDICECTOMY
EL/APPENDICECTOMY- EMERGENCY LAPARATOMY WITH APPENDICECTOMY