MANNHEIM PERITONITIS INDEX IN PREDICTING THE MORBIDITY AND MORTALITY IN PATIENTS WITH PERITONITIS DUE TO HOLLOW VISCUS PERFORATION

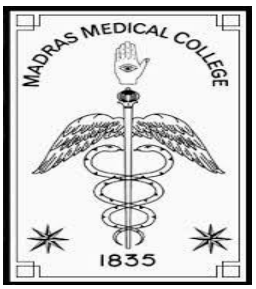
A Prospective study

DISSERTATION SUBMITTED FOR

MASTER OF SURGERY

(GENERAL SURGERY)

Branch – I



MADRAS MEDICAL COLLEGE
CHENNAI 600 003

THE TAMILNADU DR M.G.R MEDICAL UNIVERSITY

APRIL-2015

CERTIFICATE

This is to certify that the dissertation titled **"MANNHEIM PERITONITIS INDEX IN PREDICTING THE MORBIDITY AND MORTALITY IN PATIENTS WITH PERITONITIS DUE TO HOLLOW VISCUS PERFORATION"** is the original work done by **Dr.APPU MATHI ROGA RAJAN.M**, post graduate in M.S General surgery at the Department of General Surgery and Rajiv Gandhi Government General Hospital, Chennai to be submitted to The Tamil Nadu Dr.MGR Medical University, Chennai towards the partial fulfilment of the requirement for the award of M.S., degree in General Surgery, April 2015.

Prof.A. AFFEE ASMA. M.S.,
Professor of Surgery,
Institute of General Surgery,
Madras Medical College,
Chennai.

Prof.P.RAGUMANI. M.S.,
Head of the Department,
Institute of General Surgery,
Madras Medical College,
Chennai.

Dr.P.VIMALA.M.D.,
The DEAN
Madras Medical College
Chennai

DECLARATION

I **Dr. APPU MATHI ROGA RAJAN.M**, declare that this dissertation titled **"MANNHEIM PERITONITIS INDEX IN PREDICTING THE MORBIDITY AND MORTALITY IN PATIENTS WITH PERITONITIS DUE TO HOLLOW VISCUS PERFORATION"** is a record of the original work done by me in the Institute of General surgery and Rajiv Gandhi Government General Hospital, Chennai during my postgraduate course of M.S General Surgery from 2012 - 2015 under the guidance of my professor Prof. A. Affee Asma M.S., and head of the department Prof. P. Ragu Mani M.S., It is submitted to The Tamil Nadu Dr.MGR Medical University, Chennai towards the partial fulfilment of the requirement for the award of M.S., degree in General Surgery, April 2015. This record of work has not been submitted previously by me for the award of any degree from any other university.

Dr. APPU MATHI ROGA RAJAN.M

ACKNOWLEDGEMENT

I sincerely thank our **Dean Dr.P.VIMALA. M.D.,** for allowing me to do this study in this institute and Rajiv Gandhi Government General Hospital.

I wish to express my gratitude to **Prof.P.RAGUMANI. M.S, Head of the Department of General Surgery** for providing me this opportunity and having kindly permitted me to undertake this study.

I thank my unit Chief **Prof.A.AFFEE ASMA M.S.,** who with her perceptive and discerning guidance enabled me to carry out this dissertation.

I thank my Guide and former unit Chief **Prof.T.BAVANI SANKAR M.S.,** who with his perceptive and discerning guidance enabled me to carry out this dissertation

I wish to thank Assistant professors **Dr.A.Anandi M.S., Dr.S.SelvaKumar M.S., and Dr.S.Nedunchezhiyan.M.S.,** for their support and guidance provided by them to carry out this study.

I thank my Co-postgraduates, CRRIs, Medical records Officer and Medical records department staff in helping my work.

I thank all my patients for their kind co-operation in carrying out this study successfully without whom this could not be made possible

Dr.APPU MATHI ROGA RAJAN.M

Turnitin Document Viewer - Google Chrome

https://turnitin.com/dv?s=1&o=452933224&u=1032028216&student_user=1&lang=en_us&

The Tamil Nadu Dr.M.G.R.Medical ... | TNMGRMU EXAMINATIONS - DUE 15-..

Originality | GradeMark | PeerMark

MANNHEIM PERITONITIS INDEX IN PREDICTING THE MORBIDITY AND

BY 221211001.MS GENERAL SURGERY APPU MATHI ROGA RAJAN.M

turnitin

18% SIMILAR

-- OUT OF 0

MANNHEIM PERITONITIS INDEX IN PREDICTING

THE MORBIDITY AND MORTALITY IN

PATIENTS WITH PERITONITIS

DUE TO HOLLOW VISCUS PERFORATION

A Prospective study

DISSERTATION SUBMITTED FOR

MASTER OF SURGERY

(GENERAL SURGERY)
Branch – I

MADRAS MEDICAL COLLEGE
CHENNAI 600 003

Match Overview

1	el.trc.gov.om:4000 Internet source	7%
2	med-lib.ru Internet source	1%
3	E. A. Elebute. "The gra... Publication	1%
4	www.shanyarmd.com Internet source	1%
5	www.ptolemy.ca Internet source	1%
6	www.med-lib.ru Internet source	1%
7	A. Billing. "Prediction of... Publication	1%
8	www.nmcth.edu Internet source	1%

PAGE: 1 OF 124

Navigate to text-only Originality Report

14:51

ABBREVIATIONS

1. MPI - Mannheim's peritonitis index

2. ICU - Intensive care unit

3. APC - Adenomatous polyposis coli

4. W.H.O - World health orgaisation

5. ATT - Anti-tubercular treatment

6 .CAPD - Continuous ambulatory peritoneal dialysis

7 . CT - Computerized tomography

8 . SIRS - Systematic Inflammatory Response Syndrome

9 . CARS - Compensatory anti-inflammatory response syndrome

10. TNF - Tumor necrosis factor

11. ROC - Receiver operative curve

LIST OF CONTENTS

INTRODUCTION

With the advances that are being made in many areas of medicine, the surgeon must be familiar with infectious diseases of the peritoneal cavity which has increased in severity and complexity. In addition to the surgical management of secondary peritonitis from gastro intestinal perforation, the practicing surgeon may be called in to manage patient with cirrhosis with infected ascitic fluid as well as patient undergoing peritoneal dialysis with infected dialysis fluid. In addition, there is increasing recognition of a group of patients with persistent intra-abdominal sepsis or tertiary peritonitis in whom infection is associated with multi system organ failure and general depression of immune system. Peritonitis continues to be one of the major infectious problems confronting the surgeons. Despite the many advances in anti-microbial agents and supportive care, the mortality rate of diffuse suppurative peritonitis remains unacceptably high.

Its causes vary from the one requiring immediate surgical intervention to that requiring conservative management. Its accurate diagnosis and management is a challenge to every surgeon. The complex nature of infections in surgical patients, the multifaceted aspects of treatment, and the increasing complexity of ICU support make

evaluation of new diagnostic and therapeutic advances in this field very difficult. Scoring systems those provide objective details of the patient's conditions at specific stages in the disease process aid in understanding these problems. This is important in determining the course, the disease is taking in a particular patient and whether the line of management taken is appropriate or need to be changed.

The management of peritonitis patients has taken a new turn with the understanding of patho-physiological basis of the disease, the concept of sepsis syndrome and multi-organ failure. The current trend is to recognize these at the earliest and institute aggressive therapy. When the patient has already gone into multi-organ failure, the outlook appears dismal even with intensive critical care. It is here that conservative line of management, as well as newer modalities of treatment such as programmed re-laporatomy and immuno modulation is being tried. Although these newer modalities may be useful, they are expensive. Hence, proper clinical monitoring with optimum number of investigations remain the corner stone of emergency surgery and also for the better use of above methods.

The pertinent questions like Do the etiology of peritonitis influence the outcome? Do delays in presentation matter? Could this patient have been better off without surgery? Continue to question the minds of most surgeons. I seek to find answers to some of these through this study.

AIMS AND OBJECTIVES

1. Aim is to predict the risk of mortality and morbidity in patients presenting with peritonitis due to hollow viscus perforation. Assessment of surgical risk in these patients is to help in choosing the modality of management in a particular patient.

2. This study attempts to evaluate the prognostic value of Mannheim Peritonitis Index scoring in patients with peritonitis due to hollow viscus perforation, to assess it as a clinical tool in stratifying these patients according to individual surgical risk.

These are: a. Patient factors:-

i. Age of the patient

ii. Sex of the patient

b. Disease process

i. Site of perforation

ii. Duration of perforation

iii. The extent of peritoneal contamination.

c. Effect of General systemic complications like

i. Respiratory

ii. CVS system

iii. Shock

iv. Multi-organ failure.

My aim was to study the effect of above factors on Mortality and morbidity of the patients presenting with peritonitis due to hollow viscus perforation.

REVIEW OF LITERATURE

HISTORY

Physicians in antiquity dreaded abdominal complications. Despite the fact that peritonitis was extremely common, reports of successful surgical interventions were only anecdotal before the past century. Medicine's comprehension of the patho-physiology of the peritoneal cavity is still evolving. The history of our understanding of the process could be considered to be as recent as the current study. Despite this, the mortality rates for patients with secondary peritonitis have fallen in the last century from almost 100% to <10%.

One of the earliest references to peritoneum can be found in Edwin Smith Papyrus which was copies around 1700 years ago which is supposed to have been written around the time of Imhotep (the Egyptian patron god of medicine). Breasted who translated these works wrote in his translation. *"I felt as if I had been peering through a newly revealed window, opening upon the once impenetrable gloom enveloping man's earliest endeavors to understand the world he lived in. It was as if I had watched a hand slowly raising the curtain that covered this window, and then suddenly the hand had refused to lift, the curtain further".*

Since the beginning of recorded medical history, human beings have been confronted with the spectra of peritonitis. Accounts from a variety of early societies have little doubt that our ancestors recognized the value of therapeutic drainage. In a German translation of the writings of Hippocrates appears the first through description of a patient with peritonitis. “The patient looks sick and wasted. The nose is pointed, the temple sunken, the eyes lay deep are rimmed and dull. The face expresses fear, the tongue is furrowed, the skin shiny. The patient avoids all movement and breathes shallow. The abdominal wall is rigid with muscular guarding. No bowel sounds can be heard. The pulse is quick and small. A hard, tender mass in hypochondrium is a bad prognostic sign if it involves the whole area. The presence of such a mass at the beginning of the fever indicates that death is imminent”.

The above description is now known as **Hippocrates facies.** He also described septic shock as *“A protrusive nose, hollow eyes, sunken temples, cold ears that are drawn in with the lobes turned outwards, the forehead’s skin rough and tense like parchment and the whole face greenish or black or leadened”.*

In the second century A.D. Galen served as the physician to Roman citizens, gladiator and emperors. He is reported to have performed many surgeries including suturing of lacerated bowel. He wrote much about

appearance of suppuration in post-operative period. In fact, Galen believed that such suppuration was critical for proper wound healing and should not be disturbed (laudable pus). Galen's writings were revered as unshakable tenets and restrained the development of medicine and physiology for almost 1500 years.

From the time of the fall of the Roman Empire until the beginning of the 16thcentury, medicine can be characterized as magical with strong religious overtones. The fate of surgery was sealed for centuries with Pope Innocent III religious decree of 1215 known as *"Eccelsia Abhorret de Sanguine"*, literally translated as *"The Church Abhors bloodshed"*. It was only at the birth of renaissance that the mysteries of the abdominal cavity began to be known. This can be attributed to the wondrous drawings of the Michelangelo, Leonardo da Vinci and Vesalius.Peritonitis due to perforation of acute peptic ulcer was first described by Littre in 1670. The patient was a lady of high rank, Henrietta Anne, Duchess, of Oreans and daughter of Charles I of England. John Hunter, renowned for his surgical exploits, suggested that laparotomy might be possible and even useful in the treatment of peritonitis. Hertein, in 1767, reported a cure of biliary peritonitis in dogs using irrigation of abdomen.

The three developments that fostered an understanding of the peritonitis disease process included the foundation of experimental

physiology by Francois Magendie and Claude Bernard, an understanding of cellular pathology as championed by Rudolph Virchow, and the advent of the germ theory by Pasteur and Koch. George Wegner first reported in 1879, a series of experiments attempting to elucidate the normal physiology of the peritoneum. The modern era of our understanding of the peritoneum was begun by John B. Murphy of Murphy Button fame.

In 1908, he wrote

> *"There are no stomata or stigmata in the peritoneum. The endothelial lining is everywhere, continuous".*[3]

Of course, we know it is not fully true as of today. Herbert E Durham[4] analyzed fluid from peritoneal cavity and proposed a time line of cellular events, which he divided into 5 stages – (1) the stage before leukopenia, (2) the leukopenic stage, (3) the microxyphil stage, (4) the macrophage stage and (5) recovery to normal.

The experiments of Meleney[5] in the late 1926's showed that bacterial synergism existed. They showed that combinations of aerobic and anaerobic bacteria produced before sepsis than from individual strains.

Review of Current Literature

Some of the early systematic attempts to define the severity of surgical infection and risk of death derived from the observation that patients dying after surgical infection often followed a clinical course characterized by sequential organ failure. This has been called the **"Multi organ failure syndrome"**

Fry and associates showed in 1980[6] that death after major operative procedures or severe trauma was usually due to infection and became more likely as the number of failed organs increased i.e. the mortality rate with no organ failure was 3%, rising to 30% - 1Organ failure, 100% - 4 organ failure.

In 1982 Knaus and others proposed a scoring system to be used for classifying patient admitted to ICU. They devised a 2 part scale. It included physiological portion, APS-34, examines abnormality among 34 possible physiological assessments (APS-34), which obtained during the first day of admission. The second part of the score is a chronic health evaluation (CH). This examines the patient's pre-admission health by reviewing the medical history for details concerning functional status, productivity and medical attention during 6 month before admission. The

combination is called APACHE. This system is not specific for intra-abdominal infection. It was later modified using only 12 values the APACHE II.

Another approach to grade the severity of sepsis was published by Elebute and Stoner in1983[7]. These authors divided the clinical presentation of the septic state into 4 classes to which they ascribed subjective degree of severity on an analogue scale. The attributes were local effects of tissue infection, degree of temperature elevation, secondary effects of sepsis and laboratory data.

Pine and associates (1983)[8] confirmed the above findings. In addition, they looked at a number of other risk factor thought to influence the development of organ failures on death and identified clinical shock at any time, malnutrition, age and alcoholism as important predictive factors.

The papers by Pine and Knaus and their colleagues were the first to provide clear definition of **"organ failure".**

Stevens (1983)[9] recognized the need for more precision and for a greater range of potential values and devised a system of scoring to represent the severity and magnitude of organ failure. He defined 7 organ systems and assigned score of 0-5 in each system. Scores were calculated mathematically by squaring the values assigned to each organ system and

adding the 3 highest scores to arrive at **"sepsis severity score".** He based the practice of squaring the individual scores up the experimental increase in the mortality as the progressive organ system failure.

Knaus and Coworkers (1985)[10] extended these observations in a report covering 5,677 ICU admissions and 2719 patients who developed organ failure.

Teichmann and associates (1986)[11] in a report concerning scheduled reoperation for diffuse peritonitis, referred to Peritonitis Index Altermheir (PIA). This used age, extent of infection, malignancy, CVS risks and leukopenia to stratify patients.

Wacha and Coworkers (1987)[12] developed a separate peritonitis index, the Mannheim Peritonitis Index (MPI) with incorporated information with respect to age, gender, organ failure, malignancy , duration of peritonitis, involvement Of colon, extent of spread within the peritoneum and the character of peritoneal fluid to define risk. Scores range from 0 to 46.

In 1988, V. Kohli[13] and others evaluated prognostic factors in 50 cases of perforated peptic ulcer. They concluded that there is a place for prognostic scoring. They found general health status , concurrent illness, arterial hypotension at the time of admission, delay in surgery and severity

of peritoneal contaminations, some of the factors contributing to the post-operative morbidity and mortality.

In 1990, Verma and others[14] in PGI, Chandigarh, compared prognostic factors in peritonitis due to trauma. They found pre-operative shock, multiple hollow visceral injury, septicemia, and location of injury (colon and duodenum were significant prognostic factors and with high mortality).

In 1992, Bartel and other did a study of utility of programmed relaparotomy in diffuse peritonitis. It concluded that eradication of source of infection during first laparotomy, Serum Creatinine, Patients age and pre-existing hepatic disease influenced outcome.

In 1994, Demmel N[16] compared Apache II with MPI, they concluded that there was no significant difference in prognostic value between scoring systems.

Khosrovan in 1994, identified 3 important prognostic factors for high mortality – age over 70 years, admission delay in > 24 hours and pre-operative hemodynamic shock. He recommended suture of perforation and vagotomy in absence of risk factors. Simple suture of perforation in presence of single factor.

In 1994, Kriwanek S. conducted a study for prognostic factors in colonic perforation. It concluded that age over 65 years and MPI proved to be the only risk factors of significance.

In 1994, Scoanes[17] and other did a study of diverse effect of delayed treatment for perforated peptic ulcer. They concluded that delayed treatment for > 12 hrs. Increased mortality especially in elderly patient confirming finding of MPI.

In 1996, a multivariate analysis on 604 patients with intra-abdominal infection were done to compare different scores systems like Apache-II, SS of Elebute and Stoner and MPI. Results showed dominance of host-related factors over the type and source of infection on the prognosis of patients. Both MPI and Apache-II correctly graded intra-abdominal infections and were independently and strongly associated with an outcome. However, the MPI had the advantage of being easier to calculate.

SURGICAL ANATOMY OF PERITONEUM AND PERITONEAL CAVITY

Embryology of peritoneal cavity:

Peritoneal cavity is derived from the two limbs of the horseshoe shaped intraembryonic coelom, which is situated caudal to septum transversus. The 2 parts are at first separate, but fuse to form one cavity as result of lateral folding of embryonic disc. The attachment of mesentery of the primitive gut on the abdominal wall is initially in the midline. As a result of changes involving the rotation of the gut and as a result of some parts of the gut becoming retroperitoneal, the line of attachment of mesentery becomes complicated[18].The peritoneal cavity therefore comes to be subdivided into number of pockets that are separated partially by folds of peritoneum.

Parietal peritoneum:

This layer lines the inner surface of the abdominal and pelvic walls and abdominal surface of the diaphragm. It is loosely adherent to the walls by extra peritoneal connective tissue and can therefore be easily stripped. Because of somatic innervations it is pain sensitive.

Visceral peritoneum:

This layer lines the outer surface of the viscera, to which it is firmly adherent and cannot be stripped. Blood and nerve supply are same as those of underlying viscera. Because of the autonomic innervations it is pain insensitive [19].

Histologically, peritoneum consists of an outer layer of fibrous tissue, which gives strength to the membrane and an inner layer of mesothelium which secrete a serous fluid. The peritoneal cavity is the largest cavity in the human body. The surface area of its lining membrane is two square metres in adult, nearly equal to that of skin. In males, it forms a closed sac. In females, the free ends of uterine tube open into the abdominal cavity. The peritoneal cavity consists of the Greater sac and the lesser sac (omental Bursa).

The peritoneal cavity is divided into pelvic and abdominal portions. The abdominal portion is divided into supracolic and infracolic compartment by mesocolon and transverse colon. The infra colic compartment is divided into left and right by mesentery. The Right infracolic and left infracolic is divided into external and internal paracolicgutters by ascending and descending colon respectively.

Supracolic compartment is below the diaphragm and above transverse colon and mesocolon. The liver, gallbladder, stomach, duodenum 1st part and spleen lie within this space. The liver and its ligaments break this space into important sub phrenic spaces.

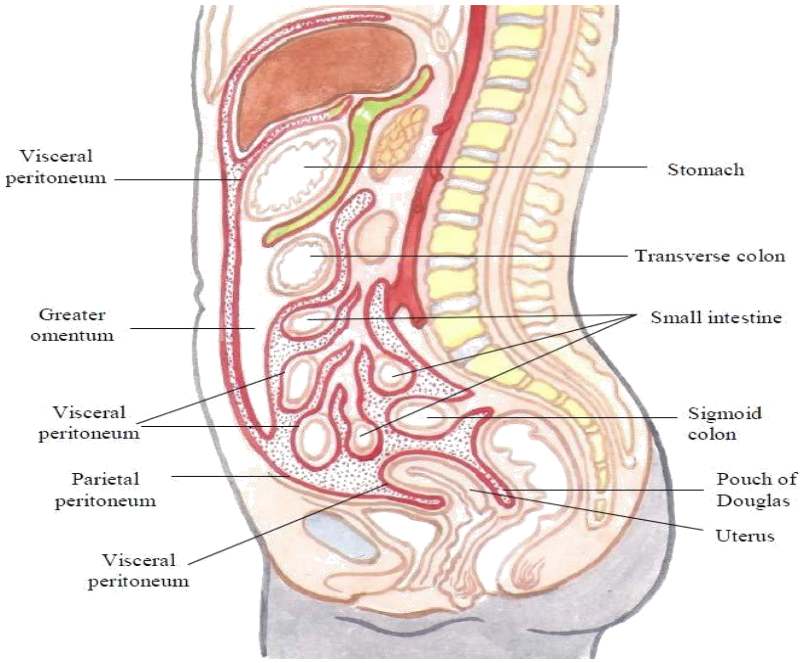


FIG: 1. PERITONEUM LAYERS – PARIETAL AND VISCERAL

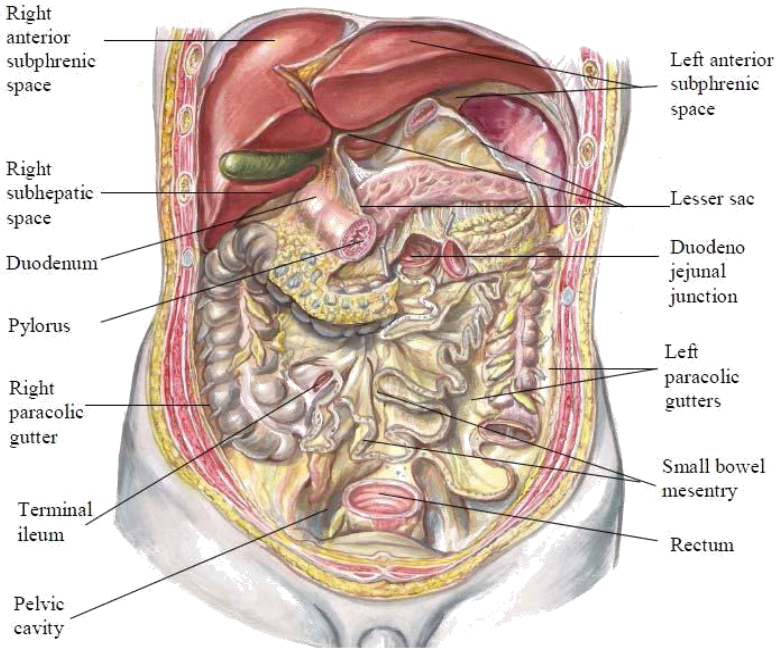


FIG:2. PERITONEAL CAVITY AND SPACES

Subphrenic spaces:

There are seven subphrenic spaces which includes four intraperitoneal spaces and three extra peritoneal spaces. It is divided into left and right by falciform ligament. The intraperitoneal spaces are:

1. *"Right anterior* (superior) (subphrenic)*space"*

2. "Right posterior (inferior) (subhepatic) *space"*

3. "Left anterior (superior) (subphrenic) *space"*

4. *"Left posterior* (inferior) (subphrenic)[20]*"*

There are three extra peritoneal spaces, which are

- *Right and left extra peritoneal space* which are the term given to perinephric spaces.
- *Midline extra peritoneal* which is another name given for the bare area of liver.

1. Right anterior (superior) intraperitoneal space (Right subphrenicspace):

It lies between the diaphragm and the right lobe of liver. It is limited posteriorly by the anterior layer of the coronary ligaments and in the right by triangular ligaments and in the left by falciform ligament.

Common causes of collection here are perforating acute cholecystitis, a perforated duodenal ulcer, a duodenal stump blow out following gastrectomy and appendicitis.

2. Right inferior (posterior) intraperitoneal space (Right sub hepatic space):

It is also called hepatorenal or Morrison's pouch. It is bordered on the right by the right lobe of the liver and the diaphragm. To the left of this space lies the foramen of Winslow and below this lies the duodenum. In front are the liver and the gallbladder and behind, the upper pole of the right kidney and diaphragm. It is bounded above by the liver and below by the hepatic flexure and transverse colon. It is the commonest site of subphrenic abscess, which usually arises from appendicitis, cholecystitis, a perforated duodenal ulcer, or following upper abdominal surgery.

3. Left anterior (superior) intraperitoneal space (subphrenic space):

It is bordered above by the diaphragm and behind by the left lobe of the liver and the left triangular ligament, the lesser omentum and anterior surface of the stomach. To the right is the falciform and to the left is the gastrosplenic omentum, spleen and diaphragm. The common cause of an abscess here is operation on the stomach, the tail of pancreas, the spleen or the splenic flexure of the colon.

4. Left inferior (posterior) intraperitoneal (left sub hepatic space):

This space is also termed as the lesser sac. The common cause of infection here is complicated acute pancreatitis. In practice a perforated gastric ulcer rarely causes a collection here because the peritoneal space is obliterated by adhesions.

Extra-peritoneal spaces.

The right and left extra-peritoneal space is the site for perinephric abscess. Midline extra peritoneal space is another name for the bare area of liver. This area may reside an abscess in amoebic hepatitis and pyogenic liver abscess. It can cause generalized peritonitis following rupture.

PHYSIOLOGY OF THE PERITONEUM

Mesothelial cells are organized in two discrete populations i.e. flattened and cuboidal cells. Gaps(stomata) between neighbouring cells of peritoneal membrane are found only among cuboidal cells. Peritonitis increases the width of these intervening stomata. Beneath mesothelial cells is a layer of basement membrane of loose collagen fibers. The basement membrane overlies a complex connective tissue layer that includes collagen and other connective tissue proteins, elastic fibers, fibroblasts, adipose cells, mast cells, eosinophils, macrophages and

lymphocytes and network of lymphatic and capillaries.[19] The mesothelial lining of the peritoneum secretes serous fluids that circulate within the peritoneal cavity and it contains 50- 100 ml of fluids with solute concentrations nearly identical to that of plasma[21] . The protein content of the peritoneal fluids is somewhat less than that of plasma about 3gm\dl. Peritoneal mesothelial lining cells and sub diaphragmatic lymphatics absorb fluid. Mesothelial cells also absorb solute by endocytosis. This bi-directional movement of fluids across peritoneal membranes has been used in peritoneal dialysis.

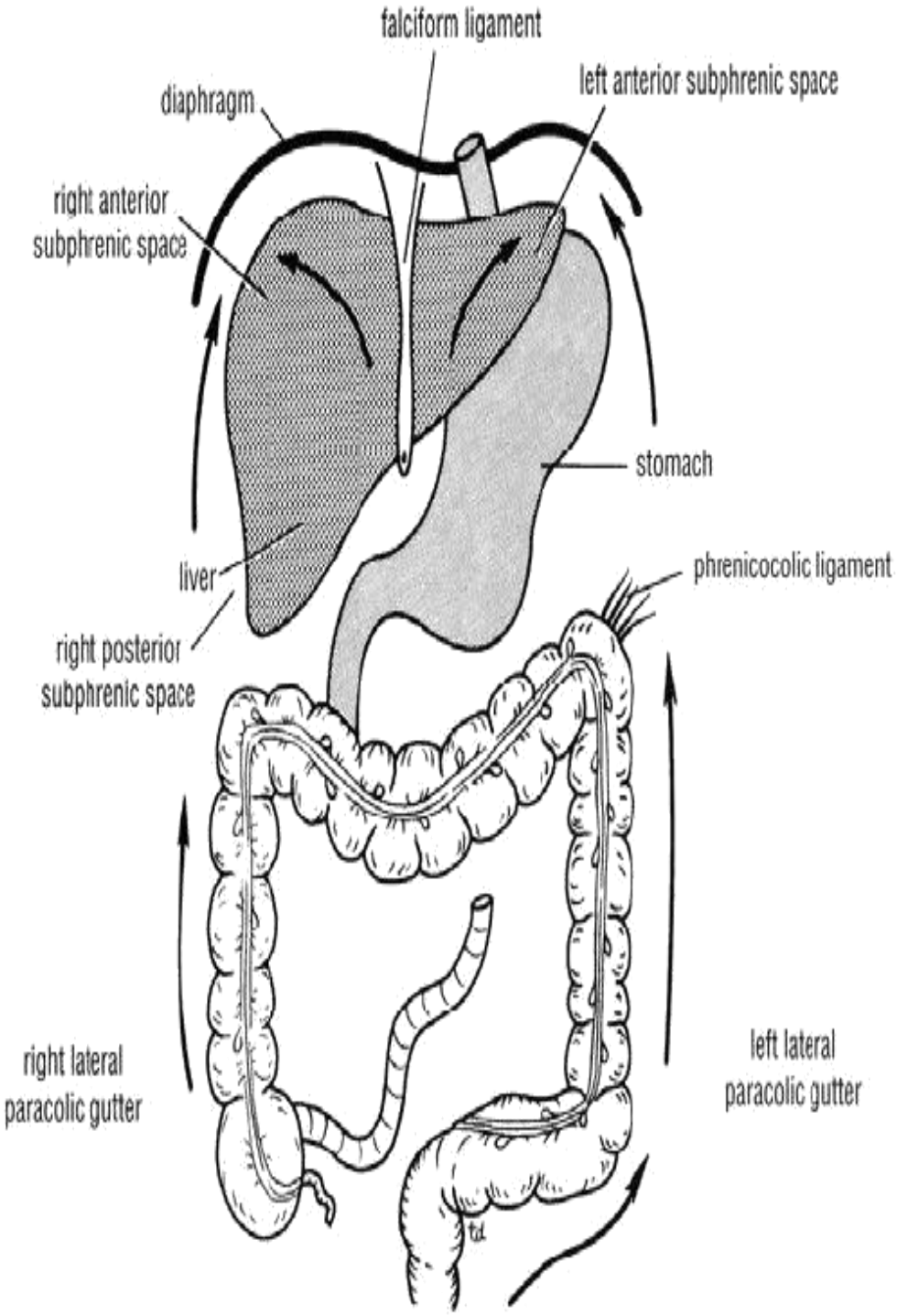
Two primary forces are responsible for the movements of fluids within the peritoneal cavity: (a) Gravity (b) Negative pressure created under the diaphragm with each normal respiratory cycle. Subphrenic collections are frequent due to relatively negative pressure is created beneath the diaphragm with each exhalation. Peritoneal fluid can enter the circulation via diaphragmatic lymphatics, which drain, into the thoracic duct.

PERITONEAL RESPONSE TO INJURY:

- Inflammatory changes in the peritoneal cavity result in the irritation of the peritoneum with loss of regional mesothelial cells. A large peritoneal defect heals in the same amount of time as a small peritoneal defect. It has been shown that after 3 days of peritoneal injury connective

tissue cells resembling new mesothelium cover wound surface. At day 5, new surface layer closely resembles adjacent normal epithelium. On day 8 mesothelium regeneration is complete. The exact origin of cells responsible for mesothelial regeneration remains unknown.

FIG: 3. NORMAL DIRECTION OF FLOW OF PERITONEAL FLUID



It is postulated, the regeneration mechanisms include

- Submesothelial cells producing new mesothelial cells.
- Surviving or floating mesothelial cells or those attached to wound edges migrating into the wound.
- Peritoneal fluid monocytes and macrophages differentiating into mesothelial cells.[19]

Normal peritoneal wound heals without adhesion formation. Adhesion develops in response to factors others than simple peritoneal wounding. Local tissue hypoxia or ischemia appears to be the most important factor in adhesion formation apart from mechanical sub peritoneal surface injury, intra-abdominal infections, and contamination of peritoneal cavity by foreign material. Deposition of fibrin following peritonitis is essential for adhesion formation. It has been shown that fibrinolytic activity is absent in healing wound until mesothelial cells are found. Fibrinolytic activity is minimal at 3 days in view of few mesothelial cells but complete at the end of 8th day, when mesothelial regeneration is complete. Therefore with intact mesothelial surface and adequate fibrinolysins, early fibrinous adhesions disappear.Formation of adhesion is both a protective response, helping to localize infection and an adoptive response to wound healing by carrying additional blood supply.

PATHOPHYSIOLOGY OF PERITONITIS

Generalized or local inflammation of peritoneum is designated as peritonitis. Each and every case of peritonitis of whatever cause, initiates a sequence of responses involving the peritoneal membrane, the bowel, and the body fluid compartments, which then produce secondary endocrine cardiac, respiratory, renal, and metabolic responses.

PRIMARY RESPONSES IN PERITONITIS:

Membrane inflammation:

Peritoneum reacts to injury by hyperemia and transudation. Edema and vascular congestion occurs in the sub peritoneal layer immediately external to peritoneal membrane. Absorption across inflamed peritoneum in early cases is increased and decreases with chronicity. Absorption of macromolecules appears to be more affected than small molecule absorption. Transudation of fluid with low protein content from the extracellularly interstitial compartment into abdomen is accompanied by diapedesis of polymorphonuclear leucocytes. During the early vascular and transudative phase of engorgement, the peritoneum acts as a TWO WAY STREET such that toxins and other materials that may be present in the peritoneal cavity are readily absorbed, enter the lymphatic and blood stream and may lead to systemic symptoms.[19] Transudation of interstitial

fluid into the peritoneal cavity across the inflamed peritoneum is shortly followed by exudation of protein rich fluid. The fluid exudates contains large amounts of fibrin and other plasma proteins in concentration sufficient to bring about clotting later, that results in agglutination of loops of bowel, other viscera and the parities in the area of peritoneal inflammation. There is increased synthesis of lipoproteins and proteolysis. Concentration of uronic acid increases reflecting the exudation of plasma proteins in the early stages of peritonitis and in later stages increased synthesis of glycosaminoglycans due to activation of fibroblasts and mesothelial cells. Changes in non-collagen and collagen protein synthesis are two events that occur in inflamed peritoneum during peritonitis. In early peritonitis non-collagen protein synthesis are increased and vice versa in later stages owing to increased protein synthesis in total. The RNA: DNA ratio, an index of protein synthesizing capability of tissues, increases during the first week of peritonitis.

Bowel response:

Initially, response of bowel to peritoneal irritation is transient hypermobility. After a short interval, motility becomes depressed and nearly complete adynamic ileus soon follows. Bowel distension with air and fluid accumulation occurs finally.

Hypovolaemia:

Peritoneum reacts to injury by hyperemia and transudation of plasma like fluid from the intracellular, extracellular and interstitial compartments into the peritoneal space. The loose connective tissue beneath the mesothelium of the viscera and mesentery traps extra cellular fluid as oedema. The atonic bowel also accumulates the fluid derived from extra cellular space. This translocation of water, electrolytes, and proteins into a "THIRD SPACE" functionally removes this volume temporarily from the body economy. The rate of functional extracellular fluid loss is proportional to the peritoneal surface area involved in the inflammatory process. With extensive generalised peritonitis, translocation of 4-6 liters or more in 24 hours is not uncommon.

SECONDARY RESPONSES IN PERTIONITIS:

Endocrine response:

There is almost an immediate adrenal medullar response, with out-pouring of epinephrine and nor-epinephrine producing systemic vasoconstriction, tachycardia and sweating. There is increased cortical hormones secretion during the first two or three days following peritoneal injury. Secretion of aldosterone and ADH is also increased in response to hypovolemia resulting in increased water and sodium conservation. Water

retention may be greater than sodium retention resulting in dilutional hyponatremia.

Cardiac response:

The effects of peritonitis and cardiac function are a reflection of both decrease in ECF volume and progress in acidosis. Volume deficit results in decreased venous return and diminished cardiac output. Heart rate increases in an attempt to increase cardiac output, but compensation is usually incomplete. Progressive acidosis results in secondary dysfunction of cardiac contractility and a further decrease in cardiac output.

Respiratory response:

Distension of abdomen, primarily due to adynamic ileus, along with restricted diaphragmatic and intercostal muscle movements because of pain, results in decreased ventilation volume and early occurrence of basilar atelectasis.

RENAL RESPONSE:

Urine volume is diminished and renal capacity to handle an excess of solute is impaired. Hypovolemia reduces cardiac output and increased secretion of ADH aldosterone in peritonitis, all acting synergistically on the kidney. Renal blood flow is reduced and in turn the GFR and tubular

urine flow. Reabsorption of water and sodium is increased often in imbalance and potassium is wasted.

Metabolic response:

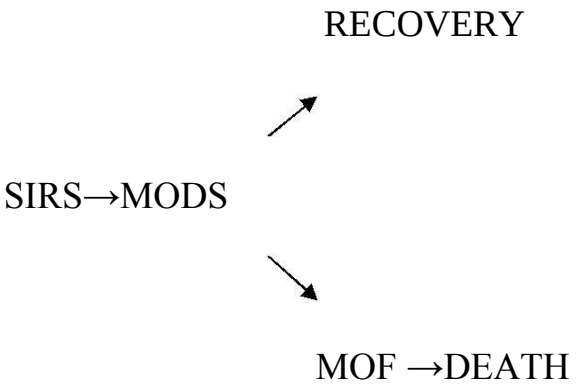
The metabolic rate is generally increased with increased peripheral O2 demand. Simultaneously the capacity of lungs and heart to deliver O2 is reduced. Poor circulation shifts from aerobic to anaerobic metabolism in muscle and other peripheral tissues. As a result, anaerobic end products of carbohydrate metabolism accumulate and lactic acidosis begins to develop.

Both ‘D’ and ‘L’ isomers of lactate are produced by bacterial metabolism and may be absorbed during peritonitis. Human beings can rapidly metabolize ‘L’ lactate, but have a relatively limited capacity to handle ‘D’ lactate. Protein catabolism begins early in peritonitis and progressively becomes severe. Plasma proteins are preferentially synthesized while muscle proteins are catabolized during peritonitis.

PATHOPHYSIOLOGY OF SEPSIS:

Osler said “Patients die not of their disease; they die of the physiological abnormalities of their disease,” which is true for sepsis. Peritoneal insult will be manifested generally as Systemic Inflammatory Response Syndrome (SIRS) which if not treated aggressively will lead on

to Multi Organ Dysfunction Syndrome (MODS). Bacteria can be experimentally demonstrated in thoracic duct in 6 minutes and in bloodstream within 12 minutes following injection of organism into peritoneal cavity[29]. Some patients succumb to death due to Multi Organ Failure (MOF) and others recover with modern day medical care.



DEFINITIONS:

1. SIRS: (*Systematic Inflammatory Response Syndrome*).

   Two or more of following clinical signs indicates SIRS

   - Temp- >38^0C or <36^0C.
   - Heart rate > 90/ min
   - Respiratory rate > 20/ min or PaCO2 < 32 mmHg
   - WBC count >12000/mm^3or <4000 mm^3 or > 10% band (immature) forms.

2. SEPSIS: SIRS + documented infection.
3. SEVERE SEPSIS: SIRS + SEPSIS + Haemodynamic compromise.
4. MODS: This is a physiological derangement in which organ function is not capable of maintaining homeostasis.

MEDIATORS OF SIRS:

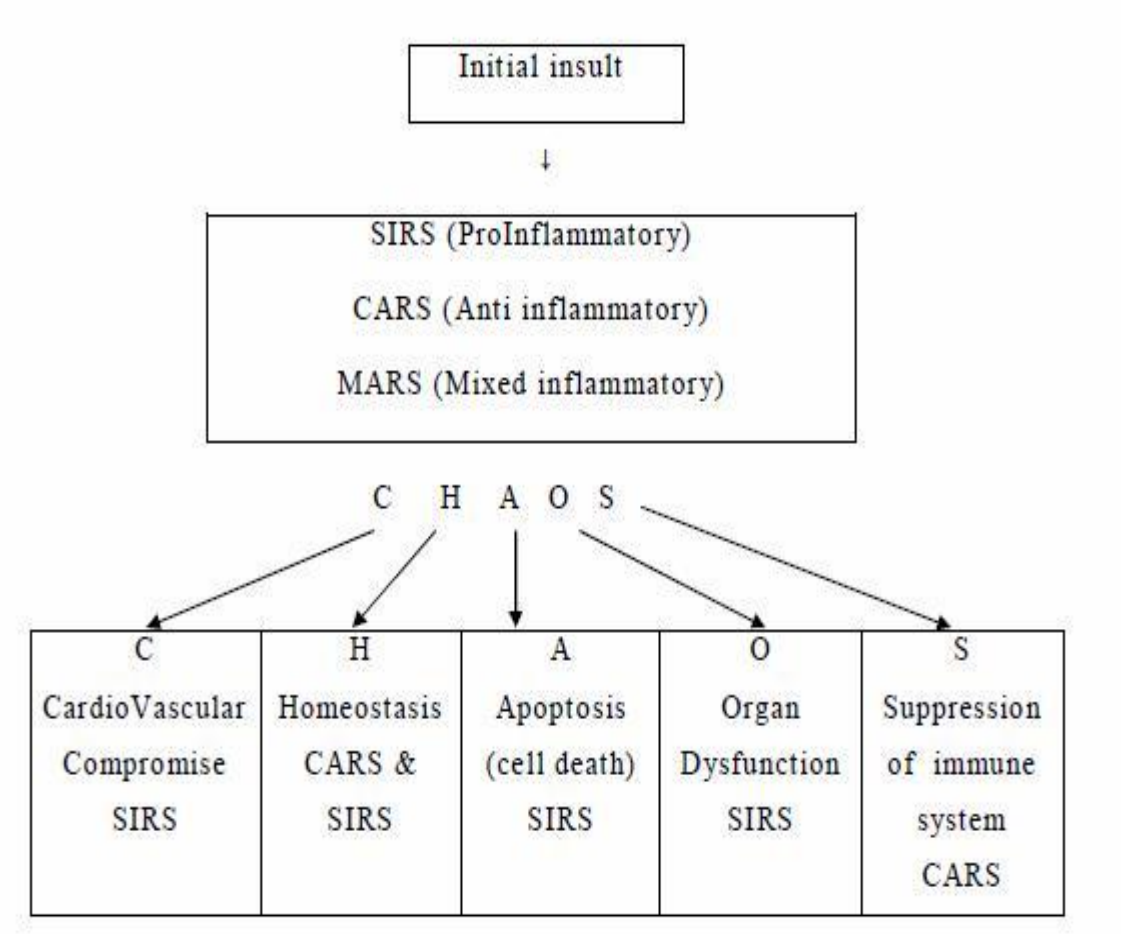
Effects of SIRS are not due to one, but many mediators. The most important one is TNF(TUMOR NECROSIS FACTOR-alpha). Others are IL-1, IL-6, Endotoxin, Endothelium, and leucocytes.

EFFECTS OF SIRS:

There will be increased peripheral vasodilatation, microvascular permeability, microvascular clotting and leukocyte or endothelial cell activation. The metabolic and nutritional effects include fever, anorexia, cachexia etc. These effects finally lead to septic shock, DIC, ARDS and MODS.

EVENTS IN SEVERE SEPSIS:

After the peritoneal insult, it is postulated that initially proinflammatory (SIRS) and later anti-inflammatory responses (CARS-compensatory anti-inflammatory response syndrome) are evoked. There is also an intermediate response i.e. MARS- mixed anti- inflammatory response syndrome. The consequences of these responses has been termed as CHAOS.



FACTORS THAT MAY FAVOUR THE DEVELOPMENT OF GENERALISED PERITONITIS:

i. **Speed of peritoneal contaminant** is a key factor in the spread of peritonitis.
ii. **Stimulation of peristalsis** by the ingestion of food hinders localization.
iii. **The virulence of the infecting organism**
iv. **Young children**, who have small omentum.
v. **Disruption of localized collections.**
vi. **Deficient natural resistance (immune deficiency)** [19]

BACTERIOLOGY OF PERITONITIS

Peritonitis as a disease process is characteristically polymicrobial in nature.

Paths of bacterial invasion of peritoneal space:

- Direct infection.
- Extension from an locally inflamed organ. E.g., Appendicitis, Cholecystitis.
- Bloodstream- part of general septicemia.

Bacteria from the alimentary canal

The number of bacteria is low within the GIT until the distal small bowel is reached, while high concentrations are found in the colon. The biliary and the pancreatic tract are normally devoid of bacteria, although they may be infected in the disease. Two or more organisms usually cause peritoneal infection. The commonest organisms isolated are Escherichia coli, aerobic and anaerobic streptococci, and the bacteriodes. Less frequently clostridium welchii is also found. Bacteroides are commonly found in peritonitis. These gram negative, non sporing organisms, although predominant in the lower intestine, often escape detection because they are strictly anaerobic and slow to grow on culture media unless there is adequate CO2 in the anaerobic apparatus[21]. Considerable interest has been focused on the bacterial interaction that results in a complex synergistic relationship among the pathogens of peritonitis.

Experimental studies have shown that, intraperitoneal injection of Bacteriodes fragilis alone resulted in no deaths and no lactic acidosis in rats. When *B.fragilis* is introduced into the peritoneal cavity with other aero tolerant microbes, the anaerobe becomes associated with an abscess phase of the peritoneal infection. When large inocula of B. fragilis are introduced, the mortality identified from the Endotoxin- bearing aerobic partner is accentuated. Mixed inocula of E.coli and B. fragilis show

synergism in models of experimental bacteremia together. The aerobic partners of the polymicrobial infection actually consume the oxygen of the microenvironment and generate a very low oxidation-reduction potential, which permits the non-aero tolerant anaerobes to survive. Peritoneal infections of greatest concern are those of the distal alimentary tract, both because of the complex aerobic-anaerobic composition of bacterial pathogens and because of the very high density of bacterial contaminants. Even in patients with nonbacterial peritonitis (e.g., intra peritoneal rupture of bladder) the peritoneum often becomes infected by transmural spread of organisms from the bowel and it is not long before a bacterial peritonitis develops[19].

BACTERIA COMMONLY ENCOUNTERED IN PERITONITIS

Facultative anaerobe and Gram-negative aerobic	Obligate Anaerobes	Facultative anaerobic gram-positive aerobic
Escherichia coli	Bacteriodes fragilis	Enterococci
Klebsiella species	Bacteriodes species	Staphylococcus
Proteus species	Fusobacterium species	Streptococcus
Enterobacter species	Clostridium species	
Morganella morganii	Peptococcus species	
Aerobic gram-negative bacilli	Peptostreptococcus species	
Pseudomonas aeruginosa	Lactobacillus species	

FACTORS INFLUENCING PERITONEAL INFLAMMATION AND INFECTION

Bacterial virulence:

The virulence of contaminating bacteria is influenced by a number of factors. Several organisms are well recognized for their innate ability to produce intra-abdominal infection in humans. Despite the massive contamination and complexity of the microbial spectrum that occurs with caecal perforation, within 24 to 48 hours, only a few isolates are recovered in peritoneal fluid culture. This indicates that only a few pathogenic bacteria survive, to predominate infection.[21]Weinstein demonstrated that E.coli and enterococcus were the predominant organisms during the peritonitis phase[22], while B. fragilis predominated during the abscess phase. Another unique pathogenicity is the remarkable ability of encapsulated anaerobic bacteria to produce abscess formation, a characteristic attributed to the capsular polysaccharide components. The ability to adhere to the mesothelial surface may also enhance the virulence of some organisms such as the enterobacteraceae and B. fragilis. Aerobic bacteria may benefit anaerobic species by lowering the redox potential of the micro environment and producing essential nutrients while anaerobic bacteria may provide the ability to inhibit neutrophil function and to develop antibiotic resistance by inactivation.

DIAGNOSIS OF PERITONITIS:

CLINICAL FEATURES:

Generalized peritonitis may present in differing ways depending on the duration of infection.

Early phase:-

Pain, which is made worse by the movement of breathing, is almost always a predominant symptom. It is initially felt at the site of original lesion. (E.g. In case of perforated gastric ulcer pain in the epigastric region).The patient usually lies still.

Pain may be sudden or gradual in onset, varying considerably in intensity, often severe and unremitting, but at times may be no more than a dull ache. In some cases, especially in feeble and aged patients, pain may be entirely absent.

Abdominal tenderness and rigidity are typically seen when inflammation involves anterior abdominal wall. Tenderness and rigidity are diminished or absent if anterior abdominal wall is unaffected as seen in pelvic peritonitis or peritonitis in lesser sac. Patients with pelvic peritonitis complain of urinary symptoms. Infrequent bowel sounds may be heard, but ceases once paralytic ileus sets in.

Pyrexia is also present in many cases. Nausea is a frequent occurrence and may be accompanied by vomiting. Fever is usually higher and more spiking in healthy young adults than infants and old aged patients. Hypothermia may occur in severely ill patients.

Vomiting may be slight at start, but as peritonitis advances, it becomes persistent. At first only the stomach contents are voided, later the fluid that is brought up is bile-stained and brownish. While finally the obstruction becomes complete, it becomes feculent. In the early stages vomiting is reflex in origin, later it becomes secondary to paralytic ileus.

A **rising pulse rate** and falling temperature are of grave significance. On the other hand, a gradually rising temperature and slowly falling pulse rate suggest localization of infection is taking place.

Intermediate phase :

Peritonitis may resolve, so that the pulse slows, the pain and tenderness diminish, leave a soft, silent abdomen. It may localize, producing one or more abscesses, with overlying swelling along with tenderness.

Terminal phase :

If localization or resolution has not occurred, the abdomen remains silent, and increasingly distends. Circulatory failure ensues, with cold, clammy extremities, sunken eyes, dry tongue, thready (irregular) pulse, drawn and anxious face (Hippocratic facies).The patient finally lapses into unconsciousness. With early diagnosis and adequate treatment, this condition is rarely seen in modern surgical practice. [22]

SIGNS OF PERITONITIS:

Inspection:

There is diminution or absence of abdominal respiratory movement. The position of patient in bed is characteristic. He lies still in bed with legs drawn up in an effort to relieve the tension on the abdominal muscles. There is uniform distension of abdomen and in early cases marked retraction of lower half of abdomen.

Palpation:

Tenderness and rigidity will be elicited. Tenderness is a constant but not a reliable sign as rigidity. Tenderness is first situated over the causative focus, but spreads with a diffusion of the peritoneal inflammation, which rapidly becomes generalized, and extreme in degree.

There are two other signs that are constantly present:

- Rebound tenderness.
- Pain experienced over the affected region by pressure on an uninvolved region.

Of all signs, rigidity of the abdominal muscles is the most important and reliable sign. Voluntary guarding following inflammation of parietal peritoneum, also by reflex spasm may be initially present. As peritonitis advances reflex spasm become so severe that board like rigidity of abdominal wall is produced.

Percussion:

Abdomen is resonant everywhere and resonant tympanic owing to the fact that the intestines are filled with gas. In certain instances, like the perforation of GIT, obliteration of liver dullness is evident.

Auscultation:

Bowel sounds are diminished from the onset. They may be absent over the area of greatest mischief, and in all established cases of peritonitis with ileus, there is often a sinister silence [22].

INVESTIGATIONS OF PATIENT WITH PERITONITIS: -

A number of investigations may elucidate doubtful diagnosis, but the clinician should mainly rely on history and physical findings to arrive at a diagnosis.

Routine Investigations:

Hemoglobin and urine analysis are done. ESR may be raised, particularly in abdominal tuberculosis affecting the peritoneum. Leukocytosis is usually seen, especially the differential counts with shift to left, are more important.

Peritoneal diagnostic aspiration:

It may be useful when sufficient peritoneal fluid is in the peritoneal cavity to be aspirated. First described by Solomon, it is done in four quadrants after infiltrating the skin with a local anesthetic. When aspiration fails, the introduction of a small quantity of sterile physiological saline, followed by aspiration after a few minutes, may produce fluid of diagnostic value. Microscopy of the fluid may show neutrophils more than 250cells/mm3 (indicator of inflammation) and bacteria (indicator of infection). Fluid is also examined for total count, differential count, $_{P}$H and gram stain and aerobic and anaerobic culture.[21]

An erect X-ray film of the abdomen:

The X-ray should include the diaphragm, lower chest and pelvis. There may be pneumoperitoneum (demonstrated by gas under the dome of diaphragm) and ground glass appearance due to edema of

peritoneum. There may be dilated gas-filled loops of bowel (consistent with paralytic ileus). Demonstration of pneumoperitoneum is seen in excess of 70% of cases of GIT origin. If the patient is too ill to stand, lateral decubitus posture can be used.

Biochemical Investigations:

- Estimation of serum electrolytes – Sodium and Potassium.
- Serum amylase levels to exclude acute pancreatitis provided it is remembered that moderately raised values are frequently found following other abdominal catastrophes and operations. For e.g., perforated peptic ulcer, Cholecystitis.
- Widal test in ileal perforation to rule out typhoid.
- Blood urea, serum creatinine to know the status of renal system
- Peritoneal fluid for culture and sensitivity: This can be done by aspiration or from fluid derived at laparotomy. It may be particularly helpful in the diagnosis of primary peritonitis.
- Laparotomy is done to diagnose and to treat peritonitis. On laparotomy, the peritoneal cavity can be cleaned by lavage.
- Biopsy can be taken wherever found necessary.[22]

Ultrasound and CT scanning:

These investigations may also be useful in some patients in identifying the cause of the peritonitis. E.g. perforated appendicitis, acute pancreatitis and also may show fluid collection in peritoneal and pelvic

cavities. It may also influence operative approach or contraindicate operation. Other investigations have to be done according to the specific etiology, which is described under the specific type of peritonitis.

Prognostic factors

Do we need scoring systems?

The complex nature of surgical infections, the multifaceted aspects of treatment, and the complexity of ICU support make evaluation of new diagnostic and therapeutic advances in this field very difficult. Scoring systems that provide objective descriptions of the patient's condition at specific points in the disease process aid our understanding of these problems[23]. The success of TNM staging for Cancer, Glasgow coma scale for head injury and acute trauma score (ATS) for trauma has prompted researchers to look for scoring system in determining the outcome of disease with regard to peritonitis. The commonly tried scoring systems are:

1. Mannheim peritonitis index
2. Sepsis score of Elebute and Stoner
3. APACHE II score.

All the systems are mainly used to predict death in patients with surgical infections. Most of the scoring systems are inappropriate for use in therapeutic decisions concerning individual patients.

In a country like India, where most of the critical care measures are unavailable and unaffordable by average citizens, it is vital that a scoring system should be evaluated which not only prognosticate accurately the outcome, but should also be simple and cost effective.

Mannheim Peritonitis Index (MPI)

MPI, was originally derived from data collected from 1253 patients with peritonitis treated between 1963 and 1979, and was developed by discriminant analysis of 17 possible risk factors, by Wacha[12], 8 of these were of prognostic relevance and is currently employed widely for predicting mortality from peritonitis. The information is collected at the time of admission and first Laparotomy.

The original reports excluded appendicitis and post-operative peritonitis, but further investigation extending to these groups did not reduce the predictive value.

Mannheim Peritonitis Index (MPI)

Risk Factors	Weighting if present
1. Age > 50 years	5
2. Female Sex	5
3. Organ Failure	7
4. Malignancy	4
5. Preoperative duration of peritonitis > 24 hr.	4
6. Origin of sepsis not colonic	6
7. Diffuse generalized peritonitis	6
8. Exudate Clear Cloudy, Purulent Faecal	 0 6 12

Definitions or organ failure

Kidney	Creatinine level ≥ 177 μ mol/l Urea level ≥ 167 m mol/l Oliguria < 20 ml / h.
Lung	PO_2 < 50 mm Hg PCO_2 > 50 mm Hg
Shock (definition according to Shoemaker)	Hypodynamic or Hyperdynamic
Intestinal obstruction (only if profound)(	Paralysis ≥ 24 h or complete mechanical ileus.

PO_2, Partial pressure of O_2, Pco_2, Partial pressure of CO_2

Detailed study of MPI was done by A. Billing [1] in 7 different centers and their data compared. They considered patients of perforated or postoperative peritonitis, peritonitis caused by pancreatitis, appendicitis and mesenteric ischemia for study.

- Each risk factor is given a weightage to produce a score used for prognostic purposes.
- Maximum score is 47
- The cut off point taken was a score of 26. Patients with higher values being classified as non-survivors.
- Patients were divided into 3 categories of severity. MPI < 21, 21 – 29, > 29.
- They found linear correlation between mean index score and mean mortality rate.

Advantage of MPI

- It is one of the easiest scores to apply
- The determination of risk is available during operation
- Surgeon can know about the possible outcome and the appropriate management can be decided.

Patient with less score can be treated with usual minimal risks, while patient with high score may need aggressive approach with critical care monitoring. Concept of programmed relaparotomy, zip technique surgery may need to be considered in these cases. It is peritonitis specific index and appears to be the best for statistical studies and comparing clinical trials. Other scores like Apache-II score are not specific for peritonitis.

Disadvantages

1. This index does not include the possibility of eradicating the source of inflammation.
2. It is a one time score; hence post-operative complications may hamper the results.
3. The index assigns peritonitis originating from colon to be a low risk. Since most of the colonic performances are usually secondary to malignancy, this may not be applicable uniformly.

Sepsis score of Elebute and Stones7

It was first published in 1983. It was primarily designed for district general hospitals, for monitoring patients suffering from peritonitis. The authors divided the clinical features of the septic state into 4 classes to which they ascribed a subjective degree of severity on an analogue scale. The attributes were local effects of tissue infection, degree of temperature elevation, secondary effects of sepsis and laboratory data.

Sepsis Score of Elebute and Stoner

Scoring of local effects tissue infection	**Score**
Attribute	
Wound infection with purulent discharge / enterocutaneous fistula	
- Requiring only light dressing changed not more than once daily	2
- Requiring to be dressed with a pack, dressing needing to be changed more than once daily, requiring application of a bag and/or requiring suction.	4
Peritonitis	
Localized	2
Generalized	6
Chest Infection	
Clinical or radiological signs of chest infection without productive cough	2
Clinical or radiological signs of chest infection with a cough producing purulent sputum	4

Full clinical manifestations of lobar /broncho pneumonia	6
Deep seated infection (e.g. subphrenic abscess,pelvic abscess, empyema Thoracis, acute or chronic osteomyelitis)	6

Scoring of Pyrexia (oral Temperature) Attribute	**Score**
Maximum daily temperature (^{o}C)	
36 – 37.4	0
37.5 – 38.4	1
38.5 – 39	2
> 39	3
< 36	3
Minimum daily temperature > 37.5 C	1
If 2 or more temperature peaks above 38.4Oc in 1 day	1
If any rigors occur in a day	1

Temperature should be recorded at least 4 times in 24 hours, record for the period is assessed as above and “pyrexia score” computed.

Scoring of secondary effects of sepsis Attribute	**Score**
Obvious jaundice in the absence of established hepatobiliary disease	2
Metabolic acidosis compensated	1
Uncompensated	2
Renal failure	3
Mental orientation	3
Bleeding diathesis	3
Scoring of laboratory data	
Blood culture single positive culture	1
Two or more positive culture separated by 24 hours	3
Single positive culture + history of invasive procedure	3
Single positive culture + cardiac murmur and/or tender enlarged spleen	

Leukocyte count (x 109 / L)	
12 – 30	1
> 30	2
< 2.5	3
Hemoglobin level (gm/dL)	
7-10	1
< 7	2
Platelet count (x 10^9/L)	
100 – 150	1
< 100	2
Plasma albumin level (gm/l)	
31 – 35	1
25 – 30	2
< 25	3

Plasma total bilirubin level in the absence of clinically obvious jaundice > 25 μ mol / L. The positive range of scores is 0 to more than 45. This system was examined in more detail by Dominioni[24]in 135 patients. The sepsis scores range from 10 to > 30. The overall accuracy was 84% for mortality.

Advantages

1. Since, this was primarily designed for district hospitals, it is more appropriate for Indian set up.
2. Since, it includes detailed clinical work up, it is more sensitive.
3. The range of lab tests is kept minimum.
4. It can be used either as a single one time score or can be used to monitor critical patients and score tabulated on regular basis.

Disadvantages

1. Most of the attributes are calculated subjectively, hence more prone for observer variations.
2. No direct attempt to score "septic shock", hence it provides indirect evidence for sepsis syndrome.

Apache – II score

This includes 2 parts: First one deals with acute physiology while second is concerned about chronic health evaluation. This was primarily designed for ICU patients. In 1984, Meakins and associates used this score to evaluate patients with peritonitis. They found striking correlation between mortality rate and increase in score. The Apache-II utilizes 12 values and determines the outcome based on this. This system even though correctly measures severity of illness, in cumbersome in surgical practice and does not give any indication regarding management modalities of patient.

Other scoring systems

John Boey in 1986 published a study of risk stratification in perforated duodenal ulcer. They included 3 criteria namely major medical illness, preoperative shock and long standing perforation (more than 24 hours). They assigned 0 if no risk factor were present and scores to 1 to 3 depending on number of risk factors present. They concluded that definitive surgery (vagotomy and drainage) can be safely performed if no risk factors are present. If any of the risk factors is present, it is preferable to do simple closure. If all 3 risk factors are present, the outcome they found was uniformly dismal whether patient was operated or treated conservatively. Hence, conservative treatment deserves reevaluation in these patients.

Which scoring is best?

Though no major studies have been conducted to compare all the studies, as most of the system requires different clinical and laboratory parameters, almost all researchers agree for a reliable, simple and easily reproducible scoring system which helps not only in decision making. Prognosticating sepsis but also can be used for comparing data at different institutes.

Billing[1] who conducted study of MPI in 2003 patients at different centers in 3 different European countries reported that is not only reliable in predicting mortality but can also be used for comparative study.

Demmel[16] conducted a study comparing MPI with Apache-II scores, and concluded that both scores were equally accurate, but MP1 was easier and disease specific.

Pacelli[3] conducted study in 1996, comparing MPI, Apache and sepsis score. They concluded that MP1 and Apache-II correctly predicted death as outcome, but MP1 was easier to calculate.

Ohmann.C[25]concluded none of the existing score was of particular use for therapeutic decision making in peritonitis. The new prognostic model should be the focus of further trials.

Deducing from above studies, it appears that MPI and sepsis score seem to be appropriate study for patients with peritonitis and sepsis syndrome, in a district hospital set up as it utilizes minimum investigations and can be used for predicting outcome of the patient. The score conceived by John Boey with reference to perforated duodenal ulcers can be utilized for decision making as regard to what surgery is to be performed whether to operate at all

.MANAGEMENT OF PERITONITIS

STANDARD TREATMENT:

Kirschner, in 1926, formulated two surgical principles for the management of peritonitis which later have become the gold standard[26].

1. **"*Plugging*"** the source of infection.
2. **"*Purging*"** the peritoneal cavity of bacteria, toxins and adjuvant.

Thus the laparotomy, repair of bowel leak and peritoneal toilet became the standard therapy, but the morbidity and mortality continued to be high.

Disadvantages of standard operative treatment:

This results in tight closure of the abdomen, where intra-abdominal pressure is already high, causing respiratory embarrassment, ventilation perfusion imbalance and its consequences. Sepsis elimination cannot be confirmed with the single laparotomy and there is no control over the intraabdominal process like anastomosis healing or bowel viability.

New operative concepts:

The era of new operative concept started in 1975 when the dissertation of Pujol from Parries University. He concludes that intraabdominal Sepsis should be treated like abscesses in any other parts of the body. He advocated leaving the abdomen open (laparostomy) and

treating like an open wound - A radically different approach. After this a number of surgeons published their experience with this new operative modality confirming definite improvement in mortality.

Treatment in general consists of

- ✓ General care of the patient
- ✓ Specific treatment for the cause
- ✓ Peritoneal lavage when appropriate

GENERAL CARE OF THE PATIENT:

Fluid resuscitation: Consists of correction of circulating volume and electrolyte imbalance. Extensive peritoneal inflammation causes fluid to shift into the peritoneal cavity and the intestinal space. Urine output has to be maintained about 30ml/hr. The plasma volume must to be restored and the plasma electrolyte concentration has to be maintained. Central Venous catheterization and pressure monitoring may be helpful in correcting fluid and electrolyte balance particularly in patients with concurrent disease. Plasma protein depletion may also need correction as the inflamed peritoneum leaks large amounts of protein. If the patient's recovery is delayed for more than 7-10 days, parenteral nutrition is required.

Gastrointestinal decompression: A nasogastric tube is passed into the stomach and aspirated. Aspiration is continued until the paralytic ileus has recovered.

Analgesia: Freedom from pain allows early mobilization. Adequate physiotherapy in the post-operative period helps to prevent basal pulmonary collapse, deep vein thrombosis and pulmonary embolism. [22]

Vital system support: If septic shock is present, special measures may be needed for cardiac, pulmonary and renal support. Oxygen is administered to overcome the mild hypoxemia that is commonly present in peritonitis because of increased metabolic demands of infection, some degree of intrapulmonary arterio-venous shunting and the mechanical impairment of pulmonary ventilation by distended, tender abdomen. Ventilatory support should be initiated whenever any of the following are present;

1. Inability to maintain adequate alveolar ventilation as evidenced by a rising PaCO2 of 50 mm Hg or greater.
2. Hypoxemia reflected in PaO2 < 55 mm Hg.
3. Evidence of shallow, rapid respiration due to muscular tiring or the use of accessory muscles of respiration.

Antibiotic therapy:

The bacterial flora is monomicrobial in nature, in primary peritonitis And polymicrobial in secondary peritonitis, an observation established by Alt emeir in 1938, in a study of appendiceal abscess[27]. When experimental peritonitis with E. coli and B. fragilis was treated with

different antibiotic regimens, clear patterns of response were seen.

Treatment with gentamicin alone improved the acute death rate in the model but had no impact on the abscess phase of the disease. Nicholas et al demonstrated improvement in the death rate of rats with polymicrobial experimental peritonitis induced with a large inoculum, by the addition of clindamycin coverage for B. fragilis. From these animal studies, combination therapy was born and became the standard for the treatment of peritonitis during the late 1970s. In the 1980s, the emergence of single antibiotics with both aerobic and anaerobic activity leads to numerous clinical studies that compared the newer antibiotics to combination therapy. With one exception, most comparative studies consistently demonstrated comparable results with single agent compared to the combination. Costs and drug toxicity reduced with the single antibiotic approach. As the infection is usually a mixed one, a single or combination therapy that have activity against aerobic and anaerobic bacteria, is used. Culturing peritoneal fluid and modifying the antibiotic subsequent to the culture sensitivity may not always influence the outcome.

SUGGESTED ANTIMICROBIAL AGENT THERAPY FOR THE TREATMENT OF ESTABLISHED SECONDARY BACTERIAL PERITONITIS:

MILD TO MODERATE INTRA-ABDOMINAL INFECTION:

- Second or third generation cephalosporin OR
- β- Lactamase inhibitor combination OR
- Monobactum + metronidazole

SEVERE INTRA-ABDOMINAL INFECTION WITHOUT RENAL DYSFUNCTION :

- Carbapenem OR
- Fluoroquinolone + metronidazole OR
- Aminoglycosides + metronidazole + ampicillin

SEVERE INTRA-ABDOMINAL INFECTION WITH RENAL DYSFUNCTION :

- Carbapenem OR
- Fluoroquinolone + metronidazole[21]

Specific treatment of the cause (operative management):

The primary therapy in the management of generalized peritonitis is surgical. This depends on the cause of generalized peritonitis e.g. perforation closure in case of perforated duodenal ulcer. Though there are other factors that affect the outcome in suppurative peritonitis,

timing of operation is an important variable that is often overlooked. In peritonitis due to pancreatitis or salphingitis or in cases of primary peritonitis of streptococcal or pneumococcal origin, non-operative management is preferred (if the diagnosis is made with certainty).

OPERATIVE PRINCIPLES:

1. Control of source of infection- Repair/Plug
2. Purge- Peritoneal lavage and toilet i.e. evacuate bacterial inoculums, pus and adjuvant.
3. Decompress- Treat or avoid intraabdominal compartmental syndrome.
4. Control- Prevent or treat persistent and recurrent infection or verify both and purge [26] .

PRINCIPLE – 1 REPAIR:

The infectious material leaking into the abdomen is to be eliminated. This involves procedures like appendicectomy, closure of duodenal or ileal perforation, resection of gangrenous viscera or necrosectomy of pancreas.The bowel ends may be anastomosed, exteriorized or simply closed.

PRINCIPLE-2 PURGE:

Infectious peritoneal fluid, pus , necrotic tissue and inflammatory exudate either contain bacteria or promote their growths and they should be removed. A large quantity of saline about 8-10 liters may be

required for wash and “radical debridement”. However, too aggressive debridement should be avoided to prevent excessive blood loss or bowel injury. Antibiotic/ betadine wash have not been proved to be any great advantage. At the end no irrigation fluid should be left in the abdomen.

PRINCIPLE-3 DECOMPRESSES:

During acute peritonitis more than 10 liters of inflammatory fluid may accumulate in the peritoneum and its sub-mesothelial loose connective tissue. The co-existent paralytic ileus, fluid accumulation in the peritoneal cavity, post resuscitation visceral and parietal edema increases the intraabdominal pressure producing a compartment syndrome. In this situation, if the abdomen is closed with tension, there will be impairment of cardiovascular, respiratory, renal and hepatic functions and also splanchnic blood flow and oxygenation. The answer to this problem lies in open abdomen or staged abdominal repair (STAR).

PRINCIPLE-4 CONTROL:

This principle aims at having control over the intra-abdominal processes like anastomotic healing, proper closure of perforation, and viability of bowel segments and formation of pus inside the abdomen. This aim is not achieved by the standard operation. This principle allows for frequent re-exploration and peritoneal toilet if required.

NEW OPERATIVE METHODS:

With the entire above complex and interesting knowledge, we can now concentrate on the new operative methods evolved for the treatment of severe intra-abdominal sepsis. In 1993, the "International society of surgery" called several experts in this field to the "International surgical week" held at Hong Kong and decided on four basically different methods.[26]

- OPA- Open abdomen (Laparostomy)
- COLA- Covered Laparostomy
- PR- Planned relaparotomy
- STAR- Staged abdominal repair

OPEN ABDOMEN (LAPAROSTOMY):

This is defined as laparotomy without re-approximation and suture closure of abdominal fasciae and skin. Abdominal cavity is left open like an open wound and dressed and finally heals by granulation. This method takes care of principles- repair, purge and decompression. The disadvantages are, there is no control over intraabdominal process, exposed viscera may perforate and huge ventral hernia results since definitive closure is not possible. Hence it has lost its popularity.

COVERED LAPAROSTOMY (COLA):

This is defined as laparotomy without re-approximation and suture closure of abdominal fasciae and covering the facial gap with materials like merles or vicryl mesh. The viscera may also be covered with skin with relaxing incision.

PLANNED REPAPAROTOMY (PR):

In this approach abdomen is left open initially and re-explored at an interval of 12-24 hours for irrigation, debridement etc. Devices used to ease re-exploration include commercially available Zipper, Ethizip, Velcro, artificial burr, PTFE mesh (Gortex) etc. this procedure allows for having control over intra-abdominal processes.

STAGED ABDOMINAL REPAIR (STAR):

This is a series of planned abdominal operations with staged re-approximation and final suture closure of the abdominal fasciae. It is planned either before or during the first operation called Index Star. The abdomen is closed temporarily with devices like Zip, Velcro etc. and controlled tension is exerted to the fascia avoiding and intra-abdominal pressure effects. Re-laparotomies are performed at 24 hour intervals at operating room. Once problem is solved abdominal cavity is formally closed.

INDICATIONS FOR STAR:

1. Diffuse peritonitis in critical patient condition.
2. Severe peritoneal edema.
3. Source of infection is not controlled.
4. Incomplete debridement of necrotic tissue.
5. When viability of bowel is uncertain, anastomosis / repair needs Re-inspection
6. Uncontrolled bleeding with packing.
7. Infected pancreatic necrosis.
8. Massive abdominal wall loss.
9. Any intra-abdominal problem that is difficult or impossible to manage with a single operation.[19]

ADVANTAGES OF STAR:

Staged abdominal repair technique allows for complete repair, debridement and purge. Anastomotic healing is monitored and any complications diagnosed early & corrected. Intra-abdominal compartment syndrome and its consequences are prevented. With the STAR technique colostomies may be avoided in favor of anatomists, abdominal drains with their disadvantages are avoided and finally this technique allows for suture closure of abdomen with sound healing.

Peritoneal lavage:

Price first advocated washing the contaminated peritoneal with large volumes of irrigant in 1905. In 1906, Torek reported that large volume irrigation reduced mortality in generalized peritonitis following appendicitis in 14%. Lavage is done on the basis that phagocytic macrophages and neutrophils cannot function unless attached to peritoneal serosa. They cannot function if they are swimming as phagocytes already dislodged from peritoneum are either dead or non-functional, in which case lavage causes no harm.

There are 3 basis principles of peritoneal lavage

1. To wash the digestive enzymes, that might have leaked into the peritoneal cavity.
2. To remove material like pus, blood and faeces that could harbor or nourish bacteria
3. To potentiate the antibiotic effect by allowing the topical application of relatively high dosage of these agents.

The majority of surgeons lavage until the fluid is clear, use more than 1 litre. In the case of the dirty abdomen (i.e. gross pus or faecal peritonitis), saline, aqueous betadine, water and antibiotic lavage can be used. Surgeons also use Intra operative Peritoneal Lavage during clean cases [28].

Drains:

The use of drains, particularly sump suction drains is an important aid in the surgical management of intra-abdominal abscesses or similarly localized collection.

CONSERVATIVE MANGEMENT

Conservative management may be advisable in following conditions

- Appendicular abscess when the infection is definitely localized and mass is subsiding.
- Gonococcal peritonitis
- Chronic pelvic abscess
- In primary primary peritonitis of children
- Moribund patients.

COMPLICATIONS OF PERITONITIS

SYSTEMIC COMPLICATION OF PERITONITIS:

1. Bacteremic/endotoxic shock
2. Bronchi pneumonia/respiratory failure
3. Renal failure
4. Bone marrow suppression
5. Multisystem failure

Bacteremic/ endotoxic shock:-

It is due to large amount of exudation from the inflamed peritoneum into the peritoneal cavity, vomiting and paralytic ileus, where the absorbing function of bowel is lost. It depends on the microbial infection in severity. Gram-negative septicemic shock is common in enteric and large bowel perforation.

Bronchopneumonia/ respiratory failure:

This occurs in early stage of peritonitis, which is severe. Hurried breathing in early stages is due to under-ventilation, which is because of abdominal distension causing restriction of diaphragmatic and intercostal muscle movement.

Renal failure:

Hypovolumia decreased cardiac output, increased secretion of ADH and aldosterone and raised intra-abdominal pressure act together in peritonitis, on the kidney. This is especially true in septic shock. Acute tubular necrosis can occur because of decreased flow and will lead to oliguria and metabolic acidosis.

ABDOMINAL COMPLICATIONS OF PERITONITIS:

1. Adhesive small bowel obstruction
2. Paralytic ileus
3. Recurrent or residual abscess
4. Portal pyemia/liver abscess.

Adhesional small bowel obstruction:-

The adhesions, when fine and minimal, are absorbed, but when dense cause intestinal obstruction at a later date. They manifest with all signs of obstruction. Failure of conservative treatment necessitates surgery, to divide the adhesions and relieve the obstruction.

Paralytic ileus: (Neurogenic obstruction)

The bacterial toxins act on neuromuscular junctions and smooth muscle of bowel producing paralytic ileus. It is beneficial as it avoids spreading of the peritoneal contents from perforated viscous to other regions but prolonged paralytic ileus may prove to be a serious setback because fluid loss from the intestine into the lumen may play a large part in protein, water and electrolyte depletion.

Abscess:

Presentation may be very vague and consist of nothing more than a lassitude, anorexia, pyrexia (often low-grade), tachycardia, leukocytosis and localized tenderness. Later on a palpable mass may develop. When palpable, an intra-peritoneal abscess should be monitored by marking out its limitations on the abdominal wall and meticulous examination. Abdominal ultrasound has been a popular method for the diagnosis of intra-abdominal abscess. It is a low cost method. Several radionuclide scans have been developed to identify abscess with in the peritoneal cavity. The gallium citrate-67 scan

achieved a certain level of popularity for the diagnosis of intra-abdominal abscess. Gallium concentrates within inflammatory foci and with use of radioactive isotope of gallium, a gamma camera should be able to identify collections of pus. More recently, indium 111-tagged leukocytes have been used as another potential imaging technique.

The diagnostic method of choice for abdominal abscesses is CT scan. The CT scan provides remarkable anatomic resolution of normal structures and of abnormal collections of fluids and pus. The use of intraluminal and in some cases, intravascular contrast agents permits differentiation of intraluminal and extraluminal collections. Abscess cavities commonly have air bubbles that augment the judgment that any fluid collection may be an abscess. The accuracy of the CT scan in the diagnosis approaches 90%.In the majority of the patients, with the aid of antibiotic treatment the abscess or mass becomes smaller and smaller and finally is undetectable.

In others, the abscess fails to resolve or becomes larger, in the event of which it must be drained. In many situations, the abscess becomes adherent to the abdominal wall, so that it can be drained without opening the general peritoneal cavity. Other modes of treatment are percutaneous drainage and open drainage of the abscess. Septic patients with evidence of severe clinical infection will usually require open laparotomy and drainage. A persistent septic response with

hyperglycemia, gastrointestinal ileus, blood culture positive for anaerobic and enteric pathogens and early evidence of respiratory failure as the initial expression of multi organ failure cascade, mean that a source of clinical infection must be identified and treated.

CLASSIFICATION OF INTRAABDOMINAL INFECTIONS

1. PRIMARY PERITONITIS

a. Spontaneous peritonitis in children.

b. Spontaneous peritonitis in adults.

c. Peritonitis in patients with CAPD.

d. Tuberculosis and other granulomatous peritonitis.

e. Other forms.

2. SECONDARY PERITONITIS

a) Acute perforation peritonitis (Acute supportive peritonitis)

b) Post-operative peritonitis

c) Post-traumatic peritonitis

3. TERTIARY PERITONITIS

a) Peritonitis without evidence for pathogens.

b) Fungal peritonitis.

c) Peritonitis with low grade pathogenic bacteria.

4. OTHER FORMS OF PERITONITIS

a. Aseptic/sterile peritonitis.

b. Granulomatous peritonitis.

c. Drug-induced peritonitis.

d. Periodic peritonitis.

e. Lead peritonitis.

f. Hyperlipidemic peritonitis.

g. Foreign-body peritonitis.

h. Talc peritonitis.

5. INTRA ABDOMINAL ABSCESS

a. Associated with primary peritonitis.

b. Associated with secondary peritonitis.

PRIMARY PERITONITIS:

Primary peritonitis is an inflammation of the peritoneum from a suspected extra peritoneal source, often via hematogenous spread. Spontaneous bacterial peritonitis is now more common in adults than in children and shows no differential sex incidence. Adults with cirrhosis or systemic lupus erythematous have replaced children with nephrosis, formerly the group most commonly affected. Spontaneous peritonitis in adults is seen most commonly in patients with ascites and is a monomicrobial infection.

Onset is more insidious in ascitic adults. Most patients complain of abdominal pain and distension, vomiting, lethargy and fever more prominent in children. Diarrhea is typical in neonates, but seldom seen in adults. The clinical picture may be non-specific. Paracentesis is the most useful diagnostic test. Fluid is examined for neutrophil cell count; pH and gram stain should be done a specimen sent for culture. The neutrophil cell count has the highest sensitivity and specificity in making the diagnosis.

A neutrophil count > 250 cells / cu mm is positive. Ascitic fluid pH is low in spontaneous bacterial peritonitis. Only one third of patients with positive fluid cultures. If the stain shows only gram-positive cocci, spontaneous peritonitis is strongly suggested; if a mixed flora of gram positive and negative is present, intestinal perforation is more likely. When the diagnosis of spontaneous bacterial peritonitis is confirmed, antibiotic therapy should be started and the patient initially managed nonoperatively.1 9, 21.

SECONDARY PERITONITIS

CHEMICAL (ASEPTIC) PERITONITIS:

Aseptic peritonitis refers to the peritoneal inflammation from substances other than bacteria. A perforated peptic ulcer provides the most severe and common form of chemical peritonitis with gastric juice and bile contaminating the peritoneal cavity. Biliary peritonitis alone

may follow gangrene and perforation of the gallbladder. Blood in the peritoneum is also a cause of peritoneal irritation after slow bleeding (e.g. a ruptured graafian follicle or following splenic injury) rather than from a catastrophic hemorrhagic event as a ruptured aneurysm where the primary pathology itself overshadows the peritoneal irritation. Meconium and urine may also precipitate chemical peritonitis.

PERITONITIS DUE TO PERFORATED PEPTIC ULCER:

The perforation generally occurs as sudden, relatively catastrophic event. The patient with a perforated peptic ulcer classically presents with abrupt onset of epigastric pain, with or without radiation to shoulder. Generalized peritonitis supervenes within hours and the patient lies motionless to minimize pain. These classic features may be absent in several circumstances. In very young or aged, immuno suppressed, quadriplegic and comatose patients, perforation may be present in a much more subtle manner. The classic presentation can be modified when gastric juice flows down the paracolic gutters, simulating acute appendicitis on the right side and acute sigmoid diverticulitis on the left. In the other forms, a perforated duodenal ulcer simulates perforated gall bladder and duodenum. [29]

Sometimes, following an ulcer perforation, the ulcer may seal rapidly before there is a spillage of gastric and duodenal contents.

Other rare presentations of perforated duodenal ulcer:

1. Perforation associated with hemorrhage is rare but a grave complication. The bleeding arises from erosion of large vessel such as gastroduodenal artery. The clinical picture is that of acute perforation of peptic ulcer with signs of hemorrhage. Perforation and pyloric stenosis, this combination is very rare. Lam and colleagues in 1978 noted that 4 out of 244 patients had this combination of perforation, hemorrhage and obstruction.
2. Retroperitoneal perforation; it usually follows blunt trauma to the abdomen in the epigastric region. It is more difficult to detect. Patient may have pain in the epigastric region and back and may develop vomiting. Later, patient may develop retroperitoneal cellulitis and succumb to it. In still some other cases, the pus may track retroperitoneally into the right iliac fossa and may present as a mass simulating appendicular abscess which on drainage may lead to duodenal fistula.

Apart from earlier mentioned investigations the following investigations are also useful

Upper gastro intestinal study with gastrograffin series:

The use of water soluble radio contrast material is advocated in diagnostic work up of the patient with duodenal ulcer perforation.

Without pneumoperitoneum it confirms diagnosis, the site, presence of ulcer crater, whether perforation is sealed off or not.

Disadvantages:

1. Pylorospasm induced by the water soluble contrast may impair clear visualization of the duodenum.
2. The time taken to perform a contrast study at odd hours.

   In retroperitoneal perforation following features may be seen in the erect abdominal X-ray.

   - Mild scoliosis, usually concave to the right.
   - Obliteration of psoas shadow.
   - Retroperitoneal air around upper pole of the right kidney along the right psoas muscle and around the transverse mesocolon.

Treatment:

The following treatment has been described for perforated ulcer.

1. Simple closure of perforation with omental patch.
2. Definitive treatment for the ulcer at the time of perforation closure . This includes – Simple closure of perforation with or without drainage procedures like gastro enterostomy and vagotomy.

Contraindications for definitive surgery include

– Unstable patient

– Perforation of more than 24 hrs duration or

– Gross contamination of the peritoneum.

For gastric perforation four quadrant biopsy has to be taken and if the patient is fit, gastric resection with ulcer has to be done unless the ulcer is juxta esophageal, in which case the ulcer should be repaired and Tanner procedure should be held in reserve as a secondary choice.

3. Laparoscopic closure of perforation

APPENDICEAL PERFORATION:

Immediate appendicectomy, has long term been the recommended treatment of acute appendicitis because of the known progression to rupture. Studies have shown that delays in presentation were responsible in majority of perforated appendices. There is no accurate way of determining when and if an appendix will rupture prior to resolution of the inflammatory process.

Appendiceal rupture occurs most frequently distal to the point of luminal obstruction along the antimesentric border of the appendix. Rupture should be suspected in the presence of fever greater than 39^0 C and a WBC count greater than 18000/mm3 . Generalized peritonitis will be present if the walling off process is ineffective in containing the rupture.

Treatment:

Treatment consists of appendectomy and peritoneal lavage and antibiotics. The skin and subcutaneous tissue should be left open and allowed to heal by secondary intention in 4 to 5 days as delayed primary closure[22].

TYPHOID PERFORATION:

Typhoid perforation is usually seen in the third week of infection with Salmonella typhi in patients with acute disease. The disease is endemic in regions with poor hygienic conditions. Typhoid bacilli are thought to pierce the peyer's patches of the intestinal wall, mainly in the distal ileum. These collections of lymphoid cells hypertrophy leading to hemorrhage and then perforation.

Perforation often is not appreciated in an already severely diseased patient and it is super infection resulting from leakage of intestinal bacteria that leads to the full-blown picture of suppurative bacterial peritonitis. Widal test will be positive in such patients. [19]

Treatment:

Surgical Management:

At laparotomy, a single perforation is found on the anti-mesentric border of the ileum in 80 per cent of the patients. Two perforations are found in 15 per cent and more than two in 5 per cent. About 90 per cent of ileal perforations are located within 60cm of the

ileo-caecal valve and caecal perforations occur in only 2 percent of the patients. Perforations at the sites other than ileum and caecum are extremely rare. A simple debridement of the margin of the perforation and meticulous closure in two layers with copious peritoneal lavage, is the procedure of choice. However, when there are more than three perforations, which are close together, it is best to resect the affected bowel and perform a primary end-to-end anastomosis. Any areas of apparent impending perforations, if not included in a resection, must be over sewn. A right hemicolectomy is undertaken only for caecal perforations.

Following peritoneal lavage, the abdominal wound is closed with drains. If there is gross faecal contamination, the skin wound may be left open to minimize wound infection. The anti-typhoid drug therapy should be continued for at least 14 days.[30]

OPERATIVE PROCEDURES IN TYPHOID PERFORATION :

Procedure	**Indication/comments**
1.Simple closure	Simple but high leak rate in some series
2.Debridement/ wedge excision+ simple closure	For single ileal perforation. Simple and effective operation, but not recommended if more than three ileal perforations close together
3.Resections Ileal resection + primary anastomosis Right hemicolectomy Simple closure or ileal resection + end-to-side ileotransverse colostomy.	Extensive operations: For multiple ileal perforations. Only for caecal perforation. Extensive operation. Has decreased morbidity but not mortality.
4. Ileostomy of perforated ileum	In extremely critical or moribund patients.
5.Simple peritoneal drainage	In extremely critical or moribund patients.
6.Oversewing	For areas of impending perforation [25].

COLONIC PERFORATION:

Perforation is less common than is obstruction, occurring in about 5 percent of patients. The site of perforation is usually within the tumor and is not associated with obstruction but is the consequence of tumor necrosis. Rapid cardiovascular collapse and endotoxaemic shock, usually signify a major leak and faecal peritonitis. About 22 percent of the cases of peritonitis have their origin in colon. More than half of these are due to inflammatory diseases, such as diverticulitis. The remaining cases are due to perforation proximal to or at stenosis caused by luminal bowel obstruction (tumor) or external bowel obstruction such as incarcerated hernia, intussusception and volvulus.A malignant growth usually does not cause peritonitis directly but may lead to bowel obstruction with either perforation of dilated segments or bowel ischemia and/or bacterial migration through the necrotic bowel wall.

Surgical treatment:

The goal of operation is to remove the diseased perforated segment of the bowel. It is possible to fashion a primary resection and end-to-end anastomosis. However, an anastomosis of unprepared bowel fashioned in a contaminated field should always be protected by proximal colostomy or ileostomy. The temporary diverting stoma can be closed about ten weeks after the emergency operation.

An alternative is to resect the perforated segment and to exteriorize the proximal and distal loops of the bowel, where the proximal opening acts as the colostomy and the distal as the mucous fistula or to use Hartman's operation for more distal lesions, where the distal end is not possible to be brought to the surface of the abdomen. In the Hartman's operation, the diseased segment is excised, end colostomy (proximal) and closure of distal stump is done. Anastomosis is done at a later date. If peritonitis is severe and the patient is not fit for surgery, three stage procedure is preferred. The first stage of the classic three –stage procedure consists of proximal colostomy (transverse). In the second stage, resection of the diseased segment and anastomosis is done. In the third stage, colostomy closure is done. There are considerable drawbacks to the three stage procedure. These include a focus of infection in the abdomen for an unduly longer period before the second stage procedure is done, also the length of time for which transverse colostomy may be present and for the patients to cope with the malodorous fluid effluent from the proximal stoma.

TUBERCULOUS PERITONITIS:

Two forms of peritonitis are seen- Acute and chronic

Acute tuberculous peritonitis:-

This type has an onset that resembles so closely acute peritonitis that the abdomen is opened straw-colored fluid escapes and tubercles are seen scattered over the peritoneum and greater omentum. Early tubercles are greyish and translucent. They soon undergo caseation, and appear white or yellow and are then less difficult to distinguish from carcinoma. Occasionally, they appear like patchy fat necrosis.

Chronic tuberculous peritonitis:-

The condition presents with abdominal pain (90%) cases, fever (60%), loss of weight (60%), ascitis (60%), night sweats (37%) and occasionally as abdominal mass.

Origin of infection:-

Infection originates from;

- Tuberculous mesenteric lymph nodes;
- Tuberculosis of ileocaecal region;
- A tuberculous pyosalphinx;
- Blood borne infection from pulmonary tuberculosis, usually the ‘miliary’, but occasionally the ‘cavitating’ forms.

Varieties of tuberculous peritonitis:-

There are four varieties of tuberculous peritonitis

a. Ascitic.

b. Encysted.

c. Fibrous.

d. Purulent.

***Ascitic form*:-**

The peritoneum is studded with tubercles and peritoneal cavity becomes filled with pale straw colored fluid. The onset is insidious. Pain is often completely absent; in other cases there is considerable abdominal discomfort, which may be associated with constipation or diarrhea. On inspection, dilated veins can be seen coursing beneath the skin of abdominal wall. Shifting dullness can be readily elicited.

***Encysted form*: (loculated)**

Encysted form is similar to the above, but one part of the abdominal cavity alone is involved. Thus a localized intra-abdominal swelling is produced, which gives rise to difficulty in diagnosis.

***Fibrous form*: (Plastic)**

Fibrous form is characterized by the production of wide spread adhesions, which cause coils of intestine, especially the ileum to become matted together and distended. These distended coils act as a 'blind loop' and give rise to steatorrhoea, wasting and attacks of

abdominal pain. On examination, the adherent intestine with omentum attached, together with the thickened mesentery, give rise to a palpable mass. The first intimation of the disease may be sub-acute or acute intestinal obstruction. The division of bands can remedy sometimes the cause of the obstruction easily. If the adhesions are accompanied by fibrous strictures of the ileum as well, it is best to excise the affected bowel, provided not too much of the small intestine needs to be sacrificed. If adhesions are only present, a plication may be performed. Chemotherapy after adequate surgery will rapidly cure the condition.

***Purulent form*:**

The purulent form is rare, and usually occurs secondary to tuberculous salphingitis. Amidst a mass of adherent intestine and omentum, tuberculous pus is present. Relatively larger cold abscesses often form and are present on the surface, commonly near the umbilicus, or burst into the bowel. In addition to prolonged general treatment, operative treatment may be necessary for the evacuation of the cold abscesses and possibly for the intestinal obstruction. The prognosis of this form of peritonitis is relatively poor.

Diagnosis:

A peritoneal fluid tap will show mostly lymphocytes. Tubercle bacilli can be retrieved from ascitic fluid in 80 percent of the time if more than one liter of fluid is cultured. The ascitic fluid has an

increased protein concentration, lymphocytic pleocytosis and glucose concentration below 30mg/dl. At laparotomy a peritoneal biopsy should be taken. The placement of drains or exteriorization of bowel should be avoided.

Treatment:

Medical line of management:

Anti-tubercular chemotherapy should be instituted in all cases of abdominal tuberculosis. At present, the anti-tuberculosis regimen recommended by W.H.O and the International Union against Tuberculosis and Lung diseases is Isoniazid (300mg daily), Rifampicin (450mg daily), Pyrazinamide (1.5gm daily orally) and Ethambutol (25mg/kg/day) or Streptomycin (0.75gm intramuscularly daily) for two months, followed by Isoniazid(600mg) and Rifampicin (600mg) twice weekly orally for four months for an individual of 40-60 kg body weight. The patient is monitored periodically especially for hepatotoxicity. Pyridoxine hydrochloride (5-10 mg/day) must be given along with Isoniazid to prevent peripheral neuropathy.

Surgical line of management:

Operation should be reserved for diagnosis if needle biopsy fails or for treatment of such complications as fecal fistula or obstruction and performed as described earlier.

Management of tuberculous perforations:

According to the site of perforation;

- Gastro-duodenal type; closure with ATT.
- Small bowel type; closure with ileo-transverse anastomosis placed proximal to perforation with ATT.
- Large bowel type; Ileo-transverse anastomosis for lesions on right side and proximal colostomy for left -sided lesions with ATT.

Definitive surgery after patient improves.

AMOEBIC PERFORATION:

Entamoeba histolytica infection of the intestine usually causes dysentery like illness, but sometimes liver abscesses or perforation of large bowel occurs. Liver abscesses also can rupture and can cause diffuse peritonitis. The clinical picture is that of bacterial peritonitis. Treatment consists of resection of the diseased bowel segment with anastomosis and, administration of metronidazole in combination with a third generation cephalosporin is carried out.[19]

MECONIUM PERITONITIS:

Meconium is a sterile mixture of epithelial cells, mucin, salts, fats and bile. It is formed when the fetus commences to swallow amniotic fluid. Meconium peritonitis is an aseptic peritonitis, which develops, late in intrauterine life or during or just after delivery. In the remainder

no cause for the perforation is discernable. It causes matting of intestinal loops and in some cases, the extruded meconium becomes calcified in a matter of weeks.[19]Meconium remains sterile until about three hours after birth; thereafter, unless the perforation has sealed, sterile meconium peritonitis gives way to acute bacterial peritonitis, which, unless treated promptly, is rapidly fatal.[22]

FOREIGN BODY PERITONITIS:

Foreign bodies may be deposited in the peritoneal cavity during operations (sponge or instrument inadvertently left behind) or may result from penetrating injuries or perforation of the intestine following ingestion. A larger foreign body can lead to the formation of an abscess in the presence of bacteria, but otherwise foreign bodies are sealed off and encapsulated.

PERIODIC PERITONITIS:

Recurrent episodes of abdominal pain, fever, and leukocytosis occur in certain population groups, notably in Americans, Arabs and Jews. The disease appears to be familial. The major point for the surgeons is that, laparotomy is not required in these episodes. Laparotomy is often performed for the first episode, since an acute intra-abdominal process requiring surgical cure cannot be ruled out. At operation, the peritoneal surfaces may be inflamed and there is free fluid but no bacteria. Colchicine is effective in preventing recurrent

attacks and a favorable response to chronic administration of colchicine is a definitive diagnostic test.

DRUG RELATED PERITONITIS:

Administration of INH and Erythromycin estolate has been reported to cause acute abdominal symptoms mimicking peritonitis but not development of true peritonitis. A number of cases have been reported in which, beta-blocking drugs have resulted striking thickening of visceral peritoneum. The most frequent clinical presentation is a typical small bowel obstruction, often subtle at onset associated with weight loss and with an abdominal mass on physical examination. The agglomeration of the small bowel produces the mass that is palpable preoperatively.

LEAD PERITONITIS:

Lead peritonitis has the same clinical picture as intermittent porphyria is associated with lead intoxication (occurring in painters, smelter workers, pica in children), and a careful history will lead to correct diagnosis.

HYPERLIPEDIMIC PERITONITIS:

Abdominal pain mimicking peritonitis may be seen in patients with type 1 and type V hyper lipoproteinemia a group of heterogeneous disorders resulting from increased concentration of chylomicrons or VLDL in the blood. If erroneously operated on during early stages, the

abdominal cavity is found to be full of chylous milky material. A careful family history will clarify the differential diagnosis.

PORPHYRIC PERITONITIS:

It is seen in patients with acute intermittent porphyries, who suffer from attacks that cause nervous system damage especially autonomic system. The pain may be localized or generalized and is often accompanied by vomiting and constipation. The diagnosis is established by the demonstration of porphobilinogen in the urine by Watson-Schwartz test.

TALCUM PERITONITIS:

Peritoneal inflammation, exudation and formation of pseudo tumor chronic inflammatory omental tumors) and formation of dense adhesion may follow contamination of peritoneal cavity by glove lubricants (talc, lycodium, mineral oil,corn starch, rice starch) or by cellulose fibers from disposable gauze pads and gowns. The reaction, particularly to rice starch, is largely a hypersensitivity response. When the diagnosis remains unclear, laparoscopy is useful. If the peritonitis is recognized, reoperation may be avoided and corticosteroids or non-steroidal anti-inflammatory drugs administered. Eventually the peritonitis resolves.

TERTIARY PERITONITIS:

Patients with peritonitis and sepsis, in whom initially have been controlled operatively and bacterial contamination have been eliminated by successful antibiotic therapy, may progress to tertiary peritonitis. It is a state in which, host immune system produces a syndrome of continued systemic inflammation. The clinical picture is one mimicking occult sepsis, as manifested by a hyper dynamic cardiovascular rate, low grade fever and general hyper metabolism. The patient had a clinical picture of sepsis, without the focus of infection. Such patients sometimes are subjected to laparotomy in an attempt to drain anticipated recurrent or residual collections of infected fluid. On operation, no pathogens are found. Empiric anti-infective therapy is of no value.

MALIGNANT PERITONITIS (CARCINOMA PERITONII)

This can produce acute and sub-acute peritonitis. It is extremely rare. Primarily, it is a mesothelioma of fibro-sarcomatous nature, which occurs in asbestos workers. Secondary tumor is common mainly from stomach, ovary and large intestine and very rarely from distant sources like breast, lung etc.

PSEUDOMYXOMA PERITONEI:

More frequently in females the abdomen is filled with yellow jelly, large quantities of which are often more or less encysted. The condition is associated with both mucinous cystic tumors of ovary and appendix. Recent studies suggest that most cases arise from primary appendiceal tumors with secondary implantation on to one or both ovaries. It is often painless and there is frequently no impairment of general health for a long time. If the abdomen seems to be distended with fluid, which cannot be made to shift, it should raise the suspicion of pseudomyxoma peritonei. At laparotomy, masses of jelly may be seen which are scooped out. The appendix, if present, should be excised with any ovarian tumor. Unfortunately, recurrence is common. Pseudomyxoma peritonei is locally malignant, but does not give rise to extra-peritoneal metastasis. Occasionally, the condition responds to radioactive isotopes or intra peritoneal chemotherapy, which may be used in recurrent cases. [32]

POST-PUERPERAL PERITONITIS:

Post-puerperal peritonitis, following puerperal infection, is more common after first deliveries. Rigidity is seldom present. This is partly due to stretched condition of the abdominal musculature. The lochia may be offensive but not necessarily so. Diarrhea is common.

Treatment:

If the infection is strictly limited to the pelvis, the correct treatment is to rest the gastrointestinal tract and provide intravenous fluid, antibiotics and correct the electrolyte imbalance. Posterior colpotomy for pelvis abscess can be done.

PERITONITIS RELATED TO PERITONEAL DIALYSIS:

- Peritonitis is a common complication of continuous ambulatory peritoneal dialysis (CAPD), in patients with end-stage renal disease.
- Peritonitis occurs frequently with CAPD than with other intermittent Peritoneal dialysis.
- Catheter related infection is the most common cause and other causes of peritonitis in CAPD are cuff extrusion and tunnel infections. Two-thirds of the positive culture patients have a gram-positive coccus as the positive organism, usually Staphylococcus aureus or Staphylococcus epidermidis. Turbidity in the dialysate is the earliest and the only finding in one-fourth of the cases.

The diagnosis is made when any of the following are present;

a. Positive culture from the peritoneal fluid.
b. Clinical signs of peritonitis
c. Cloudy dialysate effluent.

Treatment:-

The initial treatments are antibiotics administration and heparin in the dialysate as well as an increase in the dwell time of dialysate fluid. The indication for catheter removal include persistence of peritonitis after 4 to 5 days of treatment, the presence of tubercular or fungal peritonitis, faecal peritonitis or severe skin infection at the catheter site. [19]

Post operative period was monitored; intake output charts and vital charts were maintained.Drains were removed after 48 hours and sutures were removed on the 7^{th} post operative day. Most of the operated patients had uneventful recovery. Diagnosis is confirmed by histopathology reports. The patients were followed up for a variable period of time.

MATERIALS AND METHODS

The study is done in 100 patients presenting with peritonitis due to hollow viscus perforation to EMERGENCY OPD, at Rajiv Gandhi Government General Hospital**,** Chennai, from April 2014 to September 2014.

My study is a clinical, prospective, observational and open study conducted during the period of April 2014 to September 2014.

METHOD OF COLLECTION OF DATA

The study is done after obtaining a detailed history, complete general physical examination and systemic examination. The patients are subjected to relevant investigations like x-ray erect abdomen, CXR, USG and routine investigations like Hb, TC, urea, creatinine, serum electrolytes.

All investigations and surgical procedures were carried out with proper informed written consent as appropriately. The data regarding patient particulars, diagnosis, investigations, and surgical procedures is collected in a specially designed case recording form and transferred to a master chart subjected to statistical methods like mean, standard deviation, proportion, percentage calculation and wherever necessary chi square test for proportion are used.

INCLUSION CRITERIA:

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

Various aetiologies causing such features include

1. Acid peptic disease,
2. Tuberculosis,
3. Typhoid,
4. Appendicitis,
5. Gangrenous cholecystitis,
6. Malignancy

EXCLUSION CRITERIA:

Patients with

1. hollow viscus perforation due to trauma
2. associated injuries to other organs
3. associated vascular, neurogenic injuries
4. any other significant illness which is likely to affect the outcome more than the disease in study.

MODE OF STUDY:

The detail history and proper clinical findings were entered in a proforma case sheet.

Patient was subjected to methodical physical examination to assess his general condition. Local examination of abdomen was done and relevant findings were recorded. Rectal examination was done in all cases, per vaginal examination was also done in female patients.

The required and routine investigations were done to establish the diagnosis. Patients were asked to present themselves for follow up after a specific interval or at recurrence of symptoms.

MPI scoring system was done in all patients and patients were classified those with score less than 21, 21 to 29, and more than 29.

Risk Factors	Weighting if present
1. Age > 50 years	5
2. Female Sex	5
3. Organ Failure	7
4. Malignancy	4
5. Preoperative duration of peritonitis > 24 hr.	4
6. Origin of sepsis not colonic	6
7. Diffuse generalized peritonitis	6
8. Exudate Clear Cloudy, Purulent Faecal	 0 6 12

Definitions or organ failure

Kidney	Creatinine level ≥ 177 μ mol/1 Urea level ≥ 167 m mol/1 Oliguria < 20 ml / h.
Lung	PO_2 < 50 mm Hg PCO_2 > 50 mm Hg
Shock (definition according to Shoemaker)	Hypodynamic or Hyperdynamic
Intestinal obstruction (only if profound)(	Paralysis ≥ 24 h or complete mechanical ileus.

PO_2, Partial pressure of O_2, Pco_2, Partial pressure of CO_2

Preoperatively all patients received supportive treatment for correction of hypotension and electrolyte abnormalities.

During laparotomy, intra-abdominal examination of all organs was made in addition to specific pathology.

Primary closure of hollow viscous perforation, Bowel resection anastomosis, Diversion ostomies was done in cases as appropriate with thorough peritoneal lavage and abdominal drains were kept in all patients.

Post operative period was monitored; intake output charts and vital charts were maintained.

Drains were removed after 48 hours with output less than 30ml. Sutures were removed on the 7^{th} post operative day.

The patients were followed up for a variable period of time.

OBSERVATION AND RESULTS

TABLE no.1 – SITE OF PERFORATION

S.no	Site of Perforation	Frequency	Percent	Valid Percent	Cumulative Percent
1.	Duodenum	63	63.0	63.0	63.0
2.	Appendix	22	22.0	22.0	85.0
3.	Gastric	7	7.0	7.0	92.0
4.	ileum	4	4.0	4.0	96.0
5.	Colon	3	3.0	3.0	99.0
6.	jejunum	1	1.0	1.0	100.0
	Total	100	100.0	100.0	

In the study population of 100 subjects, duodenal perforation was seen in 63% of patients, followed by appendicular (22%),gastric (7%) , ileal(4%), Colon(3%) and jejunal(1%) perforation .

Chart no.1 – SITE OF PERFORATION

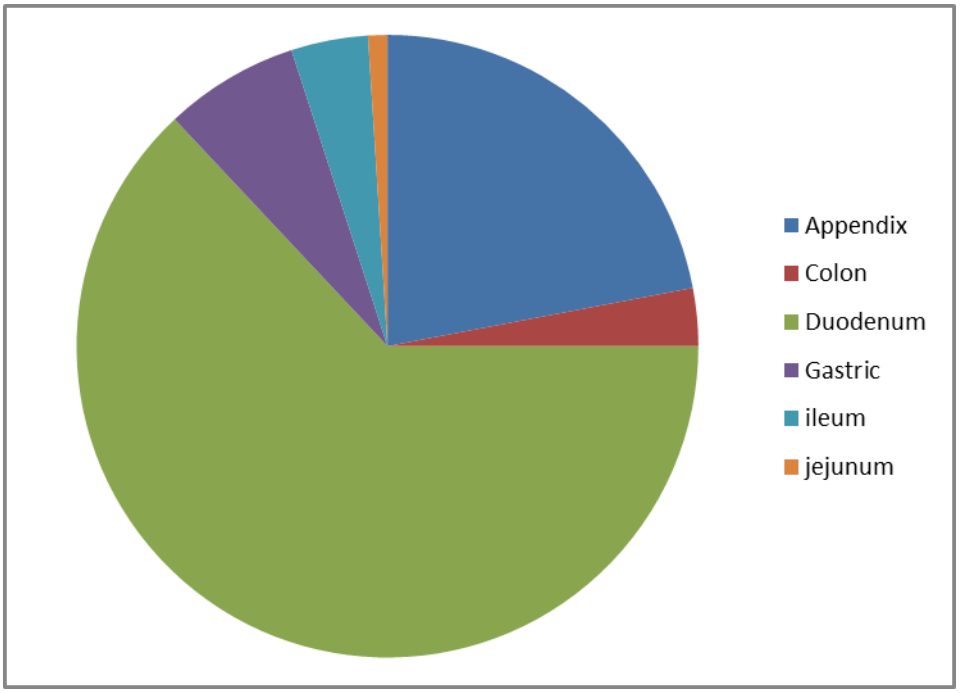


Table no .2. AGE and MPI cross tabulation

			MPI			
			<21	21-29	>29	Total
AGE	Not more than 50	Count	49	8	2	59
		% within AGE	83.1%	13.6%	3.4%	100.0%
		% within MPI	98.0%	21.1%	16.7%	59.0%
		% of Total	49.0%	8.0%	2.0%	59.0%
	More than 50	Count	1	30	10	41
		% within AGE	2.4%	73.2%	24.4%	100.0%
		% within MPI	2.0%	78.9%	83.3%	41.0%
		% of Total	1.0%	30.0%	10.0%	41.0%
Total		Count	50	38	12	100
		% within AGE	50.0%	38.0%	12.0%	100.0%
		% within MPI	100.0%	100.0%	100.0%	100.0%
		% of Total	50.0%	38.0%	12.0%	100.0%

In the total study population, among patients younger than 50 years of age 83% had MPI < 21 13.6% had MPI 21-29 and 3.4% had MPI >29 and among patients older than 50 years of age 2.4% had MPI <21 73.2% had MPI 21-29 and 24.4% had MPI >29.

Chart no .2. AGE and MPI Bar chart

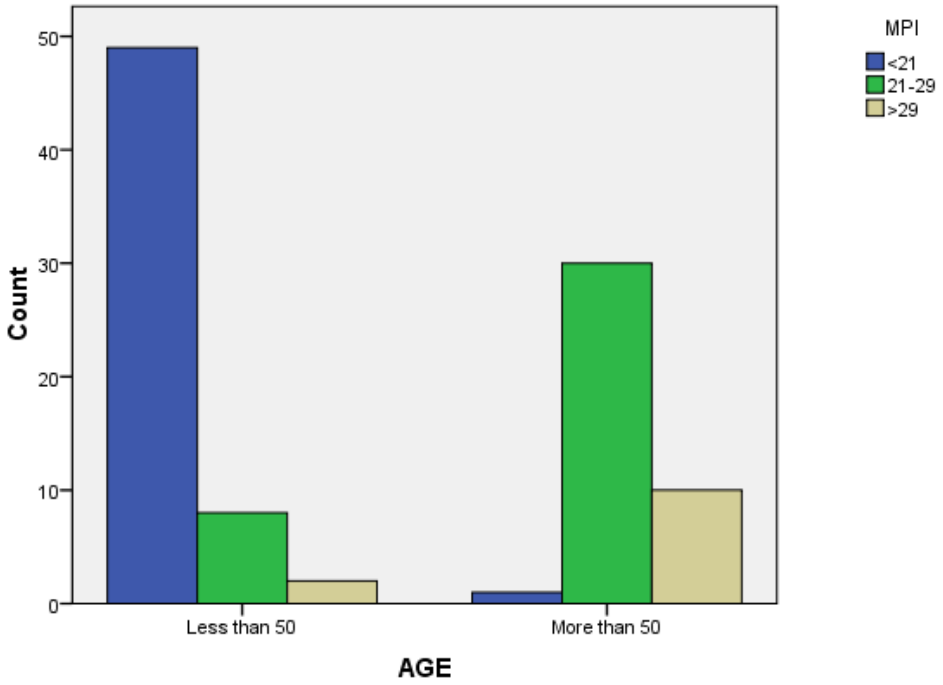


Table no.3. AGE(yrs) Statistics

N value	100
Mean	44.89
Median	43.50
Range	63
Minimum	16
Maximum	79
Std Deviation	16.201

Table no.4. SEX and MPI Cross tabulation

			MPI			
			<21	21-29	>29	Total
SEX	Male	Count	50	37	11	98
		% within SEX	51.0%	37.8%	11.2%	100.0%
		% within MPI	100.0%	97.4%	91.7%	98.0%
		% of Total	50.0%	37.0%	11.0%	98.0%
	Female	Count	0	1	1	2
		% within SEX	.0%	50.0%	50.0%	100.0%
		% within MPI	.0%	2.6%	8.3%	2.0%
		% of Total	.0%	1.0%	1.0%	2.0%
Total		Count	50	38	12	100
		% within SEX	50.0%	38.0%	12.0%	100.0%
		% within MPI	100.0%	100.0%	100.0%	100.0%
		% of Total	50.0%	38.0%	12.0%	100.0%

Among the males in the study population, 51% had MPI<21, 37.8% MPI 21-29 and 11.2% >29% and among the females 50% had MPI 21-29 and 50% had MPI >29.

Chart no.3. SEX and MPI Bar Chart

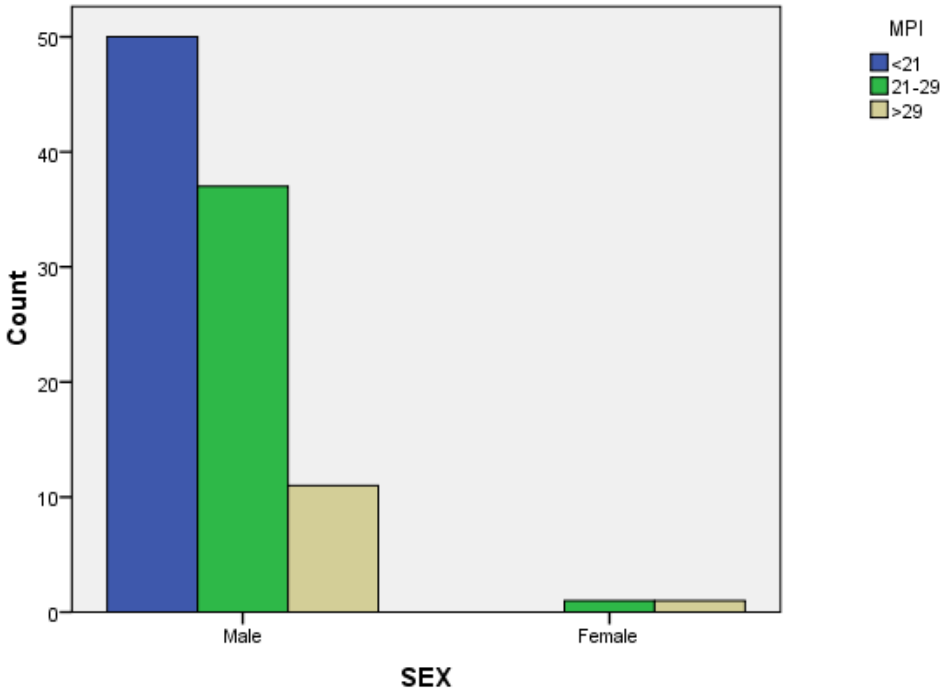


Table no.5 ORGAN FAILURE and MPI Cross tabulation

			MPI			Total
			<21	21-29	>29	
ORGAN FAILURE	No	Count	50	33	0	83
		% within ORGAN FAILURE	60.2%	39.8%	.0%	100.0%
		% within MPI	100.0%	86.8%	.0%	83.0%
		% of Total	50.0%	33.0%	.0%	83.0%
	Yes	Count	0	5	12	17
		% within ORGAN FAILURE	.0%	29.4%	70.6%	100.0%
		% within MPI	.0%	13.2%	100.0%	17.0%
		% of Total	.0%	5.0%	12.0%	17.0%
Total		Count	50	38	12	100
		% within ORGAN FAILURE	50.0%	38.0%	12.0%	100.0%
		% within MPI	100.0%	100.0%	100.0%	100.0%
		% of Total	50.0%	38.0%	12.0%	100.0%

Among those without organ failure, 60.2% had MPI <21, 39.8% had MPI 21-29, none had MPI >29 and those with organ failure, none had MPI <21, 29.4% had MPI 21-29, and 70.6% had MPI >29.

Chart no.4. ORGAN FAILURE and MPI Bar Chart

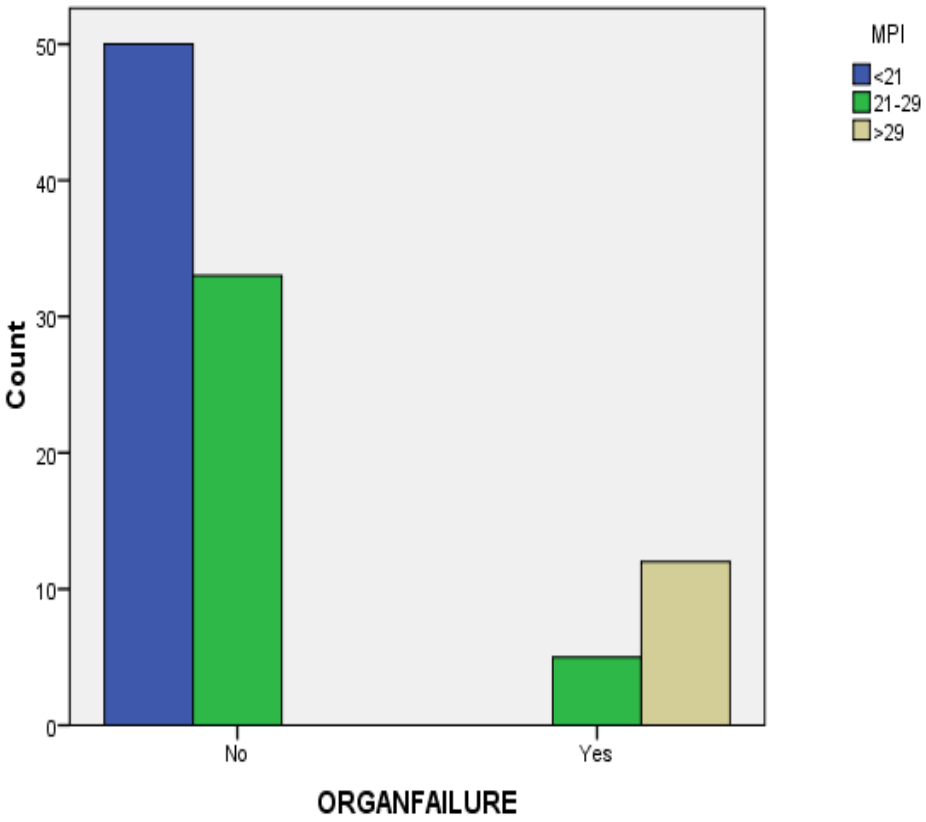


Table no.6. DURATION OF PERITONITIS and MPI Cross tabulation

			MPI			Total
			<21	21-29	>29	
DURATION OF PERITONITIS	Not more than 24 hrs	Count	4	4	0	8
		% within DURATION OF PERITONITIS	50.0%	50.0%	.0%	100.0%
		% within MPI	8.0%	10.5%	.0%	8.0%
		% of Total	4.0%	4.0%	.0%	8.0%
	More than 24hrs	Count	46	34	12	92
		% within DURATION OF PERITONITIS	50.0%	37.0%	13.0%	100.0%
		% within MPI	92.0%	89.5%	100.0%	92.0%
		% of Total	46.0%	34.0%	12.0%	92.0%
Total		Count	50	38	12	100
		% within DURATION OF PERITONITIS	50.0%	38.0%	12.0%	100.0%
		% within MPI	100.0%	100.0%	100.0%	100.0%
		% of Total	50.0%	38.0%	12.0%	100.0%

Among those with peritonitis duration < 24 hours, 50% had MPI <20 and 50% had MPI 21-29 and those with duration >24 hours, 50% had MPI <20, 37% had MPI 21-29 and 13% had MPI >29.

Chart no.5. DURATION OF PERITONITIS and MPI Bar Chart

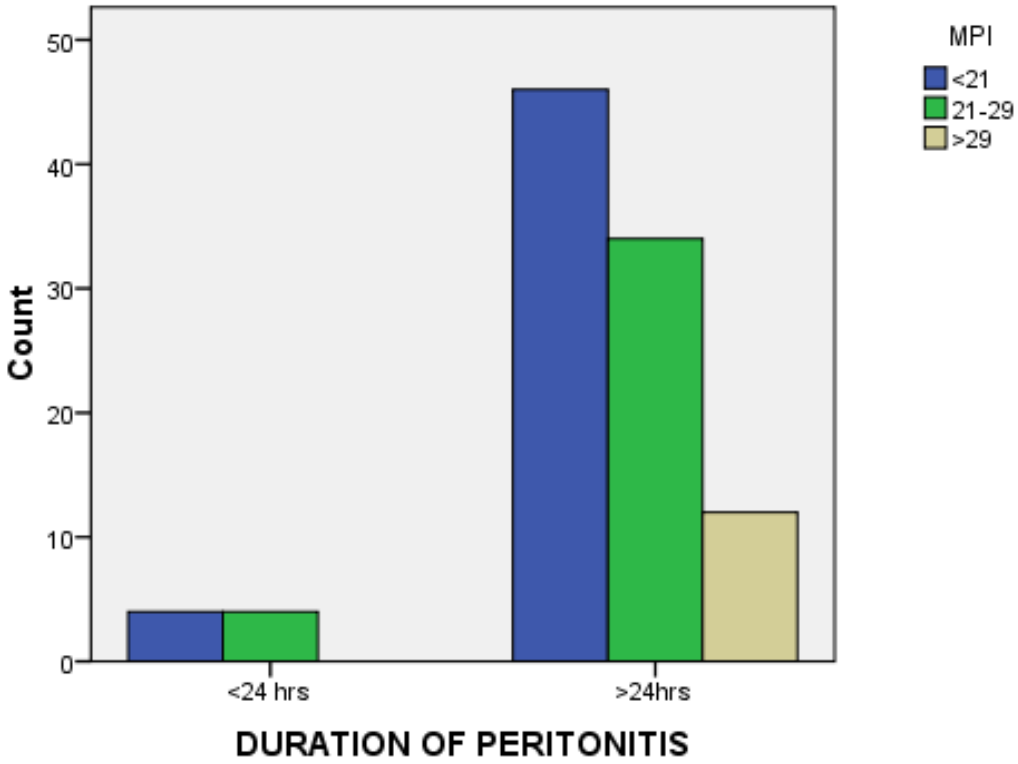


Table no.7.SITE OF PATHOLOGY and MPI Cross tabulation

			MPI			Total
			<21	21-29	>29	
SITE OF PATHOLOGY	Colonic	Count	0	1	2	3
		% within SITE OF PATHOLOGY	.0%	33.3%	66.7%	100.0%
		% within MPI	.0%	2.6%	16.7%	3.0%
		% of Total	.0%	1.0%	2.0%	3.0%
	Non Colonic	Count	50	37	10	97
		% within SITE OF PATHOLOGY	51.5%	38.1%	10.3%	100.0%
		% within MPI	100.0%	97.4%	83.3%	97.0%
		% of Total	50.0%	37.0%	10.0%	97.0%
Total		Count	50	38	12	100
		% within SITE OF PATHOLOGY	50.0%	38.0%	12.0%	100.0%
		% within MPI	100.0%	100.0%	100.0%	100.0%
		% of Total	50.0%	38.0%	12.0%	100.0%

Among those with colonic pathology none had MPI <20 and 33.3% had MPI 21-29 and 66.7% had MPI >29 and non colonic pathology 51.5% had MPI <20 and 38.1% had MPI 21-29 and 10.3% had MPI >29.

Chart no.6.SITE OF PATHOLOGY and MPI Bar Chart

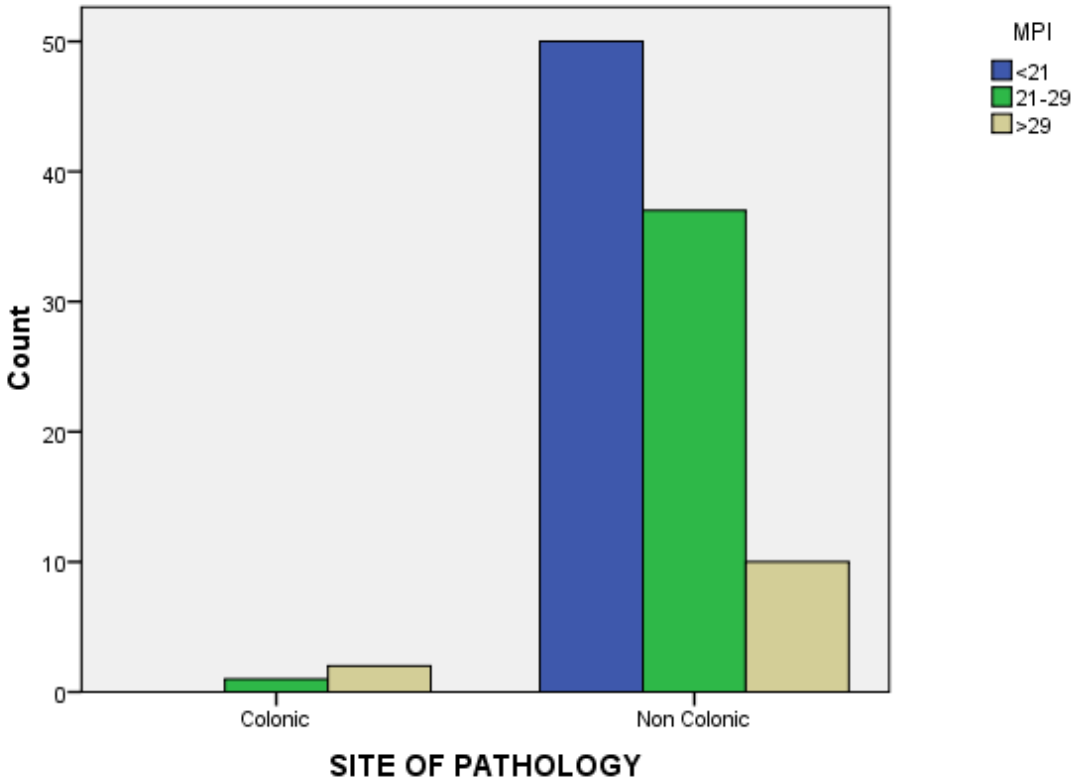


Table no.8. NATURE OF PATHOLOGY and MPI Cross tabulation

			MPI			
			<21	21-29	>29	Total
NATURE OF PATHOLOGY	Benign	Count	50	36	11	97
		% within NATURE OF PATHOLOGY	51.5%	37.1%	11.3%	100.0%
		% within MPI	100.0%	94.7%	91.7%	97.0%
		% of Total	50.0%	36.0%	11.0%	97.0%
	Malignant	Count	0	2	1	3
		% within NATURE OF PATHOLOGY	.0%	66.7%	33.3%	100.0%
		% within MPI	.0%	5.3%	8.3%	3.0%
		% of Total	.0%	2.0%	1.0%	3.0%
Total		Count	50	38	12	100
		% within NATURE OF PATHOLOGY	50.0%	38.0%	12.0%	100.0%
		% within MPI	100.0%	100.0%	100.0%	100.0%
		% of Total	50.0%	38.0%	12.0%	100.0%

Among those with benign pathology 51.5% had MPI <21 and 37.1% had MPI 21-29 and 11.3% had MPI >29 and malignant pathology none had MPI <21 and 66.7% had MPI 21-29 and 33.3% had MPI >29.

Chart no.7. NATURE OF PATHOLOGY and MPI Bar Chart

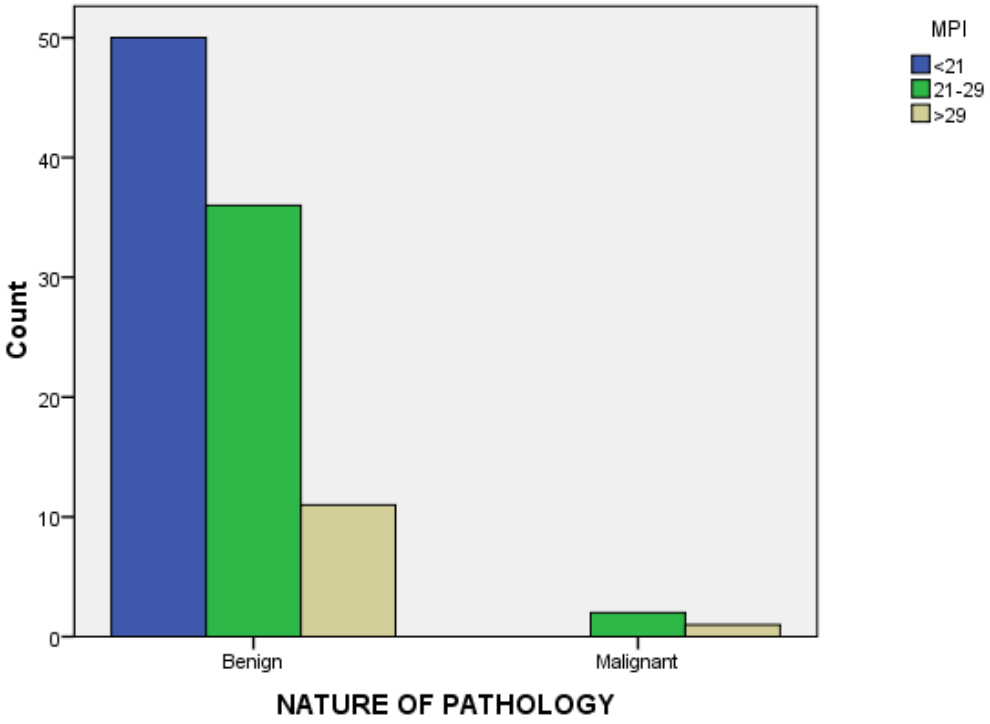


Table no.9. PERITONITIS and MPI Cross tabulation

			MPI			
			<21	21-29	>29	Total
PERITONITIS	Localised	Count	6	0	0	6
		% within PERITONITIS	100.0%	.0%	.0%	100.0%
		% within MPI	12.0%	.0%	.0%	6.0%
		% of Total	6.0%	.0%	.0%	6.0%
	Generalised	Count	44	38	12	94
		% within PERITONITIS	46.8%	40.4%	12.8%	100.0%
		% within MPI	88.0%	100.0%	100.0%	94.0%
		% of Total	44.0%	38.0%	12.0%	94.0%
Total		Count	50	38	12	100
		% within PERITONITIS	50.0%	38.0%	12.0%	100.0%
		% within MPI	100.0%	100.0%	100.0%	100.0%
		% of Total	50.0%	38.0%	12.0%	100.0%

Among those with localized peritonitis, 100% had MPI <21 and those with generalised peritonitis 46.8% had MPI <21 40.4% had MPI 21-29 and 12.8% had MPI >29.

Chart no.8. PERITONITIS and MPI Bar Chart

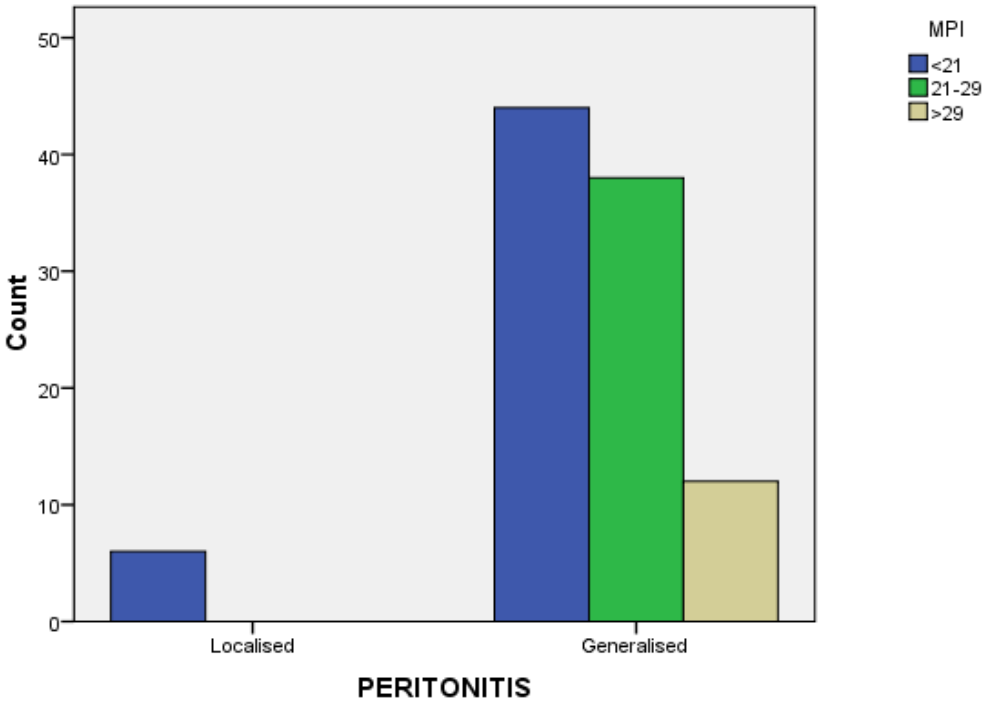


Table no.10. NATURE OF EXUDATE and MPI Cross tabulation

			MPI			Total
			<21	21-29	>29	
NATURE OF EXUDATE	Cloudy, Purulent	Count	50	37	9	96
		% within NATURE OF EXUDATE	52.1%	38.5%	9.4%	100.0%
		% within MPI	100.0%	97.4%	75.0%	96.0%
		% of Total	50.0%	37.0%	9.0%	96.0%
	Faeculent	Count	0	1	3	4
		% within NATURE OF EXUDATE	.0%	25.0%	75.0%	100.0%
		% within MPI	.0%	2.6%	25.0%	4.0%
		% of Total	.0%	1.0%	3.0%	4.0%
Total		Count	50	38	12	100
		% within NATURE OF EXUDATE	50.0%	38.0%	12.0%	100.0%
		% within MPI	100.0%	100.0%	100.0%	100.0%
		% of Total	50.0%	38.0%	12.0%	100.0%

Among those with cloudy, purulent exudates 52.1% had MPI <21, 38.5% had MPI 21-29 and 9.4% had MPI >29 and those with faeculent exudates none had MPI <21, 25% had MPI 21-29 and 75% had MPI >29.

Chart no.9. NATURE OF EXUDATE and MPI Bar chart

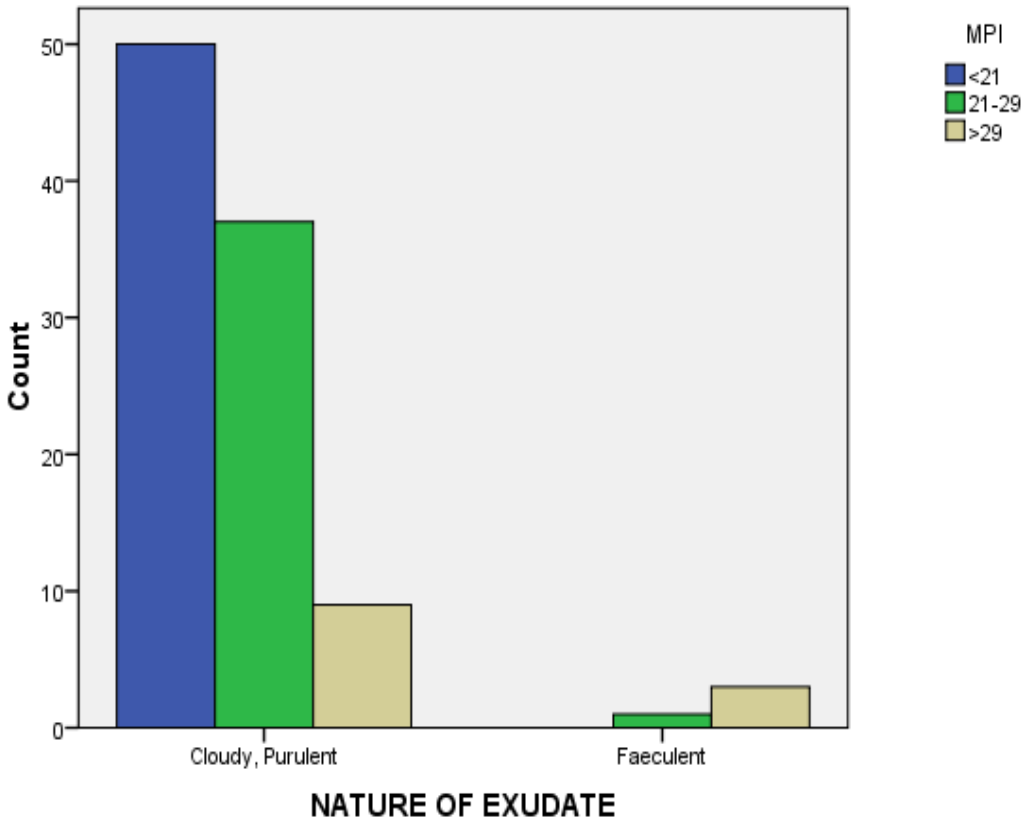


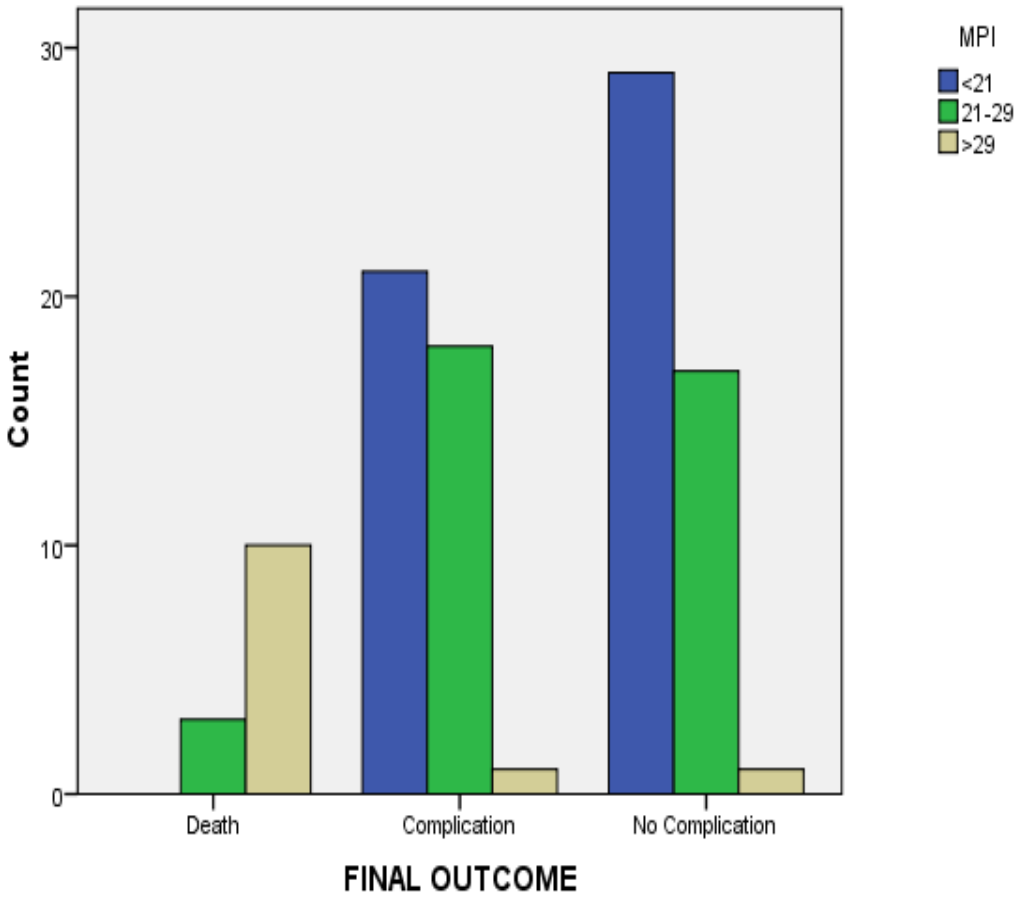
Table no.11. FINAL OUTCOME and MPI Cross tabulation

			MPI			
			<21	21-29	>29	Total
FINAL OUTCOME	Death	Count	0	3	10	13
		% within FINAL OUTCOME	.0%	23.1%	76.9%	100.0%
		% within MPI	.0%	7.9%	83.3%	13.0%
		% of Total	.0%	3.0%	10.0%	13.0%
	Complication	Count	21	18	1	40
		% within FINAL OUTCOME	52.5%	45.0%	2.5%	100.0%
		% within MPI	42.0%	47.4%	8.3%	40.0%
		% of Total	21.0%	18.0%	1.0%	40.0%
	No Complication	Count	29	17	1	47
		% within FINAL OUTCOME	61.7%	36.2%	2.1%	100.0%
		% within MPI	58.0%	44.7%	8.3%	47.0%
		% of Total	29.0%	17.0%	1.0%	47.0%
Total		Count	50	38	12	100
		% within FINAL OUTCOME	50.0%	38.0%	12.0%	100.0%
		% within MPI	100.0%	100.0%	100.0%	100.0%
		% of Total	50.0%	38.0%	12.0%	100.0%

Pearson Chi-Square value - 61.64. p value – 0.0001

Among the total population, 40% had complications, 47% had no complications and 13% had expired. Amongst those who expired there was no patient with MPI <21 23.1% had MPI 21-29 and 76.9% had MPI > 29. Amongst those who had complications 52.5 % had MPI <21, 45% had MPI 21-29, 2.5% had MPI >29. Amongst those without complications 61.7% had MPI <21, 36.2% had MPI 21-29, and 2.1% had MPI >29.

Chart No.10. FINAL OUTCOME and MPI Bar chart



DISCUSSION

Peritonitis remains a hot spot for the surgeons despite advancements in surgical technique and intensive care treatment. Various factors like age, sex, duration, site of perforation, extent of peritonitis and delay in surgical intervention are associated with morbidity and mortality. A successful outcome depends upon early surgical intervention, source control and exclusive intraoperative peritoneal lavage. Also various methods and scoring systems are used to identify the risks and morbidity and mortality in those patients.

In the present study, hundred cases of peritonitis those attended RGGGH emergency department from April 2014 to September 2014 were included with age ranging from 16 to 79 years. . The mean age of the patients was 44.89 (SD 16.2) years. There was male preponderance (98%) in this study and the most common etiology of peritonitis was duodenal perforation seen in 63% of patients, followed by appendicular perforation (22%),gastric (7%), ileal(4%), Colon(3%) and jejunal perforation(1%).

Most patients presented with history of abdominal pain, abdominal distension and fever with varying duration, most (92%) presenting after 24 hours of onset of symptoms. MPI scoring system done in all patients depending on preoperative and intra-operative

finding and patients were categorized into three categories those <21, 21 to 29, >29. Majority(50%) of patients had MPI less than 21. 52.5% of patients with MPI score less than 21 developed complications. 45% of patients had complications with MPI score 21-27. Complications include minor(wound infection) and major(Respiratory, Renal, Circulatory,Post operative leak) categories. There was no mortality in patients with MPI less than 21, whereas those patients with MPI score more than 29 had the highest mortality rate of 76.9%. Patient with MPI score with from 21 to 29 had mortality rate of 23.1%. The outcome of the study is statistically significant by chi-square test with p Value <0.0001. This study is compared to available literature and other studies.

R Függer, M Rogy, F Herbst, M Schemper, F Schulz. 113 patients suffering from purulent peritonitis entered this retrospective study for evaluation of the prognostic value of the Mannheim Peritonitis-Index. There was no lethality below an index x = 21, between x = 21 and x = 29, it was 29% and lethality increased to 100% in patients with an index x greater than or equal to 30. Statistical validation showed that prognosis was correct in 93% for the index x = 27, with a sensitivity and specificity of also 93%. Between x = 21 and x = 29 prognosis of the MPI was correct in at least 65%. The MPI is

shown as a prognostic index for peritonitis with high accuracy in individual prognosis, that could be easy routinely documented.[38]

A S Ermolov, V E Bagdat'ev, E V Chudotvortseva, A V Rozhnov. A retrospective analysis of 100 case histories of patients with diffuse peritonitis was made in order to evaluate the prognostic significance of the Mannheim Peritonitis Index (MPI). The patients were divided into 3 groups according to the amount of scores: in the first group (12-20 scores) there were no lethal issues, in the second group (21-29 scores) 42% of the patients died, 100% lethality was noted in the third group when MPI was 30 scores or more. [39]

Kusumoto yoshiko and nakagawa masayuki et al. evaluated the reliability of the Mannheim Peritonitis Index (MPI) in predicting the outcome of patients with peritonitis. Method: Subjects were 108 patients operated on for intraabdominal infection and excluded subjects with appendicitis. Results: Overall mortality was 5.3% in men and 15.2% in women, with death occurring only in patients older than 50 years. A comparison of MPI and mortality showed patients with a MPI score of 26 or less to have mortality of 3.8%, where as those with a score exceeding 26 had mortality of 41.0%.[40]

Qureshi AM, Zafar A, Saeed K, Quddus A. et.al. One hundred and twenty-six patients who presented to the department with secondary peritonitis were included in the study. Mortality rate for MPI score > or = 26 was 28.1% while for scores less than 26 it was 4.3%. For MPI scores pound 20 mortality rate was 1.9%, for scores 21-29 it was 21.9% and for score 30 or more it was 28.1%. Chi-square showed significant association between mortality and increasing MPI score ($p < 0.01$). Odd ratios calculated were significant for age > 50 years, malignancy, organ failure, pre-operative duration of peritonitis > 24 hours and cloudy, purulent exudate.[43]

CONCLUSION

- Peritonitis remains a hot spot for the surgeons despite advancements in surgical technique and intensive care treatment. Various factors like age, sex, duration, site of perforation, extent of peritonitis and delay in surgical intervention are associated with morbidity and mortality.
- Duodenal perforation is the most common etiology of peritonitis followed by appendicular perforation, gastric, ileal, Colon and jejunal perforation in this study.
- Males are commonly affected compared to females in this study.
- Emergency laparotomy and primary repair of the hollow viscus perforation is more effective in patients with secondary and tertiary peritonitis.
- In the management of patients with generalized peritonitis, scoring the patients into various risk groups can be beneficial.
- MPI scoring system is easy score to apply, the determination of risk is available during operation and surgeon can know about the possible outcome and the appropriate management can be decided.
- MPI is more effective in predicting the mortality in peritonitis due to hollow viscous perforation.

FIG 4: PRE-OPERATIVE PHOTOGRAPH OF A PERITONITIS PATIENT

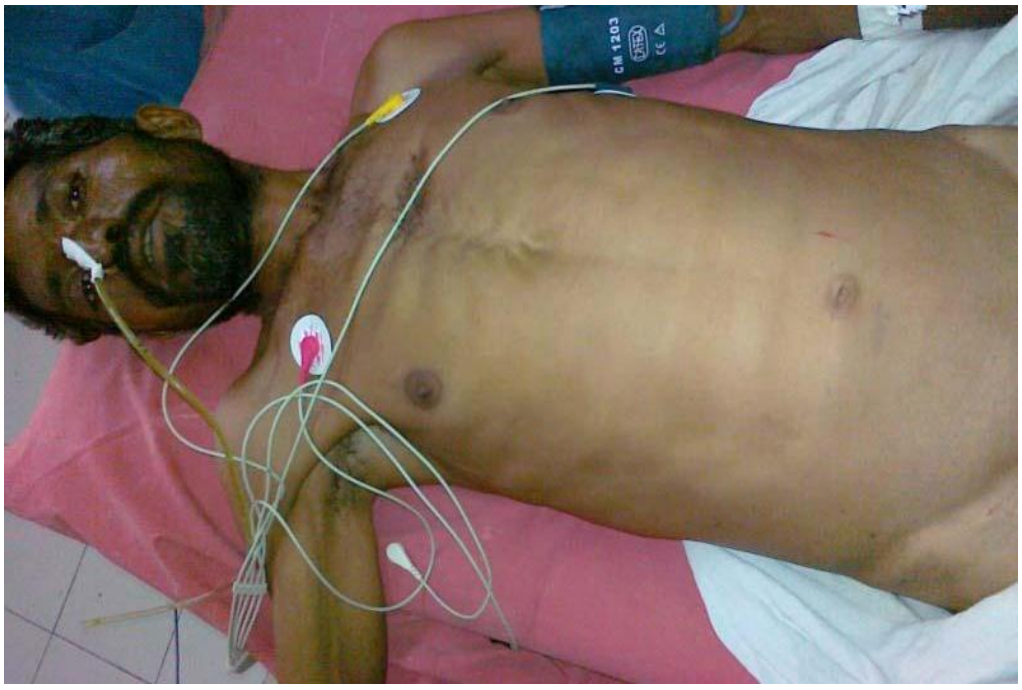


FIG 5: PLAIN RADIOGRAPH PHOTO OF PERITONITIS PATIENT

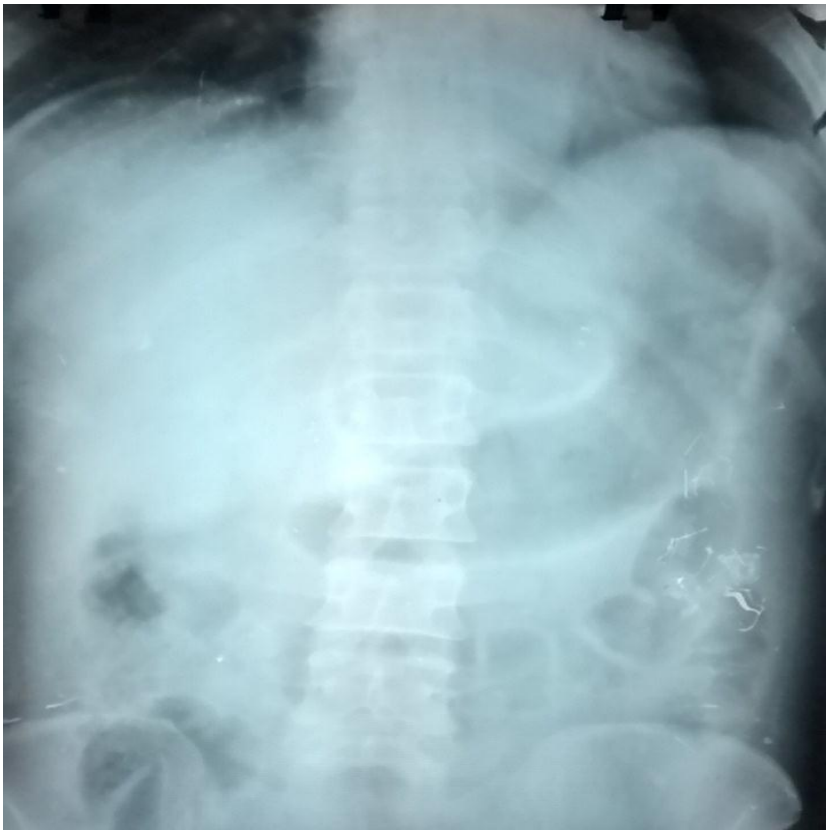


FIG 6: INTRA-OP PHOTO OF DUODENAL PERFORATION

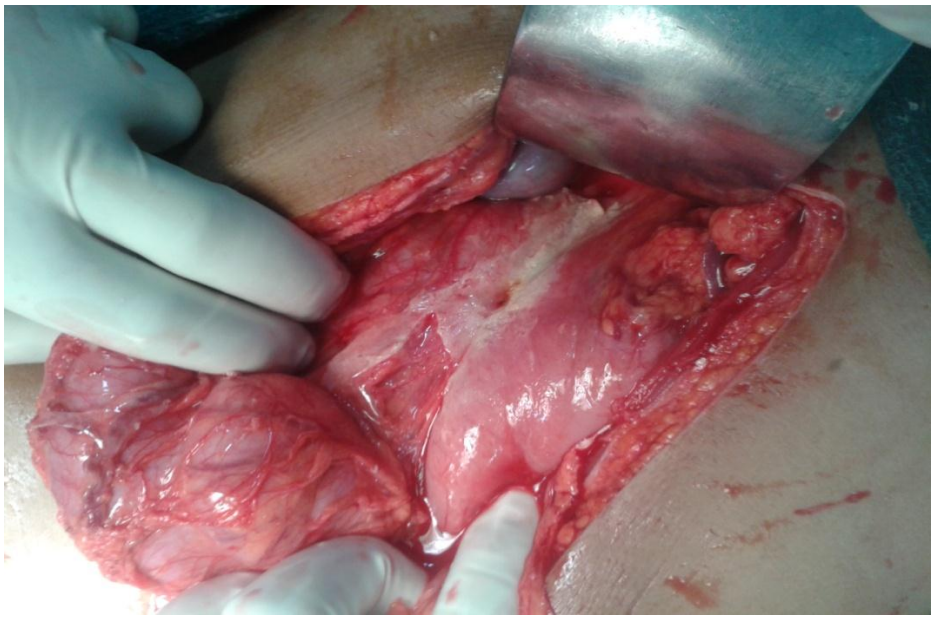


FIG 7: INTRA-OP PHOTO OF CLOSURE OF DUODENAL PERFORATION

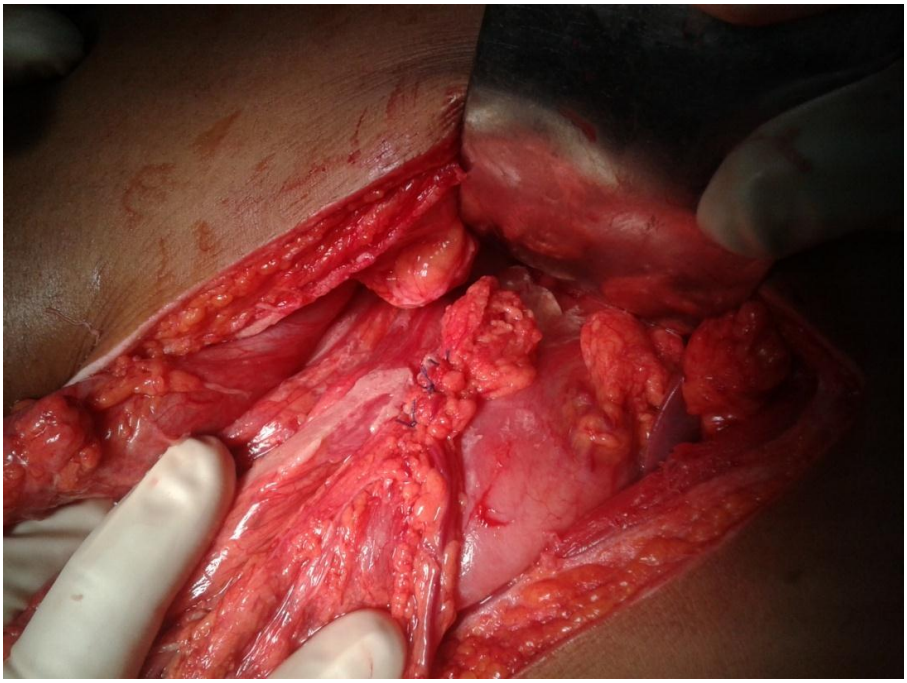


FIG 8: POST-OP PHOTO OF WOUND INFECTION

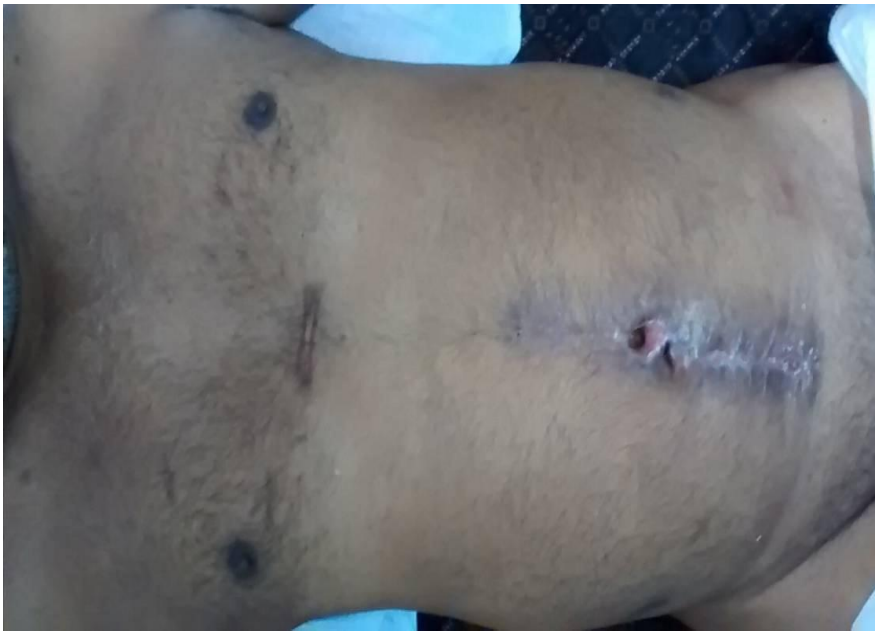
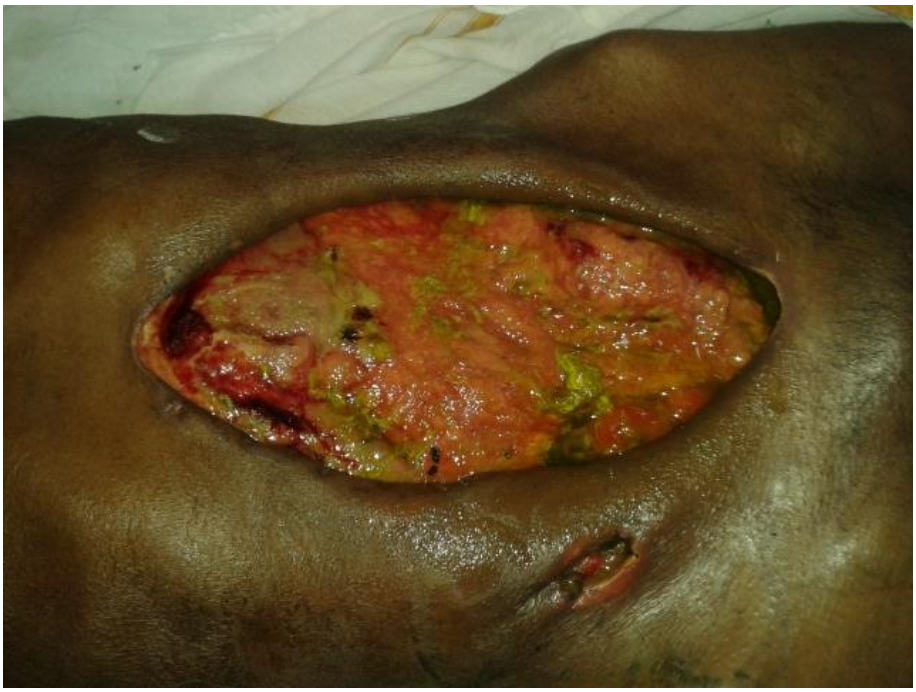


FIG 9: POST-OP PHOTO OF ENTERO CUTANEOUS FISTULA



BIBLIOGRAPHY

1. Billing A, Frohlich D, Schildberg F.W., Prediction of outcome using the Mannheim peritonitis index in 2003 patients. Peritonitis study group. Br. J. Surg 1994 Feb: 81(2): 209-13.

2. Delinger P.E. et al. Surgical infection stratification system for intra-abdominal infection. Arch. Surg. 1985 Jan; 120:21.

3. Pacelli F, et al. Prognosis in intra-abdominal infectins. Multivariate analysis on 604 patients. Arch Surg. 1996 June; 131(6): 641-5

4. Durham H. The mechanism of reaction to peritoneal infection.J. Pathol. Bacteriol. 1897;4:338-82.

5. MelaneyF.L. Olip J, et al. Peritonitis: II. Synergism of bacteria commonly found in peritoneal exudates. Arch Surg. 1932;25:709.

6. Fry D.E. Garrison R.N. et al. Determinants of death in patients with intra-abdominal abscess. Surgery.1980;88:517.

7. Elebute E.A., Stoner H.B. The grading of sepsis.Br. J. Surg. 1983;70:29-31.

8. Pine R.W. Wertz M.J. et. Al. Determinants of organ malfunction or death in patients with intra-abdominal sepsis. Arch Surg. 1983; 118:242-249.

9. Stevens L.E. Gauging the severity of surgical sepsis. Arch Surg.1983;118:1190-1192.

10. KnausW.A , Draper E.A, Wagner D.P. et al. Prognosis in acute organ – system failure. Ann. Surg. 1985;202:685-693.

11. Teichmann W. Wittmann D.H, et al. Scheduled reoperations (ettappenlavage) for diffuse peritonitis. Arch. Surg. 1986; 121: 147-152.

12. Wach. H, Linder M.M, et al. Mannheim peritonitis index – prediction of risk of death from peritonitis; construction of a static and validation of an empirically based index. Theoritical Surgery 1987; 1: 169-77.

13. Kohli V, et al. Evaluation of prognostic factors in perforated peptic ulcer. Indian Journal of Surgery. 1988 May-June; 50:184.

14. Verma G.R. et al. Gastro-intestinal injuries in abdominal trauma. Trop Gastroenterol 1990 Oct-Dec; 11(4):206-10.

15. Demnel N. Muth G., Maag K., Osterholzer G. Prognostic scores in peritonitis: the Mannheim peritonitis index or APACHE II? Langenbecks Arch Chir 1994; 379(6):347-52.

16. Svanes C, et al. Adverse effects of delayed treatment for perforated peptic ulcer. Ann. Surg. 1994 August;220(2):168.

17. Autio V. The spread of intraperitoneal infection.Acta ChirSc and Suppl 1981; 91-98.

18. Inderbir Singh, Human Embryology. 6t h edit ion.

19. Dietmer H Whittmann, Alonzo P. Walker and Robert E. London. Peritonitis and intra abdominal infection.Principles of Surgery by Schwartz. Page No. 1449–1480. 6th ed, McGraw Hill, 1999.

20. Peter L. Williams & Roger Warwick. Gray Anatomy. 36t h edit ion. Splanchnology. Pg. 1332, Churchill Livingstone 1980.

21. Michael J Zinner, Robert S. Bennion. Peritonit is and Intraabdomina l abscess. Maingot's abdominal operat ions. 10th ed, page no.633– 650, Appleton and Lange A Simon and Schuster Company.1997.

22. Christopher J Bulstrode and R.C.G Russel, Jermy Thompson. The peritoneum, omentum, mesentry and retroperitoneal space. Bailey and Love's Short Practice of Surgery. Page No. 1133 –1152, 24t h edit ion, 2004.

23. Dominioni L, Dionigi R, Zanello M, et al. Sepsis score and acute-phase proteinresponse as predictors of outcome in septic surgical patients. Arch Surg 1987;122:141-146.

24. Ohmann C, Hau T, et al. Prognostic modeling in peritonitis. Peritonitis study group of the surgical infection society, Europe. Eur. J. Surg. 1997 Jan; 163(1) : 53-60. 1999.

25. Dumont A.K, Mass W.K, et al. Increased survival from peritonitis after blockade of trans-diaphragmatic absorption of bacteria. Surg. Gynecol Obstet. 1966; 162:248.

26. Liyod M. Nyhus. Robert J Baker. Mastery of Surgery, 3r d edit ion, Page No. 146 – 152.

27. Alt emeierW.A : The bacterial flora of acute perforated appendicitis withperitonit is. A bacteriological study based upon 1000 cases. *Ann Surg*1938 ; 107 : 517 – 528.

28. Whiteside, Tytherleigh, Thrush, Farouk, Galland, et al. Intraoperative peritoneal lavage – who does it and why ? *Annals of Royal College of Surgeons of England*.87(4): 255 – 258, July2005.

29. Michael J Zinner. Complicat ion of peptic ulcer. Maingot's abdominal operations. 10th edition, page no. 981-994, Appleton and Lange a Simon and Schuster Company 1997.

30. Peter J. Morris and William C. Wood, Cristopher S. Grant, William J.K. Huizinga and A.S. Daar. Typhoid fever and salmonella infection. Oxfort Text Book of Surgery. 2nd ed, Vol.3, Pg.3231 Oxford University Press 2000.

31. Christopher J Bulstrode and R.C.G Russel, Jermy Thompson. The peritoneum, omentum, mesentry and retroperitoneal space. Bailey and Love's Short Practice of Surgery. Page No.1143– 1144, 24t h edit ion, 2004.

32. Dietmer H Whittmann, Robert E. Rogers and Gregory P. Sutton. Gynaecology.Principles of Surgery by Schwartz. Pg.1804. 6t h ed. McGraw Hill

33. Ohmann C, Wittmann DH, Wacha H. Prospective evaluation of prognostic scoring systems in peritonitis. *Eur J Surg* 1993 159: 267-74.

34. Kachroo R, Ahmad MN, Zargar HU. Peritonitis – an analysis of 90 cases. *Indian J Surg* 1984; 46: 204-9.

35. Ermolov AS, Bagdat'ev VE, Chudotvortseva EV, Rozhnov AV. Evaluation of the Mannheim Peritonitis Index. [Article in Russian] Vestn Khir Im I I Grek.1996;155:22-3. [Abstract]

36. Függer R, Rogy M, Herbst F, Schemper M, Schulz F. Vali dation study of the Mannheim Peritonitis Index. [Article in German] Chirurg 1988;59:598-601. [Abstract]

37. Notash AY, Salimi J, Rahimian H, Fesharaki MH, Abbasi A. Evaluation of Mannheim peritonitis index and multiple organ failure score in patients with peritonitis. Indian J Gastroenterol 2005;24:197-200.

38. Der Chirurg Zeitschrift fur alle Gebiete der operativen Medizen (1988) Volume:59, Issue: 9, Pages: 598-601

39. A S Ermolov, V E Bagdat'ev, E V Chudotvortseva, A V Rozhnov Evaluation of the Mannheim Peritonitis Index Vestnik Khirurgii Imeni I I Grekova (1996) Volume: 155, Issue: 3, Pages: 22-23

40. Kusumoto yoshiko and nakagawa masayuki et al. Study of Mannheim Peritonitis Index to Predict Outcome of Patients with Peritonitis. Japanese Journal of Gastroenterological Surgery. VOL.37;NO.1;PAGE.7-13(2004)

41. Demmel N, et al. prognostic scores in peritonitis : the mannheim peritonitis index or APACHE II? Langenbecks arch chir. 1994;379(6): 347-52.

42. Gedik E, Girgin S, Taçyildiz IH, Akgün Y et.al. Risk factors affecting morbidity in typhoid enteric perforation. Langenbecks Arch Surg. 2008 Nov;393(6):973-7. Epub 2007 Nov 20.abstract.

43. Qureshi AM, Zafar A, Saeed K, Quddus A et. al. Predictive power of Mannheim Peritonitis Index. J Coll Physicians Surg Pak. 2005 Nov;15(11):693-6.abstract.

44. Mulari K, Leppäniemi A et. al. Severe secondary peritonitis following gastrointestinal tract perforation. Scand J Surg. 2004;93(3):204-8. abstract.

45. Kologlu M, Elker D, Altun H, Sayek I. et. al. Validation of MPI and PIA II in two different groups of patients with secondary peritonitis. Hepatogastroenterology. 2001 Jan-Feb;48(37):147-51.abstract.

46. Liverani A, Correnti SF, Paganelli MT, Antonini G, Mercati U et.al. Mannhein index in the prognosis and treatment of acute peritonitis Minerva Chir. 1998 May;53(5):385-9. abstract.

47. Bosscha K, Reijnders K, Hulstaert PF, Algra A, van der Werken C. et. al. Prognostic scoring systems to predict outcome in peritonitis and intra-abdominal sepsis. Br J Surg. 1997 Nov;84(11):1532-4. abstract.

48. Ermolov AS, Bagdat'ev VE, Chudotvortseva EV, Rozhnov AV.et. al. [Evaluation of the Mannheim Peritonitis Index]. Vestn Khir Im I I Grek. 1996;155(3):22-3.Abstract

49. Rogy M, Függer R, S3chemper M, Koss G, Schulz F. et,al . [The value of 2 distinct prognosis scores in patients with peritonitis. The Mannheim Peritonitis Index versus the Apache II score]. Chirurg. 1990 Apr;61(4):297- 300. Abstract

50. Barrera Melgarejo E, Rodríguez Castro M, Borda Luque G, Najar Trujillo N. et.al. [Predictive mortality value of the peritonitis index of Mannheim].Rev Gastroenterol Peru. 2010 Jul-Sep;30(3):211-5. Abstract

51. Bracho-Riquelme RL, Reyes-Romero MA, Torres-Valenzuela A, Flores-García AI. et.al. The grade-response relation between severity of peritonitis and serum cytokine concentrations explains Mannheim Peritonitis Index threshold. . Surg Infect (Larchmt). 2010 Aug;11(4):379-86. Abstract

52. Malik AA, Wani KA, Dar LA, Wani MA, Wani RA, Parray FQ. Et.al. Mannheim Peritonitis Index and APACHE II--prediction of outcome in patients with peritonitis. Ulus Travma Acil Cerrahi Derg. 2010 Jan;16(1):27-32. Abstract

53. Tan KK, Bang SL, Sim R.et.al. Surgery for small bowel perforation in an Asian population: predictors of morbidity and mortality. J Gastrointest Surg. 2010 Mar;14(3):493-9. Epub 2009 Dec 9. Abstract

54. Abrar Maqbool Qureshi; Afsheen Zafar; Khurram Saeed ; Predictive power of Mannheim Peritonitis Index. Department of General Surgery, Griffith Base Hospital, Noorebar Avenue, Griffith, Australia. Journal of the College of Physicians and Surgeons--Pakistan : JCPSP Volume: 15 ISSN: 1022-386X ISO Abbreviation: J Coll Physicians Surg Pak, 2005 NoV.

<u>ஆராய்ச்சி ஒப்புதல் படிவம்</u>

<u>ஆராய்ச்சி தலைப்பு</u>

குடலில் ஓட்டை ஏற்பட்டு பாதிப்புக்குள்ளாகும் நோயாளிகளின் ஆரோக்கியம் குன்றிய நிலை மற்றும் உயிரிழப்பு முடிவினை தீர்மானிக்கும் காரணிகளை மேன்ஹிம் குடல் சவ்வு அழற்சி அளவீட்டைக் கொண்டு முன்னறிவது பற்றிய ஆய்வு

பெயர்	:	தேதி	:
வயது	:	உள் நோயாளி எண்	:
பால்	:	ஆராய்ச்சி சேர்க்கை எண்	:

இந்த ஆராய்ச்சியின் விவரங்களும் அதன் நோக்கமும் முழுமையாக எனக்கு தெளிவாக விளக்கப்பட்டது.

எனக்கு விளக்கப்பட்ட விஷயங்களை புரிந்துகொண்டு நான் எனது சம்மதத்தை தெரிவிக்கிறேன்.

இந்த ஆராய்ச்சியில் பிறரின் நிர்பந்தமின்றி என் சொந்த விருப்பத்தின்பேரில் நான் பங்கு பெறுகின்றேன். இந்த ஆராய்ச்சியில் இருந்து நான் எந்நேரமும் பின் வாங்கலாம் என்பதையும் அதனால் எந்த பாதிப்பும் ஏற்படாது என்பதையும் நான் புரிந்துகொண்டேன்.

இந்த ஆராய்ச்சியினால் ஏற்படும் நன்மைகளையும் சில பக்க விளைவுகளையும் பற்றி தெளிவாக மருத்துவர் மூலம் தெரிந்துகொண்டேன்.

நான் என்னுடைய சுய நினைவுடனும் மற்றும் முழு சுதந்திரத்துடனும் இந்த மருத்துவ ஆராய்ச்சியில் என்னை சேர்த்துக்கொள்ள சம்மதிக்கிறேன்.

ஆராய்ச்சியாளர் கையொப்பம் பங்கேற்பாளர் கையொப்பம்

நாள் :
இடம் :

<u>ஆராய்ச்சி தகவல் தாள்</u>

தலைப்பு **குடலில் ஓட்டை ஏற்பட்டு பாதிப்புக்குள்ளாகும் நோயாளிகளின் ஆரோக்கியம் குன்றிய நிலை மற்றும் உயிரிழப்பு முடிவினை தீர்மானிக்கும் காரணிகளை மேன்ஹிம் குடல் சவ்வு அழற்சி அளவீட்டைக் கொண்டு முன்னறிவது பற்றிய ஆய்வு**

சென்னை இராஜீவ்காந்தி அரசு பொது மருத்துவனையில், குடலில் ஓட்டை ஏற்பட்டு பாதிப்புக்குள்ளாகும் நோயாளிகளின் ஆரோக்கியம் குன்றிய நிலை மற்றும் உயிரிழப்பு முடிவினை தீர்மானிக்கும் காரணிகளை மேன்ஹிம் குடல் சவ்வு அழற்சி அளவீடு கொண்டு முன்னறிவது பற்றிய ஓர் ஆய்வு இங்கு நடைபெறுகிறது.

இந்த ஆராய்ச்சியில் குடலில் ஓட்டை ஏற்பட்டு வலியுடன் வரும் நோயாளிகளின் வயது, பாலினம், நோயின் தன்மை, நோயின் காரணம், சிறுநீரக, குடல் மற்றும் பல உறுப்புகள் செயலிழப்பு, குடல் சவ்வு அழற்சியின் நேரம், அறுவை சிகிச்சையின்போது காணப்படும் ஓட்டையின் இடம், குடல் சவ்வு அழற்சியினால் ஏற்படும் நீரின் தன்மை, தமனியின் இரத்த பிராணவாயு அளவு மற்றும் கரியமிலவாயு அளவு ஆகியவற்றை அளந்து மேன்ஹிம் குடல் சவ்வு அழற்சி அளவீட்டைக்கொண்டு நோயாளியின் ஆரோக்கியம் குன்றிய நிலை மற்றும் உயிரிழப்பு முடிவினை முன்னறிவதே இதன் நோக்கமாகும். இதனால் தங்களது நோயின் ஆய்வறிக்கையோ அல்லது சிகிச்சையோ பாதிப்பு ஏற்படாது என்பதையும் தெரிவித்துக்கொள்கிறோம்.

முடிவுகளை அல்லது கருத்துகளை வெளியிடும்போதோ அல்லது ஆராய்ச்சியின் போதோ தங்களது பெயரையோ அல்லது அடையாளங்களையோ வெளியிடமாட்டோம் என்பதையும் தெரிவித்துக் கொள்கிறோம்.

இந்த சிறப்பு சிகிச்சையின் முடிவுகளை ஆராய்ச்சியின்போது அல்லது ஆராய்ச்சியின் முடிவின் போது தங்களுக்கு அறிவிக்கப்படும் என்பதையும் தெரிவித்துக் கொள்கிறோம்.

இந்த ஆராய்ச்சியில் பங்கேற்பது தங்களுடைய விருப்பத்தின் பேரில் தான் இருக்கிறது. மேலும் நீங்கள் எந்நேரமும் இந்த ஆராய்ச்சியிலிருந்து பின்வாங்கலாம் என்பதையும் தெரிவித்துக் கொள்கிறோம்.

ஆராய்ச்சியாளர் கையொப்பம் பங்கேற்பாளர் கையொப்பம்

நாள் :
இடம் :

PATIENT CONSENT FORM

STUDY TITLE : "**MANNHEIM PERITONITIS INDEX IN PREDICTING THE MORBIDITY AND MORTALITY IN PATIENTS WITH PERITONITIS DUE TO HOLLOW VISCOUS PERFORATION"**

STUDY CENTRE: Institute of General surgery, Rajiv Gandhi Government General Hospital, Madras Medical College.

PARTICIPANT NAME: **AGE:** **SEX:** **I.P. NO :**

I confirm that I have understood the purpose of interventional procedure for the above study. I have the opportunity to ask the question and all my questions and doubts have been answered to my satisfaction.

I have been explained about the possible complications that may occur during the interventional and interventional procedure. I understand that my participation in the study is voluntary and that I am free to withdraw at any time without giving any reason.

I understand that the investigator, regulatory authorities and the ethical committee will not need my permission to look at my health records both in respect to the current study and any further research that may be conducted in relation to it, even if I withdraw from the study. I understand that my identity will not be revealed in any information released to third parties or published, unless as required under the law. I agree not to restrict the use of any data or results that arise from the study.

I hereby consent to participate in this study of the

"**MANNHEIM PERITONITIS INDEX IN PREDICTING THE MORBIDITY AND MORTALITY IN PATIENTS WITH PERITONITIS DUE TO HOLLOW VISCOUS PERFORATION"**

Date:

Place:

Patient's name:

Signature / thumb impression of patient

Signature of the Investigator:

Name of the investigator:

INFORMATION SHEET

We are conducting a study on "**MANNHEIM PERITONITIS INDEX IN PREDICTING THE MORBIDITY AND MORTALITY IN PATIENTS WITH PERITONITIS DUE TO HOLLOW VISCOUS PERFORATION"**

among patients attending Rajiv Gandhi Government General Hospital, Chennai and for that your information is valuable to us.

The purpose of this study is to find out the beneficial aspects including the Early diagnosis , definitive treatment, reduction of morbidity, hospital stay & financial implications of surgical management of patients with secondary peritonitis due to hollow viscus perforation.

We are selecting certain cases and if you are found eligible, we may be using your information which in any way do not affect your final report or management.

The privacy of the patients in the research will be maintained throughout the study. In the event of any publication or presentation resulting from the research, no personally identifiable information will be shared.

Taking part in this study is voluntary. You are free to decide whether to participate in this study or to withdraw at any time; your decision will not result in any loss of benefits to which you are otherwise entitled.

The results of the special study may be intimated to you at the end of the study period or during the study if anything is found abnormal which may aid in the management or treatment.

Signature of the Participant Signature of the Investigator

Date

Place

PROFORMA

Name : _____ IP. No: **Age :**

Address: _____ **Sex :**

_____

Occupation:		
DOA & Time:		
DOS & Time:		
DOD:		
Chief complaints:	Abdominal pain site	
	Started on & Time:	
Treatment history:	For present illness: Yes/No	
	Type of treatment:	
	Duration:	
Past history:	Peptic ulcer disease:	
	Drugs used:	
	Surgery for peptic ulcer:	
Personal history:	Smoking, duration:	
	Alcohol, duration:	
Co morbid illness:	HT / DM / CLD /CRF / TB/COPD/CVA	
General examination		
	Consciousness	
	Orientation	
	Hydration	
	Fever	

Jaundice	
Anemia	
Respiratory distress	
Vitals	
PR:	
BP:	
Temp:	
RR:	
Temp:	
Systemic examination::	
CVS:	
RS:	
Abdomen :	
Investigations : CXR	
USG	
Biochemistry Glucose	
Urea	
Creatinine	
Na+	
K+	
ABG pO2	
pCO2	
CBC TC:	
DC:	
Platelets:	

DIAGNOSIS	
Treatment:	
PRE operative: **Urine output (ml/hr)**	
Intestinal Obstruction (duration)	
Shock (SBP/MAP)	
Duration between pain and surgery:	
PER operative:	
Surgery : open / lap	
Exudate : (Clear/Cloudy-Purulent/Faeculent)	
Site of pathology	
: <1cm / 1 – 3cm / Size >3cm	
Malignancy or Benign	
Peritonitis (generalised / localized)	
Procedure done	
POST Operative period	
Respiratory support	
Circulatory support	
Renal function	
Complications :	
Leakage	
Fluid collection	
Paralytic ileus	
Intestinal obstruction	
Bleeding	
Wound complication	

Pulmonary complication:	
Cardiac complication	
Renal complication	
Hepatic complication	
Multi organ failure	
Others	
FINAL OUTCOME :	

S.No	NAME	AGE	SEX	ABDOMINAL PAIN	PAIN DURATION in DAYS	TACHYCARDIA	SYSTOLIC BP (mm/Hg)	DIASTOLIC BP(mm/Hg)	MEAN ARTERIAL BP(mm/Hg)	LIVER DULLNESS OBLITERATION	ABDOMEN GUARDING/ RIGIDITTY	BOWEL SOUNDS	AIR UNDER THE DIAPHRAGM	UREA (mg/dl)	CREATININE(mg/dl)	pO2(mmHg)	pCO2(mmHg)	Urine output (ml/hr)	DURATION BETWEEN SYMPTOMS & SU	NATURE OF EXUDATE	NATURE OF PATHOLOGY	PERITONITIS	SITE OF PATHOLOGY	PROCEDURE DONE	RESPIRATORY SUPPORT	CIRCULATORY SUPPORT	Post op perforation LEAK	FLUID COLLECTION	PARALYTIC ILEUS	INESTINAL OBSTRUCTION	WOUND INFECTION	Wound dehiscence	RESPIRATORY complication	RENAL Complication	FINAL OUTCOME
1	Paneer selvam	##	M	Y	3	Y	80	40	53	Y	Y	NH	Y	102	2.8	43	52	10	74	Faeculent	B	Generalised	Colon	PC & DO	Y	Y	N	N	N	N	Y	N	LRI	AKI	Death
2	Kanimozhi	##	F	Y	3	Y	74	42	52	Y	Y	H	Y	88	1.8	84	47	15	76	Cloudy	B	Generalised	Duodenum	MG	Y	Y	N	N	N	N	Y	N	N	AKI	Death
3	Palani	##	M	Y	7	Y	82	52	62	Y	Y	NH	Y	93	3.2	88	48	15	175	Faeculent	M	Generalised	Colon	R & A	N	Y	N	N	N	N	Y	Y	N	AKI	Death
4	Venkatesh	##	M	Y	2	Y	90	40	56	N	Y	H	N	110	2.6	38	53	10	52	Faeculent	B	Generalised	ileum	PC	Y	Y	N	N	N	N	N	N	ARDS	AKI	Death
5	Arumugam	##	M	Y	3	Y	68	30	42	Y	Y	H	Y	40	1.2	40	51	40	78	Cloudy	B	Generalised	Duodenum	MG	Y	Y	N	N	N	N	N	N	ARDS	N	Death
6	Mani	##	M	Y	3	Y	82	42	55	Y	Y	H	Y	38	0.8	39	40	50	75	Cloudy	B	Generalised	Duodenum	MG	Y	N	N	N	N	N	N	N	ARDS	n	Death
7	Rangachari	##	M	Y	1	N	90	50	63	Y	Y	H	Y	54	2.3	42	53	15	28	Cloudy	B	Generalised	Duodenum	MG	Y	N	N	N	N	N	Y	Y	LRI	AKI	Death
8	Rajagopal	##	M	Y	2	Y	100	60	73	Y	Y	H	Y	90	2.4	88	42	10	56	Cloudy	B	Generalised	Duodenum	MG	Y	Y	N	N	N	N	N	N	ARDS	AKI	Death
9	Siva	##	M	Y	3	Y	70	50	56	Y	Y	NH	Y	78	2.6	48	52	15	73	Cloudy	B	Generalised	Duodenum	MG	Y	Y	-	-	-	N	N	N	ARDS	AKI	Death
10	Ettiyappan	##	M	Y	4	Y	90	46	60	Y	Y	NH	Y	100	1.8	85	40	20	97	Cloudy	B	Generalised	Duodenum	MG	Y	Y	N	N	N	N	N	N	LRI	AKI	Death
11	Devaraj	##	M	Y	4	Y	70	36	47	Y	Y	NH	Y	110	3.5	90	45	5	97	Cloudy	B	Generalised	Duodenum	MG	Y	Y	-	-	-	-	-	-	-	AKI	Death
12	Vishalkumar	##	M	N	2	Y	80	50	60	Y	Y	H	Y	120	3.2	78	44	5	53	Cloudy	B	Generalised	Duodenum	MG	Y	Y	-	-	-	-	-	-	ARDS	AKI	Death
13	Krishnapillai	##	M	Y	3	Y	86	40	55	N	Y	H	N	20	0.7	90	37	15	79	Cloudy	B	Generalised	Duodenum	MG	Y	Y	-	-	-	-	-	-	ARDS	AKI	Death
14	Gopal	##	M	Y	3	Y	110	70	83	Y	Y	H	Y	22	0.8	98	36	30	77	Cloudy	B	Generalised	Duodenum	MG	Y	Y	N	N	N	N	Y	N	LRI	AKI	Discharge
15	Pattammal	##	F	Y	3	Y	146	80	##	Y	Y	H	Y	25	0.7	89	38	40	74	Cloudy	B	Generalised	Duodenum	MG	Y	Y	N	N	N	N	Y	N	N	N	Discharge
16	Ravikumar	##	M	Y	7	Y	150	92	##	Y	Y	NH	Y	30	0.9	92	41	35	180	Cloudy	B	Generalised	Duodenum	MG	Y	Y	N	N	N	N	Y	Y	N	N	Discharge
17	Shankar	##	M	Y	3	Y	90	50	63	Y	Y	H	Y	27	0.6	94	40	55	70	Cloudy	B	Generalised	Duodenum	MG	Y	N	n	N	N	N	N	N	N	N	Discharge
18	Sabiq	##	M	Y	1	N	130	70	90	N	Y	H	N	21	1.1	88	40	60	27	Cloudy	B	Generalised	ileum	PC	Y	N	N	N	N	N	Y	N	LRI	Rena	Discharge
19	Ganapathy	##	M	Y	4	Y	128	74	92	Y	Y	H	Y	26	0.7	87	42	25	98	Cloudy	B	Generalised	Duodenum	MG	Y	Y	N	N	N	N	N	N	LRI	N	Discharge
20	Muruganant	##	M	Y	4	Y	160	80	##	N	Y	H	N	23	0.8	95	39	40	100	Purulent	B	Localised	Appendix	PL & AP	Y	Y	N	N	N	N	N	N	N	n	Discharge
21	Abdulla	##	M	Y	1	Y	120	70	86	N	Y	H	N	25	0.6	88	36	40	21	Cloudy	B	Generalised	Duodenum	MG	Y	Y	N	N	N	N	N	N	LRI	REN	Discharge
22	Inbaraj	##	M	Y	2	Y	110	68	96	Y	Y	H	Y	30	0.8	93	39	55	25	Cloudy	B	Generalised	Duodenum	MG	Y	Y	N	N	N	N	N	N	N	AKI	Discharge
23	Smith	##	M	Y	3	Y	130	80	96	N	Y	H	N	34	0.9	96	40	65	82	Cloudy	B	Generalised	Appendix	PL & AP	Y	Y	N	N	N	N	N	N	N	AKI	Discharge
24	RajaSekar	##	M	Y	1	N	130	70	90	N	Y	NH	N	31	0.8	91	41	65	26	Cloudy	B	Generalised	Appendix	PL & AP	N	N	N	N	N	N	N	N	N	N	Discharge
25	Arulraj	##	M	Y	3	N	118	78	91	N	Y	NH	N	32	1.2	85	38	50	76	Cloudy	B	Localised	Appendix	PL & AP	N	N	N	Y	Y	N	N	N	LRI	N	Discharge
26	Periyasamy	##	M	Y	2	N	120	68	85	Y	Y	H	Y	25	1.1	89	43	85	56	Cloudy	B	Generalised	Duodenum	MG	N	N	N	N	Y	N	N	N	N	N	Discharge
27	Kumar	##	M	Y	1	N	100	68	78	Y	Y	NH	Y	20	0.8	89	36	55	27	Cloudy	B	Generalised	Duodenum	MG	N	N	N	N	N	N	N	N	N	N	Discharge
28	Paramasivam	##	M	Y	2	Y	102	64	76	N	Y	NH	N	22	0.7	88	43	65	57	Cloudy	B	Generalised	Appendix	PL & AP	N	N	N	N	N	N	N	N	N	N	Discharge
29	Masilamani	##	M	Y	1	N	138	70	92	Y	Y	H	Y	25	0.9	87	43	60	28	Cloudy	M	Generalised	Gastric	R &A	N	N	N	N	N	N	Y	N	N	N	Discharge
30	Prakash	##	M	Y	3	Y	124	68	86	Y	Y	H	Y	30	1.1	95	47	65	75	Cloudy	B	Generalised	Duodenum	MG	N	N	N	N	N	N	Y	Y	N	N	Discharge
31	Saravanan	##	M	Y	2	Y	120	70	86	Y	Y	H	Y	27	1	88	51	55	51	Cloudy	B	Generalised	Duodenum	MG	Y	Y	N	N	N	N	Y	N	Y	AKI	Discharge
32	Sivakumar	##	M	Y	2	N	110	80	90	Y	Y	H	Y	21	1.2	93	43	60	56	Cloudy	B	Generalised	Duodenum	MG	N	N	N	N	N	N	N	N	N	N	Discharge
33	Abshiek kumar	##	M	Y	2	N	110	60	76	N	Y	H	N	26	0.8	96	43	65	60	Cloudy	B	Generalised	Appendix	PL & AP	N	N	N	N	N	N	N	N	N	N	Discharge
34	Gangan	##	M	Y	3	N	120	78	92	N	Y	H	N	23	1.2	90	43	50	76	Purulent	B	Generalised	Appendix	PL & AP	N	N	N	N	N	N	N	N	N	N	Discharge
35	Elumalai	##	M	Y	2	Y	110	80	90	N	Y	H	N	25	0.8	98	42	45	49	Cloudy	B	Generalised	Appendix	PL & AP	N	N	N	N	N	N	Y	N	N	N	Discharge
36	Manikandan	##	M	Y	1	N	146	68	94	Y	Y	H	Y	30	1.1	89	47	50	26	Cloudy	B	Generalised	Duodenum	MG	N	N	N	N	N	N	N	N	N	N	Discharge

S.No	NAME	AGE	SEX	ABDOMINAL PAIN	PAIN DURATION in DAYS	TACHYCARDIA	SYSTOLIC BP (mm/Hg)	DIASTOLIC BP(mm/Hg)	MEAN ARTERIAL BP(mm/Hg)	LIVER DULLNESS OBLITERATION	ABDOMEN GUARDING/ RIGIDITTY	BOWEL SOUNDS	AIR UNDER THE DIAPHRAGM	UREA (mg/dl)	CREATININE(mg/dl)	pO2(mmHg)	pCO2(mmHg)	Urine output (ml/hr)	DURATION BETWEEN SYMPTOMS & SU	NATURE OF EXUDATE	NATURE OF PATHOLOGY	PERITONITIS	SITE OF PATHOLOGY	PROCEDURE DONE	RESPIRATORY SUPPORT	CIRCULATORY SUPPORT	Post op perforation LEAK	FLUID COLLECTION	PARALYTIC ILEUS	INESTINAL OBSTRUCTION	WOUND INFECTION	Wound dehiscence	RESPIRATORY complication	RENAL Complication	FINAL OUTCOME
37	Kumar	##	M	Y	6HR	Y	136	64	88	Y	Y	H	Y	34	1.8	92	45	70	9	Cloudy	B	Generalised	Duodenum	MG	N	N	N	N	N	N	N	N	N	N	Discharge
38	Pintu	##	M	Y	3	N	90	70	76	N	Y	H	N	31	1.5	89	46	55	80	Cloudy	B	Generalised	Appendix	PL & AP	N	N	N	N	N	N	N	N	N	N	Discharge
39	Kumar	##	M	Y	3	Y	130	68	88	Y	Y	H	Y	32	1.2	88	39	60	82	Cloudy	B	Generalised	Duodenum	MG	N	N	N	N	N	N	N	N	N	N	Discharge
40	Babu	##	M	Y	1	Y	128	70	89	Y	Y	H	Y	33	0.7	87	38	60	28	Cloudy	B	Generalised	Duodenum	MG	N	N	N	N	N	N	N	N	N	N	Discharge
41	Murugan	##	M	Y	1	N	120	70	86	N	Y	H	N	27	0.8	95	43	60	28	Cloudy	B	Generalised	Appendix	PL & AP	N	N	N	N	N	N	N	N	N	N	Discharge
42	Santhanam	##	M	Y	3	Y	120	68	85	Y	Y	H	Y	24	0.7	88	37	65	79	Cloudy	B	Generalised	Duodenum	MG	N	N	N	N	N	N	Y	N	N	N	Discharge
43	Arumugam	##	M	Y	8hrs	Y	110	80	90	N	Y	H	N	30	0.9	93	34	70	26	Cloudy	B	Generalised	Appendix	PL & AP	N	N	N	N	N	N	N	N	N	N	Discharge
44	Fakrudeen	##	M	Y	1hr	Y	130	50	76	Y	Y	H	Y	28	0.6	96	38	65	14	Cloudy	B	Generalised	Duodenum	MG	Y	Y	N	N	N	N	N	N	LRI	REN	Discharge
45	Anbarasan	##	M	Y	5hr	Y	84	70	74	Y	Y	H	Y	22	1.1	91	44	70	18	Cloudy	B	Generalised	Duodenum	MG	N	N	N	N	N	N	N	N	LRI	N	Discharge
46	Jeyachandran	##	M	Y	8hrs	Y	118	74	88	Y	Y	H	Y	18	0.7	85	47	70	10	Cloudy	B	Generalised	Duodenum	MG	N	N	N	N	N	N	N	N	N	N	Discharge
47	Murugesan	##	M	Y	10hr	N	104	80	88	N	Y	H	N	19	0.8	87	46	65	23	Cloudy	B	Localised	Appendix	PL & AP	N	N	N	N	N	N	N	N	N	N	Discharge
48	Muna	##	M	Y	2	N	100	70	80	N	Y	H	N	22	0.6	95	37	55	52	Cloudy	B	Localised	Appendix	PL & AP	N	N	N	Y	N	N	Y	Y	LRI	n	Discharge
49	Kasi	##	M	Y	2	Y	102	68	79	Y	Y	H	Y	25	0.8	88	45	60	51	Cloudy	B	Generalised	Duodenum	MG	N	N	N	N	N	N	N	N	N	N	Discharge
50	Baskar	##	M	Y	10	N	120	80	93	N	Y	H	N	18	0.9	93	36	60	244	Cloudy	B	Generalised	Appendix	PL & AP	N	N	N	N	N	N	N	N	N	N	Discharge
51	Bala	##	M	Y	2	N	110	70	83	Y	Y	H	Y	18	0.8	96	45	55	52	Cloudy	B	Generalised	Duodenum	PL & AP	N	N	N	N	Y	N	N	N	LRI	N	Discharge
52	Mani	##	M	Y	10	N	110	70	83	Y	Y	NH	Y	22	0.9	98	36	85	250	Cloudy	B	Generalised	Duodenum	MG	N	N	N	N	N	N	N	N	N	N	Discharge
53	Ramadoss	##	M	Y	1	Y	124	68	86	N	Y	H	N	20	0.7	85	36	60	27	Cloudy	B	Generalised	ileum	R &A	N	N	N	N	N	N	N	N	LRI	N	Discharge
54	Lakshmanan	##	M	Y	2hr	Y	124	80	94	Y	Y	H	Y	22	0.8	88	34	70	28	Cloudy	B	Generalised	Duodenum	MG	N	N	N	N	N	N	N	N	N	N	Discharge
55	Krishnamoorthy	##	M	Y	2	Y	100	50	66	Y	Y	H	Y	25	0.7	87	41	75	50	Cloudy	B	Generalised	Gastric	PC	Y	Y	N	N	N	N	Y	Y	LRI	prer	Discharge
56	Selvam	##	M	Y	7	Y	110	70	83	Y	Y	H	Y	30	0.9	95	38	80	172	Cloudy	B	Generalised	Duodenum	MG	Y	N	N	N	N	N	Y	Y	LRI	prer	Discharge
57	Loganathan	##	M	Y	2	Y	110	74	86	Y	Y	H	Y	27	0.6	88	37	80	51	Cloudy	B	Generalised	Duodenum	MG	N	N	N	N	N	N	N	N	N	AKI	Discharge
58	Radhakrishnan	##	M	Y	10	N	120	80	93	N	Y	H	N	21	1.1	93	39	85	243	Cloudy	B	Generalised	jejunum	PC	N	N	N	N	N	N	N	N	N	N	Discharge
59	Appuraj	##	M	Y	2	N	110	70	83	N	Y	H	N	26	0.7	96	36	60	52	Cloudy	B	Generalised	Duodenum	MG	N	N	N	N	Y	N	N	N	LRI	N	Discharge
60	Purusothaman	##	M	Y	3	N	146	68	94	Y	Y	H	Y	23	0.8	91	36	65	75	Cloudy	B	Generalised	Duodenum	MG	N	N	N	N	N	N	N	N	N	N	Discharge
61	Subramani	##	M	Y	2	Y	136	80	98	N	Y	H	N	25	0.6	85	41	70	42	Cloudy	B	Generalised	Appendix	PL & AP	N	N	N	N	N	N	N	N	N	N	Discharge
62	Hussain	##	M	Y	10	N	90	52	64	Y	Y	NH	Y	30	0.8	94	40	60	245	Cloudy	M	Generalised	Gastric	R & A	N	N	N	N	N	N	N	N	N	N	Discharge
63	Narayanasamy	##	M	Y	2	N	130	78	95	Y	Y	NH	Y	34	0.9	89	36	60	51	Cloudy	B	Generalised	Duodenum	MG	N	N	N	N	Y	N	N	N	LRI	N	Discharge
64	Gnanamoorthy	##	M	Y	2	Y	128	80	96	Y	Y	H	Y	31	0.8	88	43	60	49	Cloudy	B	Generalised	Duodenum	MG	Y	Y	N	N	N	N	Y	Y	LRI	prer	Discharge
65	Moorthy	##	M	Y	7	Y	120	68	85	Y	Y	NH	Y	32	1.2	87	35	65	180	Cloudy	B	Generalised	ileum	PC	Y	N	N	N	N	N	Y	Y	LRI	prer	Discharge
66	Yesuraj	##	M	Y	1	N	120	64	82	N	Y	H	N	22	0.8	95	47	55	28	Cloudy	B	Localised	Appendix	PL & AP	N	N	N	N	N	N	N	N	N	N	Discharge
67	Jeyaraman	##	M	Y	2	N	110	70	83	Y	Y	H	Y	20	0.8	88	49	65	54	Cloudy	B	Generalised	Duodenum	MG	N	N	N	Y	N	N	Y	y	LRI	N	Discharge
68	Balaji	##	M	Y	2	Y	130	68	88	Y	Y	H	Y	22	0.7	93	37	60	53	Cloudy	B	Generalised	Duodenum	MG	N	N	N	N	N	N	N	N	N	N	Discharge
69	Lingaiyan	##	M	Y	3	Y	84	70	74	Y	Y	H	Y	25	0.9	96	34	70	78	Cloudy	B	Generalised	Duodenum	MG	N	N	N	N	N	N	Y	Y	N	N	Discharge
70	Srinivasan	##	M	Y	1	Y	118	70	86	Y	Y	H	Y	30	1.1	90	38	75	27	Cloudy	B	Generalised	Duodenum	MG	Y	N	N	N	N	N	Y	N	LRI	N	Discharge
71	Bindy	##	M	Y	3	Y	104	68	80	Y	Y	NH	Y	27	1	98	44	80	77	Cloudy	B	Generalised	Gastric	PC	N	N	N	N	N	N	N	N	N	N	Discharge
72	Maruthu	##	M	Y	3	N	100	80	86	Y	Y	NH	Y	21	1.2	89	47	80	76	Cloudy	B	Generalised	Duodenum	MG	N	N	N	N	N	N	N	N	N	N	Discharge

S.No	NAME	AGE	SEX	ABDOMINAL PAIN	PAIN DURATION in DAYS	TACHYCARDIA	SYSTOLIC BP (mm/Hg)	DIASTOLIC BP(mm/Hg)	MEAN ARTERIAL BP(mm/Hg)	LIVER DULLNESS OBLITERATION	ABDOMEN GUARDING/ RIGIDITTY	BOWEL SOUNDS	AIR UNDER THE DIAPHRAGM	UREA (mg/dl)	CREATININE(mg/dl)	pO2(mmHg)	pCO2(mmHg)	Urine output (ml/hr)	DURATION BETWEEN SYMPTOMS & SU	NATURE OF EXUDATE	NATURE OF PATHOLOGY	PERITONITIS	SITE OF PATHOLOGY	PROCEDURE DONE	RESPIRATORY SUPPORT	CIRCULATORY SUPPORT	Post op perforation LEAK	FLUID COLLECTION	PARALYTIC ILEUS	INESTINAL OBSTRUCTION	WOUND INFECTION	Wound dehiscence	RESPIRATORY complication	RENAL Complication	FINAL OUTCOME
73	Dhamodaran	##	M	Y	2hr	Y	102	50	67	Y	Y	H	Y	26	0.8	92	46	85	20	Cloudy	B	Generalised	Duodenum	MG	N	N	N	N	N	N	N	N	LRI	N	Discharge
74	Rangan	##	M	Y	3	N	138	70	92	Y	Y	NH	Y	23	1.2	89	37	60	82	Cloudy	B	Generalised	Duodenum	MG	N	N	N	N	N	N	N	N	N	N	Discharge
75	Kesavan	##	M	Y	3	Y	124	74	90	Y	Y	NH	Y	25	0.8	88	45	65	79	Cloudy	B	Generalised	Gastric	PC	N	N	N	N	N	N	N	N	N	N	Discharge
76	Premkumar	##	M	Y	1	N	120	80	93	Y	Y	H	Y	30	1.1	87	36	70	26	Cloudy	B	Generalised	Duodenum	MG	N	N	N	N	N	N	N	N	N	N	Discharge
77	Ahamed	##	M	Y	3	N	130	70	90	Y	Y	H	Y	34	1.8	95	45	60	89	Cloudy	B	Generalised	Gastric	PC	N	N	N	Y	Y	N	n	N	LRI	n	Discharge
78	Devanathan	##	M	Y	2	Y	110	80	90	Y	Y	H	Y	34	1.8	92	46	80	52	Cloudy	B	Generalised	Duodenum	MG	N	N	N	N	N	N	N	N	N	AKI	Discharge
79	Manoharan	##	M	Y	1	N	146	80	##	N	Y	H	N	32	1.2	93	36	60	28	Cloudy	B	Generalised	Duodenum	MG	N	N	N	N	N	N	N	N	N	N	Discharge
80	Chandran	##	M	N	2	Y	136	52	80	Y	Y	H	Y	33	0.7	96	34	65	51	Cloudy	B	Generalised	Duodenum	MG	N	N	N	N	N	N	N	N	N	N	Discharge
81	Tarunkumar	##	M	Y	1	N	90	78	82	Y	Y	H	Y	27	0.8	91	41	55	27	Cloudy	B	Generalised	Duodenum	MG	N	N	N	N	N	N	Y	N	N	N	Discharge
82	Rajendran	##	M	Y	3	Y	130	79	96	N	Y	NH	N	24	0.7	85	38	65	80	Cloudy	B	Generalised	Appendix	PL & AP	N	N	N	N	N	N	Y	Y	N	N	Discharge
83	Suriyakumar	##	M	Y	2	Y	128	68	88	Y	Y	H	Y	30	0.9	87	37	70	57	Cloudy	B	Generalised	Duodenum	MG	Y	Y	N	N	N	N	Y	N	Y	AKI	Discharge
84	Sivaraj	##	M	Y	2	N	120	64	62	N	Y	H	N	28	0.6	95	39	75	52	Cloudy	B	Generalised	Appendix	PL & AP	N	N	N	N	N	N	Y	N	N	N	Discharge
85	Gokul	##	M	N	2	N	120	70	86	N	Y	H	N	22	1.1	88	36	80	55	Cloudy	B	Localised	Appendix	PL & AP	N	N	N	N	N	N	N	N	N	N	Discharge
86	Pandian	##	M	Y	3	N	110	68	82	Y	Y	NH	Y	18	0.7	93	36	80	83	Cloudy	B	Generalised	Duodenum	MG	N	N	N	N	N	N	N	N	N	N	Discharge
87	Manimaran	##	M	Y	2	Y	130	70	90	Y	Y	H	Y	19	0.8	96	41	85	54	Cloudy	B	Generalised	Duodenum	MG	N	N	N	N	N	N	Y	N	N	N	Discharge
88	Sandhanagopal	##	M	Y	1	N	84	40	54	N	Y	H	N	22	0.6	91	46	60	28	Cloudy	B	Generalised	Appendix	PL & AP	N	N	N	N	N	N	N	N	N	N	Discharge
89	Ganesan	##	M	Y	6HR	Y	118	80	92	Y	Y	H	Y	25	0.8	85	36	65	9	Cloudy	B	Generalised	Duodenum	MG	N	N	N	N	N	N	N	N	N	N	Discharge
90	Sriram	##	M	Y	2	Y	104	68	80	Y	Y	H	Y	18	0.9	98	43	70	56	Cloudy	B	Generalised	Duodenum	MG	N	N	N	N	N	N	N	N	n	N	Discharge
91	Nagendran	##	M	Y	3	Y	100	64	76	Y	Y	H	Y	18	0.8	89	35	60	86	Cloudy	B	Generalised	Gastric	PC	N	N	N	N	N	N	Y	Y	N	N	Discharge
92	Bijanlal	##	M	Y	1	Y	102	70	80	Y	Y	H	Y	22	0.7	92	47	60	27	Cloudy	B	Generalised	Duodenum	MG	N	N	N	N	N	N	N	N	N	N	Discharge
93	Nithyanandham	##	M	Y	1	N	138	68	91	N	Y	H	N	25	0.9	89	49	60	27	Cloudy	B	Generalised	Duodenum	MG	N	N	N	N	N	N	N	N	N	N	Discharge
94	Rajesh	##	M	Y	3	Y	120	70	86	Y	Y	H	Y	30	1.1	88	41	65	87	Cloudy	B	Generalised	Appendix	PL & AP	N	N	N	N	N	N	Y	N	N	N	Discharge
95	Ramkumar	##	M	Y	1	Y	120	70	86	Y	Y	H	Y	27	1	87	38	55	27	Cloudy	B	Generalised	Duodenum	MG	N	N	N	N	N	N	N	N	N	N	Discharge
96	Batchidoss	##	M	Y	1	Y	110	68	82	Y	Y	H	Y	21	1.2	95	37	65	26	Cloudy	B	Generalised	Duodenum	MG	N	N	N	N	N	N	N	N	N	N	Discharge
97	Anduraj	##	M	Y	1	Y	130	80	96	Y	Y	H	Y	26	0.8	88	39	60	26	Cloudy	B	Generalised	Duodenum	MG	N	N	N	N	N	N	N	N	LRI	N	Discharge
98	Prabakaran	##	M	Y	1	Y	84	50	61	N	Y	H	N	23	1.2	93	36	70	29	Cloudy	B	Generalised	Appendix	PL & AP	N	N	N	N	N	N	N	N	N	N	Discharge
99	Kamaraj	##	M	Y	1	Y	118	70	86	Y	Y	H	Y	25	0.8	96	36	75	28	Cloudy	B	Generalised	Duodenum	MG	Y	N	N	N	N	N	Y	Y	LRI	N	Discharge
100	Annappan	##	M	Y	4	Y	104	74	84	Y	Y	NH	Y	30	1.1	91	41	80	98	Faeculent	B	Generalised	Colon	O	N	N	N	N	N	N	N	N	N	N	Discharge

KEY TO MASTER CHART

M	-	Male
F	-	Female
H	-	Heard
NH	-	Not Heard
B	-	Benign
M	-	Malignant
PC	-	Primary Closure
DO	-	Diversion Ostomy
O	-	Ostomy
MG	-	Modified Graham's Live Omental Patch Closure
R & A	-	Resection and Anastomosis
PL	-	Peritoneal Lavage
AP	-	Appendectomy
LRI	-	Lower respiratory tract Infection
ARDS	-	Acute respiratory distress syndrome
AKI	-	Acute Kidney Injury
Y	-	Yes
N	-	No