EFFECTIVENESS OF MANNHEIM PERITONITIS INDEX SCORING SYSTEM IN PREDICTING THE MORBIDITY AND MORTALITY IN PERITONITIS DUE TO HOLLOW VISCOUS PERFORATION

Dissertation submitted to

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For the award of the degree of

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GOVERNMENT KILPAUK MEDICAL COLLEGE,

CHENNAI

MAY 2018

CERTIFICATE BY THE GUIDE

This is to certify that the dissertation entitled **"EFFECTIVENESS OF MANNHEIM PERITONITIS INDEX SCORING SYSTEM IN PREDICTING THE MORBIDITY AND MORTALITY IN PERITONITIS DUE TO HOLLOW VISCOUS PERFORATION"** is a bonafide research work done by **Dr. PETA PAVAN KUMAR,** under my direct guidance and supervision in the Department of General Surgery, Govt Kilpauk Medical College, Chennai during his **M.S. (General Surgery)** course from May 2015 to May 2018.

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DECLARATION

I, **DR PETA PAVAN KUMAR** hereby declare that this dissertation "**EFFECTIVENESS OF MANNHEIM PERITONITIS INDEX SCORING SYSTEM IN PREDICTING THE MORBIDITY AND MORTALITY IN PERITONITIS DUE TO HOLLOW VISCOUS PERFORATION"** is a bonafide and genuine work carried out by me under the direct guidance of **Prof. Dr. M. ALLI, DGO, M.S (GS),** Professor and Chief, SU-1 AND **Prof. Dr. R. KANNAN, M.S** Professor and Head of Department of General Surgery at Govt Kilpauk Medical College, Chennai – 600 010.

This dissertation or any part there of has not been submitted by me to any other university for award of any degree or diploma.

Date: **Dr. PETA PAVAN KUMAR**

Place: Chennai

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INSTITUTIONAL ETHICS COMMITTEE
GOVT. KILPAUK MEDICAL COLLEGE,
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Protocol ID. No.10/2017 Meeting held on 17.04.2017

The Institutional Ethical Committee of Govt. Kilpauk Medical College, Chennai reviewed and discussed the application for approval "**Effectiveness of Mannheim Peritonitis index scoring system in predicting the morbidity and mortality in peritonitis due to hollow viscous perforation"** submitted by Dr.Peta Pavan Kumar, M.S. (General Surgery), PG Student, GKMC, Chennai-10

The Proposal is APPROVED

The Institutional Ethical Committee expects to be informed about the progress of the study any Adverse Drug Reaction Occurring in the Course of the study any change in the protocol and patient information /informed consent and asks to be provided a copy of the final report.

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PLAGIARISM EVALUATION OF MODIFIED ALVARADO SCORING AND ULTRASONOGRAM IN THE DIAGNOSIS OF ACUTE APPENDICITIS.docx (D31044275)

Instances where selected sources appear:

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LIST OF ABBREVIATIONS

MPI	-	Mannheims peritonitis index
ICU	-	Intensive care unit
APC	-	Adenomatous polyposis coli
WHO	-	World health organisation
ATT	-	Anti tubercular treatment
CAPD	-	Continuous ambulatory peritoneal dialysis
CT	-	Computerized tomography
SIRS	-	Systematic inflammatory response syndrome
CARS	-	Compensatory anti inflammatory response syndrome
TNF	-	Tumour necrosis factor

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INTRODUCTION

Acute generalized peritonitis from gastrointestinal hollow viscous perforation is a potentially life threatening condition. The prognosis of peritonitis remains poor despite development in diagnosis and management. Early identification of patients with severe peritonitis may help in selecting patients for aggressive surgical approach[1-3].

Grading the severity of acute peritonitis has assisted in no small way in decision making and has improved therapy in the management of severely ill patients[4]. Empirically based risk assessment for important clinical events has been extremely useful in evaluating new therapies, in monitoring resources for effective use and improving quality of care[5-6].

Many scoring systems have been designed and used successfully to grade the severity of acute peritonitis like, Acute physiology and chronic health evaluation (APACHE) II score, simplified acute physiology score (SAPS), Sepsis severity score (SSS), Ranson score, Imrite score, Mannheim peritonitis index (MPI)[7,8]. MPI was developed by Wacha and Linder in 1983[9]. It was developed based on the retrospective analysis of data from 1253 patients with peritonitis, in which 20 possible risk factors were considered. Of these only 8 proved to be of prognostic relevance and were entered into the Mannheim Peritonitis Index, classified according to their predictive power. Patients with a score exceeding 26

were defined as having a high mortality rate. The Mannheim Peritonitis Index (MPI) is a specific score, which has a good accuracy and provides an easy way to handle with clinical parameters, allowing the prediction of the individual prognosis of patients with peritonitis[9].

AIMS AND OBJECTIVES

1) Aim is to predict the risk of morbidity and mortality in patients with peritonitis due to hollow viscous 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 MPI scoring system in patients with peritonitis due to hollow viscous perforation, to assess it as a clinical tool in stratifying these patients according to individual surgical risk.

REVIEW OF LITERATURE

Peritonitis due to hollow viscous perforation has been documented by many historians. Previously the disease was inevitably fatal due to lack of knowledge of surgical procedures, lack of availability of good quality post operative care. With ages, management of this condition has undergone various changes in surgical procedures for the specific conditions and also the level of post op care increasing the survival rates to a significant level.

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).

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.

In 1926 operative role of treatment of hollow viscous perforation was first documented. Kirschner et al 1926 reported that mortality decreased from >90% to <40% with the introduction of operative procedure.

In 1980 Fry et al showed that mortality after major operative procedures increased as the number of failed organs increase. Mortality

was 3% with no organ failure, which increased to 30% with 1 organ failure and to 100% with 4 major organ failures[10].

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 grading the severity of sepsis was published by Elebute and Stoner in 1983[11] . These authors divided the clinical features of the septic state into 4 classes to which they described 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 lab data.

Pine and associates (1983)[12] 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, alcoholism and age 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)[13] recognized the need for more precision and for a greater range of potential values and devised a scoring system to represent the magnitude and severity of organ failure. He defined 7 organ systems and assigned score of 0-5 in each system. Scores were calculated 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)[14] extended these observations in a report covering 5,677 ICU admissions and 2719 patients who developed organ failure.

Teichmann and associates (1986)[15] 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)[16] developed a separate peritonitis index, the Mannheim Peritonitis Index (MPI) with incorporated information regarding age, gender, organ failure, cancer, 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[17] and others evaluated prognostic factors in 50 cases of perforated peptic ulcer. They concluded that there is a place for prognostic scoring. They found Gen. Health, 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[18] 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[19] compared Apache II with MPI, they concluded that there was no significant different in prognostic value between scoring systems. Khosrovanin 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[20] 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 factor over the type and source of infection on the prognosis of patients. Both Apache-II and MPI correctly graded intra-abdominal infections and were strongly and independently associated

with an outcome. However, the MPI has the advantages of being easier to calculate.

In a study done in Columbia over a span of 10 years which included 267 patients concluded that commonest site of perforation was colon, mortality was 20% and mean hospital stay was 22 days [21].

In a clinical study done by Ali Yaghoobi Notash, overall hospital mortality rate was 17.5% including 80% of patients with MPI >29, in non survivors the mean score was 33.9, survivors had the mean score of 19.9%[22].

In a study by the Japanese workers published in 2004 the sensitivity of MPI score more than 26 was 77.7 and specificity was 97.9[23].

Study by Dr A. Billing , D. Fröhlich[24], The Peritonitis Study Group showed for a threshold index score of 26, the sensitivity was 86 (range 54-98) percent specificity 74 (range 58-97) percent and accuracy 83 (range 70-94) percent in predicting death.

For patients with a score less than 21 the mean mortality rate was 2.3 (range 0-11) percent, for score 21-29, 22.5 (range 10.6 - 50) percent and for score greater than 29, 59.1 (range 41-87) percent.. The Mannheim

peritonitis index provides an easy and reliable means of risk evaluation and classification for patients with peritoneal inflammation.

Ajaz Ahmad Malik conducted a prospective study in patients having generalized peritonitis over 2 years, results showed mortality of 82.3% with score of MPI >25[25] .

Study done by F.Ntirenganya [26], conducted a prospective study on the outcome of peritonitis using Mannheim peritonitis index , results showed that when MPI> 29 points , predictive power of MPI for morbidity was 0.896 with a sensitivity of 66.7% and specificity of 99.04%.

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[27] .The peritoneal cavity therefore comes to be subdivided into number of pockets that are separated partially by folds of peritoneum.

Parietal peritoneum:

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

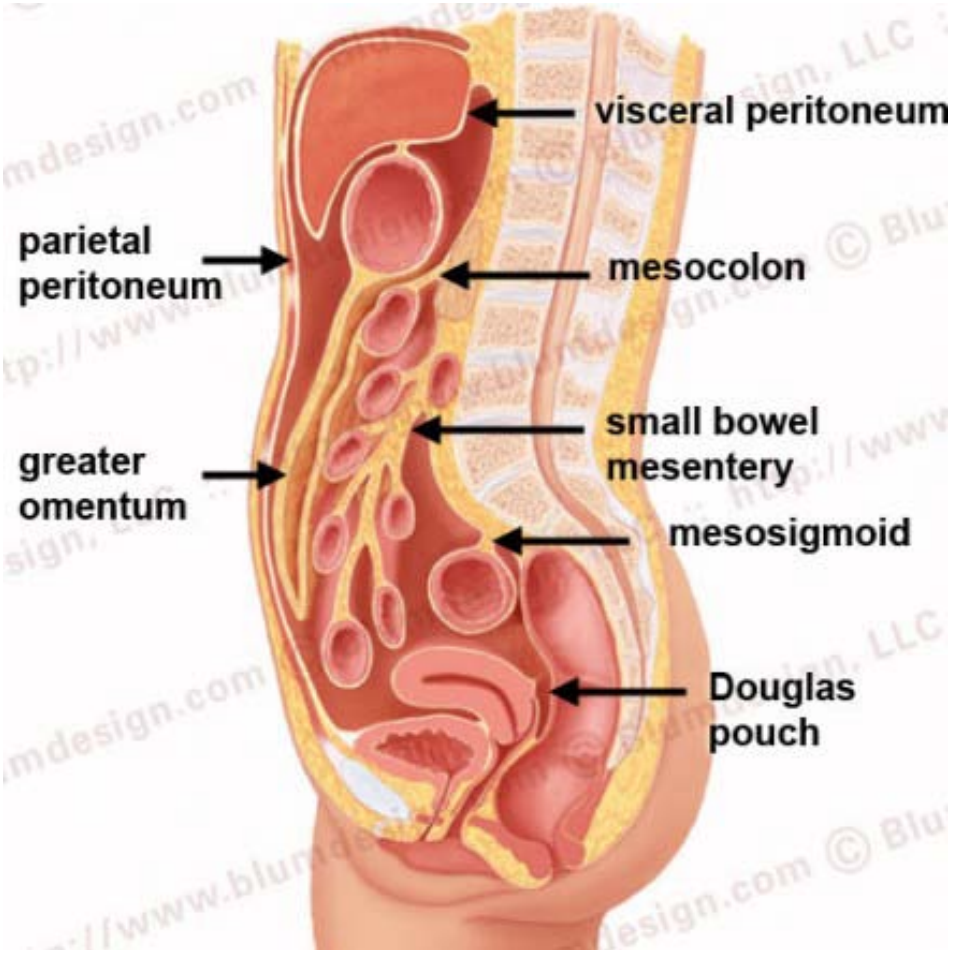


Fig.no.1: Peritoneum –parietal and visceral layers

Visceral peritoneum:

It 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[28]. Histologically, peritoneum is composed of an outer layer of fibrous tissue, which gives strength to the and an inner layer of mesothelial cells which secrete a serous fluid.

The peritoneal cavity is the largest cavity in the 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 a main region termed 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 transverse colon and mesocolon. The infra colic compartment is divided into right and left by mesentery.

The Right infracolic and left infracolic is divided into external and internal paracolic gutters by ascending and descending colon respectively. Supracolic compartment is below the diaphragm and above transverse colon and mesocolon. The liver, gallbladder, stomach, first

part of the duodenum and spleen lie in this space. The liver and ligaments break this space into important sub phrenic space.

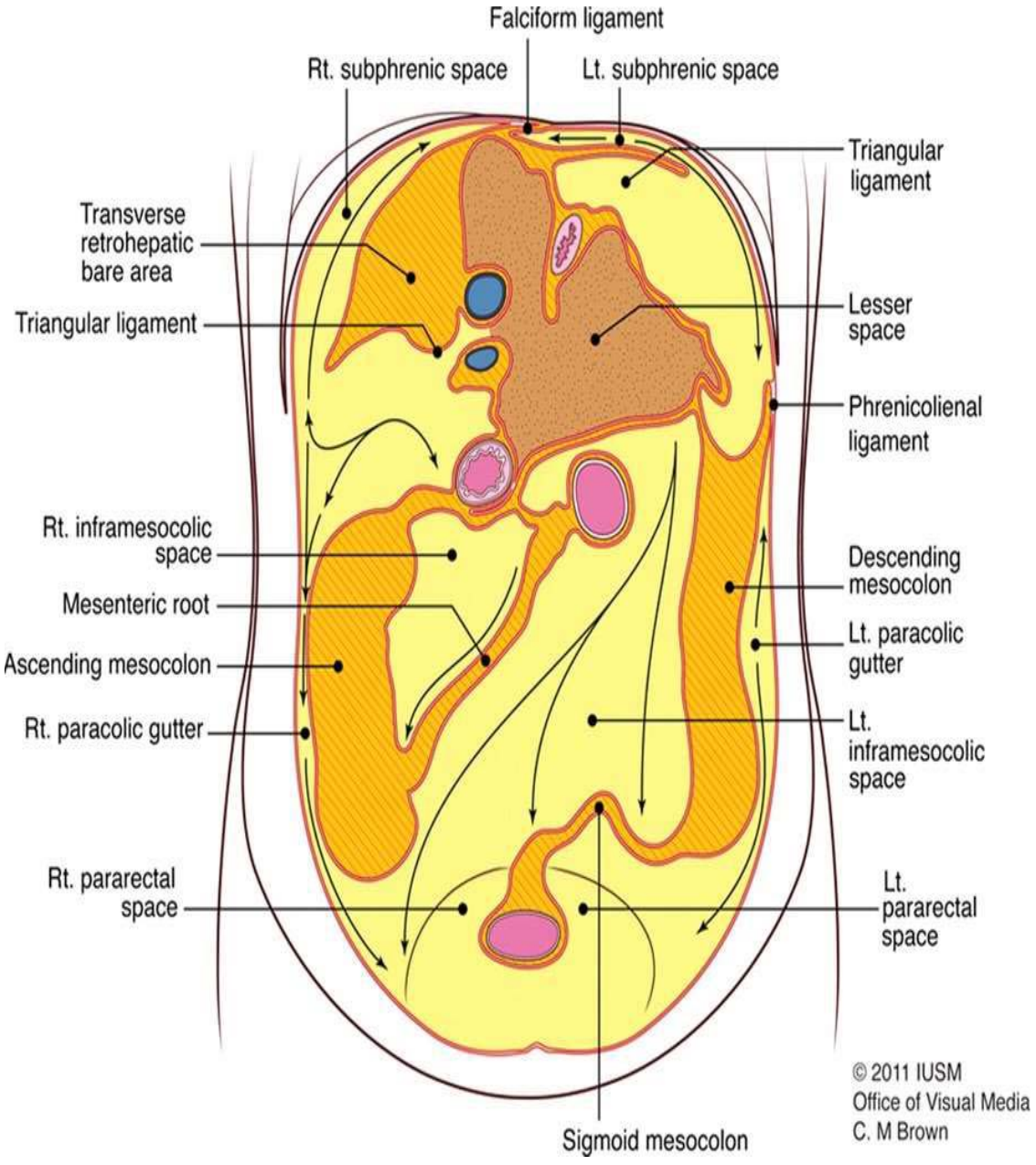


Fig.no.2: Peritoneal cavity and spaces

Subphrenic spaces:

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

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

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

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

4. Left posterior (inferior) (subphrenic)[29]

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 subphrenic space):

It lies between the right lobe of liver and the diaphragm. It is limited posteriorly by the anterior layer of the coronary and the right triangular ligaments and to 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 Morrison's or hepatorenal pouch. It is bounded on the right by the right lobe of the liver and the diaphragm. To the left is situated the foramen of Winslow and below this lies the duodenum. In front are the liver and the gallbladder and behind, the upper part of the right kidney and diaphragm. The space is bounded above by the liver and below by the transverse colon and hepatic flexure. It is the deepest space and 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 bounded above by the diaphragm and behind by the left triangular ligament and the left lobe of the liver, the gastrohepatic omentum and anterior surface of the stomach. To the right is the falciform and to the left the spleen, gastrosplenic omentum 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):

It is another name for the lesser sac. The commonest 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.

Extraperitoneal spaces

The right and left extraperitoneal space is the site for perinephric abscess. Midline extra peritoneal space is another name for the bare area of the liver. This area may develop 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. cuboidal and flattened cells. Gaps (stomata) between neighbouring cells of peritoneal membrane are found only among cuboidal cells. Peritonitis increases the width of these stomata. Beneath mesothelial cells is a 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[28.]

The mesothelial lining cells of the peritoneum secretes serous fluids that circulate within the peritoneal cavity. The peritoneal cavity contains 50- 100 ml of fluids with solute concentrations nearly identical to that of plasma[30]. 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 process of endocytosis. This bi-directional movement of fluids across peritoneal membranes has been used in peritoneal dialysis.

Two primary forces govern the movements of fluids within the peritoneal cavity. (a) Gravity (b) Negative pressure created beneath the diaphragm with each normal respiratory cycle. Subphrenic collections occur frequently because a 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.

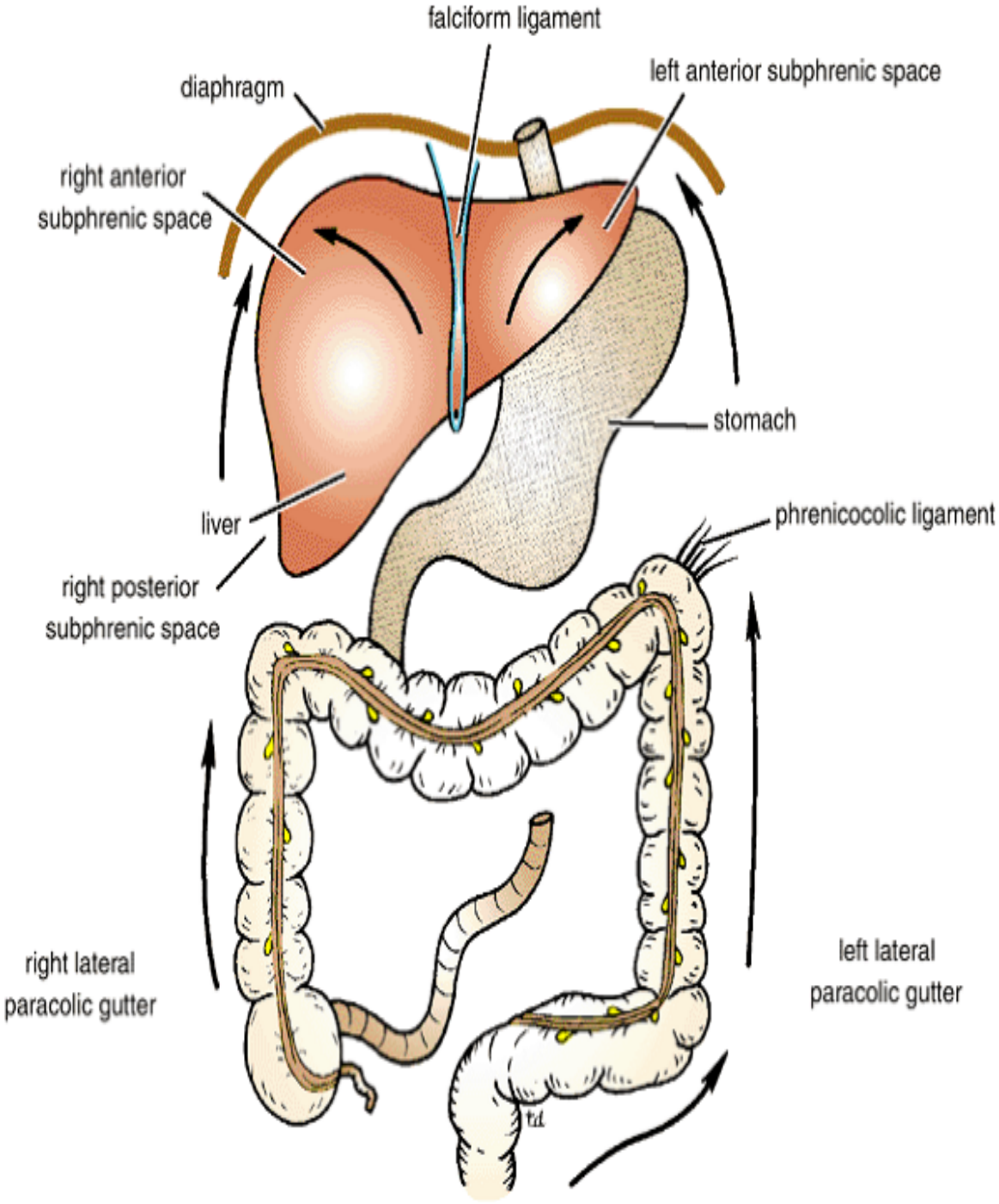


Fig.no . 3: Normal direction of flow of peritoneal fluid

PERITONEAL RESPONSE TO INJURY:

Any inflammatory event in the peritoneal cavity results in the peritoneal irritation with loss of regional mesothelial cells. A large peritoneal defect heals in the same amount of time as a small 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. 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[28]

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[28]. 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.

HYPOVOLEMIA:

Peritoneum reacts to injury by hyperemia and transudation of plasma like fluid from the extracellular, intracellular, and interstitial compartments into the peritoneal space. The loose connective tissue beneath the mesothelium of the viscera, mesentery and parities trap extra cellular fluid as edema. The atonic bowel also accumulates the fluid

derived from extra cellular space. This translocation of water, electrolytes, and proteins into a sequestered “THIRD SPACE” functionally removes this volume temporarily from the body economy. The rate of functional extracellular fluid loss is proportional to the surface area of peritoneum involved in the inflammatory process. With extensive 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 secretion of cortical hormones 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, both of 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 brings about secondary dysfunction in cardiac contractility and a further decrease in cardiac output.

RESPIRATORY RESPONSE:

Abdominal distension, primarily due to adynamic ileus, coupled with restricted diaphragmatic and intercostal muscle movements because of pain, results in decrease in ventilator volume and early appearance 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 oxygen demand. Simultaneously the capacity of lungs and heart to deliver oxygen is reduced. Poor circulation leads to shift 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- >380C or <360C
   - Heart rate > 90/ min
   - Respiratory rate > 20/ min or PaCO2 < 32 mmHg
   - WBC count >12000/mm3 or <4000 mm3 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-a). Others are IL-1, IL-6, Endotoxin, Endothelium, and leucocytes.

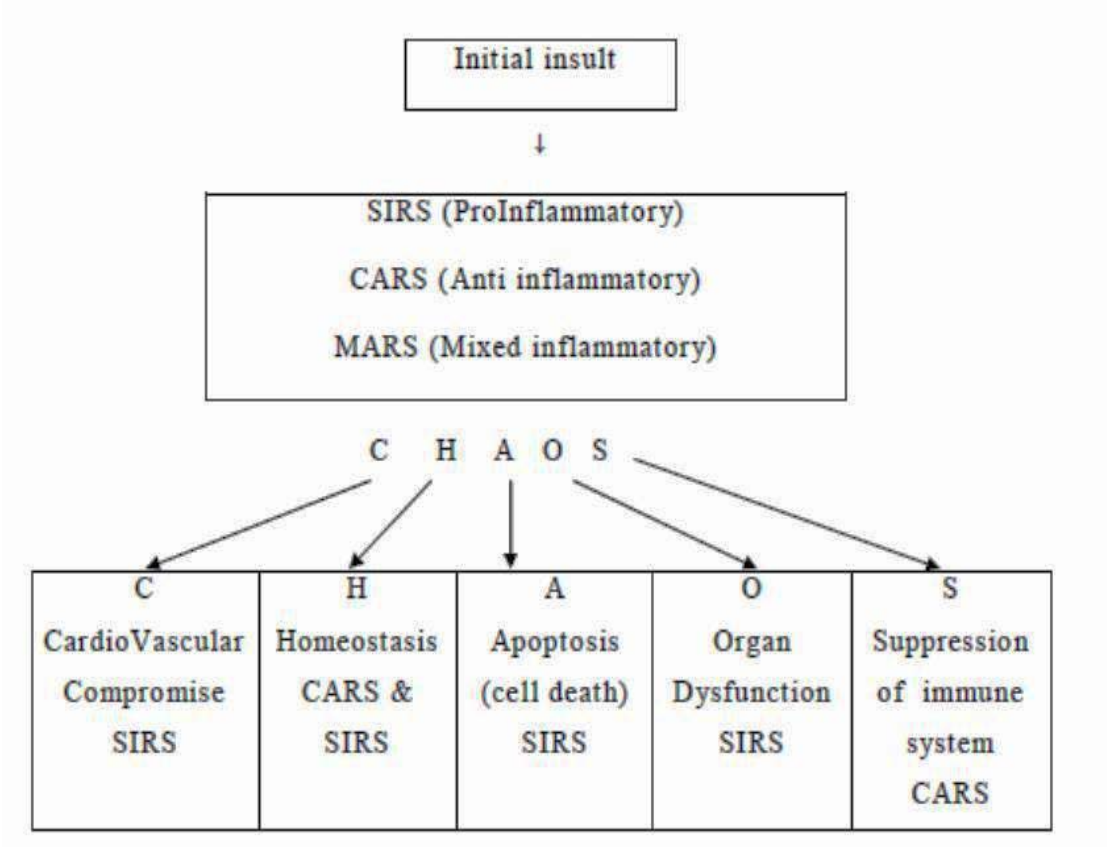
EFFECTS OF SIRS

There will be increased peripheral vasodilatation, microvascular permeability, microvascular clotting and leukocyte/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 spectrum of consequences of these responses has been termed CHAOS.



FACTORS THAT MAY FAVOUR THE DEVELOPMENT OF GENERALISED PERITONITIS:

- Speed of peritoneal contaminant is a prime factor in the spread of peritonitis.
- Stimulation of peristalsis by the ingestion of food hinders localization.
- The virulence of the infecting organism.
- Young children, who have small omentum.
- Disruption of localized collections.
- Deficient natural resistance (immune deficiency)[28].

BACTERIOLOGY OF PERITONITIS

Peritonitis as a disease process is characteristically polymicrobial in nature

Paths of bacterial invasion of peritoneal space:

- Direct infection.
- Local extension from an 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 bacteroids . Less frequently clostridium welchii is also found. Bacteroids 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[30]. 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.fragilisis introduced into the peritoneal cavity with other aero tolerant microbes, the anaerobe becomes associated with an abscess phase of the peritoneal infection. When largeinocula 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.

Table 1 : Bacteria commonly encountered in peritonitis

Facultative anaerobes and Gram-negative aerobes	**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 first experienced 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 frequent 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 gravest 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 silent, soft abdomen. The condition may localize, producing one or more abscesses, with overlying swelling and tenderness.

Terminal phase:

If resolution or localization 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[31]

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 involvement of parietal peritoneum by inflammation, also by reflex spasm may be initially present. As peritonitis advances reflex spasm may 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[31].

INVESTIGATIONS OF PATIENT WITH PERITONITIS: -

A number of diagnosis may elucidate doubtful diagnosis, but in the diagnosis, the clinician should rely on history and physical findings mainly.

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/mm^3 (indicator of inflammation) and bacteria (indicator of infection). Fluid is also examined for cell count, differential, PH and gram stain and aerobic and anaerobic culture[30].

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 right dome of diaphragm) ground glass appearance, obliteration of peritoneal pad of fat line and psoas shadow 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.
- 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[31]

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 patients condition at specific points in the disease process aid our understanding of these problems[32]. 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) 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 Wacha16 , 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 post-operative peritonitis and appendicitis, but further investigation that extension to these groups did not reduce the predictive value.

MPI

Risk factor	Score
Age >50 years	5
Female sex	5
Organ failure*	7
Malignancy	4
Preoperative duration of peritonitis >24 h	4
Origin of sepsis not colonic	4
Diffuse generalized peritonitis	6
Exudates:	
Clear	0
Cloudy, purulent	6
Fecal	12

*Kidney failure: Creatinine level > 177 mmol/L or urea level > 167 mmol/L or oliguria < 20 mL/h; pulmonary insufficiency: PO_2 < 50 mmHg or PCO_2 > 50 mmHg; intestinal obstruction/paralysis > 24 h or complete mechanical ileus; shock hypodynamic or hyperdynamic

MPI scoring system

Detailed study of MPI was done by A. Billing[24] 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 cutoff 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 monitory. 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 perforation are usually secondary to malignancy, this may not be applicable uniformly.

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

BOEY SCORING SYSTEM[33]

(a) Shock at admission (systolic blood pressure <90 mmHg),

(b) Severe medical illness (ASA III–V), and

(c) Delayed presentation (duration of symptoms >24 h).

Risk Factors	No. of Risk Factors	Risk of Mortality Boey
	0	0
Preoperative BP < 100 mmHg	1	10%
Delayed presentation > 24 h	2	45.5%
Major medical illness present	3	100%

Table no.2: Boey scoring system

Advantages

Simple, easy to remember and apply.

Disadvantages

1. Does not consider various other physiological factors which do have a significant role in predicting the patients condition.

2. Less accurate.

Haceteppe score[34] – used in peptic ulcer perforation

The four variables in the study

- The presence of a serious coexisting medical illness,
- Acute renal failure,
- White cell count of more than 20 × 109/l, and
- Male sex.

There has been no study to revalidate this score or test its accuracy against others.

Sickness assessment[35]

Kennedy *et al* - first described this scoring system.

- Hypotension;
- Severe chronic disease and
- Whether or not the patient was independent and self-caring.

These conditions were clearly defined. In the group of patients with a SA score of zero, there were no deaths. Mortality in patients with one, two and three parameters present was 52%, 60% and 100% respectively. Not widely used.

Fitness score[36]

Playforth *et al* in 1987 introduced this scoring system .The 26 risk factors were chosen by the authors and weighted arbitrarily from 1 – 4.

In addition to the difficulty of scoring 26 variables preoperatively, some, such as the presence of perforation or obstruction and diagnosis of cancer, may not be available before surgery

Reiss index:[37]

Factors considered :

- Age
- Urgency of surgery
- ASA

- Presence of malignancy and
- Diagnosis

An emergency laparotomy where the diagnosis was unknown could not be scored with this system, which has been shown to be inferior to the ASA classification in predicting postoperative morbidity and mortality. Scoring systems such as the Reiss Index or Fitness Score can be used pre-operatively if there is time to gain enough data to complete the scoring.

Sepsis scores

As well as the APACHE score, several other scoring systems have been developed for intra-abdominal sepsis.

These scores include the Simplified Acute Physiology Score (SAPS), Sepsis Score, Multiple Organ Failure Score and Mannheim Peritonitis Index (MPI)

Scores predicting morbidity

Veltkamp score - 11 patient, disease and surgery-related variables are used .Minor complications are less-successfully predicted hence less commonly used.

VA respiratory failure prediction index - The VA study was modelled on over 80, 000 men who developed respiratory failure (defined

as mechanical ventilation for 48 hours or more) after (non-cardiac) surgery.

Weighted scores are given for type of surgery, emergency surgery (less than 12 hours after admission), albumin, urea, pre-morbid functional status, respiratory function history and age. A score over 40 predicts a risk of respiratory failure of 31%.

POSSUM scoring – Physiological and operative severity score for the enumeration of mortality based on Copeland, Jones and Walters Br J Surg (1991)

Scores calculated taking into consideration 2 parameters

1. Physiological severity –Age, cardiac signs, respiratory signs, systolic blood pressure, pulse, Glasgow coma scale, hemoglobin, total count, urea, sodium, potassium and ECG.

2. Operative severity – Multiple procedures, total blood loss, peritoneal soiling, malignancy, operative severity and mode of surgery.

Considered to be midway between too simple ASA scoring and too complex APACHEII scoring system.

Uses 12 physiological variants and 6 operative variants

Drawbacks

- Tends to overestimate the mortality in low risk patients
- Tends to overestimate if used in other specialties.

P POSSUM –Portsmouth predictor equation for mortality –

Prytherch et al Br J Surg 1998 introduced the corrected version of the scoring system. This scoring is more accurate than the original POSSUM scoring but it still overestimates the mortality in low risk patients. Higher the risk more is the accuracy of the scoring system. There have been new versions of this scoring system like V-POSSUM used specifically for specialties.

Predicted death rate=1/ (1+ e-R)

Where R is (0.1692×physiological score) + (0.1550×oerative score) -9.065 in POSSSUM R = (0.13×physiogical score) + (0.16 ×operative score)-7.04 in P-POSSUM

MANAGEMENT OF PERITONITIS

STANDARD TREATMENT:

Kirschner, in 1926, formulated two surgical principles for the management of peritonitis which later have become the gold standard[38].

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 many abscesses in 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[31].

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[39].

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.

Antimicrobial agent therapy for established secondary bacterial peritonotis

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[30]

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 salpingitis 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 infect ion or verify both and purge[38]

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, resect ion 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 adjuvant either contain bacteria or promote their growths and they should be removed. A large quantity of saline about 8-10 litres 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 litres 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[38]

- 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: It is indicated in the following conditions:-

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[28]

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 favour 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 lt. 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 IOPL during clean cases[40].

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 MANAGEMENT

Conservative management may be advisable in following conditions

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

COMPLICATIONS OF PERITONITIS

SYSTEMIC COMPLICATION OF PERITONITIS:

1. Bacteremic/endotoxic shock
2. Broncho 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:

Hypovolemia 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. Adhesional 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. Paracentecis 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[28,30].

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 graffian 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[41].

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.

2) 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.

3) 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 drainage procedures like gastro-enterostomy with or without 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 a 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 390 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 appendicectomy 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[31].

TYPHIOD PERFORATION:

Typhoid perforation is usually seen in the third week of infect ion 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 patients28 .

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, usually without 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[42].

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-coloured 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%), ascites (60%), night sweats (37%) and occasionally as abdominal mass.

Origin of infection:-

Infect ion originates from;

- Tuberculous mesenteric lymph nodes;
- Tuberculosis of ileocaecal region;
- A tuberculous pyosalphinx;
- Blood borne infection from pulmonary tuberculosis, usually the “military”, 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 coloured fluid. The onset is insidious. Pain is often completely absent; in other cases there is considerable abdominal discomfort, which may be associated with constipation of diarrhoea. 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 salpingitis. Amidst a mass of adherent intestine and omentum, tuberculous pus is present. Sizable 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 litre 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.

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[28].

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[28]. 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[31].

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 favourable 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 intermit tent 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 tumour (chronic inflammatory omental tumours) 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 fibres 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, in whom peritonitis and sepsis initially have been controlled operatively and in whom bacteria have been eliminated by

successful antibiotic therapy, may progress to tertiary peritonitis. It is a state in which, host defense 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 provide drainage of anticipated recurrent or residual collections of infected fluid. On operation, no pathogens are present. Empiric anti-infective therapy is of no value.

MALIGNANT PERITONITIS (CARCINOMA PERITONEI):

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 tumours of ovary and appendix. Recent studies suggest that most cases arise from primary appendiceal tumours 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 tumour. 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.[43]

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

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

a. Positive culture from the peritoneal fluid.

b. Cloudy dialysate effluent.

c. Clinical signs of peritonitis.

Treatment:

The initial treatment is administration of antibiotics 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 fungal or tubercular peritonitis, faecal peritonitis or severe skin infection at the catheter site[28]. 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 7th 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

Source of Data

This consists of all the patients who will get admitted with hollow viscous perforation in General Surgery department, Govt. Kilpauk Medical College and Hospital, Chennai were included in the study. Necessary data was collected; MPI score was calculated for each patient and analysis done.

Method of collection of data (including sampling procedure)

A. Study design: Cohort study

B. Place of study: Govt. Kilpauk Medical College and Hospital, Chennai.

C. Study sample size

64 cases of peritonitis due to hollow viscous perforation are followed up over one month to assess morbidity and mortality.

When mortality is 50% in those with MPI score $\geq$ 29 and Confidence level is 95%,

Power study at 80%, 14% mortality rate in those with MPI score 21-29.

D. Study period: April 2017 to September 2017

E. Method of sampling: Random sampling

F. Inclusion criteria:

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

Various aetiologies causing such features include,

1. Acid peptic disease
2. Typhoid
3. Tuberculosis
4. Appendicitis
5. Malignancy

G. Exclusion criteria:

1. Patients with hollow viscous perforation due to trauma
2. Patients with associated vascular diseases
3. Patients with any other significant illness which is likely to affect the outcome more than the disease in study.
4. Patients age greater than 70 years.

Once diagnosis of peritonitis had been determined by operative findings, the patient was enrolled into the study. Using history, clinical examination, and lab values risk factors found in MPI were classified according to values indicated and individual variable scores were added to establish MPI score. The cases were first grouped into three, as described by Billing: those below 21 pts, between 21-29 pts, and those above 29 pts.

In addition to personal data such as name, age, sex, etc., the following information was registered: file number; dates of admission and discharge from the hospital; days hospitalized; date of surgery and information related to illness (surgical findings, medical treatment and evolution of illness).

Patient evolution was followed, occurrence of complications and discharge due to improvement or death. Time elapsed from initial diagnosis to moment of event (death or discharge from hospital) was determined.

Out-patient follow-up was continued for 30 days to establish perioperative morbidity and mortality. The minimum possible score was zero, if no adverse factor were present, and maximum was 47 if presence of all were confirmed. Analysis was done with each variable in the scoring system as an independent predictor of morbidity or mortality and the scoring system as a whole.

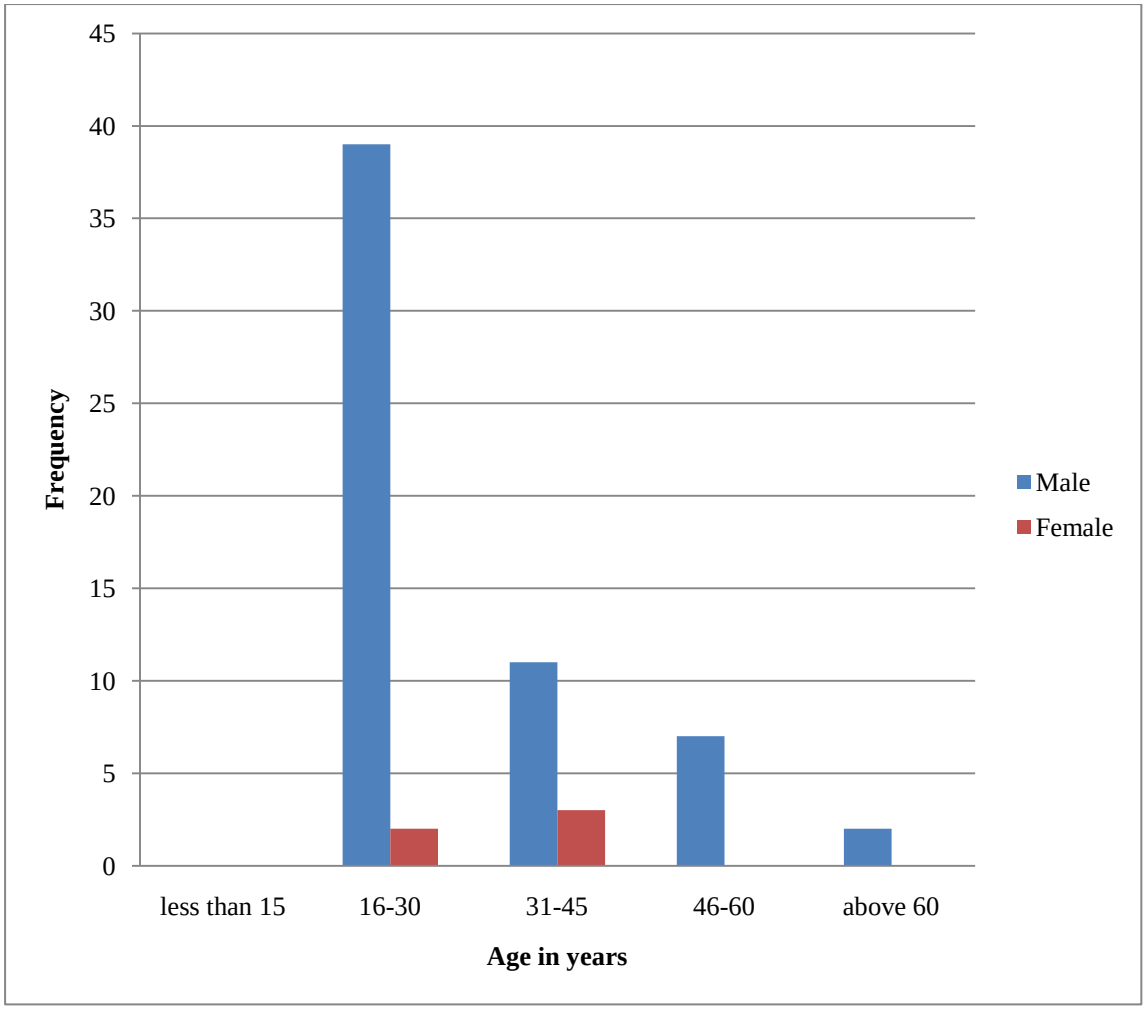
OBSERVATION AND RESULTS

In this study 64 cases of secondary and tertiary peritonitis who attended surgical emergency unit were selected over a period of six months from April2017 to September 2017.

TABLE 1

	Sex		**Total**
Age	**Male**	**Female**	
Less than 15			
16-30	39	2	41 (64.06%)
31-45	11	3	14 (21.9%)
46-60	7		7 (10.93%)
Above 60	2		2 (3.12%)
Total	59	5	64 (100%)

In the study, 64 patients with diagnosis of secondary peritonitis were included. The mean age of patients was 29.86 (SD 12.27) years ranging from 16 to 75 and majority of patients (64.06%) belonged to age group of 16-30 years. There was male preponderance (92.1%)



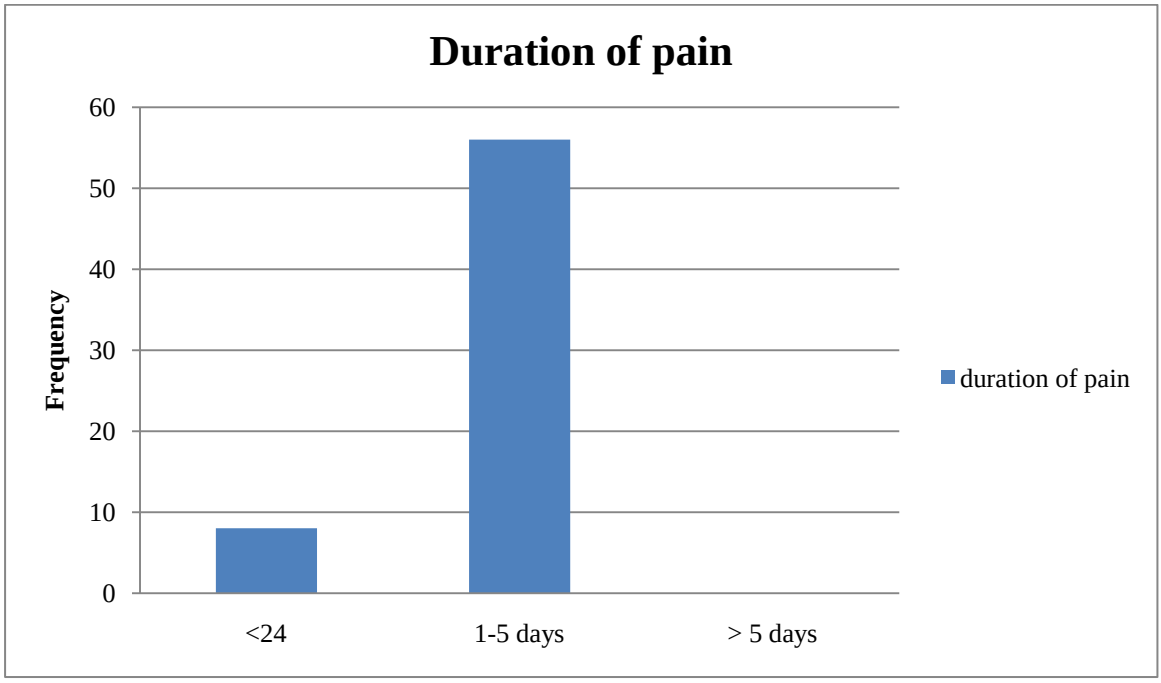
Graph no 1.: Age and sex wise distribution of subjects

Table 2

Time of presentation of study subjects

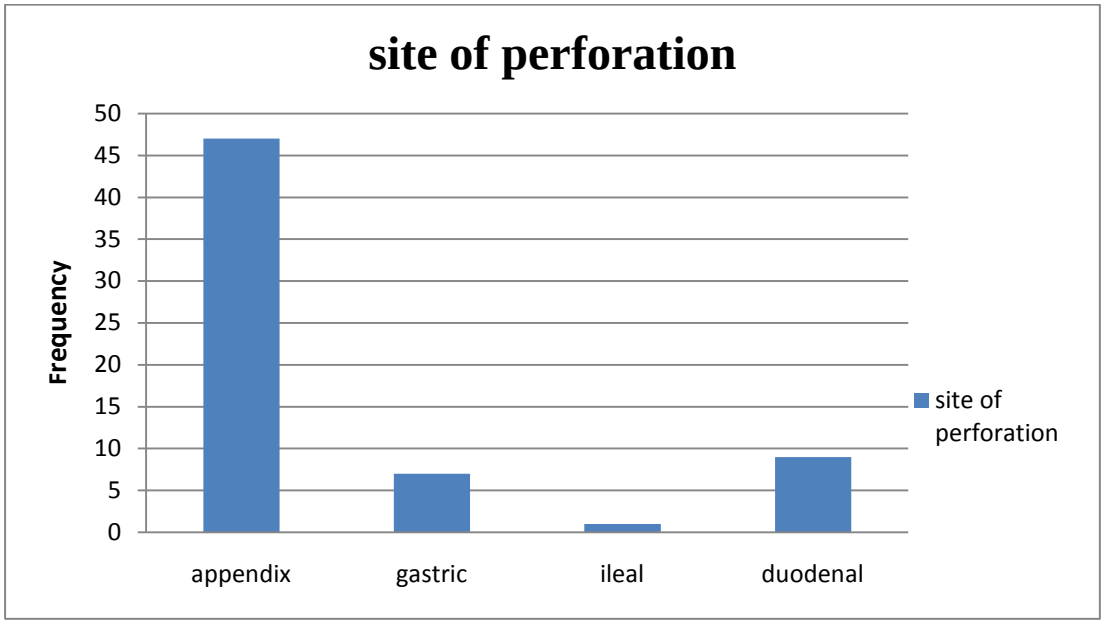
Duration of pain	Patients	Percent
<24 hrs	8	12.5%
1-5 days	56	87.5%
>5 days		
Total	64	100%

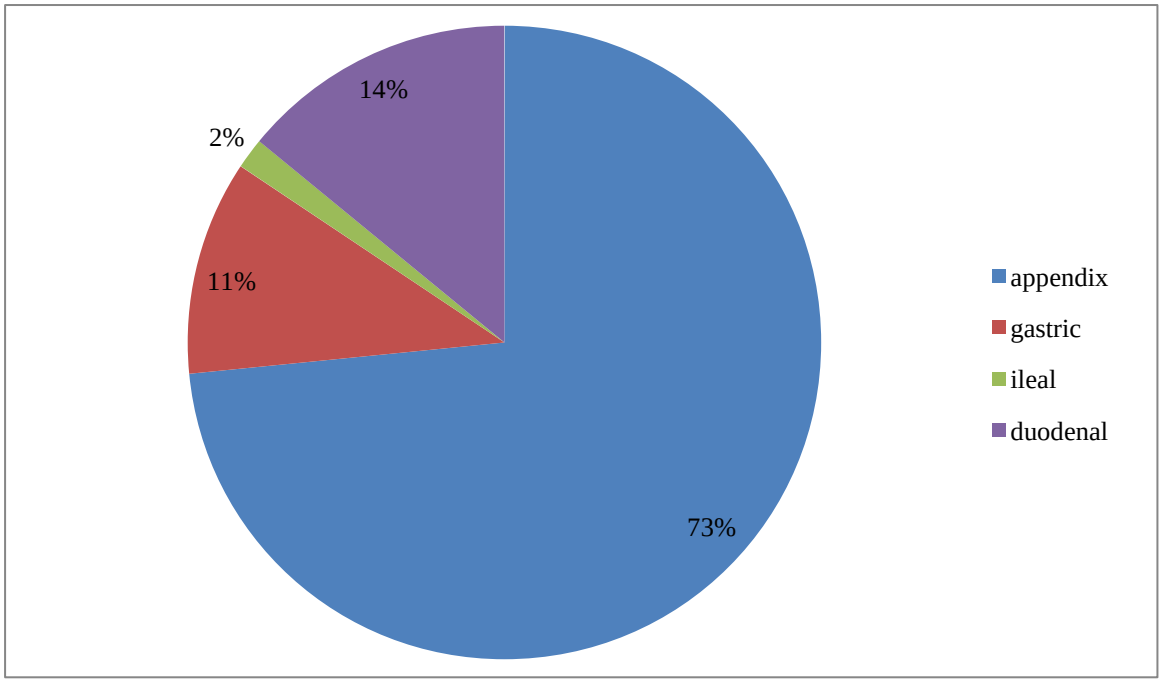
In the study group of 64 patients, majority of patients presented to the hospital after 24 hrs of onset of symptoms and the mortality of those patients who presented within 1 to 5 days and was higher than as compared to mortality in patients who presented on the first day of onset of symptoms.



Graph no 2 . Duration of pain

Site of perforation



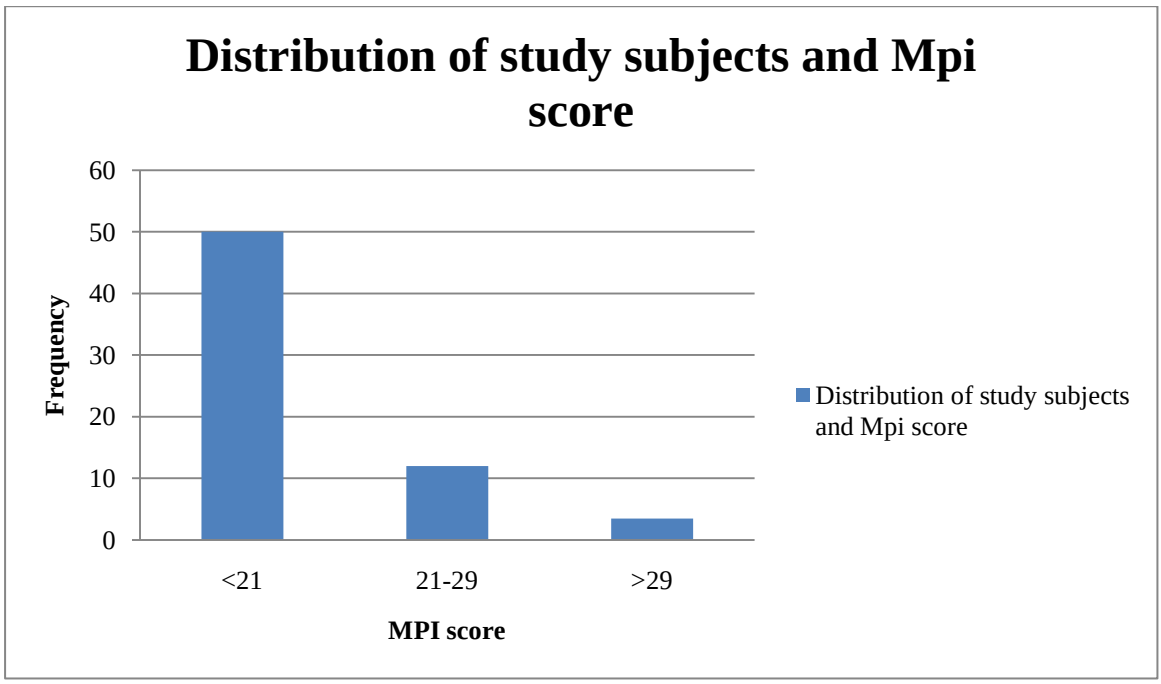


Graph no 3 .:Etiological distribution

Table no 3 . Distribution of study subjects and MPI

MPI score	Frequency	Percent
<21	50	78.12%
21-29	12	18.76%
>29	2	3.12%
Total	64	100%

In the study group 78.12% population was in the low risk group (<21) and 18.75% were in the moderate risk group , 3.12% were in the high risk group (>29). Patients with organ failure on admission , longer duration of illness before the surgery , diffuse peritonitis , feculent exudates were more likely to have higher scores and hence fall into high risk group than their counterparts.

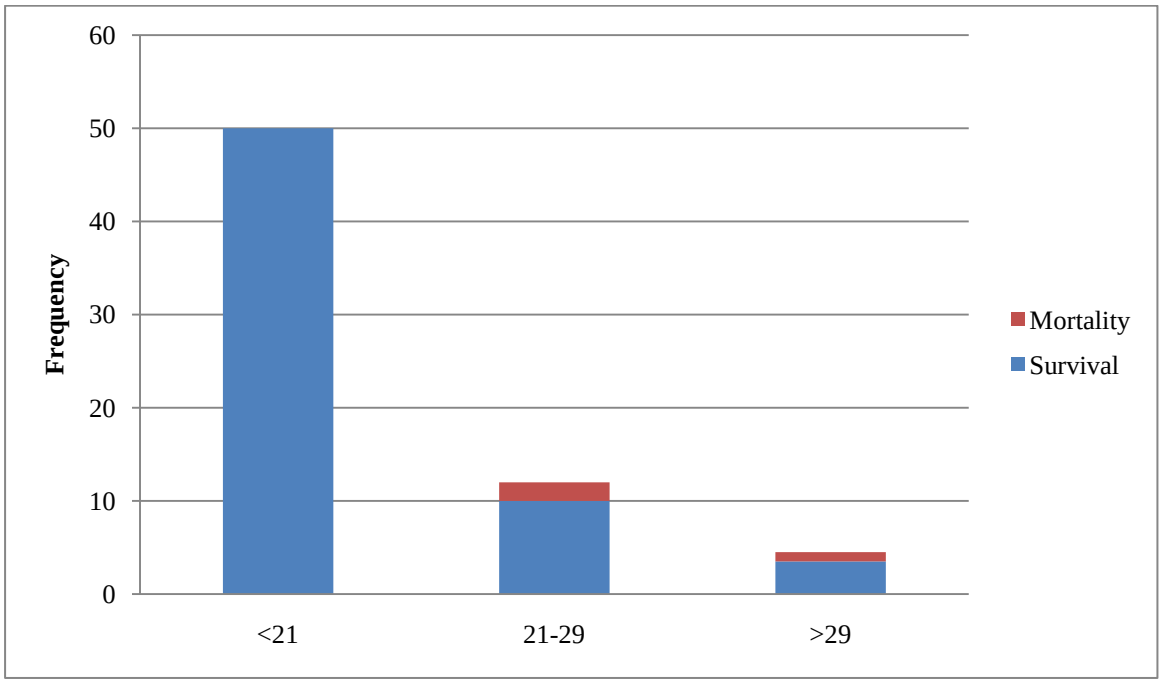


Graph no 4 : Distribution of study subjects and Mpi score

Table no 4: Mortality and MPI score

Mpi score	Patients	Mortality	Percent
<21	50		
21-29	12	2	16.67%
>29	2	1	50%
Total	64	3	

In the study group, 50% of patients had mortality among patients with MPI score more than 29 and none of the patients died with MPI score less than 21, 16.67% of patients had mortality in mpi score 21-29.

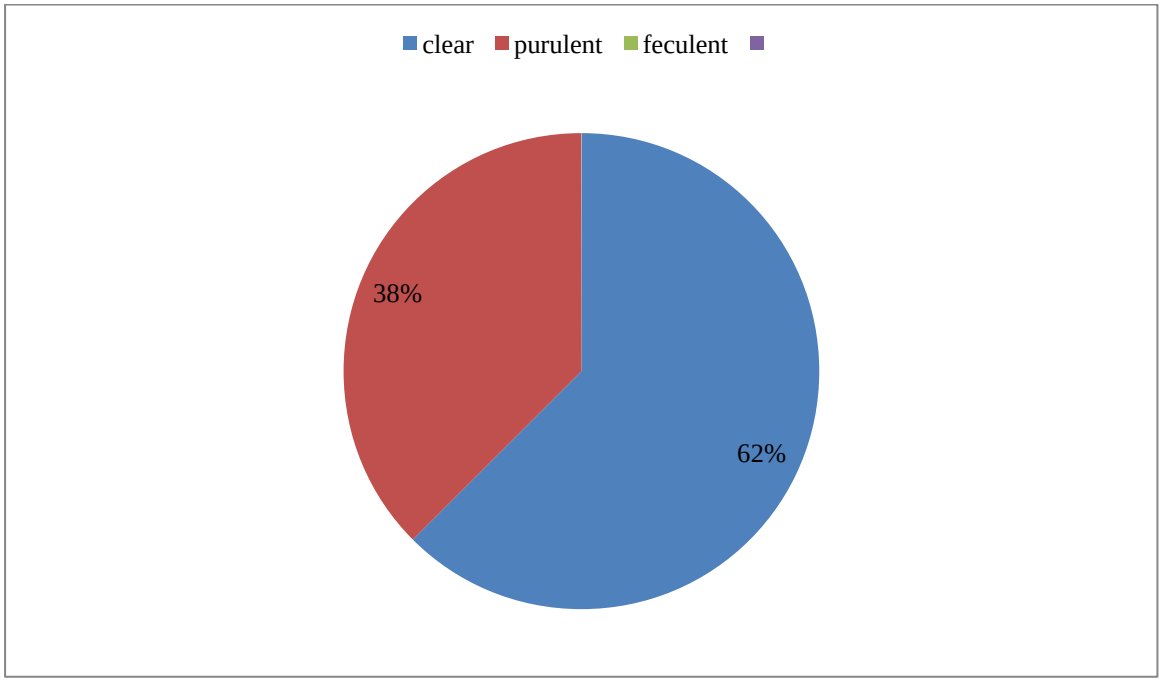


Graph no 5 : MPI Score

Table no 5 : Exudates

Exudates	Frequency	Percent
Clear	40	62.5
Purulent	24	37.5
Feculent	-	-
	64	100%

Presence of feculent or purulent exudates was reflected in higher eventual scores. Feculent and purulent exudates were associated with significantly increased post op complications requiring increased hospital stay. Patients with clear exudates had no post op complications.

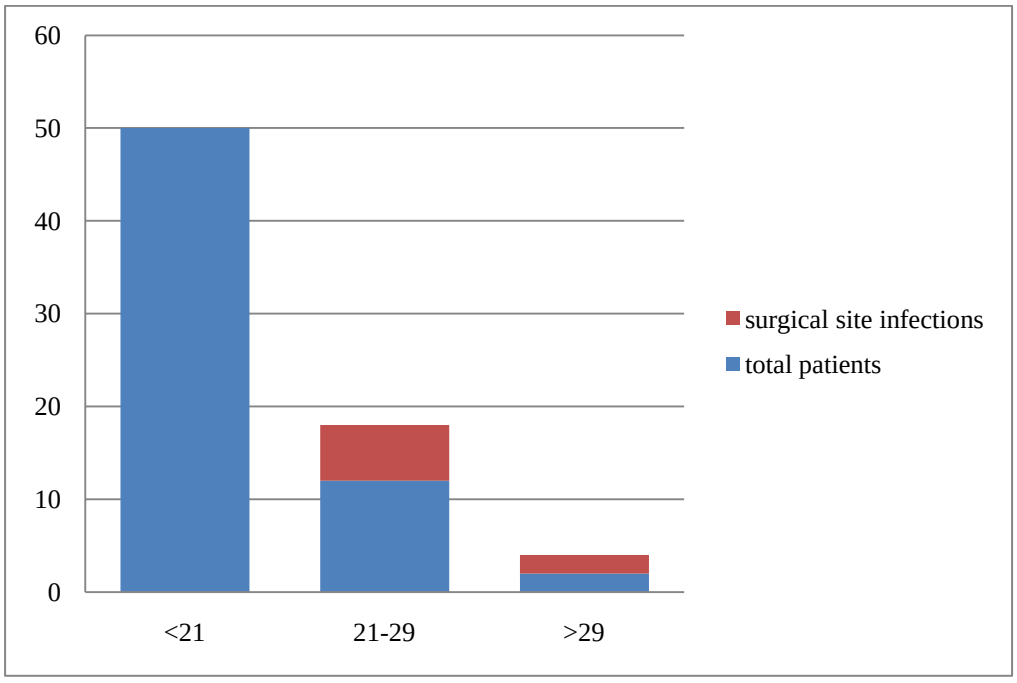


Graph no 6 : Exudate

Table no 6 : Development of wound complications

		Score			Total
SSI		**<21**	**21-29**	**>29**	
	No	50	6	-	56
	Yes	-	6	2	8
Total		50	12	2	64

100% of the patients with scores > 29 developed wound related complications in the post op period which was about 50% in patients with score 21-29 and patients with scores <21 did not develop wound related complications. The post op complications were significantly higher in the group with score>29. This included the surgical site infections, pulmonary, renal complications and development of multi organ failure.



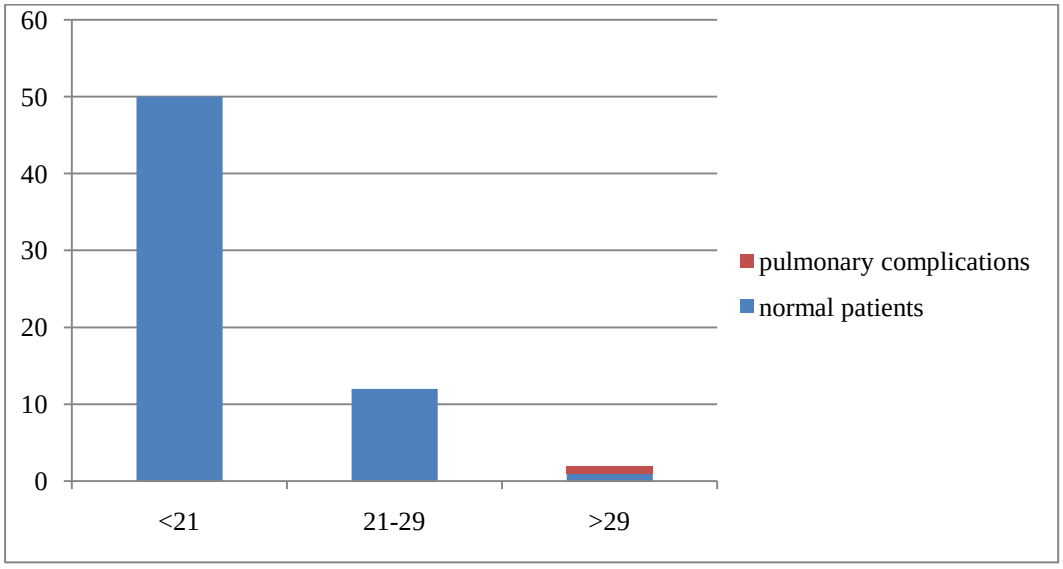
Graph no:7 Wound complications

Table no 7: Pulmonary complications

		MPI score			
Pulmonary		**<21**	**21-29**	**>29**	
	No	50	12	1	63
	Yes			1	1
Total		50	12	2	64

The pulmonary complications in the form of post op pneumonia, pleural effusion which required continuous monitoring of oxygen saturation, nebulisation and hence lead to longer post op recovery were significantly higher as the score increased.

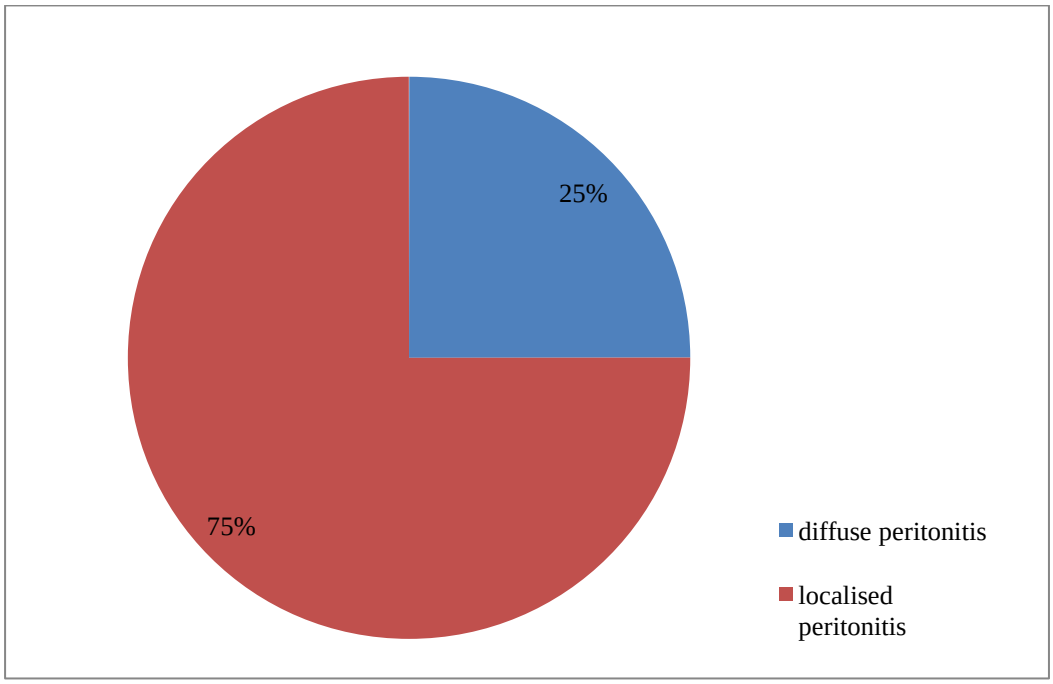
50% of patients with >29 had some form of pulmonary complications.



Graph no 8 : Pulmonary complications

Peritonitis

25% of the study population presented with diffuse peritonitis and 75% with localized peritonitis.



Graph no 9 .: Peritonitis

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 to morbidity and mortality in those patients.

In the present study 64 cases of peritonitis those attended Government Kilpauk medical hospital from April 2017 to September 2017 were included with age ranging from 15 to 70 years. The mean age of the patients was 29.86 (SD 12.27) years. There was male preponderance (92.1%) with male to female ratio of 14.7:1. In our study the most common etiology of peritonitis was appendicular perforation(73%). duodenal perforation was seen in 14% of patients, followed by gastric (11%), and ileal (2%),.

Ohmann et al. reported duodenal ulcer perforation as the commonest cause for peritonitis in his series while Kachroo et al. found appendicular perforation as the commonest cause. The overall diagnostic accuracy for peritonitis was 97.3%.

In the study group of 64 patients, majority of patients presented to the hospital after 24 hrs of onset of symptoms and the mortality of those patients who presented after 24 hrs of onset of symptoms was higher than as compared to mortality in patients who presented on the first day of onset of symptoms.

In the study group of 64 patients, 78.1% of patients had MPI score less than 21, did not develop wound related complications with 0 % mortality and 50% of patients had morbidity (wound infection) and 16.67% mortality with MPI score 21 to 29 and those patients with MPI score than 29 had the highest mortlity i.e. 50%.

In Billing A, Fröhlich D, Schildberg FW., patients with scores of less than 21 had a mortality rate ranging from 0-2.3% and those with MPI between 21 and 29 had a mortality rate of approximately 65%. MPI score of more than 29 had the highest mortality, up to more than 80% in some studies.

Notash AY, Salimi J, Rahimian H, Fesharaki MH, Abbasi A. have shown important cut-off points to be 21 and 29 when using the MPI, with mortality of 60%, and up to 100% for scores more than 29.

Kusumoto yoshiko et al., evaluated the reliability of the MPI in predicting the outcome of patients with peritonitis in 108 patients. 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% [19].

In a study conducted by Qureshi AM et al., score of < 21 had mortality of 1.9%, score of 21-29 had 21.9% and score > 30 had mortality of 28.1%. Mortality rate for MPI score more than 26 was 28.1% while for scores less than 26 it was 4.3% [20].

Malik AA et al., did prospective study using 101 consecutive patients having generalized peritonitis over a two-year period. In the MPI system, mortality was 0 in the group of patients with a score of less than 15, while it was 4% in the patients scoring 16-25 and 82.3% in those with scores of more than 25.

What draws one's attention when comparing the results of studies of the MPI conducted in the last 30 years is the repetition of the most important risk factors in a significant number of studies, namely: organ failure, age above 50 years, faecal nature of fluid in the peritoneal cavity, neoplastic cause, exit site outside of the colon, diffuse peritonitis and presence of symptoms more than 24 h before the procedure.

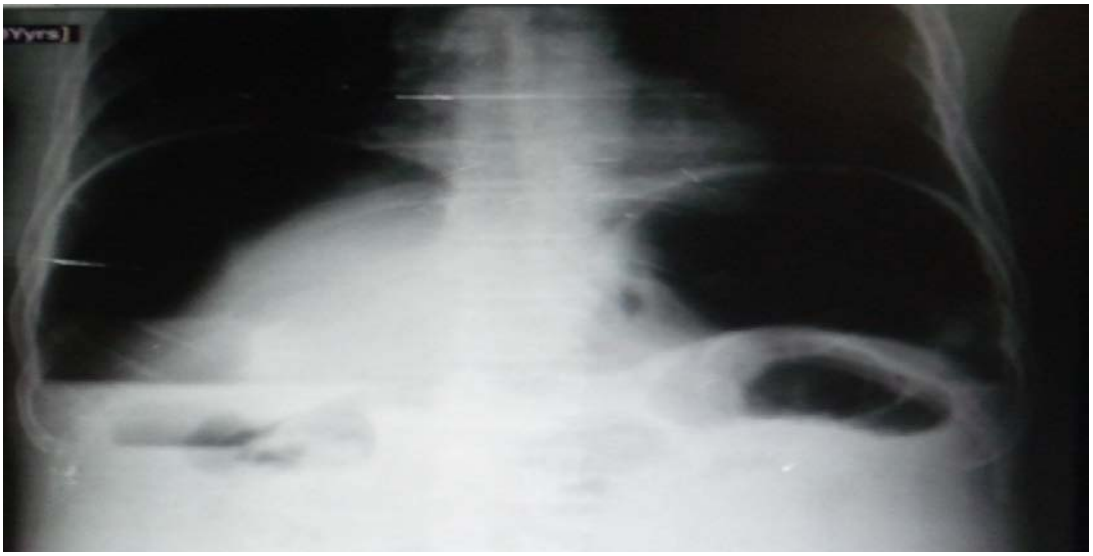
Analysis of the collected material revealed that division of patients based on the obtained MPI score may help assess the risk of developing serious disturbances of the general condition in the postoperative period as well as the necessity of continued treatment of the patient in an intensive care unit or relaparotomy. Sensible use of the score will facilitate identification of patients in the high-risk group, thus possibly raising awareness of their increased risk of postoperative complications, such as: cardiorespiratory failure, acidosis, electrolyte disorders and postoperative wound complications.

Despite the fact that the Mannheim score is easy to use and effective in predicting mortality, it cannot be used as a preoperative system used at admission to stratify patients based on the risk of death, since it requires consideration of intraoperative assessment, such as the nature of fluid in the peritoneal cavity and anatomical exit site as well as histopathological assessment (a cause of neoplastic or non-neoplastic origin). Other disadvantage of the score is the fact that it does not take into account chronic diseases and major systemic disorders, which are very important risk factors for death and serious complications.

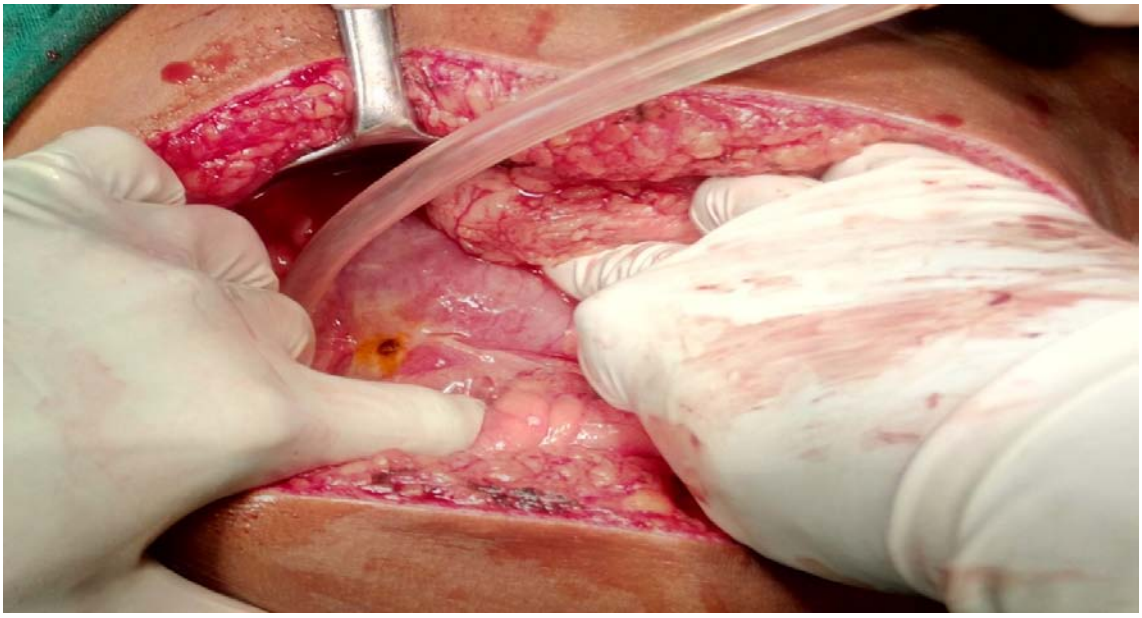
CONCLUSION

- There have been several attempts at creating a scoring system to predict mortality and morbidity risk after emergency surgery.
- Some scoring systems provide a prediction that approximates to the observed mortality rate for a cohort, but none is sufficiently accurate to rely upon when considering an individual patient.
- This is a validation study of the MANNHEIM PERITONITIS INDEX scoring system for predicting the morbidity and mortality in patients with peritonitis due to hollow viscous perforation.
- The results of this study proves that MPI scoring system is a simple and effective tool for assessing this group of patients, and can be used as a guiding tool to decide on the management of the patient.
- Among the various variables of the scoring system duration of pain, organ failure on presentation had a significant hand in predicting the eventual outcome of the patient.

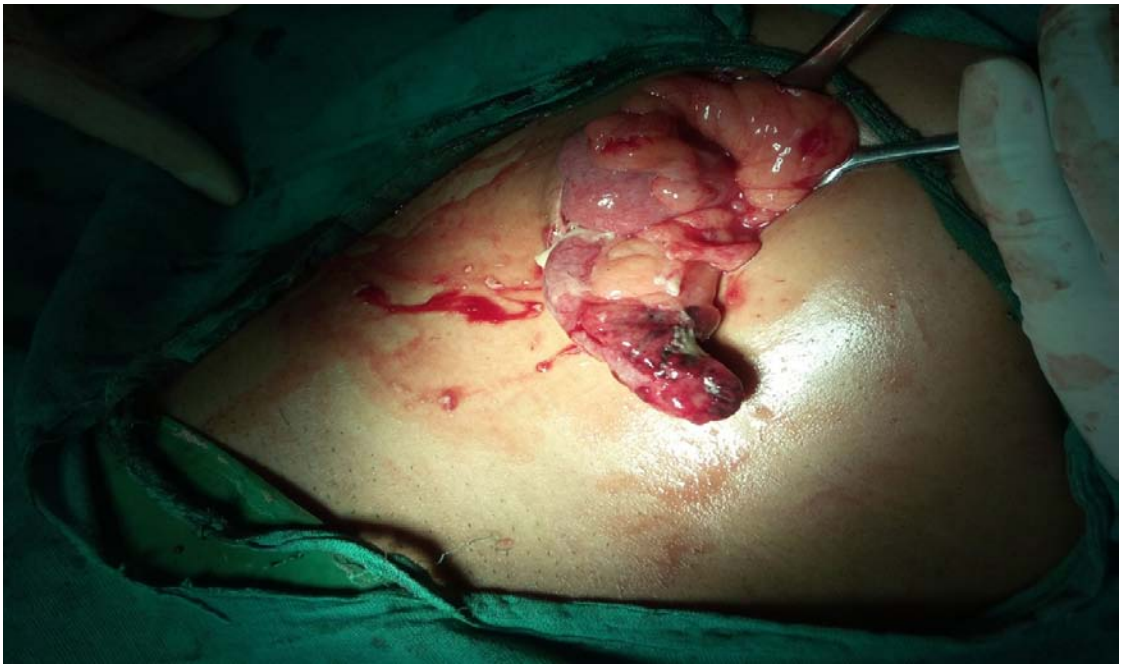
PHOTOGRAPHS



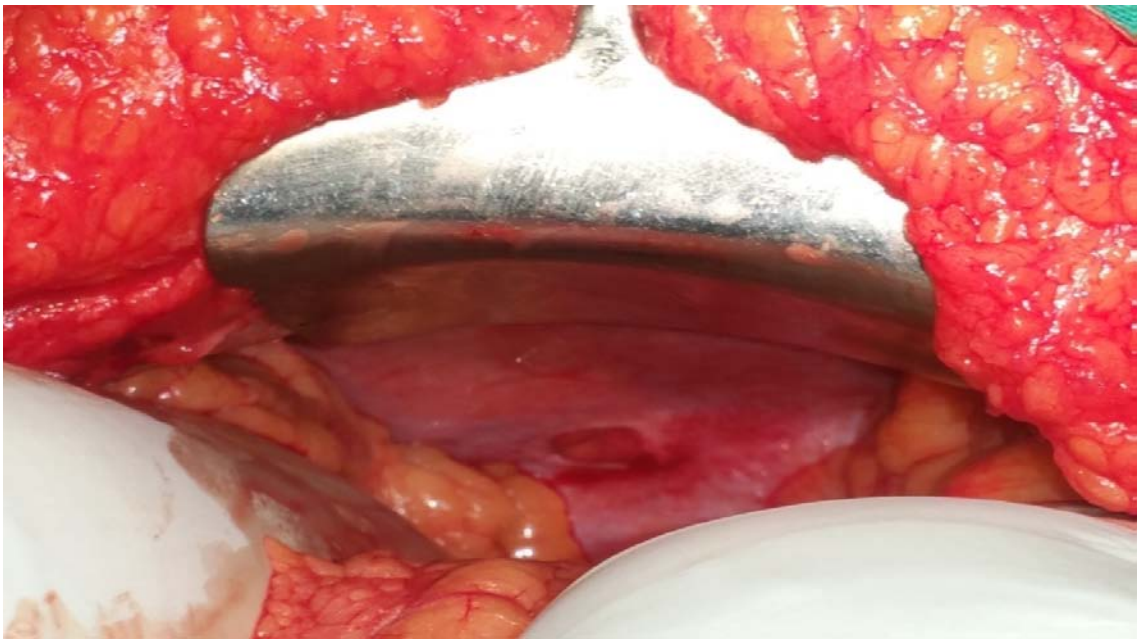
Plain radiograph photo of peritonitis patient



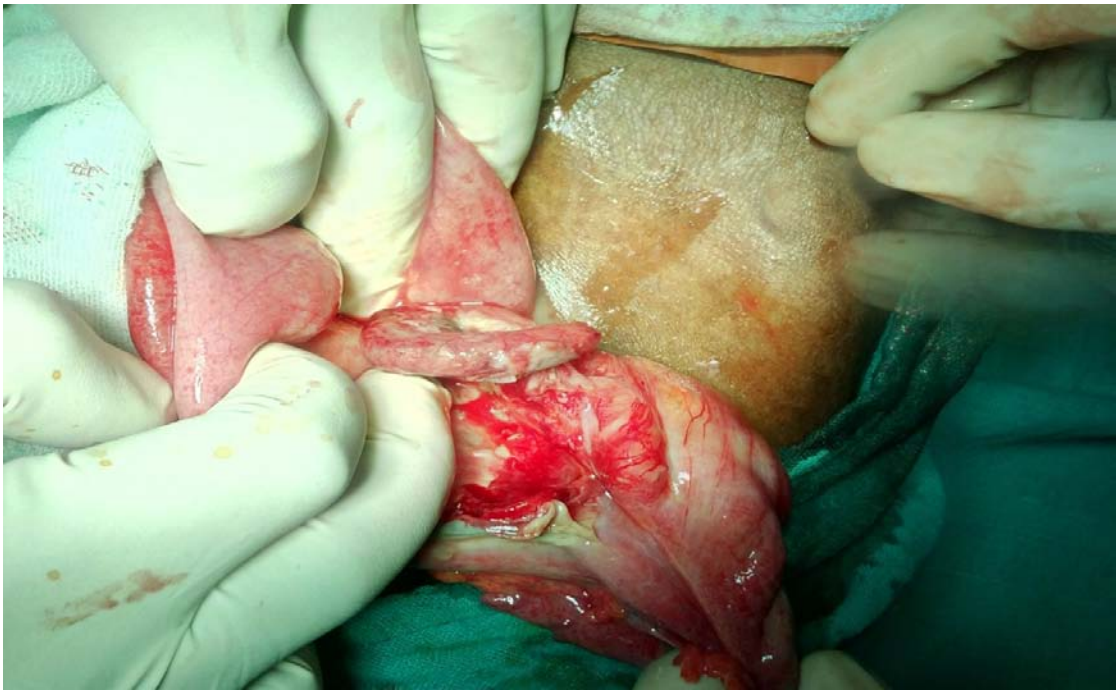
Intra operative photo of Duodenal perforation



Intra operative photo of Appendicular perforation



Intra operative photo of Gastric perforation



Intra operative photo of Appendicular perforation

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ANNEXURES

PROFORMA

Name	I.P.No
Age	DOA
Sex	
Marital status :	DOD
Occupation	Unit
Address	

A. CHIEF COMPLAINTS

Pain abdomen

Fever

Vomiting

Indigestion

Loss of appetite

Abdominal distension :

Bowel Disturbances :

Urinary Disturbances :

Loss of weight

Any other

B. HISTORY OF PRESENTING ILLNESS

1. Pain abdomen

Site

Duration

Mode of onset Insidious / Sudden

Severity

Nature - Aching / burning / stabbing / constricting

Throbbing / colicky / distending

Relieving factors

Exacerbating factors :

Radiation

2. Fever

Duration

Type - Continuous / intermittent

Associated features : High / low / moderate

Grade

3. Vomiting

Duration

Frequency

Spontaneous / Induced:

Nature - Food particles / Digested food / clear acidic fluid Bilious /coffee ground / feculent

4. Indigestion - Discomfort after food / fullness

5. Loss of appetite - Yes / No

6. Abdominal distension : Onset, Progress, Associate factors

Pain, Relieving factors

7. Bowel disturbances: Frequency , Constipation / diarrhea

Tenesmus, H/o passing worms

Physical characters

8. Urinary disturbance: Frequency, Quantity

Pain, Haematuria, Color

9. Loss of weight Yes

Percentage

Duration

10. Any other

C. PAST HISTORY

Similar illness**,**Any other illness

Any history ofsurgeries,

Tuberculosis,Diabetes,Hypertension

D. FAMILY HISTORY

Malignancies

Similar illness

E.PERSONAL HISTORY

Smoking**,**Alcohol

Type of diet,Any other habits

Bowel habits,Bladder habits

F.DRUG HISTORY

ATT, Steroids ,Insulin

G. MENSTRUAL HISTORY

Menarche,Menstrual cycles,Menopause ,Any other disturbances

H. SOCIAL HISTORY

Marital status

Socio-economic status

I. GENERAL PHYSICAL EXAMINATION

Built - Well / Moderate / Poor

Nourishment - Well / Moderate / Poor

Pallor - Mild / Moderate / Severe

Icterus - Mild / Deep

Pedal edema - Pitting / Non Pitting

Febrile - Yes / No

Dehydration - Yes / No

Gen. Lymphadenopathy - Yes / No,,Group involved

Tender / non tender

Consistency – Soft / Firm / Rubbery / Hard Matted / Discrete,Mobility: Yes / No

Pulse - Rate,Rhythm,Volume

Blood Pressure

Other

J. LOCAL EXAMINATION OF ABDOMEN

1. INSPECTION

a) Shape - Flat / Scaphoid / distended

b) Any mass / fullness

- Site
- Number
- Extent
- Shape
- Surface

• Borders

• Movement with respiration :

• Head raising test :

c) Umbilicus - Shape,Position

d) Visible veins - Yes / No, Site

e) Visible peristalsis - Yes / No, Type

f) Flanks

g) Hernial orifices

h) All quadrants moving equally with respiration

i) Scars - No / site / nature of healing

j) Sinuses - No / site / surrounding skin / nature of discharge

k) Fistula

l) Any others

2. PALPATION

a) Feel of the abdomen

• Soft / Doughy

• Guarding

• Rigidity - Localized / generalized

• Tenderness - , Present / Absent

b) Free fluid Fluid thrill

Shifting dullness

3. PERCUSSION

a) Dullness continuous with :Liver,Spleen,Extents

b) Free fluid - Puddle‟s sign

Shifting dullness

c) Bladder - Yes / No

d) Renal angle - Normal / dull

4. AUSCULTATION

Bowel sound - Yes / No, Frequency, Character

P/R

Wall

Lumen

Nature of finger stain

P/V

RS

CVS

PROVISIONAL DIAGNOSIS

K. INVESTIGATIONS

a) Blood - Hb%,TC,DC,ESR,Blood group,FBS,Blood urea,Serum creatinine

b) Urine - Sugar, Albumin, Microscope

c) Stools - Gross,Microscopy,Occult blood

d) Chest X-ray

e) Plain X-ray abdomen:

f) Ultrasound :

L.CLINICAL DIAGNOSIS

M.TREATMENT

Conservation

Operative: Simple / Radical

MPI SCORE

N. POST-OP-PERIOD - Complications

O.FOLLOW UP - Good / Fair / Poor

P.MORTALITY

MASTER CHART

SL NO	Name	IP number	Age(yrs)	Sex	Organ failure	Exudate	Peritonitis	Duration of pain	Site of perforation	Complications		MPI score	Mortality
					B urea/ S creatinine					Pulmonary	Wound infection		
1	Bharathan	4599	21	Male	Absent	clear	localised	10 hrs	appendicular			<21	
2	sudhakar	5266	38	Male	absent	purulent	diffuse	2 days	appendicular			<21	
3	sivaprakash	5031	60	Male	absent	clear	localised	1 day	appendicular			<21	
4	sanaullah	6818	22	Male	present	purulent	diffuse	5 days	duodenal		present	21-29	
5	Raffick	5942	18	Male	absent	clear	localised	2 days	appendicular			<21	
6	paramesvaran	7161	17	Male	absent	clear	localised	1 day	appendicular			<21	
7	Karuna	7774	27	female	absent	clear	localised	12 hrs	appendicular			<21	
8	karthik	8758	24	Male	absent	purulent	localised	3 days	appendicular			<21	
9	Ranjith	10661	17	Male	absent	clear	localised	1 day	appendicular			<21	
10	Neelakandan	11530	21	Male	absent	clear	localised	2 days	appendicular			<21	
11	Srinivasan	8764	35	Male	present	purulent	diffuse	3 days	gastric		present	21-29	
12	Vijay	12270	26	Male	absent	clear	localised	2 days	appendicular			<21	
13	Venkatraman	16243	57	Male	absent	clear	localised	1 day	appendicular			<21	
14	Sridhar	18723	19	Male	absent	clear	localised	3 days	appendicular			<21	
15	Banu	17271	21	female	absent	clear	localised	10 hrs	appendicular			<21	
16	Ashok kumar	9250	22	Male	present	purulent	diffuse	3 days	gastric			21-29	
17	Patchaiyyapan	11717	30	Male	present	purulent	diffuse	5 days	duodenal		present	21-29	
18	sridhar	18723	18	Male	absent	clear	localised	2 days	appendicular			<21	
19	Saran raj	20726	28	Male	absent	clear	localised	2 days	appendicular			<21	
20	Sathya prakash	20921	25	Male	absent	clear	localised	2 days	appendicular			<21	
21	kamal	20972	23	Male	absent	clear	localised	2 days	appendicular			<21	
22	Sathyamurthi	11621	33	Male	present	purulent	diffuse	3 days	gastric			21-29	
23	Ramalingam	12384	65	Male	present	purulent	diffuse	3 days	duodenal		present	21-29	
24	Bala shankar	21279	29	Male	absent	clear	localised	2 days	appendicular			<21	
25	Karthik Raja	21417	20	Male	absent	clear	localised	2 days	appendicular			<21	
26	Pawn raj	21484	48	Male	absent	clear	localised	2 days	appendicular			<21	
27	Manikandan	11464	25	Male	absent	purulent	localised	2 days	appendicular			<21	

MASTER CHART

SL NO	Name	IP number	Age(yrs)	Sex	Organ failure	Exudate	Peritonitis	Duration of pain	Site of perforation	Complications		MPI score	Mortality
					B urea/ S creatinine					Pulmonary	Wound infection		
28	Manasa	23917	35	female	Absent	clear	localised	10 hrs	appendicular			<21	
29	Thirumalai	27474	23	Male	absent	purulent	diffuse	2 days	appendicular			<21	
30	Boopalan	26438	18	Male	absent	purulent	localised	1 day	appendicular			<21	
31	Pradeep	21599	16	Male	present	purulent	diffuse	5 days	duodenal		present	21-29	
32	Mohan	28600	29	Male	absent	clear	localised	2 days	appendicular			<21	
33	Kasinathan	21601	47	Male	absent	clear	localised	1 day	appendicular			<21	
34	Marimuthu	24250	36	Male	absent	clear	localised	12 hrs	appendicular			<21	
35	Govindan	25556	35	Male	absent	purulent	localised	3 days	duodenal			<21	
36	Saktara	28351	50	Male	absent	purulent	localised	1 day	appendicular			<21	
37	Tharakodi	27751	61	Male	absent	purulent	localised	2 days	Ileal			<21	
38	Vadivel	28047	36	Male	present	purulent	diffuse	3 days	duodenal		present	21-29	
39	Vignesh	30884	19	Male	absent	clear	localised	2 days	gastric			<21	
40	Yokesh	26415	20	Male	absent	clear	localised	1 day	appendicular			<21	
41	Thimmalai	27474	23	Male	absent	clear	localised	3 days	appendicular			<21	
42	Balaramjana	30475	22	Male	absent	clear	localised	10 hrs	appendicular			<21	
43	charulatha	32811	34	female	present	clear	localised	10 hrs	appendicular			<21	
44	Ragavan	30419	55	Male	present	purulent	diffuse	5 days	duodenal		present	>29	
45	Vinodkumar	29416	23	Male	absent	purulent	localised	2 days	appendicular			<21	
46	Kannan	33187	26	Male	absent	clear	localised	2 days	appendicular			<21	
47	Kishore	33707	18	Male	absent	clear	localised	2 days	appendicular			<21	
48	Saravanan	33863	22	Male	absent	clear	localised	2 days	appendicular			<21	
49	Subash	34713	22	Male	present	purulent	diffuse	3 days	gastric			21-29	
50	Murugan	34708	40	Male	present	purulent	diffuse	3 days	duodenal			21-29	
51	Akshay	34700	31	Male	absent	clear	localised	2 days	appendicular			<21	
51	Pemmal	34661	19	Male	absent	clear	localised	2 days	appendicular			<21	
53	Guna	35170	24	Male	absent	clear	localised	2 days	appendicular			<21	
54	Tharun balaji	35217	19	Male	absent	purulent	localised	2 days	appendicular			<21	
55	Vel murugan	35590	30	Male	absent	clear	localised	2 days	appendicular			<21	

MASTER CHART

SL NO	Name	IP number	Age(yrs)	Sex	Organ failure	Exudate	Peritonitis	Duration of pain	Site of perforation	Complications		MPI score	Mortality
					B urea/ S creatinine					Pulmonary	Wound infection		
56	Udhayan	34180	58	Male	present	purulent	diffuse	5 days	gastric	present	present	>29	death
57	Saravanan	38457	32	Male	absent	clear	localised	2 days	appendicular			<21	
58	Vani R	37905	32	female	absent	clear	localised	11hrs	appendicular			<21	
59	Ganesh	37904	18	Male	absent	clear	localised	1 day	appendicular			<21	
60	Raguraman	16691	33	Male	present	purulent	diffuse	4 days	duodenal			21-29	death
61	Santhosh	36734	22	Male	absent	clear	localised	2 days	appendicular			<21	
62	Rakesh	32785	28	Male	absent	clear	localised	1 day	appendicular			<21	
63	Manoj	38902	26	Male	absent	clear	localised	2 days	appendicular			<21	
64	Balu	8990	40	Male	present	purulent	diffuse	4 days	Gastric			21-29	death