EFFECTIVENESS OF MANNHEIM PERITONITIS INDEX IN PREDICTING THE MORBIDITY AND MORTALITY OF PATIENTS WITH HOLLOW VISCOUS PERFORATION

TAMIL NADU Dr. M.G.R MEDICAL UNIVERSITY.

CHENNAI

In partial fulfillment of the requirements for the degree of

MASTER OF SURGERY

In

GENERAL SURGERY

Under the guidance of

PROF. DR. KANNAN M.S

DEPARTMENT OF GENERAL SURGERY

GOVT. KILPAUK MEDICAL COLLEGE

CHENNAI

YEAR-2014

CERTIFICATE BY THE GUIDE

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Dr.RAJKUMAR.R

ABSTRACT

BACKGROUND AND OBJECTIVES

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.

METHODS

30 patients admitted in govt royapettah hospital & kilpauk medical college between april- sept 2014were included in the study. Necessary data was collected; MPI score was calculated for each patient and analysis done.

RESULTS AND INTERPRETATION

The number of postoperative complications, duration of ICU and hospital stay proportionately increased with the MPI score. Out of the 8 variables used in this scoring system, duration of pain, intra peritoneal fluid and organ failure on admission carried more significance in predicting the morbidity in the post op period than the other variables.

CONCLUSION

MANNHEIM PERITONITIS INDEX is a simple and effective method in predicting the morbidity of patients with hollow viscous perforation

KEY WORDS: Peritonitis, Scoring systems, Outcome predictors, morbidity

INSTITUTIONAL ETHICAL COMMITTEE
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Ref.No.2212/ME-1/Ethics/2014 Dt:03.04.2014.
CERTIFICATE OF APPROVAL

The Institutional Ethical Committee of Govt. Kilpauk Medical College, Chennai reviewed and discussed the application for approval "A Study on effectiveness of Mannheim peritonitis index in predictin the morbidity and mortality of patients with hollow viscous perforation" – For Project Work submitted by Dr.R.Rajkumar, MS (GS), PG Student, KMC, 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.

GOVT. KILPAUK MEDICAL COLLEGE
30 MAY 2014
Kilpauk, Chennai-10.

30/5/14

CHAIRMAN,
Ethical Committee
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LIST OF ABBREVIATIONS USED

Sl. No.	Abbreviations	Full form
1.	MPI	Mannheim's peritonitis Index
2.	ICU	Intensive care unit
3.	APC	Adenomatous polyposis coli
4.	W.H.O	World health organization
5.	ATT	Anti-tubercular treatment
6.	CAPD	Continuous ambulatory peritoneal dialysis
7.	CT	Computerized tomography
8.	SIRS	Systematic Inflammatory Response Syndrome
9.	CARS	compensatory anti-inflammatory response syndrome

INTRODUCTION

With the advancements in the field of medicine and technology, the surgeon must be well aware of the infectious diseases of the peritoneal cavity which has increased in severity and complexity. In addition to management of hollow viscous perforation. Surgeon may also be called for management of patients with cirrhosis or patients undergoing peritoneal dialysis with infected ascitic fluid. Or there is increased recognition of group of critically ill patients with persistent intra-abdominal sepsis or tertiary peritonitis in whom peritonitis is associated with multi organ failure and immune system failure. Despite the advancement in anti-biotics and ICU care, the mortality rate of patients presenting with diffuse suppurative peritonitis is very high

Its etiology may vary from the one requiring immediate surgery to that of a simple conservative management. Its early identification detection and management remains a challenge to every surgeon.

The complex nature of etio-pathogenesis, the multifaceted aspects of treatment, and increasing complexity of ICU management makes the evaluation of new diagnostic and therapeutic advancement in this field very complex. Scoring system which will provide the exact objective descriptions of the patient's

condition at specific points in disease process aid our understanding of these problems. This is very important in determining the course of the disease and whether appropriate management given to the patient is appropriate or needs to be changed.

With the betterment in understanding the patho-physiology of the disease, sepsis syndrome and multi-organ failure. The current practice is to recognize the earliest and institute aggressive treatment. Patients who are already into multi organ failure the outlook is poor whatever the management is. It is here the conservative approach as well as newer modalities of management like programmed relaprotomy, immune modulation is tried. These newer modalities may be effective they are very expensive. Hence, a proper clinical examination and monitoring with optimum number of investigations remains the corner stone

The questions in the mind of every surgeons managing these kinds of patients are

Does the etiology of peritonitis influence the outcome?

Do delays in presentation matters?

Could this patient managed better without surgery?. Continue to dog the minds of surgeons. I seek to find answers to some of these questions through my study

AIM & OBJECTIVE

1. To study the validity of scoring systems that are most widely used. These are:

    a. Mannheim peritonitis index.

    b. Sepsis score of Elebute & Stoner

    c. APACHE-II

2. Factors determining the outcome of the disease:

    a. Patient factors

        i. Age

        ii. Sex of the patient

        iii. General health of the patient- Nutrition, anemia

    b. Disease process

        i. Site of perforation

        ii. Duration of perforation

        iii. The extent of peritoneal contamination.

    d. Effect of General systemic complications like

        i. Respiratory

        ii. CVS system

        iii. Shock

        iv. Multi-organ failure

My aim in this study is to find out the role of these factors on morbidity & mortality of the patients

REVIEW OF LITERATURE

HISTORY:

Peritonitis is a dreaded complication for physicians since antiquity. Though the occurrence of peritonitis was common, reports suggest that successful surgical interventions were only anecdotal before the past century.because our knowledge in understanding the etio-pathogenesis of sepsis & multi organ failure the mortality rates of patients with secondary peritonitis have fallen from nearly 100% to <10%.

One of the earliest documentation can be found in Edwin Smith Papyrus 1700 B.C. proposed to be written during the time of Imhotep (The Egyptian patron god of medicine). Breasted who translated these works in his translation are. “I felt as if I had been peering through a newly revealed window, opening upon the once impenetrable gloom enveloping man’s earliest endeavors to understand the world he lived in. it was as if I had watched a hand slowly raising the curtain that covered this window, and then suddenly the hand had refused to lift, the curtain further”. The curtain may have mean peritonitis.

Since the beginning of documented medical history, Physicians have confronted with a variety of causes for peritonitis. Hippocrates appears to be the first description of a peritonitis patient. “The patient looks sick and wasted., the eyes lay deep and dull. the tongue is furrowed, The nose is pointed, the temple is sunken the skin shiny The face express fear, The abdomen is rigid with guarding. The patient avoids movements and breathes rapid and shallow. pulse is quick and thready. tender mass in hypochondrium is a bad prognostic sign if it also involves the whole area. The presence of such mass along with fever indicates the death is imminent- Hippocratic facies

He also described septic shock as " a protrusive nose, hollow eyes, sunken temples, cold ear lobes drawn outwards, whole face greenish , the forehead skin tense and rough like a parchment".

In the second century A.D Galen, the physician of The Roman empire reported many surgeries including suturing of bowel laceration. He also reported suppurative peritonitis in the following post-operative period. He also believed that such a suppuration is an essential part in wound healing and should be left undisturbed (laudable pus). His writings were revered unshakable tenets & restrained the development of further treatment for almost 1600 years.

From the fall of Roman empire till 16th century medicine was plagued with superstitious beliefs and strong religious overtones. Fate of medicine was sealed for many years with pope Innocent III religious decree of 1215 called as "EccelsiaAbhorret de Sanguine" which literally means "The Church prohibits Blood shed". It was later in the 18th century the mysteries inside the abdomen begin to be known because of the wonderful drawings of Vesalius, Leonardo Da vinci.

Peritonitis following perforation of acute peptic ulcer was first recorded by Littre in 1670. The patient was, Henritta anne, Duchess of Oreans & daughter of king Charles I England. Heretin, in 1767, observed a cure in biliary peritonitis in dogs just by irrigation of the abdomen.

The three major development in understanding came after the works done by Francois Magendie in experimental physiology. Advent of germ theory by Koch and pastuer. An understanding of cellular pathology as championed by Rudolph Virchow.

In 1879 George wegner conducted a series of experiments to demonstrate the normal physiology of peritoneum. In 1908 John.B.Murphy wrote “the endothelium of peritoneum is continuous there is no stoma or stigmata”. Of course we know today its not fully true.

The experimental study conducted by Meleney in 1926 showed that synergism existed between aerobic and anaerobic bacteria resulting in sepsis than from individual strains.

Hippocrates (460BC-371BC)

REVIEW OF CURRENT LITERATURE

Some of the early attempts to assess the risk of death in patients dying from peritonitis or severe septicemia followed a similar clinical course characterized by sequential organ failure called as “Multiple organ failure syndrome or Multi organ dysfunction (MODS)”

In 1980 fry and his colleagues made a study that showed that death after major surgery or severe trauma due overwhelming infection became more likely as the number of organs failed. I.e. the death rate was only 3% with no organ failure rising to 30% for single organ failure to 100% for more than 4 organ failure.

APACHE scoring was proposed by knaus in 1982 was originally a 2 part scale classifying the patients admitted in I.C.U. which includes physiological part assessing 34 vital physiological assessment (APS 34) obtained on the first day of admission. And the second part about chronic health evaluation(CH). This is combined to form the APACHE scoring . which was later reduced involving only 12 parameters called APACHE II. This scoring system is not specific for intra-abdominal sepsis.

In 1983 Elebute and Stoner proposed a scoring system into 4 classes. Criteria considered was secondary effects of sepsis, degree of temperature elevation, local effects of infection & laboratory values. This scoring system was not found to be accurate

Stevens 1983 recognized the importance of organ failure and devised a more accurate scoring system which includes 7 organ system and assigned a score 0-5 according to severity. scores were then obtained by squaring the values assigned to organs systems adding 3 highest scores. This is called as “sepsis severity score”

Tiechmann and colleagues in 1987 proposed Peritonitis Index Altermheir (PIA) which used age, severity, cvc risks, malignancy and leucopenia to assess the severity

Wacha and his co-workers (1987) developed Mannheim peritonitis index age, sex, malignancy, duration, colonic sepsis, organ failure, character of peritoneal fluid to assess risk.its score ranges from 0 to46

Verma (1990) from PGI chandigarh, assessed the factors influencing in peritonitis due to trauma. They included shock, hollow visceral injuries, septicemia and location of perforation(duodenum and colon perforation had significant mortality)

Scoanes(1994) conducted a study of diverse effects of treatment of neglected peptic ulcer. They found that delay more than 12hours with old age is associated with a higher mortality rates thus confirming the findings of MPI scoring

In 1996 a large multivariate clinical trial invoving 602 patients admitted in ICU with intra abdominal sepsis was conducted and several scoring systems were compared. In that both APACHE II and MPI correctly assessed the severity & mortality and morbidity with MPI scoring being more specific for intra abdominal sepsis and also easier to calculate.

SURGICAL ANATOMY OF PERITONEAL CAVITY

EMBRYOLOGY:

During development peritoneal cavity develops from two limbs of horse shoe shaped intraembryonic coelom,just below the septum tranversum. As the result of lateral folding of the embryo these two limbs fuse to form a single cavity. Later as the result of rotation of the gut several organs become retro peritoneal and attachments of mesentry which was in the midline becomes complicated and several pockets that are sub divided by the folds of peritoneum are created.

Peritoneal cavity:

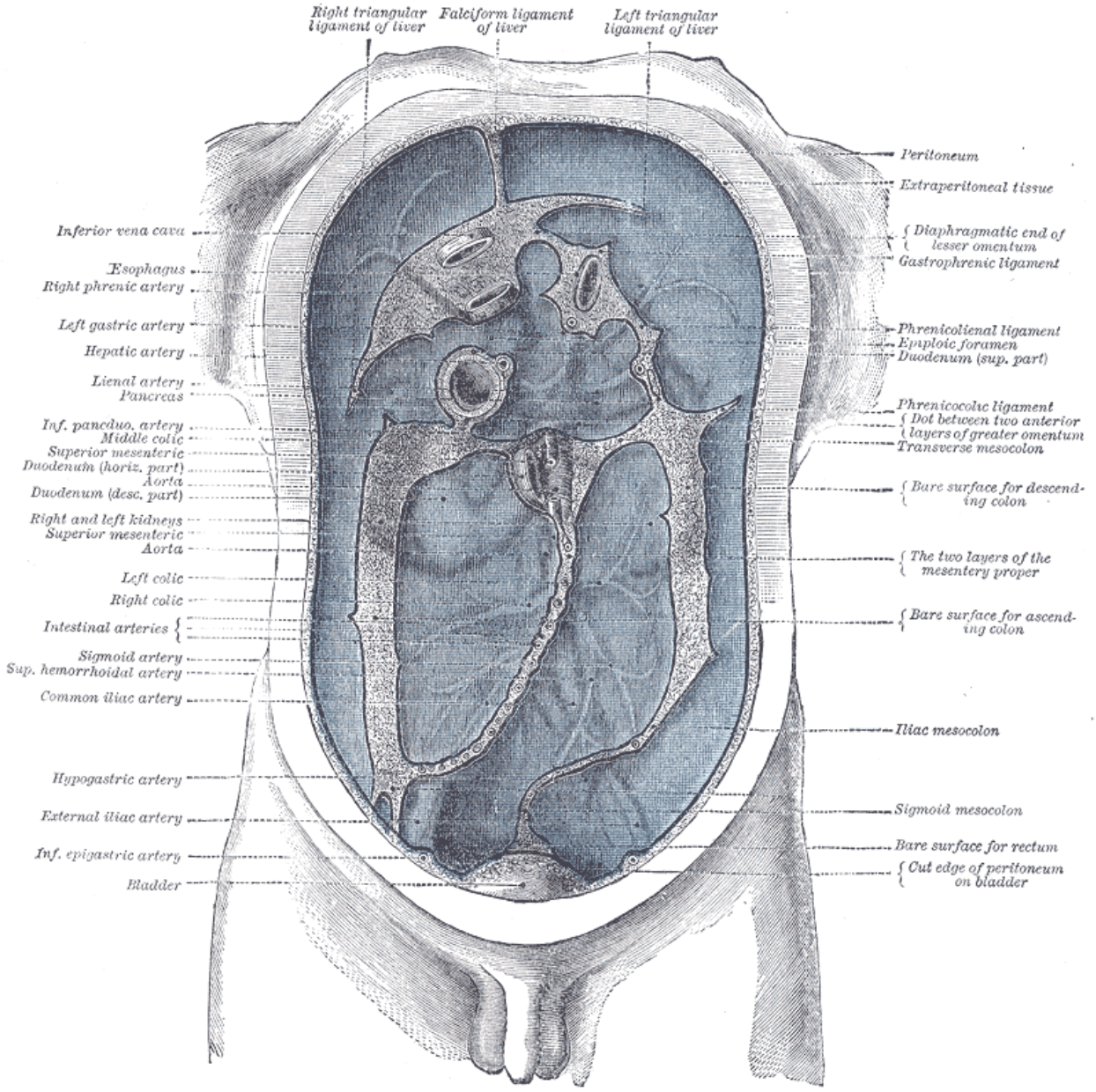
Peritoneal cavity is one of the largest cavity in the body. With the surface area of about 2sq.m almost equal to that of skin. Peritoneal cavity is closed in males and in females it opens to the exterior through the uterine tubes

General peritoneal cavity is divided into a larger greater sac and a smaller lesser sac called the omental bursa

Peritoneal cavity can be divided into a larger abdominal and a smaller pelvic portions. The abdominal portions can be divided into supra colic and infra colic portions by the attachment of transverse colon. The supracolic compartment is between the diaphragm and the transverse colon which contains the liver, stomach, gall bladder, spleen and first part of duodenum.the liver and its attachments to the peritoneum sub divides this into important sub-phrenic space. The infra colic portion can be sub divided into right and left by the attachment of the root of mesentry. Which can be further divided into external and internal paracolic gutters by the attachment of ascending and descending colon respectively.

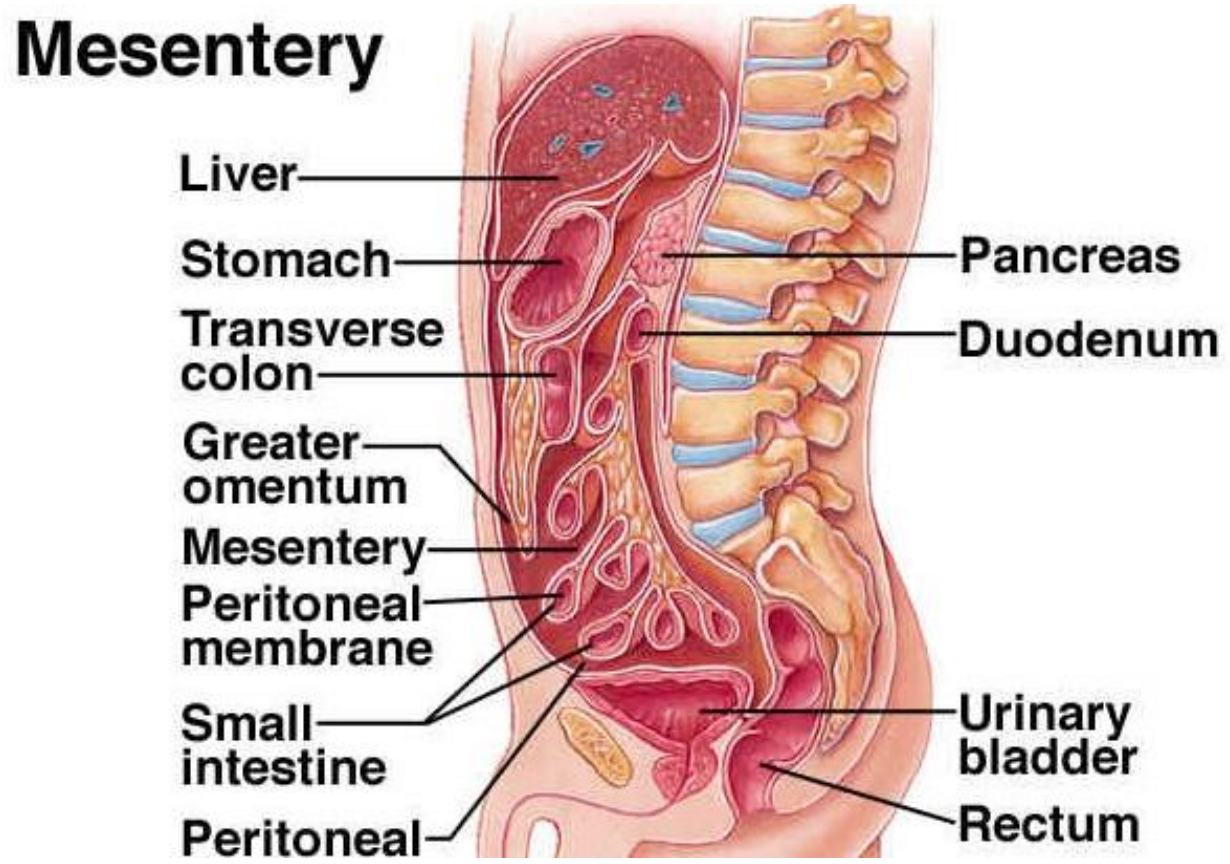
Parietal peritoneum:

It's the inner lining of the abdominal cavity lined by a single layer of mesothelial cells which is innervated by somatic nerves and is pain sensitive. It can be easily stripped off because of the presence of extra peritoneal connective tissue.



Visceral peritoneum:

This covers the outer surface of the viscera and is pain insensitive layer.it cannot be stripped easily and derives the same blood supply as that of the viscera. just like the parietal peritoneum it is lined by a single layer of mesothelial cells.



Subphrenic spaces:

There are about 7 sub phrenic spaces in which 4 are intra peritoneal and 3 extra peritoneal spaces. Which sub divided into right and left by the falciform ligament. They are namely;

1. Right anterior space
2. Right posterior space
3. Left anterior space
4. Left posterior space

} Intra peritoneal spaces

Extra peritoneal spaces are
1.right extra peritoneal space
2.left extra peritoneal space
3.midline extra peritoneal space

} or perinephric space

1. Right anterior intra peritoneal space-

This lies between the right lobe of liver and diaphragm.posteriorly by the coronary and right triangular ligament and falciform ligament in the left. Collection in this space is most commonly due to perforated gall bladder, perforated duodenal ulcer or a stump blow out , appendicular perforation.

2. Right posterior intra peritoneal space-

This space is also called the hepato-renal pouch or Morrison's pouch. Its boundaries are right by the diaphragm left by the foramen of winslow superiorly by right lobe of liver and inferiorly by the transverse colon. This is the deepest space and most common site of collection

of free fluid and sub diaphragmatic abscess due to perforation of hollow viscous in the upper abdominal cavity.

3. Left anterior intra peritoneal space:

This space is bounded right by the falciform ligament and left by the spleen and diaphragm. Above by the diaphragm and below by the left lobe of liver.collection noted in this space are usually due to operations involving the stomach, pancreatic tail,splenic flexure

4. Left posterior intra peritoneal space:

This space is also called the lesser sac or the omental bursa.the is the most commonest site for collections following pancreatitis or due to a rupture pseudocyst.

Midline extra peritoneal space:

Extra peritoneal space is the otherwise called as the bare area of liver. Abcess due to rupture of bacterial or amoebic liver abcess can collect in this space or can cause generalized peritonitis.

PHYSIOLOGY

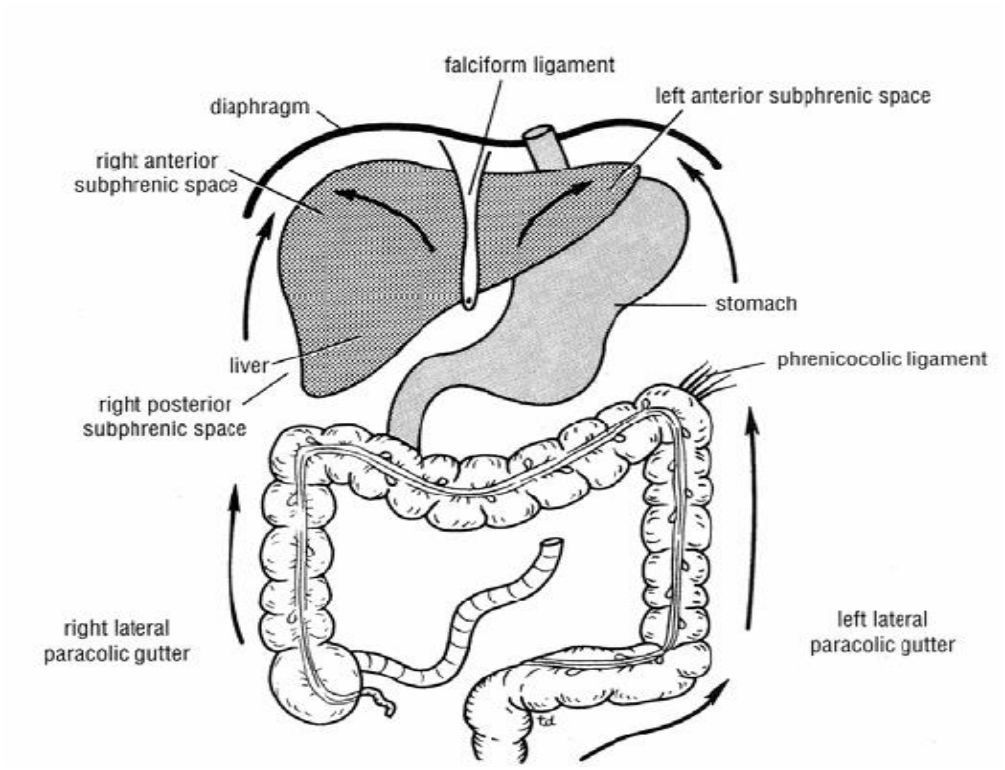
Two primary factors that govern the peritoneal fluid absorption are

(i) Gravity

(ii) Negative pressure created by the diaphragm during respiration

Peritoneal fluid enters the diaphragmatic lymphatics which in turn enter the thoracic duct

Peritoneum is lined by the mesothelial cells overlying the basement membrane. Cells are arranged in two different population's i.e flattened cells and cuboidal cells. Gap junctions exist only between the cuboidal cells during peritonitis these gap junctions increase in size. The mesothelial cells secrete the protein rich peritoneal fluid similar to composition of plasma (normal range 50-100ml) The basement membrane is made up of collagen and contains elastic fibres, proteins, fibroblast, mast cells and other inflamatory cells.



NORMAL DIRECTION OF PERITONEAL FLUID FLOW

PERITONEAL REACTION TO INJURY:

Any inflammatory event or injury in the peritoneal cavity results in the peritoneal irritation and irritation with loss of mesothelial cells in that region. Unlike epithelial cell healing which heals from the edges slowly by cell migration. A large peritoneal defect heals in same amount of time as minor defect.

Studies shows that

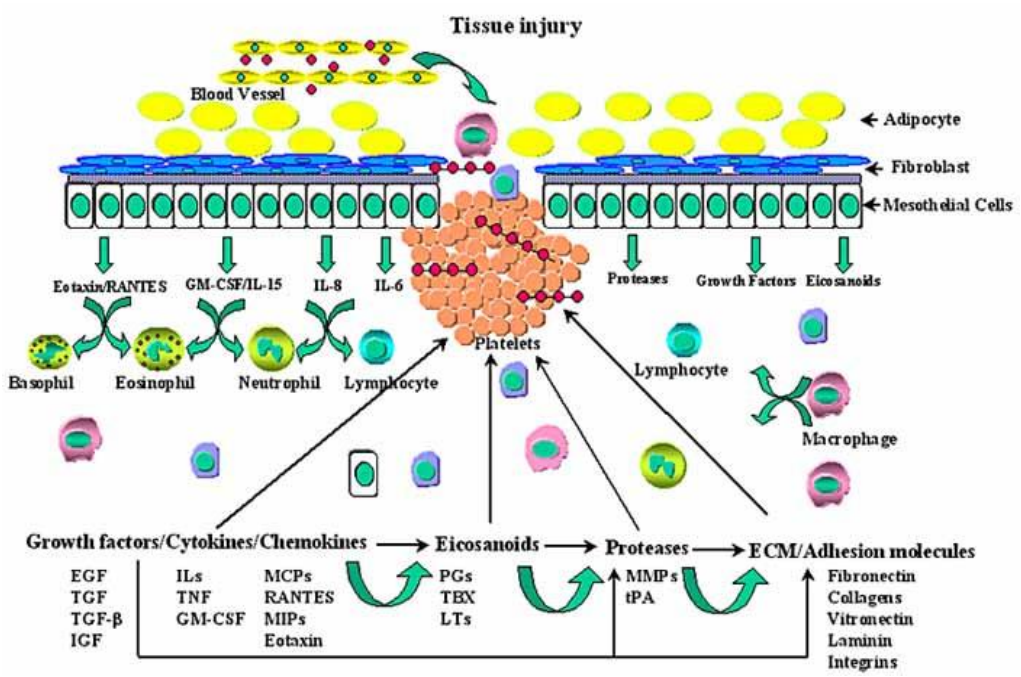
- ✓ After 3days of peritoneal injury connective tissue cells from actively dividing fibroblast resembling new mesothelium cover wound.
- ✓ At day 5 new layer closely resembles adjacent normal mesothelium.
- ✓ On day 8-10 healing is complete mesothelium regeneration is complete.

The accurate process of cells for mesothelial regeneration remains unknown. It is postulated, the mechanisms are

- ✓ 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.

Normal peritoneal wound heals with no adhesion. Adhesion develops in response to Local tissue hypoxia or ischemia or presence of foreign bodies like suture or latex from gloves, mechanical sub peritoneal surface injury, intra-abdominal infections (Deposition of fibrin following peritonitis). The fibrin lytic activity is absent in healing wound until mesothelial cells are found. Fibrin lytic Activity starts at 3 days and complete at the end of 8th day. So adhesions

can be minimized by proper surgical methods having a intact mesothelial surface and proper use of suture materials.


Tissue injury
Blood Vessel
Adipocyte
Fibroblast
Mesothelial Cells
Eotaxin/RANTES
GM-CSF/IL-15
IL-8
IL-6
Proteases
Growth Factors
Eicosanoids
Basophil
Eosinophil
Neutrophil
Lymphocyte
Platelets
Lymphocyte
Macrophage
Growth factors/Cytokines/Chemokines
Eicosanoids
Proteases
ECM/Adhesion molecules
EGF
TGF
TGF-β
IGF
ILs
TNF
GM-CSF
MCPs
RANTES
MIPs
Eotaxin
PGs
TBX
LTs
MMPs
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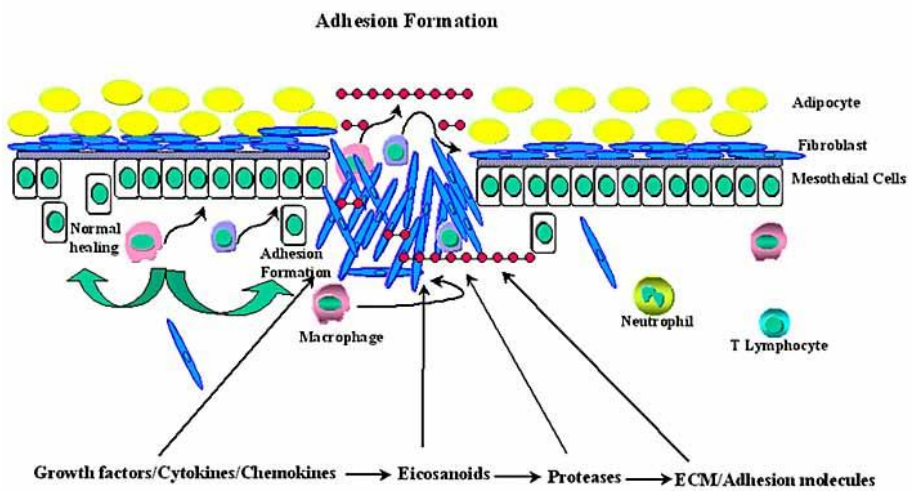
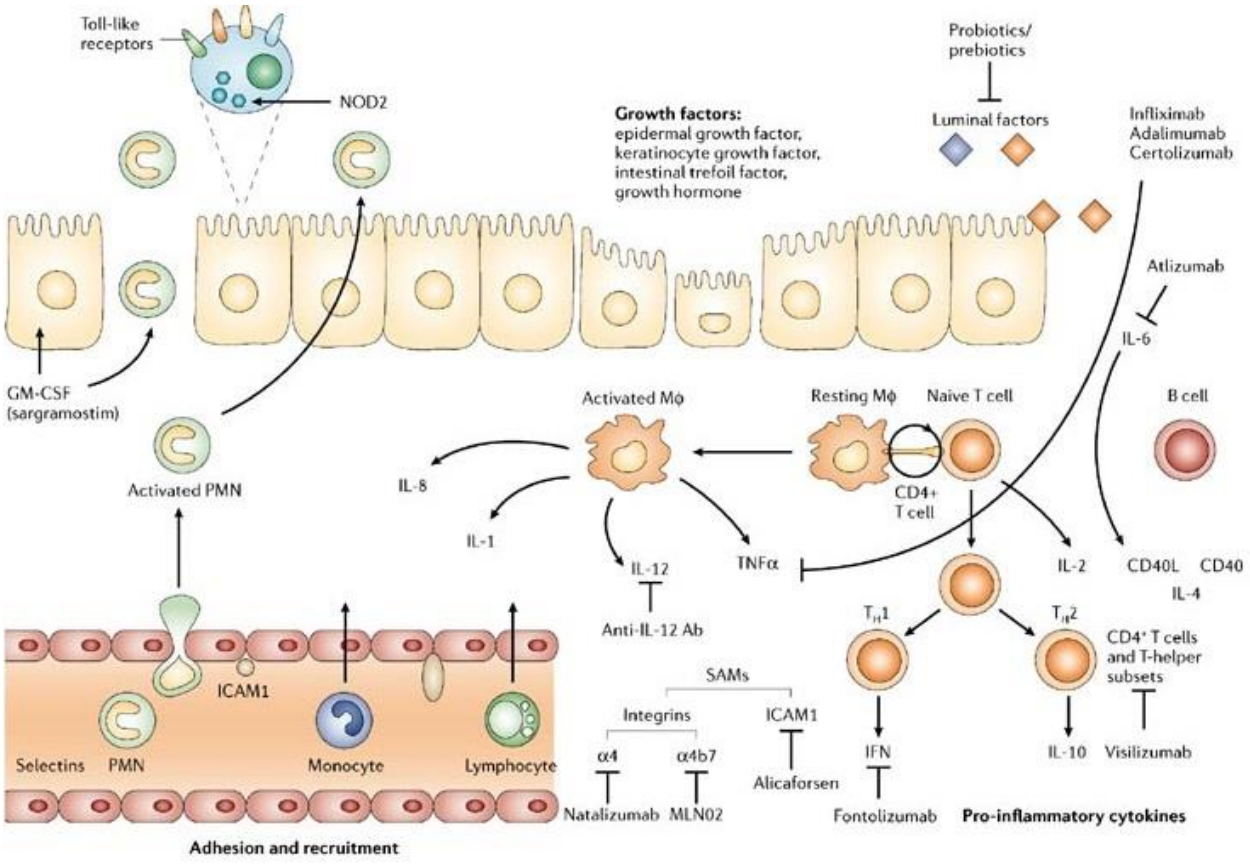
Collagens
Vitronectin
Laminin
Integrins


Adhesion Formation
Adipocyte
Fibroblast
Mesothelial Cells
Normal healing
Adhesion Formation
Macrophage
Neutrophil
T Lymphocyte
Growth factors/Cytokines/Chemokines
Eicosanoids
Proteases
ECM/Adhesion molecules

PATHOPHYSIOLOGY OF PERITONITIS

Localized or generalised inflamation of the peritoneum is called peritonitis.Peritonitis can be due to various causes, which initiates a cascade of reactions involving not only the peritoneum but the bowel,body fluid compartments producing secondary cardiac respiratory endocrine,renal and metabolic responses.



PRIMARY REACTION:

MEMBRANE INFLAMMATION:

Primary reaction of the peritoneum for inflammation is hyperemia vasodilatation and transudation. Edema and vasodilatation occurs immediately in the sub peritoneal layer. Transudation of fluid with low molecular weight proteins occurs from the extra cellular interstitial spaces into the peritoneal cavity occurs along with diapedesis of the polymorphs .During the acute phase, both the absorption and transudation increases rapidly. Peritoneum acts as a "TWO WAY BIOLOGICAL MEMBRANE" through toxins and bacteria's are absorbed systemically which may lead to septic shock .on the other hand the exudates is very rich in fibrin and other plasma proteins which are sufficient to bring about clotting later which later results in agglutination of bowel loops and viscera.

Early Stages- Concentration of uronic acid increases

Later stages- Glysoaminoglycans increases (increased activity of fibroblast and mesothelial cells.)

Changes in the synthesis of non-collagen and collagen protein 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. During the first week of peritonitis the ratio of RNA: DNA, an index of protein synthesizing capability increases

BOWEL RESPONSE:

The initial response of the bowel is hypermobility. Later, as the peritonitis progress the mobility gets depressed followed by a complete adynamic ileus and finally bowel distension and air fluid levels occur.

HYPOVOLEMIA:

The loose connective tissues beneath the peritoneum and visceral peritoneum and the mesentery starts secreting large amounts of extra cellular fluid as edema. This sudden translocation of fluid into the "THIRD SPACE" reduces the circulatory volume in the body and deprives body fluid from the body economy. With extensive peritonitis translocation of more than 4-6 Liters of fluid in a day is not uncommon causing septic shock.

SECONDARY RESPONSES IN PERITONITIS

Septic shock can be divided into compensatory and decompensatory stages.in compensatory stage the counter defensive mechanisms kick in and tries to maintain homeostasis & B.P but if the sepsis is not controlled the compensatory mechanisms finally fails leading to a irreversible down spiral called the decompensatory shock

ENDOCRINE RESPONSE

There is immediate adrenal surge with release of large amount of adrenaline and nor-adrenaline and other catecholamines causing tachycardia, pupillary dilatation, sweating, vasoconstriction to skin and kidneys and vasodilatation to heart muscles liver and brain maintaining circulation to vital organs. For the first two or three days there is increase in cortisol secretion. Finally secretion of ADH and aldosterone increase causing water and sodium conservation. Water retention is more than sodium retention causing dilutional hyponatremia

CARDIAC & VASCULAR RESPONSE

Because of the hypovolemia reduction in ECF and progressive acidosis results in reduced venous return and diminished cardiac output. Heart in compensatory stage of shock reacts by increase in rate and stroke volume but as the progresses and decompensatory stage starts the heart cannot cope up alonf with progressive acidosis brings about secondary dysfunction in cardiac contraction and cardiac output reduces and finally cardiac failure occurs.

There is severe vasospasm in the arterioles compensating the reduction in B.P due to ECF reduction conserving blood only to vital organs

RESPIRATORY RESPONSE

Fluid and inflammatory cells not only accumulates in the peritoneal cavity but also infiltrates and inflammate the alveoli causing reduction in oxygenation coupled with increased abdominal pressure due to ileus and pain causing reduction in diaphragmatic action , all finally leads to reduction and respiratory failure

RENAL RESPONSE

Kidneys conserve water and sodium in response to reduction in GFR due to ADH and aldosterone stimulation. Potassium is wasted. With reduction in GFR the juxta-glomerular cells activates renin-angiotensin mechanism causing a raise in BP

METABOLIC RESPONSE

The oxygen demand of the tissues increase significantly but the capacity of heart and lungs are reduced. Poor circulation and reduced oxygen supply leads to shift from the aerobic to anareobic metabolism in muscles leading to Lactic acid accumulation further leading to metabolic acidosis which further aggravates the condition leading to a vicious cycle.

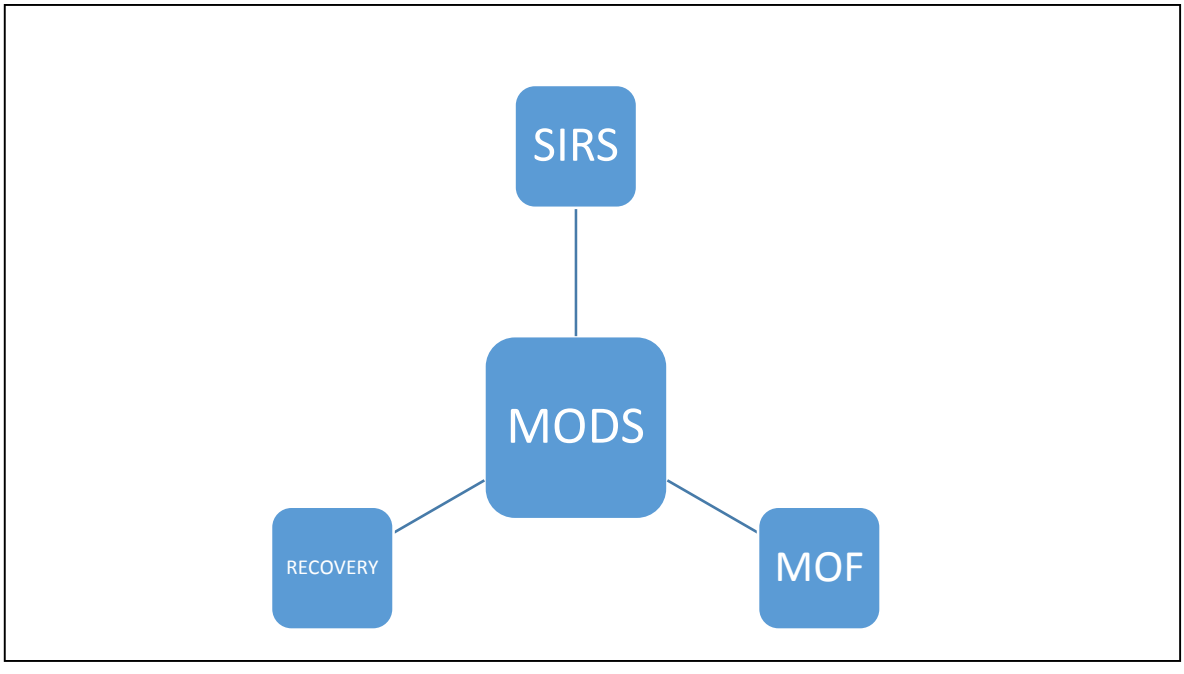
Over all there is a breakdown of proteins in the muscles but there is a preferential increase in the production of plasma proteins. There is lipolysis. Adipose tissues are broken down to produce more energy supplying for the increased demand but increased fat lysis produces ketoacidosis

FACTORS FAVOURING THE DEVELOPMENT OF GENERALISED PERITONITIS:

1. Areas of perforation- colonic perf. has worst prognosis
2. Speed of peritoneal containment
3. Peristalsis- ingestion of food hinders localization
4. Virulence and type of infecting organism
5. Young children who have smaller omentum
6. Deficient host resistance

PATHOPHYSIOLOGY OF SEPSIS:

As Sir. Osler mentioned primary cause of death is not their disease; but their physiological abnormalities. When a bacteria is injected in the peritoneum it can be identified as early as 6minutes in the blood stream and 12 minutes in the blood stream. The peritoneal injury will be initially manifested As Systemic Inflammatory Response Syndrome (SIRS) which when not identified quickly ll lead to Multi Organ Dysfunction Syndrome (MODS).From many patients succumb to death due to Multi Organ Failure (MOF) or many with proper treatment recover



DEFINITIONS:

1. SIRS: (Systematic Inflammatory Response Syndrome).

   Two or more of following clinical signs indicates SIRS

   - ✓ Temperature- >38C or <36C.
   - ✓ Heart rate > 100/ min
   - ✓ Respiratory rate > 20/ min or PaCO2 < 32 mmHg
   - ✓ WBC count >12000/mm or <4000 mm 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:

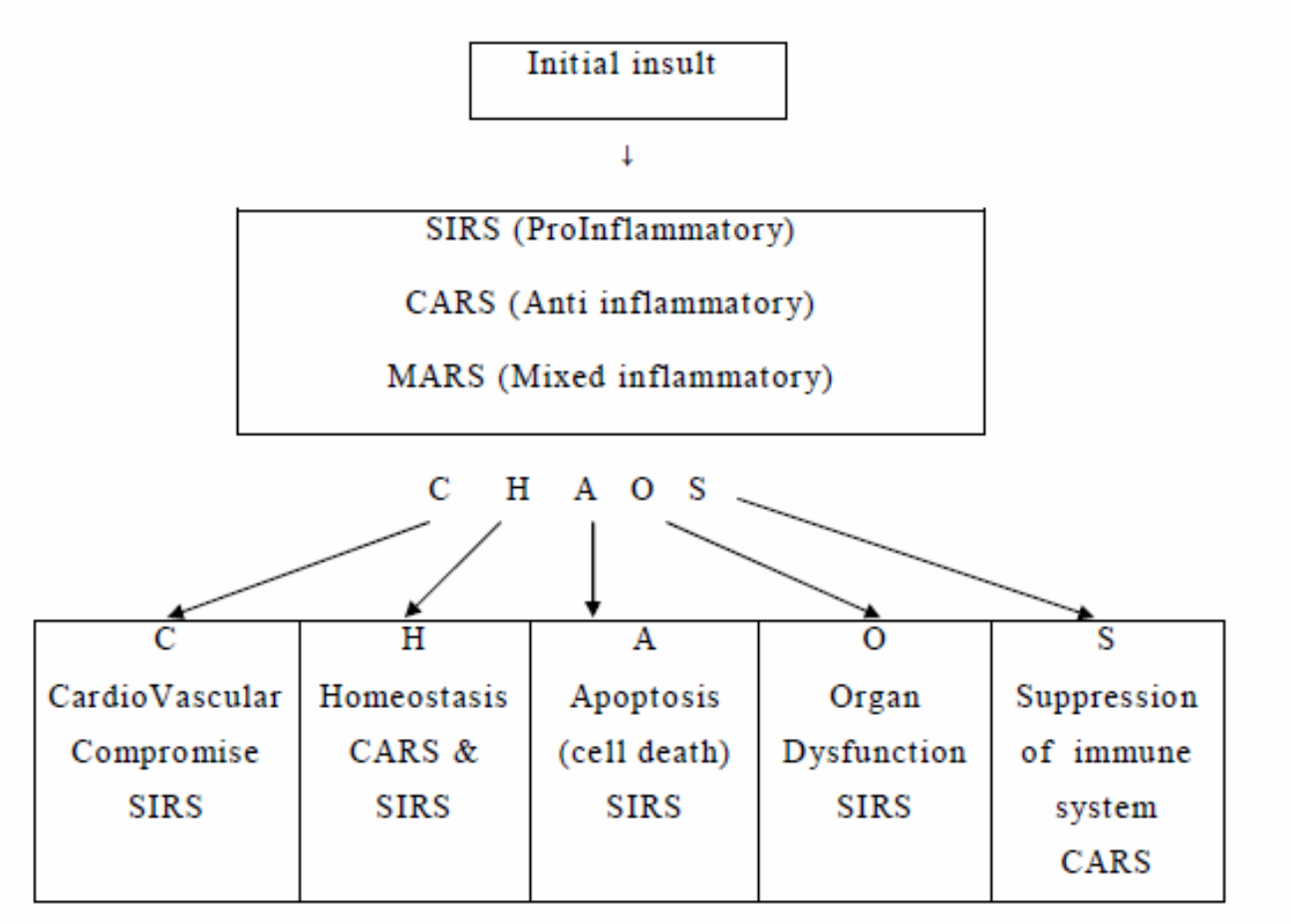
SIRS is not due to a single mediator.various mediators are newlt discovered and added to the long list. The most important one is Histamine, TNF(TUMOR NECROSIS FACTOR-a). Others are IL- 1, IL-6, Endotoxin, Endothelium derived toxins,and bradykinins and prekallikerins

EFFECTS OF SIRS:

There will be increased in the peripheral vasodilatation, microvascular permeability,& clotting and leukocyte/endothelial cell activation. Other effects include fever, anorexia, cachexia etc. All these finally lead to septic shock, DIC, ARDS and Multi organ failure.

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.



BACTERIOLOGY IN PERITONITIS:

Peritonitis following perforation of hollow viscera is usually poly microbial in nature Paths of invasion of peritoneal cavity

1. Direct spread
2. Local extension from an infected organ like appendicitis or cholecystitis
3. Via blood stream

Table 1 : Bacteria commonly encountered in peritonitis

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

4.

BACTERIA FROM THE ALIMENTARY TRACT

The bacterial counts are usually less until the distal small bowel is reached, after the distal ileum and colon the bacterial counts ranges about 107-108/ml of stools. The biliary and pancreatic tracts are usually devoid of bacterias or stasis where they get secondarily infected. Even though peritonitis is polymicrobial the usual organisms that are isolated most commonly are E.coli & aerobic or anaerobic streptoccoci & Bacteroids sp. less commonly clostridium.welchii. These gram negative organisms although more frequent in the colon they often fail to be isolated in culture because they are slow growing and very low oxygen tension.21even though peritonitis is poly microbial only a certain species of bacterias are repeatedly isolated in peritoneal cultures.This indicates only a few bacterias survive to cause peritoneal infection Weinstein demonstrated that E.coli and enterococccus predominated in the peritonitis phase and B.fragilis predominated in the abscess phase. 21Experimental studies have shown that there exist a synergistic effect between aerobic and anaerobic bacterias. Studies have shown that innoculation of B.fragilis alone resulted in no deaths or lactic acidosis in rats. But when a inoculum of E.coli and B.fragilis is injected there appears abscess and death resulted from the Endo-toxin bearing aerobic partner.the aerobic partner actually uses all the oxygen available and creates a very low oxygen-redox potential region which permits the anaerobes to survive. Therefore it is of greatest concern on peritonitis due to distal GI tract pathology because of the high bacterial load. Even in case of nonbacterial peritonitis(Intra peritoneal Rupture of bladder) the peritoneum soon gets infected with trans mural spread of bacterias and its not long before a full blown bacterial peritonitis develops.

CLINICAL FEATURES

EARLY STAGES:-

PAIN:

Pain which is made worse on respiration and movements. Patients prefer to lie still on bed without moving. The origin of site of pain usually corresponds to the site of lesion sometimes, the site of pain may be referred.gradually the pain increases not responding to conservative management and analgesics over a period of 6-8hours. Later guarding and involuntary muscle spasm called guarding sets in and the abdomen of the patients become Board like Rigidity.the pain may be insignificant in case of very feeble or extremes of ages or in immune compromised patients.anterior abdominal wall tenderness sometimes will not be elicited in peritonitis involving the pelvis or the lesser sac. In such cases, patients usually compliant of urinary symptoms initially there may be infrequent bowel sound later its not heard as the ileus sets in.

PYREXIA:

Both hyper and hypothermia can occur in peritonitis. In young Fever usually occurs in high grade and in spikes later as the shock sets in the peripheries become cold.in extremes of ages or terminally ill patients or immune compromised patients usually respond as hypothermia as the first symptom. Nausea may be found inconsistently.

VOMITING

Nausea and vomiting may occur infrequently, initially the stomach contents are voided later the bile and intestinal contents are voided.in case of obstruction only the bilious output is present in vomitus later it becomes faeculent due to bacterial translocation and contamination. In the early stages it is reflex in nature later it is due to paralytic ileus.

TACHYCARDIA

A raise in pulse rate in response to inflammatory mediator release and reduced ECF occurs. A continuous raise in pulse rate and fall in temperature is a grave sign.in contrast a raise in temperature and falling pulse rate usually signifies a localization of infection.

TERMINAL PHASE:

If the peritonitis is not contained a generalized peritonitis ensues resulting in full blown, and circulatory collapse& shock .the patient presents with cold, clammy extremities, sunken eyes, dry tongue, thready (irregular) pulse, drawn and anxious face (Hippocratic facies). The patient finally lapses into unconsciousness. With adequate resuscitation and modern day intensive care patients presenting early will improve dramatically with adequate care.

SIGNS OF PERITONITIS
INSPECTION

The position of patient on bed is characteristic, he lies still on bed with legs drawn upwards in an effort to relax the peritoneum and abdominal muscles.abdomen is uniformly distended in the early stage more pronounced in lower region

PALPATION

Tenderness and rigidity are the prominent sign. Tenderness is a constant but not reliable as guarding or rigidity. Tenderness is first localized to the region, but later spreads as the infection spreads, which rapidly becomes generalized, and extreme in degree.

There are two other signs of clinical significance:

1. Rebound tenderness- sudden release of pressure causes springing of parital peritoneum back into position causing severe pain
2. Rigidity- initial voluntary guarding due to peritoneal inflammation is later replaced by diffuse abdominal muscles spasm resulting in a board like rigidity and pain. This usually prevents the clinician in palpating further or identifying mass

PERCUSSION:

Abdomen is usually uniformly resonant owing to air and fluid filled edematous bowel. Rarely there may be obliteration of liver dullness

Auscultation:

In the initial phase of obstruction there is increased bowel sounds but later paralytic ileus sets in and the bowel sounds slowly reduces to sluggish to finally total paralytic ileus.

INVESTIGATIONS

BLOOD INVESTIGATIONS:

Complete blood count, renal function test with electrolytes is measured .there may be raise in ESR along with increase in leukocytosis predominantly polymorphs. Renal parameters may show hypoglycemia with elevated urea and creatinine levels with or without dyselectronemia . may times septic shock presents along with DIC in such cases the is marked raise in PT, aPTT , INR values

BIOCHEMICAL INVESTIGATIONS:

1. 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.

2. Widal test in ileal perforation to rule out typhoid.
3. 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.

DIAGNOSTIC PERITONEAL LAVAGE:

It is useful when adequate free fluid is present in peritoneum. Solomon described, it is done in four quadrants after infiltrating the skin with a local anesthesia. When aspiration is dry then sterile saline may be injected and aspiration tried after sometime. Fluid is examined for cell, differential count, PH and gram stain and aerobic and anaerobic culture21 Microscopy of the fluid showing neutrophils more than 250cells/mm3 (indicator of inflammation) and bacteria (indicator of infection) is significant.

AN ERECT FILM OF ABDOMEN:

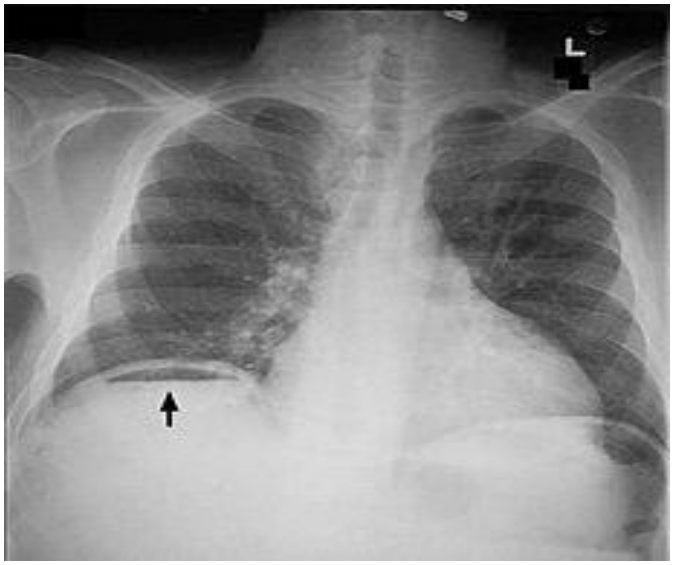
Plain X-ray should include the diaphragm, lower chest abdomen and pelvis. There may be pneumoperitoneum (demonstrated by gas under the dome of diaphragm) sensitivity 70%,ground glass appearance, obliteration of peritoneal pad of fat line and psoas shadow47 due to edema of peritoneum. There may be dilated air-gas-filled loops of bowel (consistent with paralytic ileus). If the patient is too sick to stand, Left lateral decubitus Posture is helpful.

Laparotomy: To diagnose and to treat peritonitis. On laparotomy, the peritoneal cavity lavage is done.

Biopsy: can be taken wherever found necessary22

Ultrasound and CT scanning:

These investigations are not routinely done. They are done in stable patients when diagnosis is in doubt or in certain conditions. E.g. perforated appendicitis, acute pancreatitis may show fluid collection in peritoneal and pelvic cavities which may influence operative approach or contraindicate operation. Other investigations have to be done according to the specific etiology and patient condition, which is described individually[48]



NEED FOR A SCORING SYSTEM:

The complex nature of peritonitis, the modern aspects of treatment, and the complexity of ICU support make evaluation of new diagnostic and therapeutic advances in this field very cumbersome. Scoring systems that provide objective descriptions of the patient's condition at specific points in the disease process aid our23 understanding of these problems is absolutely necessary

Many scoring systems have been devised for assessing patient risk of mortality and morbidity during peritonitis. So a reliable, reproducible and cost effective scoring system is needed to

1. Assess the effectiveness of different treatment regimen
2. Adequate utilizisation of surgical ICU
3. Identify patients who require more aggressive management
4. Assess the patient and predict outcome
5. Be able to inform patient's relatives with greater objectivity

Most of the scoring systems are created from large operative databases, using statistical and research to predict which outcomes are strongly associated with outcome. When a patient presents to the hospital requiring emergency surgery the surgeon and anesthetist must be able to assess the risks of anesthesia and surgery by establishing the patient's pre-morbid condition, quality of life and to weigh the risk benefit ratio. Surgery may not be advised if the chance of success is minimal and the risk of causing a fatal outcome is considered more.

EXISTING SCORING SYSTEM:

Scores	Predicting Mortality	Predicting Morbidity
Scores does not require operative information	ASA APACHE-II Boey Score Sickness assessment Hacetteppe Score Physiological POSSUM	APACHE-II VA pneumonia prediction index VA Respiratory failure score Veltkamp score
Scores that require operative information	Mannheim peritonitis index Fitness score Reiss index POSSUM,P-POSSUM Cleveland colorectal model surgical Risk Score	POSSUM,P-POSSUM

ASA SCORING:

This score is routinely used in surgical emergency cases and was not originally designed to predict mortality but it has been shown to give a good estimate of mortality risk with great advantage of being simple. Down side being it is subjective and inter observer variation in measuring. The fact that ASA scores vary between observers suggest that it is really an expert assessment of risk and not actually a scoring system at all

Table 2b: Summary of the 6 studies observing mortality after emergency surgery in the elderly

	ASA I	ASA II	ASA III	ASA IV	ASA V
Total deaths	31	62	143	41	
Total cases	511	338	329	46	
Mortality	**6%**	**18%**	**44%**	**89%**	

ELEBUTE & STONES SEPSIS SCORE:

It was devised in 1983 primarily designed for district general hospitals for monitoring patients affected with peritonitis. It consist of 4 classes to which they ascribed a subjective degree of severity in analogue scale. The 4 parameters that are studied are namely, local effects of tissue infection, degree of temperature elevation, secondary effects of sepsis & lab data

ADVANTAGE:

- ✓ Since it is designed for district hospitals it is more appropriate for indian set up
- ✓ More sensitive since it includes detailed clinical work up
- ✓ Can be used as a one time assessment or can be used in continuous monitoring critical care patients
- ✓ Range of lab data is minimum

DISADVANTAGE:

- ✓ Subjective , more prone for observer variation
- ✓ No direct attempt to scor SEPTIC SHOCK, provided indirect evidence only

BOEY SCORING SYSTEM

a) Shock on the day of admission (SBP <90mm Hg)

b) Associated severe co-morbid illness(ASA III-IV)

c) Late presentation(>24hrs)

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%

ADVANTAGES

Simple, easy to remember and apply

DISADVANTAGES

a) Less accurate

b) Many vital parameters predicting the outcome of the disease are missing.

HACETEPPE SCORE:- used in acid peptic disease and perforation

Factors considered are

1. Male sex

Serious co-morbid illness

Acute renal failure

White cell count of more than 20x109/L

No study has been conducted to this score revalidating it other scoring system.

SICKNESS ASSESSMENT[37]

First described by Kennedy in 1993

1. Hypotension
2. Severe Co morbid Illness
3. Patient ambulatory or bedridden.

Patients presenting with SA score 0 there has been no deaths. With SA involving one, two or three parameters the mortality was found to be 52%,60%, and 100% respectively.

FITNESS SCORE:

Playforth and his colleagues in 1987 introduced this system.26 risk factors are identified and weighted arbitrarily from 1 to 4. In addition to difficulty in assessing 26 variables pre operatively some, such as the presence of perforation or obstruction and diagnosis of cancer, may not be available before laparotomy is done.

Reiss Index:

1. Age
2. urgency of surgery
3. ASA
4. Malignant perforation
5. Diagnosis

An emergency laparotomy where the diagnosis was not known could not be assessed with this system, which has shown to be ASA classification in predicting post-op mortality and morbidity

SCORES PREDICTING MORBIDITY:

1. **VELTKAMP SCORE:**

   It considers 11 criteria, minor complications are less commonly predicted so not widely used

2. **VA respiratory failure prediction index**

   VA study was conducted in 80000 men those developed respiratory failure (mechanical ventilation for more than 48 hours) for non-cardiac surgery. Score were given on age, type of surgery, emergency surgery or not, albumin, urea, co-morbid illness, respiratory function. A score of more than 40 predicts a risk of resp. failure of 31%

POSSUM SCORING:

Jones & walters(1991) from Copeland proposed this physiological & operative severity score. Uses up to 12 Physiological criteria and 6 operative criteria.

PHYSIOLOGICAL SEVERITY:

Age, cardiac and respiratory status, Systolic blood pressure, pulse, Glasgow come scale, Hb ,total count, urea, creatinine, sodium & ECG

OPERATIVE SEVERITY:

Peritoneal soiling, total blood loss, duration and mode of surgery, multiple procedures, malignancy

Drawbacks:

Tends to overestimate mortality and less specific when used in other specialties P POSSUM- Portsmouth predictor equation for mortality Prytherch 1998 proposed a corrected version of original POSSUM scoring system. it is accurate compared to POSSUM. Higher the risk more is the accuracy of the scoring system. There has been several modifications like V POSSUM used in specilities

APACHE II:

it is one of the most widely used scoring system which integrates various parameters during the first 24hrs of ICU admission. The original APACHE II was designed to predict mortality risk stratification by assessing the patient independent effects of medical or surgical treatment

DISADVANTAGES:

- ✓ The primary intra op findings are not considered
- ✓ The primary surgery would have altered the variables used to calculate APACHE II score
- ✓ Mortality prediction is less accurate
- ✓ Difficult to calculate since numerous variables are considered
- ✓ Complex and time consuming

MANNHEIM PERITONITIS INDEX:

Wacha & Linder in 1983collected data from 1253 patients admitted with peritonitis between 1963 & 1979 and identified 17 discrete factors affecting the outcome in these patients. From these 8 have been chosen to have prognostic relevance and included in calculating the mortality. Informations are collected at two stages one at the time of admission other at the time of first laparotomy.

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

Definitions or organ failure

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

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

A. Billing conducted a detailed study in 7 different centers and their data were compared and was found to be convincing. They considered patients admitted with perforated or post-operative peritonitis in this;

- ✓ maximum score is 47
- ✓ each risk factor or operative finding is given a weightage to produce a score
- ✓ The cut off point is taken as 29 above which the outcome is very poor patients were divided into 3 categories:

I- MPI<21
II- MPI 21-29
III- MPI >29

Studies shows that, there exists a linear relationship between mean index score and mean mortality rate

ADVANTAGES:

- ✓ Fast & easiest
- ✓ Determination of risk is available during operation
- ✓ Possible outcome and appropriate management can be initiated

Patients presenting with lower peritonitis score are treated with low risk and can be manage in post-op ward in contrast to patients who present with high score needs a continuous intensive care management. And these patients are not candidates for prolonged and major resection procedures. These patients are managed with Damage Control Procedures or programmed relaparotomy, zip technique surgery may considered.

DISADVANTAGES:

- It is a one time score; hence postoperative follow up it cannot be used
- Since the index considers operative findings also. surgeon cannot predict the outcome pre operatively

WHICH SCORING SYSTEM IS BEST?

Though there is no major comparative study or meta-analysis of data is done to compare all the studies almost all researchers agree for a reliable, reproducible, simple and less timing and less cost effective scoring system which helps not only in decision making . Prognosticating sepsis but also used for comparing data at different institutes.

Demmel[16] conducted a study comparing MPI scoring and APACHE II scoring and concluded both scoring systems predicted the outcome accurately but MPI was easier and disease specific and cost effective

Billing[1] who conducted MPI scoring system in 3 centres in 3 different European countries concluded that MPI not only reliable but also can be used in comparative study

Ohmann.C[25] concluded that none of the present scoring system accurately predicted the outcome and new prognostic model should be the focus of further trials

In 1996 Pacelli3 compared MPI, APACHE and sepsis score & concluded both APACHE & MPI rightly predicted death as outcome.

From the above studies, it appears that MPI and APACHE scoring system seems to be appropriate for patients presenting with sepsis. with MPI being easy and cost effective can be used in district hospitals also for predicting the outcome

MANAGEMENT OF PERITONITIS
STANDARD TREATMENT:

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

1. “Plugging”-the source of infection.
2. “Purging”-the peritoneal cavity of bacteria, toxins and adjuvant.Thus the laparotomy, repair of bowel leak and peritoneal toilet became the standard therapy, but the morbidity and mortality continued to be high.

Disadvantages of standard operative treatment:

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

New operative concepts:

The era of new operative concept started in 1975 when the dissertation of Pujol from Parries University. He concludes that intraabdominal Sepsis should be treated like 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.[22]

Vital system support:

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

1. Inability to maintain adequate alveolar ventilation as evidenced by a rising $PaCO_2$ of 50 mm Hg or greater.

2. Hypoxemia reflected in $PaO_2 < 55$ mm Hg.

3. Evidence of shallow, rapid respiration due to muscular tiring orthe use of accessory muscles of respiration.

Antibiotic therapy:

The bacterial flora is monomicrobial in nature, in primary peritonitis and polymicrobial in secondary peritonitis, an observation established by Alt emeir in 1938, in a study of appendiceal abscess.[27] When experimental peritonitis with E. coli and B. fragilis was treated with different antibiotic regimens, clear patterns of response were seen. Treatment with gentamicin alone improved the acute death rate in the model but had no impact on the abscess phase of the disease. Nicholas et al demonstrated improvement in the death rate of rats with polymicrobial experimental peritonitis induced with a large inoculum, by the addition of clindamycin coverage for B. fragilis. From these animal studies, combination therapy was born and became the standard for the treatment of peritonitis. during the late 1970s. In the 1980s, the emergence of single antibiotics with both aerobic and anaerobic activity leads to numerous clinical studies that compared the newer antibiotics to combination therapy. With one exception, most comparative studies consistently demonstrated comparable results with single agent compared to the combination. Costs and drug toxicity reduced with the single antibiotic approach.

As the infection is usually a mixed one, a single or combination therapy that have activity against aerobic and anaerobic bacteria, is used. Culturing peritoneal fluid and modifying the antibiotic subsequent to the culture sensitivity may not always influence the outcome.

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

MILD TO MODERATE INTRA-ABDOMINAL INFECTION

Second or third generation cephalosporin OR

ß- Lactamase inhibitor combination OR

Monobactum + metronidazole

SEVERE INTRA-ABDOMINAL INFECTION WITHOUT RENAL DYSFUNCTION

Carbapenem OR

Fluoroquinolone + metronidazole OR

Aminoglycosides + metronidazole + ampicillin

SEVERE INTRA-ABDOMINAL INFECTION WITH RENAL DYSFUNCTION

Carbapenem OR

Fluoroquinolone + metronidazole[21]

SPECIFIC TREATMENT OF THE CAUSE (OPERATIVE MANAGEMENT):

The primary therapy in the management of generalized peritonitis is surgical. This depends on the cause of generalized peritonitis e.g. perforation closure in case of perforated duodenal ulcer. Though there are other factors that affect the outcome in suppurative peritonitis, timing of operation is an important variable that is often overlooked. In peritonitis due to pancreatitis or salphingitis or in cases of primary peritonitis of streptococcal or pneumococcal origin, non-operative management is preferred (if the diagnosis is made with certainty).

OPERATIVE PRINCIPLES:

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

PRINCIPLE – 1 REPAIR:

The infectious material leaking into the abdomen is to be eliminated. This involves procedures like appendicectomy, closure of duodenal or ileal perforation, 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 liters may be required for wash and "radical debridement". However, to aggressive debridement should be avoided to prevent excessive blood loss or bowel injury. Antibiotic/ betadine wash have not been proved to be any great advantage. At the end no irrigation fluid should be left in the abdomen.

PRINCIPLE-3 DECOMPRESSES:

During acute peritonitis more than 10 liters of inflammatory fluid may accumulate in the peritoneum and its sub-mesothelial loose connective tissue. The co-existent paralytic ileus, fluid accumulation in the peritoneal cavity, post resuscitation visceral and parietal edema increases the intraabdominal pressure producing a compartment syndrome. In this situation, if the abdomen is

closed with tension, there will be impairment of cardiovascular, respiratory, renal and hepatic functions and also splanchnic blood flow and oxygenation. The answer to this problem lies in open abdomen or staged abdominal repair (STAR).

PRINCIPLE-4 CONTROL:

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

NEW OPERATIVE METHODS:

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

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

OPEN ABDOMEN (LAPAROSTOMY):

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

COVERED LAPAROSTOMY (COLA):

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

PLANNED REPAPAROTOMY (PR):

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

STAGED ABDOMINAL REPAIR (STAR):

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

INDICATIONS FOR STAR:

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.[19]

ADVANTAGES OF STAR:

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

PERITONEAL LAVAGE:

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

There are 3 basis principles of peritoneal lavage

1. To wash the digestive enzymes, that might have leaked into the peritoneal cavity.
2. To remove material like pus, blood and faeces that could harbor or nourishbacteria
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 l. 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[28].

DRAINS:

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

CONSERVATIVE MANGEMENT

Conservative management may be advisable in following conditions

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

COMPLICATIONS OF PERITONITIS
SYSTEMIC COMPLICATION OF PERITONITIS:

1. Bacteremic/endotoxic shock
2. Bronchi pneumonia/respiratory failure
3. Renal failure

4. Bone marrow suppression
5. Multisystem failure

BACTEREMIC/ ENDOTOXIC SHOCK:-

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

BRONCHOPNEUMONIA/ RESPIRATORY FAILURE:

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

RENAL FAILURE:

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

ABDOMINAL COMPLICATIONS OF PERITONITIS:

1. 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.1 9, 21.
SECONDARY PERITONITIS
CHEMICAL (ASEPTIC) PERITONITIS:

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

PERITONITIS DUE TO PERFORATED PEPTIC ULCER:

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

perforated gall bladder and duodenum.[29] Sometimes, following an ulcer perforation, the ulcer may seal rapidly before there is a spillage of gastric and duodenal contents. Other rare presentations of perforated duodenal ulcer:

1. Perforation associated with hemorrhage is rare but a grave complication. The bleeding arises from erosion of large vessel such as gastroduodenal artery. The clinical picture is that of acute perforation of peptic ulcer with signs of hemorrhage.

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.

- ✓ Simple closure of perforation with omental patch.
- ✓ 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.

- ✓ Laparoscopic closure of perforation

APPENDICEAL PERFORATION:

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

Appendiceal rupture occurs most frequently distal to the point of luminal obstruction Along the antimesentric border of the appendix. Rupture should be suspected in the presence of fever greater than[39] 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.[22]

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 patients.[19]

Treatment:
Surgical Management:

At laparotomy, a single perforation is found on the anti-mesentric border of the ileum in 80 per cent of the patients. Two perforations are found in 15 per cent and more than two in 5 per cent. About 90 per cent of ileal perforations are located within 60cm of the ileo-caecal valve and caecal perforations occur in only 2 percent of the patients. Perforations at the sites other than ileum and caecum are extremely rare. A simple debridement of the margin of the perforation and meticulous closure in two layers with copious peritoneal lavage, is the procedure of choice. However, when there are more than three perforations, which are close together, it is best to resect the affected bowel and perform a primary end-to-end anastomosis. Any areas of apparent impending perforations, if not included in a resection, must be over sewn. A right hemicolectomy is undertaken only for caecal perforations.

Following peritoneal lavage, the abdominal wound is closed, 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.[30]

COLONIC PERFORATION:

Perforation is less common than is obstruction, occurring in about 5 percent of patients. The site of perforation is usually within the tumor and is not associated with obstruction but is the consequence of tumor necrosis. Rapid cardiovascular collapse and endotoxaemic shock, usually signify a major leak and faecal peritonitis.

About 22 percent of the cases of peritonitis have their origin in colon. More than half of these are due to inflammatory diseases, such as diverticulitis. The remaining cases are due to perforation proximal to or at stenosis caused by luminal bowel obstruction (tumor) or external bowel obstruction such as incarcerated hernia, intussusception and volvulus... A malignant growth usually does not cause peritonitis directly but may lead to bowel obstruction with either perforation of dilated segments or bowel ischemia and/or bacterial migration through the necrotic bowel wall.

SURGICAL TREATMENT:

The goal of operation is to remove the diseased perforated segment of the bowel. It is possible to fashion a primary resection and end-to-end anastomosis. However, an anastomosis of unprepared bowel fashioned in a contaminated field should always be protected by proximal colostomy or ileostomy. The temporary diverting stoma can be closed about ten weeks after the emergency operation. An alternative is to resect the perforated segment and to exteriorize the proximal and distal loops of the bowel, where the proximal opening acts as the colostomy and the distal as the mucous fistula or to use Hartman’s operation for more distal lesions, where the distal end is not

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

TUBERCULOUS PERITONITIS:

Two forms of peritonitis are seen- Acute and chronic

ACUTE TUBERCULOUS PERITONITIS:-

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

CHRONIC TUBERCULOUS PERITONITIS:-

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

ORIGIN OF INFECTION:-

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

VARIETIES OF TUBERCULOUS PERITONITIS:-

There are four varieties of tuberculous peritonitis

a. Ascitic.

b. Encysted.

c. Fibrous.

d. Purulent.

ASCITIC FORM:-

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

ENCYSTED FORM: (LOCULATED)

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

FIBROUS FORM: (PLASTIC)

Fibrous form is characterized by the production of wide spread adhesions, which cause coils of intestine, especially the ileum to become matted together and distended. These distended coils act as a 'blind loop' and give rise to steatorrhoea, wasting and attacks of abdominal pain. On examination, the adherent intestine with omentum attached, together with the thickened mesentery, give rise to a palpable mass. The first intimation of the disease may be sub-acute or acute intestinal obstruction. The division of bands can remedy sometimes the cause of the obstruction easily. If the adhesions are accompanied by fibrous strictures of the ileum as well, it is best to excise the affected bowel, provided not too much of the small intestine needs to be sacrificed. If adhesions are only present, a plication may be performed. Chemotherapy after adequate surgery will rapidly cure the condition.

PURULENT FORM:

The purulent form is rare, and usually occurs secondary to tuberculosis salphingitis. 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 liter of fluid is cultured. The ascitic fluid has an increased protein concentration, lymphocytic pleocytosis and glucose concentration below 30mg/dl. At laparotomy a peritoneal biopsy should be taken. The placement of drains or exteriorization of bowel should be avoided.

TREATMENT:

MEDICAL LINE OF MANAGEMENT:

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

SURGICAL LINE OF MANAGEMENT:

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

MANAGEMENT OF TUBERCULOUS PERFORATIONS:

According to the site of perforation:

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

AMOEBIC PERFORATION:

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

MECONIUM PERITONITIS:

Meconium is a sterile mixture of epithelial cells, mucin, salts, fats and bile. It is formed when the fetus commences to swallow amniotic fluid. Meconium peritonitis is an aseptic peritonitis, which develops, late in intrauterine life or during or just after delivery. In the remainder no cause for the perforation is discernable. It causes matting of intestinal loops and in some cases, the extruded meconium becomes calcified in a matter of weeks. Meconium remains sterile until[19] about three hours after birth; thereafter, unless the perforation has sealed, sterile meconium peritonitis gives way to acute bacterial peritonitis, which, unless treated promptly, is rapidly fatal.[22]

FOREIGN BODY PERITONITIS:

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

PERIODIC PERITONITIS:

Recurrent episodes of abdominal pain, fever, and leukocytosis occur in certain population groups, notably in Americans, Arabs and Jews. The disease appears to be familial. The major point for the surgeons is that, laparotomy is not required I 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 attack and a favorable response to chronic administration of colchicine is a definitive diagnostic test.

DRUG RELATED PERITONITIS:

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

LEAD PERITONITIS:

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

HYPERLIPEDIMIC PERITONITIS:

Abdominal pain mimicking peritonitis may be seen in patients with type 1 and type V hyper lipoproteinemia a group of heterogeneous disorders resulting from increased concentration of chylomicrons or VLDL in the blood. If erroneously operated on during early stages, the abdominal cavity is found to be full of chylous milky material. A careful family history will clarify the differential diagnosis.

PORPHYRIC PERITONITIS:

It is seen in patients with acute 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 tumor (chronic inflammatory omental tumors) and formation of dense adhesion may follow Contamination of peritoneal cavity by glove lubricants (talc, lycodium, mineral oil, corn starch, rice starch) or by cellulose fibers from disposable gauze pads and gowns.

The reaction, particularly to rice starch, is largely a hypersensitivity response. When the diagnosis remains unclear, laparoscopy is useful. If the peritonitis is recognized, reoperation may be avoided and corticosteroids or non-steroidal anti-inflammatory drugs administered. Eventually the peritonitis resolves.

TERTIARY PERITONITIS:

Patients, 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 PERITONII):

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

PSEUDOMYXOMA PERITONEI:

More frequently in females the abdomen is filled with yellow jelly, large quantities of which are often more or less encysted. The condition is associated with both mucinous cystic tumors of ovary and appendix. Recent studies suggest that most cases arise from primary appendiceal tumors with secondary implantation on to one or both ovaries. It is often painless and there is frequently no impairment of general health for a long time. If the abdomen seems to be distended with fluid, which cannot be made to shift, it should raise the suspicion of pseudomyxoma peritonei. At laparotomy, masses of jelly may be seen which are scooped out. The appendix, if present, should be excised with any ovarian tumor. Unfortunately, recurrence is common.

Pseudomyxoma peritonei is locally malignant, but does not give rise to extra-peritoneal metastasis. Occasionally, the condition responds to radioactive isotopes or intra peritoneal chemotherapy, which may be used in recurrent cases.32

POST-PUERPERAL PERITONITIS:

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

TREATMENT:

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

PERITONITIS RELATED TO PERITONEAL DIALYSIS:

- ✓ Peritonitis is 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:

Positive culture from the peritoneal fluid.

a. Cloudy dialysate effluent.

b. 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[19] Post operative period was monitored; intake output charts and vital charts were maintained. Drains were removed after 48 hours and sutures were removed on the 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:

30 patients with hollow viscous perforation admitted in Government Royapettah Hospital, Kilpauk Medical college Hospital from April 2014 to September 2014 were included in the study Necessary data to be collected; MPI score were to be calculated for each patient and analysis to be done

METHOD OF COLLECTION OF DATA

The study is done after obtaining a detailed history, complete general physical Examination and systemic examination. The patients are subjected to relevant investigations like x-ray erect abdomen, CXR, USG and routine investigations like Hb, TC, urea, creatinine, serum electrolytes. All investigations and surgical procedures were carried out with proper informed written consent as appropriately. The data regarding patient particulars, diagnosis, investigations, and surgical procedures is collected in a specially designed case recording form and transferred to a master chart subjected to statistical methods like mean, standard deviation, proportion, percentage calculation and wherever necessary chi square test for proportion are used.

Inclusion Criteria:

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

Various etiologies causing such features include

Acid peptic disease,

Typhoid,

Tuberculosis,

Gangrenous cholecystitis,

Appendicitis,

Malignancy

Exclusion criteria:

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

MODE OF STUDY:

The detail history and proper clinical findings were entered in a proforma case sheet. Patient was subjected to methodical physical examination to assess his general condition. Local examination of abdomen was done and relevant findings were recorded. Rectal examination was done in all cases, per vaginal examination was also done in female patients. The required and routine investigations were done to establish the diagnosis. Patients were asked to present themselves for follow up after a specific interval or at recurrence of symptoms.

MPI scoring system was done in all patients and patients were classified those with score less than 21, 21 to 27, and more than 27. Preoperatively all patients received supportive treatment for correction of hypotension and electrolyte abnormalities. During laparotomy, intra-abdominal examination of all organs was made in addition to specific pathology. Primary closure of hollow viscous perforation was made in all cases with thorough peritoneal lavage and abdominal drains were kept in all patients. Post-operative period was monitored; intake output charts and vital charts were maintained. Drains were removed after 48 hours and sutures were removed on the 7^{th} post-operative day. Most of the operated patients had uneventful recovery, 18 patients had morbidity in terms of wound infection and intensive care, 28 patients had mortality. The patients were followed up for a variable period of time.

OBSERVATION AND RESULTS

In this study 30 cases of patients presenting with peritonitis who attended surgical emergency unit from paril 2014 to September 2014 were selected and studied and the following datas were collected

TABLE 1. AGE & SEX WISE DISTRIBUTION OF STUDY SUBJECTS

	MALE (%)	FEMALE (%)	TOTAL (%)
LESS THAN 25	5 (16.6%)	2(6.66%)	7
25-50	11(36.6%)	4(13.3%)	15
More than 50	6(20%)	2(6.66%)	8
TOTAL	22	8	30

In this study, the mean age of peritonitis was 41.3yrs (S.D 18.55yrs) & majority of patients belong to the age group 25-50 years (49.9%). The is a male preponderance with male to female ratio 2.75:1

FIGURE 1

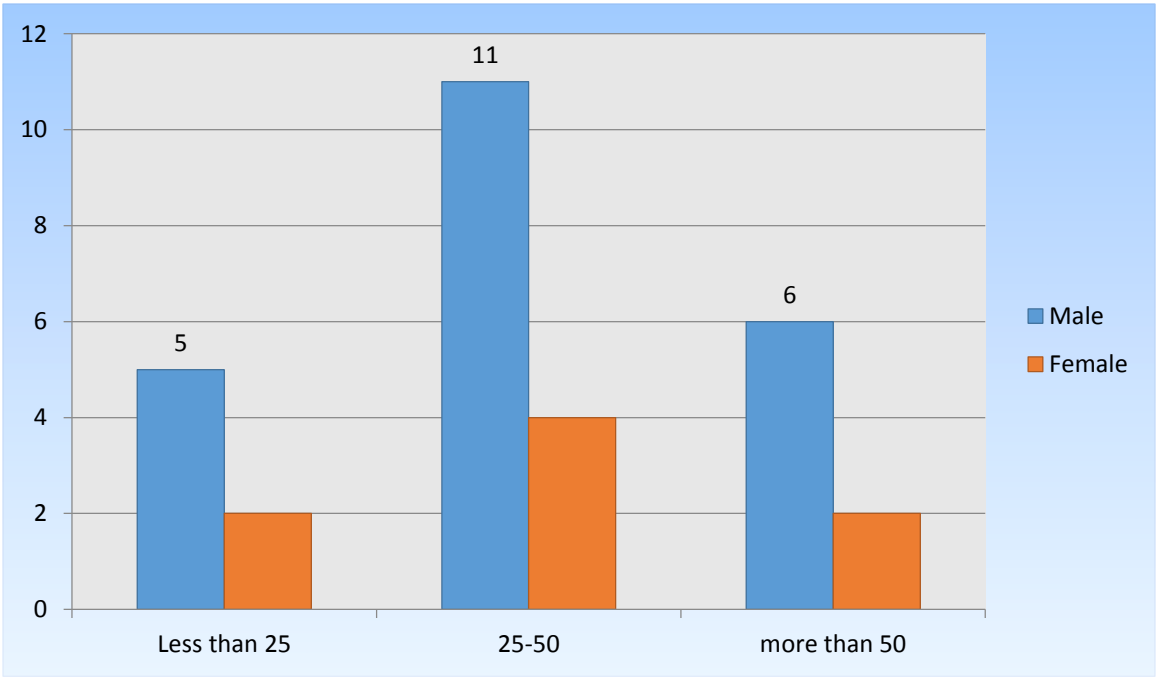


TABLE2.TIME OF PRESENTATION & MORTALITY ASSOCIATION

Duration	Mortality (%)	Survival(%)	Total(%)
One day	2 (20%)	8 (80%)	10
2 to 5 days	3 (15.7%)	16 (84.3%)	19
More than 5 days	1 (100%)	-	1

In this study with 30 patients , most of them presented after 24hrs to the hospital (63.3% & 3.3%) and the mortality and survival rate was 15.7% % 84.3% for presentation between 2to5 days and mortality rate was 100% for the patient presenting after 5 days. In contrast to patients presenting early within 24hrs had better survival rate of 80%.

FIGURE 2

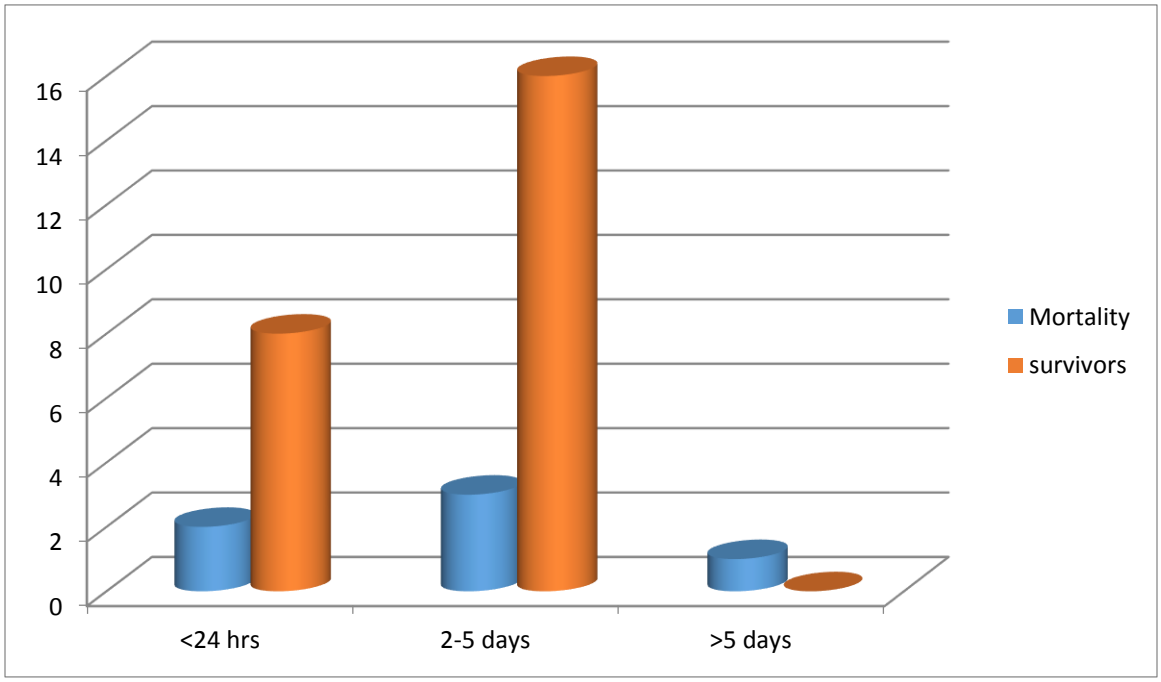


Table3. Distribution of study subjects and MPI score

MPI SCORE	DEAD %	SURVIVORS %	TOTAL %
<21	0	13 (100%)	13(43.3%)
21-27	4 (33.3%)	8 (66.6%)	12(40%)
>27	5 (100%)	0	5(16.67%)
TOTAL	9	21	30 (100%)

In this study involving 30 patients, 43.3% of patients had a MPI score of less than 21 of which survival rate was 100% with no mortality . for 40% of the patients with MPI score 21-27 the mortality score noted was 33.3% and survival rate noted was 66.6%. and in patients with MPI score of more than 27 mortality rate was found to be 100% these patients are otherwise called as non survivors

FIGURE 3:

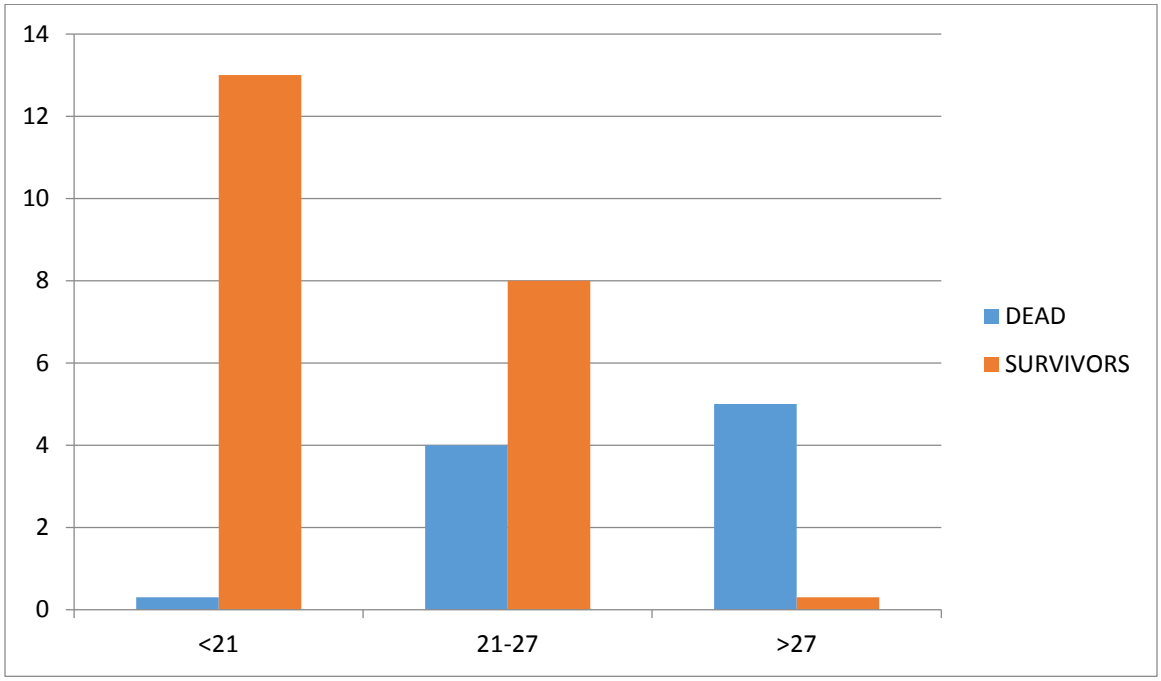
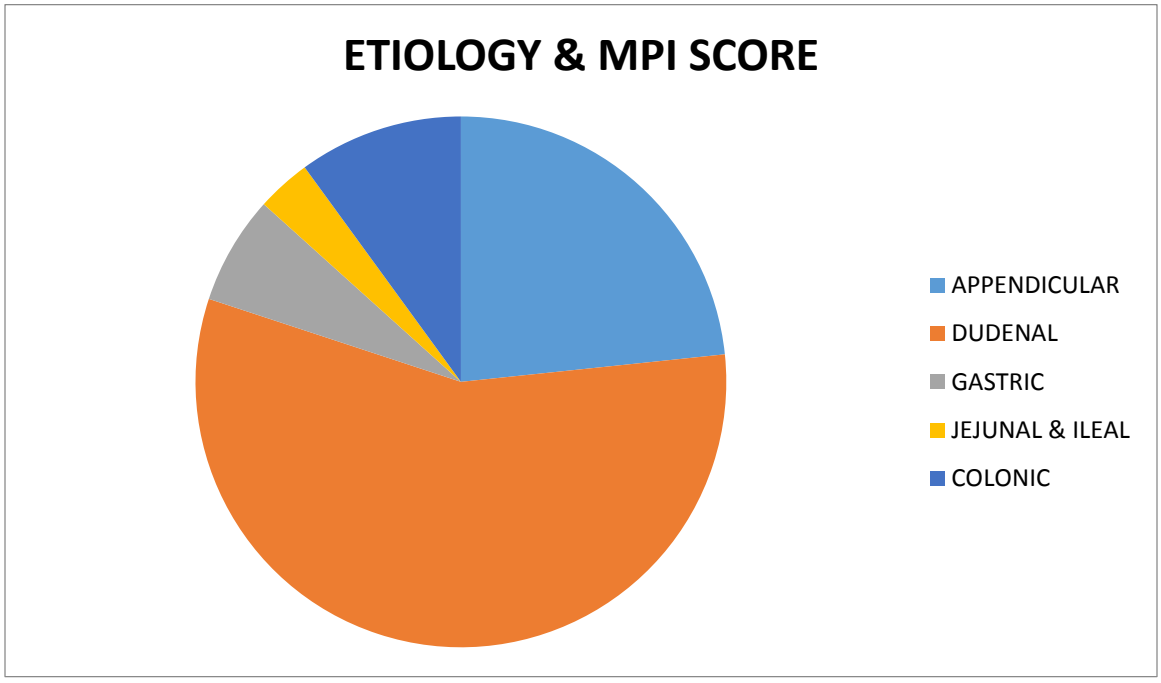


Table4. Etiology and MPI score

MPI SCORE	<21	21-27	>27	PERCENTAGE
APPENDICULAR	1	6	-	23.3%
DUDENAL	11	4	2	56.6%
GASTRIC	2	-	-	6.6%
JEJUNAL & ILEAL	-	1	-	3.3%
COLONIC	-	-	3	10%

In this study involving 30 patients , duodenal perforation was most commonly found in about 56.6%, followed by appendicular 23.3%, colonic 10%, gastric 6.6% and jejunal and ileal 3.3%. Among the duodenal perforation group 6.6% had a score of more than 27 due to late presentation and had very poor survival rate. The colonic perforation patients had MPI score of more than 27 and had very poor survival rate due to faecal contamination.

FIGURE 4:



DISCUSSION

Management of peritonitis remains a challenge for surgeons despite the advancements in investigations surgical techniques and intensive care management. Several factors like age , sex, duration, site of perforation, size of perforation, extent of peritonitis, delay in presentation all contribute to mortality and morbidity. A successful intervention depends upon early diagnosis and surgery, source control & through intra peritoneal lavage

R.Fugger,M .Rogy F .Schulz et all 113 patients suffering from peritonitis entered this retrospective study for evaluation of prognostic value of Mannheim peritonitis index. They found no mortality for scores below 21. Between 21-29 it was 29% and mortality increased to 1005 wen scores were more than 30. Statistical analysis showed that the prognosis were correct in 93% . Between x=21 and x=29 prognosis of MPI was correct in at least 65%. The MPI is shown as a prognostic index for peritonitis with high accuracy in individual prognosis. That could be easily documented.[38]

Billing ,D.Frohlich, W.Schildberg et all. The reliability of MPI was & its predictive power was studied in 2003 patients in seven centres in three European

countries. The prevalence of risk factors varied considerably in the study groups. For a threshold index of 26, the sensitivity was 86, specificity was 74% and accuracy was 83% in predicting death. For the patients with a score less than 21 the mean mortality rate was 2.3% for score 21-29, 22.5(range 10.6-50) %& for score greater than 29, 59.1 (range 41-87) percent. The mean index score & mean mortality rate correlated in the dufferent groups, reflecting a homogenous standard of therapy for peritonitis.

Ohmann et all[33] reported that duodenal ulcer as the most common cause for peritonitis in his series while **Kachroo et all**[33] found that appen. Perforation as the commonest cause. The over all diagnostic accuracy for peritonitis was 97.3%

In that study, most of the patients presented to the hospital after 24hrs., between 2 to 5 days and the mortality were 26.9% and 75% respectively as compared mortality (7.1%) in patients who presented on the first day of onset of symptoms.

In the study group of 100 patients, 52% of patients had MPI score less than 21 of which 5.8% of patients developed wound infection with 0% mortality and 94.2% of patients being normal, 41.4% of patients had morbidity and mortality MPI score 21 to 27 and those patients with MPI score more than 27 had the highest mortality of 84.2%.

Billing A, Frohlich D Schildberg FW [1,35,36] analyzed patients with scores <21 had a mortality ranging from 0-2.3% and those with MPI between 21 and 29 had a mortality rate of approximately 65%[36] MPI score of more than 29 had the highest mortality up to 80% in some studies[37]

In the study group, 75% of the patients had morbidity in terms of infection and gaping with scores more than 21. The positive predictive value of MPI score for morbidity is 75% with sensitivity 83.3%. specificity 90.74%. three patients required ICU care for three to four days.

In the study group, 84.8% of patients had mortality among patients with MPI score more than or equal to21 and none of the patients died with MPI score <21. The PPV of MPI score for mortality 84.8% sensitivity 100% & specificity 90.74%.

Barrera Melgarejo E, Rodríguez Castro M, Borda Luque G, Najar Trujillo N. et. al. A prospective study appears, of 103 patients, greater of 14 years, with I diagnose of peritonitis, between November 2004 to April 2005. a mortality of 50% in patients with greater index of 26 points was obtained.

One was a sensitivity 95.9%, a specificity of 80%, with positive a predictive value 98.9% and a negative predictive value of 50%. When considering 3 groups, < 21, 21-29 and > 29 points, was a mortality of 60% in patients with greater index of 29. I am made a survival curve obtaining itself a significant difference with a p=0, 0098.[50]

Mulari K, Leppäniemi A et. al. Retrospective analysis of 66 consecutive Patients with secondary peritonitis caused by gastrointestinal tract perforation and Requiring postoperative treatment in an intensive care unit was performed using univariate and multivariate analysis to identify risk factors for hospital mortality. The overall hospital mortality rate was 36 %. Significant risk factors in the univariate analysis included advanced age (p = 0.000), pre-existing illness (p = 0.000), chronic medication (p = 0.028), hospital transfer (p = 0.036), non-traumatic cause of perforation (p = 0.031), high Mannheim peritonitis index (MPI) score (p = 0.001), and high C-reactive protein (CRP) level in the early postoperative phase (p = 0.015). In a multivariate analysis, only advanced age (odds ratio 1.1008, p =

0.000) and high postoperative CRP level (odds ratio 1.0095, p = 0.008) were identified as independent prognostic factors for hospital mortality.[44]

Gedik E, Girgin S, Taçyildiz IH, Akgün Y et.al. Ninety-six patients with typhoid enteric perforation were reviewed. Nine variables were applied the univariate analysis, which were greater than 30 years (P = 0.218), male gender (P = 0.02), preoperative treatment (P = 0.147), less than or equal to 48 h perforation-operation interval (P =0.013), greater than 4,000 K/UL WBC (P = 0.388), less than 8 g/dL Hgb (P = 0.026), greater than 29 Mannheim Peritonitis Index (P < 0.0001), multiple perforation number (P = 0.614), and primary repair (P = 0.105). Logistic regression analysis showed that Mannheim Peritonitis Index (P = 0.014) and perforation-operation interval (P = 0.047) were defined as independent risk factors affecting morbidity.

Ermolov AS, Bagdat'ev VE, Chudotvortseva EV, Rozhnov AV. Et. al. A retrospective analysis of 100 case histories of patients with diffuse peritonitis was made in order to evaluate the prognostic significance of the Mannheim Peritonitis Index (MPI). The patients were divided into 3 groups according to the amount of scores: in the first group (12-20 scores) there were no lethal issues, in the second group (21-29 scores) 42% of the patients died, 100% lethality was noted in the

third group when MPI was 30 scores or more. MPI allows not only making a retrospective evaluation of results of treatment of peritonitis and comparing outcomes of different surgical accesses; it may be one of criteria for programmed relaparotomy or for using laparostomy in patients of the 2nd and 3rd groups.[48]

Kologlu M, Elker D, Altun H, Sayek I. et. al.A total of 473 patients were included in the study; 75 of them had postoperative peritonitis (POSTOP group) and the remaining 398 had secondary peritonitis due to other causes (OTHER group). Using multiple logistic regression, MPI and PIA II were combined in an equation and this new variable was called combined peritonitis score (CPS). Overall mortality was17.8% in OTHER group and 33.3% in POSTOP group ($P = 0.0018$). Higher MPI scores, lower PIA II scores and higher CPS scores were associated with higher mortality in both groups ($P < 0.0001$). Mean MPI values were higher, mean PIA II values were lower and mean CPS values were higher in POSTOP group ($P < 0.001$).

The areas under ROC curves of CPS were bigger than MPI and PIA II in both groups. Sharpness of CPS was higher in both groups compared to MPI and PIA II ($P < 0.05$). Proportion of correct predictions of outcome was highest in CPS among the three scores ($P = 0.0074$). CPS had the best correlation with observed mortality.[45]

Malik AA, Wani KA, Dar LA, Wani MA, Wani RA, Parray FQ. Et. al.A prospective study was conducted using 101 consecutive patients (69 male, 32 female) 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. Similarly, in the APACHE II system, no mortality was noted in patients with scores less than 10. Mortality was 35.29% and 91.7% in the groups scoring 10-20 and more than 20,respectively.[52]

Bracho-Riquelme RL, Reyes-Romero MA,Torres-Valenzuela A, Flores-García AI. Et. al. A statistically significant association was found between the medians of severity of peritonitis and IL-6 ($p < 0.025$), TNF-alpha ($p < 0.01$), CRP ($p < 0.033$), IL-10 ($p < 0.0001$), and IL-13 ($p < 0.004$). Both TNF-alpha and IL-10 had a direct, and IL-13 an indirect, relation to severity, whereas CRP and IL-6 tended toward linear behavior in equilibrium. A significant association persisted between individual MPI scores and IL-6 ($p < 0.002$), TNF-alpha ($p < 0.002$), CRP ($p < 0.002$), and IL-10 ($p < 0.001$), but not IL-13 ($p = 0.646$).[51]

SUMMARY

- ✓ A study of MPI scoring system in 30 cases done in patients presenting with clinical features of peritonitis which was later confirmed peroperatively in govt royapettah hospital and kilpauk medical college over the period of six months from april2014 to september 2014. And necessary datas are collected and analyzed.
- ✓ Most of the patients presented with history of pain abdomen, abdominal distension, and fever with varying duration, most of the patients 66.6% presenting after 24 hours
- ✓ Relavent investigations were done in all patients and most patients diagnosed with plain x ray erect abdomen and ultra sonogram
- ✓ All the patients were managed by emergency laparotomy and primary repair if hollow viscous perforation is done
- ✓ Post operatively patients were managed with appropriate antibiotics, sicu management was required in 5 patients
- ✓ MPI scoring system done in all patients depending on preoperative and intra operative findings and patients were categorized under three, those with scores <21, 21-27,>27
- ✓

- ✓ Men are most commonly affected in the ratio of 2.7:1. With mean age group of 41.3 years.with SD 18.55yrs
- ✓ Those with scores <21 (43.3%) had no mortality and those with score >27 (16,67%) had 100% mortality and those with scores 21-27 (40%) had a mortaility in 2/3rds of the patients
- ✓ Etiologically duodenal perforation was found most commonly around 56.6% followed by appendicular (23.3%), colonic(10%), Gastric (6.6%) ileal (3.3%)

CONCLUSION

- ✓ Despite the advances in the investigations ICU care and surgical methods the hollow viscous perforation and peritonitis remains a hot spot for treating surgeons. Various factors like age, sex, duration, site of perforation and delay in surgical management are associated with morbidity and mortality.
- ✓ Males are more commonly affected than females
- ✓ Fecal contamination carries worst outcome
- ✓ Older age group individuals fight sepsis poorly and succumb to infections more easily
- ✓ Duodenal perforation remains the most common cause followed by appendicular perforation
- ✓ Emergency laparotomy and primary repair of the hollow viscous perforation and through peritoneal lavage remains the primary mode of management
- ✓ In the management of patients with generalized peritonitis scoring and categorizing the patients into various groups is beneficial. Because people who have scores between 21-27 needs more aggressive care and ICU management

- ✓ MPI scoring system is easy to apply, the risk determination is available during operation and the treating surgeon can know the possible outcome and the appropriate management can be instituted.
- ✓ MPI scoring his the best scoring system available in predicting the mortality of patients presenting with peritonitis

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ANNEXURES

ANNEXURE 1. 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

Progress : Steady / gradually declining / gradually/worsening /

Fluctuating / associated with appearance of swelling

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 of surgeries :

Tuberculosis :

Diabetes :

Hypertension :

D. FAMILY HISTORY

Tuberculosis :

Diabetes :

Hypertension :

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 sounds : Yes / No

Frequency

Character

EXAMINATION OF BACK AND SPINE

a) Renal angle

Fullness : Yes / No

Tenderness : Yes / No

Percussion : Res / dull

b) SpineDeformity: Yes / No

Tenderness : Yes / No

Paraspinal rigidity : Yes / No

Fullness : Yes / No

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

c)Occult blood

d) Chest X-ray :

e) Plain X-ray abdomen:

f) Ultrasound **:**

L.CLINICAL DIAGNOSIS

TREATMENT

Operative Simple / Radical

OPERATIVE FINDING:

MALIGNANCY: YES/NO

GENERALISED PERITONITIS:

ORGIN OF SEPSIS:

Nature of Exudates:

M.MPI SCORE

N. POST-OP-PERIOD: Complications

O.FOLLOW UP:

P.MORTALITY:

ANNEXURE 2

CONSENT FORM FOR ANAESTHESIA / OPERATION

Name: **Guardian: Relationship**

Full Address **Designation:**

I ……. Hosp. No…….. in my full senses hereby give my complete consent for …….partcipating in the study EFFECTIVENESS OF MANNHEIM PERITONITIS INDEX IN PREDICTING THE MORBIDITY AND MORTALITY OF PATIENTS WITH HOLLOW VISCOUS PERFORATION or any other intervention deemed fit which is and diagnostic/ biopsy / transfusion / operation to be performed on me /my son/my daughter …….. Age …….. under any anesthesia deemed fit. The nature and risks involved in the procedure have been explained to me to my satisfaction. For academic and scientific purpose, the operation / procedure may be televised or photographed.

Date: Signature of the Patient / Guardian

ANNEXURE 3. KEY TO MASTER CHART

A- age

AB-Absent

ABH-Altered bowel habits

A/D- Air under diaphragm

AD -Abdominal distension

APACHE- Acute physiology and chronic health evaluation.

AXR-Abdominal X-ray

BS-Bowel sounds

CVS-Cardiovasular system

Co-Coolie

CREAT-Creatinine

CXR- Chest X-ray

D -Days

DEC -Decreased

Diag -Diagnosis

DU-Duodenal

Dur- Duration

EmLap-: Emergency laparotomy

F -Female

F/ up -Follow up

Fe : Febrile

FF : Free fluid

Fw : Factory worker

G/R : Guarding/Rigidity

H/W : House wife

Hb : Hemoglobin

HT : Hypo tension

HyNa: Hyponatremia

IP no : In patient number

LA : Loss of appetite

M : Male

MPI : Mannheim's peritonitis index

N : Normal

Occ: Occupation

Olig : oliguria

Op finding: Operative findings

PERI: peritonitis

P/R: Per Rectal examination

PA: Pain abdomen

PHTB: Past h/o Tuberculosis

Post op: Post operative period

R/S : Respiratory system

SICU : Surgical intensive care unit.

SE: serum electrolytes

TC: total count

Ten: Tenderness

U: Urea

USG: Ultrasonography

Vom: Vomiting

WI : Wound infection

WL: Weight loss

Yrs: Years

Ip no	AGE	sex	duration of peritonitis	origin colonic
19860	62	f	>24hr	y
798	21	m	>24hr	n
1228	35	m	<24hr	n
3586	42	f	>24hr	n
2759	17	f	>24hr	y
3101	80	m	<24hr	y
2648	37	m	>24hr	n
4839	24	m	<24hr	n
5839	60	m	>24hr	n
3708	27	m	>24hr	n
2890	18	m	>24hr	y
3947	23	m	>24hr	n
5920	44	m	<24hr	n
6038	36	f	>24hr	n
16829	73	m	>24hr	n
25980	32	f	>24hr	y
19035	28	m	>24hr	n
29053	29	m	<24hr	n
18905	79	m	<24hr	n
39048	50	m	>24hr	n
10958	49	f	>24hr	y
23409	32	m	>24hr	n
19304	67	f	<24hr	n
9029	40	m	>24hr	n
8495	60	m	>24hr	n
7291	18	m	>24hr	n
3905	21	f	<24hr	y
4905	36	m	>24hr	n
19302	54	m	>24hr	n
27890	45	m	>24hr	n
	41.3			

organ failure	malignancy	diffuse peritonitis	nature of exudate
n	y	y	faeculant
n	n	n	clear
n	n	y	clear
n	n	y	purulent
n	n	n	feculent
y	y	y	feculent
n	n	y	purulent
n	n	n	clear
y	n	y	purulent
n	n	y	clear
n	n	y	purulent
n	n	y	purulent
n	n	y	clear
y	y	y	clear
y	y	y	purulent
n	n	n	faeculant
n	n	y	clear
n	n	y	clear
y	y	y	clear
n	n	y	purulent
n	n	n	faeculant
n	n	n	clear
y	n	y	purulent
n	n	n	clear
n	y	y	purulent
n	n	y	clear
y	n	y	faeculant
n	n	y	clear
y	n	y	clear
n	n	y	clear

MPI Score	category	morbidity	no of adm days	air un dia	guarding
42	3	y	2	present	present
4	1	n	7	present	present
6	1	n	7	present	present
21	2	n	10	present	present
22	2	n	14	absent	present
40	3	y	2	present	present
16	2	n	9	present	present
0	1	n	1	present	present
27	2	y	21	present	present
10	1	n	5	present	present
22	2	n	8	present	present
16	1	n	8	present	present
6	1	n	7	present	present
26	2	n	9	present	present
32	3	y	3	present	present
26	2	n	7	present	present
12	1	n	7	present	present
6	1	n	7	present	present
22	2	y	9	present	present
21	2	n	8	present	present
22	2	n	8	present	present
4	1	n	7	present	present
29	3	y	5	present	present
4	1	n	7	present	present
25	2	n	8	present	present
10	1	n	7	present	present
36	3	y	4	present	present
10	1	n	7	present	present
22	2	y	1	present	present
10	1	n	5	present	present