EVALUATION OF PROGNOSTIC SCORING SYSTEM IN PERFORATION PERITONITIS – COMPARITIVE STUDY BETWEEN APACHE II AND MANHEIM'S PERITONITIS INDEX SYSTEMS

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CERTIFICATE

This is to certify that the dissertation entitled **"EVALUATION OF PROGNOSTIC SCORING SYSTEM IN PERFORATION PERITONITIS – COMPARATIVE STUDY BETWEEN APACHE II AND MANHEIM'S PERITONITIS INDEX SYSTEMS"** is the bonafide work done by **Dr.Sudharsan. S. B.,** Post Graduate student (2010 – 2013) in the Department of General Surgery, Government Stanley Medical College and Hospital, Chennai under my direct guidance and supervision, in partial fulfilment of the regulations of The Tamil Nadu Dr. M.G.R Medical University, Chennai for the award of M.S., Degree (General Surgery) Branch - I, Examination to be held in April 2013.

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

Prof. P. DARWIN M.S.
Professor and Head of surgery,
Dept. of General Surgery,
Stanley Medical College,
Chennai - 600001.

PROF. S. GEETHA LAKSHMI, M.D., PhD,
The Dean,
Stanley Medical College,
Chennai

DECLARATION

I, solemnly declare that this dissertation titled "**Evaluation of Prognostic Scoring System in Perforation Peritonitis – Comparative Study between APACHE II and Manheim's Peritonitis Index Systems**" is a bonafide work done by me in the Department of General Surgery, Government Stanley Medical College and Hospital, Chennai under the guidance and supervision of my unit chief **Prof.A. RAJENDRAN, M.S.,** and Head of the Department **Prof.P. DARWIN, M.S.**

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CONTENTS

Introduction

INTRODUCTION

Gastrointestinal perforation is a common abdominal emergency and is still a dreaded condition with high mortality. Perforation of any part of the intestine is life threatening, which is most commonly managed by general surgeons. The vast majority of perforations are duodenal and gastric in origin, precipitated by alcohol and drugs. Malignancy and traumatic perforations are on the rise.

Evaluation and management of gastro-intestinal perforations provide one of the most challenging experiences for a surgeon. Ever since the "**Hippocratic facies**" was identified and attributed to peritonitis, there has been a continuous and remarkable change in the diagnosis and management. It is indeed true to mention here that ***"Only the changes are permanent in the field of medicine".*** Surgeons must continually reassess the standard of treatment and be receptive to new ideas.

The present thesis focuses on the prediction of mortality with APACHE II AND Manheim's Peritonitis Index in cases of perforation peritonitis and to identify the better among the two. To predict the prognosis and survival of a patient is indeed difficult as different patients

respond differently and hence the emphasis and need for development of an objective score.

Review of Literature

PERITONITIS AND INTRA ABDOMINAL INFECTION

"Peritonitis and intra abdominal infection are not synonymous"

Peritonitis[1] denotes inflammation of the peritoneal cavity and this may be caused by bacteria or by irritation of extravasated secretions. It is synonymous with systemic inflammatory response that occurs after trigger of any inflammation. Intra-abdominal infection refers to peritonitis caused by bacteria. It is regarded as a localized equivalent of systemic sepsis.

Intra-abdominal infection is defined as an inflammatory response of the peritoneal cavity to micro-organisms and their toxins, which results in production of purulent exudates within the cavity.

Intra-abdominal abscess[2] is the culmination of the on-going inflammatory process, where the infection gets localized. It begins as a focal accumulation of neutrophils in an area of liquefactive necrosis around bacteria. It comprises of a localized collection of pus within a pyogenic membrane. Pus consists of necrotic leukocytes and tissue cells.

The pyogenic membrane is composed of an inner layer of neutrophils and an outer layer of granulation tissue.

CLASSIFICATION OF PERITONITIS

1. **PRIMARY (1^o) PERITONITIS - Bacterial peritonitis without disruption of intra abdominal hollow viscera**

   a. Spontaneous bacterial peritonitis

   b. Peritonitis associated with Chronic Ambulatory Peritoneal Dialysis

   c. Tuberculosis / granulomatous peritonitis

2. **SECONDARY (2^o)PERITONITIS - Localized (abscess) or diffuse peritonitis with disruption of an abdominal viscus**

   a. Acute perforation peritonitis

      1. Hollow viscus perforation
      2. Intestinal ischemia/ bowel gangrene

   b. Post-operative peritonitis due to

      1. Anastomotic leak
      2. Accidental perforation and devascularisation

   c. Post-traumatic peritonitis following

      1. Blunt injury abdomen
      2. Penetrating injury abdomen

3. **TERTIARY (3^o) PERITONITIS- Peritonitis that occurs late due to disturbance in the host's immune response**

   a. Peritonitis with no evidence of pathogens

b. Peritonitis with low grade bacteria/ fungi/ opportunistic pathogens

EPIDEMIOLOGY

In India, perforation peritonitis still remains the most common surgical emergency. Its incidence is estimated to be anywhere between **8-13 %** of cases presenting as an acute abdomen. The presentation to the hospital in our population is usually very late with well established generalized peritonitis and purulent/ faecal contamination with septicaemia. Despite the recent developments in surgical techniques, intensive antibiotic regimens and supportive care, management of peritonitis continues to be difficult and complex.

The **proximal gastrointestinal tract** perforations are **six times** as common as perforations of distal gastrointestinal tract in **India**[3], as against the studies from the western world where the distal gastrointestinal tract perforations are more common.

Duodenal ulcer perforation and **appendicular perforations** are the leading causes of generalized peritonitis in India.[3] Hollow visceral perforations due to abdominal trauma, both blunt and penetrating injury are on the rise and require appropriate management to reduce the morbidity and mortality. Despite delays in presentation, the overall

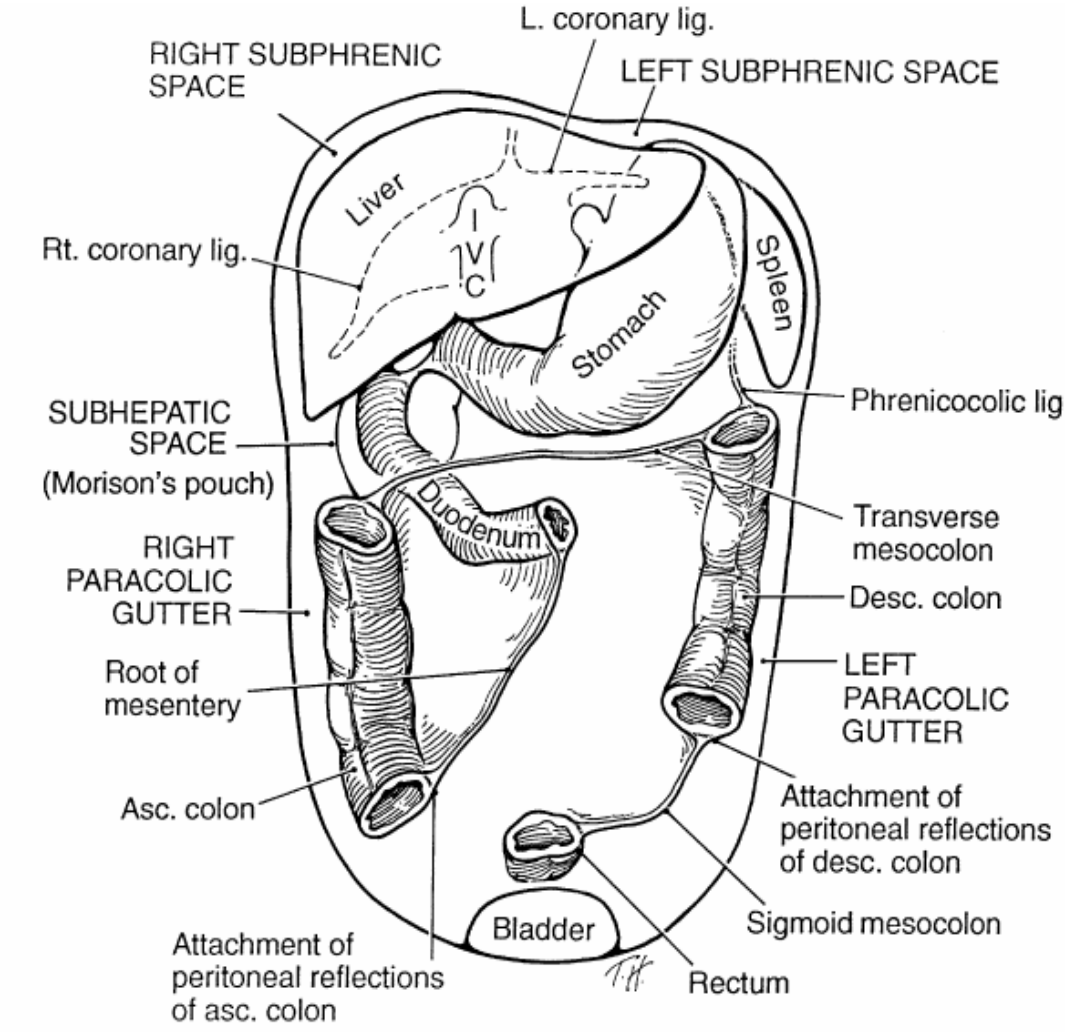
prognosis remains comparable to western figures. A comparatively younger age group involved may be responsible for the better outcome.[3]

ANATOMY OF PERITONEAL CAVITY

The peritoneum consists of a single sheet of simple squamous epithelium of mesodermal origin, termed ***mesothelium***, lying on a thin connective tissue stroma[4]. The surface area is **1.0 to 1.7 m^2**, about that of the total body surface area[5]. In males, the peritoneal cavity is sealed, whereas in females, it is open to the exterior through the ostia of the fallopian tubes. The peritoneal membrane is divided into parietal and visceral components. The parietal peritoneum covers the anterior, lateral, and posterior abdominal wall surfaces as well as the inferior surface of the diaphragm and the pelvis. The visceral peritoneum covers most of the surface of the intra peritoneal organs namely the stomach, jejunum, ileum, transverse colon, liver, and spleen and the anterior aspect of the retroperitoneal organs (i.e., the duodenum, left and right colon, pancreas, kidneys, and adrenal glands).

The peritoneal cavity is subdivided into interconnected compartments or spaces by **11 ligaments** and **mesenteries**.[2] The peritoneal ligaments or mesenteries include the coronary, gastro-hepatic, hepato-duodenal, falciform, gastro-colic, duodeno-colic, gastrosplenic,

splenorenal, and phrenicocolic ligaments and the transverse mesocolon and small bowel mesentery. These structures partition the abdomen into **nine potential spaces**: right and left Subphrenic, subhepatic, supramesenteric and inframesenteric, right and left paracolic gutters, pelvis, and lesser space.



Subphrenic spaces on either side extend between the corresponding lobes of the liver and the diaphragm. The **subhepatic** space is bounded by the liver above, duodenum and the hepato-duodenal

ligament medially, hepatic flexure and mesocolon below and the parietal wall laterally. This space is in communication with the most dependent space of the peritoneal cavity in recumbent position, the **Morrison pouch**.

The **paracolic gutters** are bounded between the ascending or the descending colon medially in the right and left sides respectively, and the parietal wall laterally. The right space communicates freely with the subhepatic space and pelvis, whereas the left side space is limited by the sigmoid colon and the phrenico-colic ligament.

The **lesser sac** is the space that lies posterior to the stomach bounded superiorly by the caudate process of liver and the mesocolon inferiorly. The grater sac is the name given to the general peritoneal cavity with which the lesser sac communicates at the ***Foramen of Winslow***.

The **pelvic cavity** is the intra peritoneal space within the confines of the true pelvis. It is the most dependent portion of the abdominal cavity in upright position.

These ligaments, mesenteries, and peritoneal spaces direct the circulation of fluid in the peritoneal cavity and thus this knowledge of

anatomy is useful in predicting the route of spread of infectious and malignant diseases. For example, perforation of the duodenum from peptic ulcer disease may result in the movement of fluid (and the development of abscesses) in the sub hepatic space, the right paracolic gutter, and the pelvis.

The blood supply to the **visceral peritoneum** is derived from the **splanchnic** blood vessels, whereas the **parietal peritoneum** is supplied by branches of the **intercostals**, **sub costal**, **lumbar**, and **iliac vessels**.

According to the anatomic spread of infection, peritonitis can be classified as:

1. **Diffuse peritonitis**
2. **Localized peritonitis**
    - Intra abdominal abscess
    - Inter loop abscess
    - Douglas abscess (pelvic abscess)
    - Subphrenic abscess
    - Retrocolic abscess
    - Pancreatic abscess
3. Other abscesses

PHYSIOLOGY OF PERITONEAL CAVITY

The peritoneum is a **single layer of mesothelial cells** with a basement membrane supported by an underlying highly vascularised connective tissue layer. It has been estimated that a 1 mm increase in thickness of the peritoneum by fluid accumulation can result in sequestration of nearly 18 L of fluid, which accounts for the massive fluid shifts seen with diffuse peritonitis[6].

The peritoneum covers the **entire interior surface** of the abdominal wall, diaphragm, retro peritoneum and the pelvic surfaces which comprise the abdominal cavity, in addition to the intra-abdominal viscera. Nearly one half of the peritoneum functions as a passive, semi-permeable membrane to the diffusion of water, electrolytes and macromolecules. About **50 ml or less of sterile fluid** is present within the peritoneal cavity under normal conditions. The fluid is secreted from the visceral peritoneal surfaces and circulates through the peritoneal cavity (*Autio et al*). This fluid normally has a low specific gravity, protein content and < 3000 cells per cubic mm, resembling lymph fluid[7].

Contrast material introduced into the peritoneal cavity in the paracecal area primarily transmigrates towards the right sub phrenic area and the pelvis[7]. It is thought that the cephalad movement is produced by

the creation of **negative pressure** in the **sub phrenic space** by diaphragmatic motion. Most of the peritoneal fluid is absorbed through the lymphatics of the parietal peritoneal surfaces, while a minor amount is absorbed through the diaphragmatic lymphatics.[8] The clearance of particulate matter, cells and micro-organisms contained in the peritoneum may be largely dependent on diaphragmatic lymphatics.[9] ***Intercellular gaps*** or ***stomata*** are specifically situated between peritoneal mesothelial cells of the diaphragmatic surface. The diameters of these stomata vary between **4 to 12 micrometres**.[10,11] Fluid and substances not amenable to absorption are channelled via the stomata and through the fenestrations in the basement membrane, and into specialized diaphragmatic lymphatics called ***lacunae.***

During the respiratory cycle, diaphragmatic relaxation in expiration opens the stomata and promotes rapid filling of the lacunae.[9] Contraction of the diaphragm at inspiration empties the lacunae into efferent lymphatic channels. Negative intra thoracic pressure during inspiration facilitates the movement of fluid into thoracic lymphatic channels, which eventually is delivered into the central circulation via the thoracic duct. Animal observations have revealed this process of particulate clearance via the diaphragmatic lymphatics to be rapid. Following intra-peritoneal

injection, bacteria can be recovered as early as **6 minutes** from the right thoracic duct, and within **12 minutes** from the blood.[12]

A number of factors influence the diaphragmatic clearance mechanism or "**pump**".

- Blockage of the stomata can be effected by introduction of **platelets**, or **application of talc** to occlude stomata in animal models. This reduces the particulate clearance process.[13,14]

- **Body positioning** can also influence the effectiveness of the pump. ***Steinberg*** has noted that placing the animals in head up position delayed, but did not prevent the appearance of bacteria in circulation after peritoneal inoculation.[10]

- Elimination of the pump by chemical paralysis of the diaphragm results in reduced clearance from the peritoneum. Reducing the spontaneous respiration during general anaesthesia has been observed to reduce the particulate clearance.[14] In studies in pigs, the incidence of bacteraemia was observed to be substantially low in those undergoing **muscular paralysis** and **mechanical ventilation**.[15] Similarly, application of positive end expiratory

pressure which impedes the thoracic lymphatic flow has been shown to decrease the peritoneal bacterial clearance.[16]

Clearly, this diaphragm pump acts as a primary local defence mechanism by effecting clearance of bacteria from the peritoneal cavity. The benefit of this mechanism in terms of host survival is uncertain, though the incidences of bacteremia and mortality have been reduced in a number of animal and human studies. ***Dumont et al*** reported a reduction in mortality in a rat model, when platelets or scarification were used to block the stomata.[14] ***Skau et al*** reported reduced bacteraemia in pigs using only muscular paralysis and mechanical ventilation.[15] Clinical observations in humans have revealed that the mortality from peritonitis is reduced in patients placed in semi-upright position.[17] ***Dunn et al*** have noted that one half of intra peritoneal inoculum are cleared by diaphragmatic lymphatics and another one-third undergo phagocytosis by resident macrophages.[18] The above two are the first line of clearance after bacterial contamination.[19]

LOCAL RESPONSE TO PERITONEAL INFECTION

The primary objective of local response to infection is the containment or removal of micro-organisms from the peritoneal cavity. The inflammatory response is similar to inflammation that occurs

elsewhere in the body. It is characterized by hyperaemia, influx of fluid, phagocytic cell recruitment and fibrin deposition.

Any noxious stimuli that causes vascular endothelial or mesothelial cell injury is capable of initiating peritonitis. Though gram negative bacterial endo toxin is considered the classical stimulating agent of peritonitis based upon experimental models, a number of other agents are capable of inducing similar responses. ***Wiles et al*** has noted similar physiological effects with organisms such as gram positive bacteria, *Bacteroides* species, and yeasts that have no endotoxins, or biologically inactive endotoxins.[20] This implies that exoenzymes or capsular polysaccharides also act as stimulators of inflammation. In addition, the similarity in systemic response to both gram negative and fungal peritonitis, namely fever, hypotension, leukocytosis, and shock, suggests that the systemic action is not direct, but mediated by mediators such as **interleukin-1 (IL 1) and Tumour Necrosis factor (TNF).**[21,22] This suggests that the overall inflammatory response, both local and systemic is a general one and not specific for any single infectious agent. Non-infectious irritants such as gastric juice, pancreatic juice, bile salts, meconium and urine are well recognized causes of sterile peritonitis and

probably initiate the inflammatory cascade by inciting mesothelial cell damage or by direct activation of complement system.

Alteration in blood flow and vascular permeability

An alteration in local blood flow and influx of fluid to the site of inflammation are the two earliest observed physiological changes. **Histamine**, released from tissue mast cells and basophils, is an early chemical mediator of these changes. Similar effects are exerted by bradykinin, a product of the contact activation system.

The release of histamine is triggered by **mesothelial cell injury**, antigen antibody complexes, **complement cascade** products (C3a and C5a) and platelet activating factors. Both histamine and bradykinin causes pain, vasodilatation and increased permeability of the peritoneal vessels.[23] Other vasoactive substances such as prostaglandin E2α (PGE2α) and Leukotriene C4 are produced by bradykinin and they contribute to the observed vascular effects too.

Normally, the peritoneum allows bi-directional flow of fluid, but with inflammation, there occurs an uni-directional flow of fluid from the extracellular space into the peritoneal cavity. Depending upon the extent of inflammation, fluid volumes as high as 10 L or more may be accumulated in the peritoneal cavity.[24] The initial fluid that accumulates is a transudate of low protein content. With increasing permeability, there

occurs exudation of copious amounts of fluid rich in immunoglobulins, complement factors, coagulation factors and cytokines. This process clearly succeeds in delivering the essential humoral mediators of inflammation to the site of infection.

Thus, massive third-space fluid loss and loss of plasma proteins can result in hypovolemic shock. In addition, continued accumulation of fluid may eventually impair bacterial phagocytosis by diluting opsonins and impeding migration of neutrophils.

The perturbations of normal biological functions that accompany all cases of peritonitis are well established. There occurs drastic reduction in the concentrations of opsonic molecules IgG and C3. The normal phagocytic functions are thus greatly hampered inside the peritoneal cavity.

Peritoneal Macrophages are the first line of cellular defence against bacterial invasion. Early clinical experience demonstrated that several hours had to elapse before cell counts became elevated, PMNs appeared in the abdominal cavity, and clinical infection was established. Moreover, not every patient who contaminated the abdominal cavity developed clinical peritonitis. Studies in animal models of peritonitis also suggest that the resident peritoneal macrophages are responsible for the

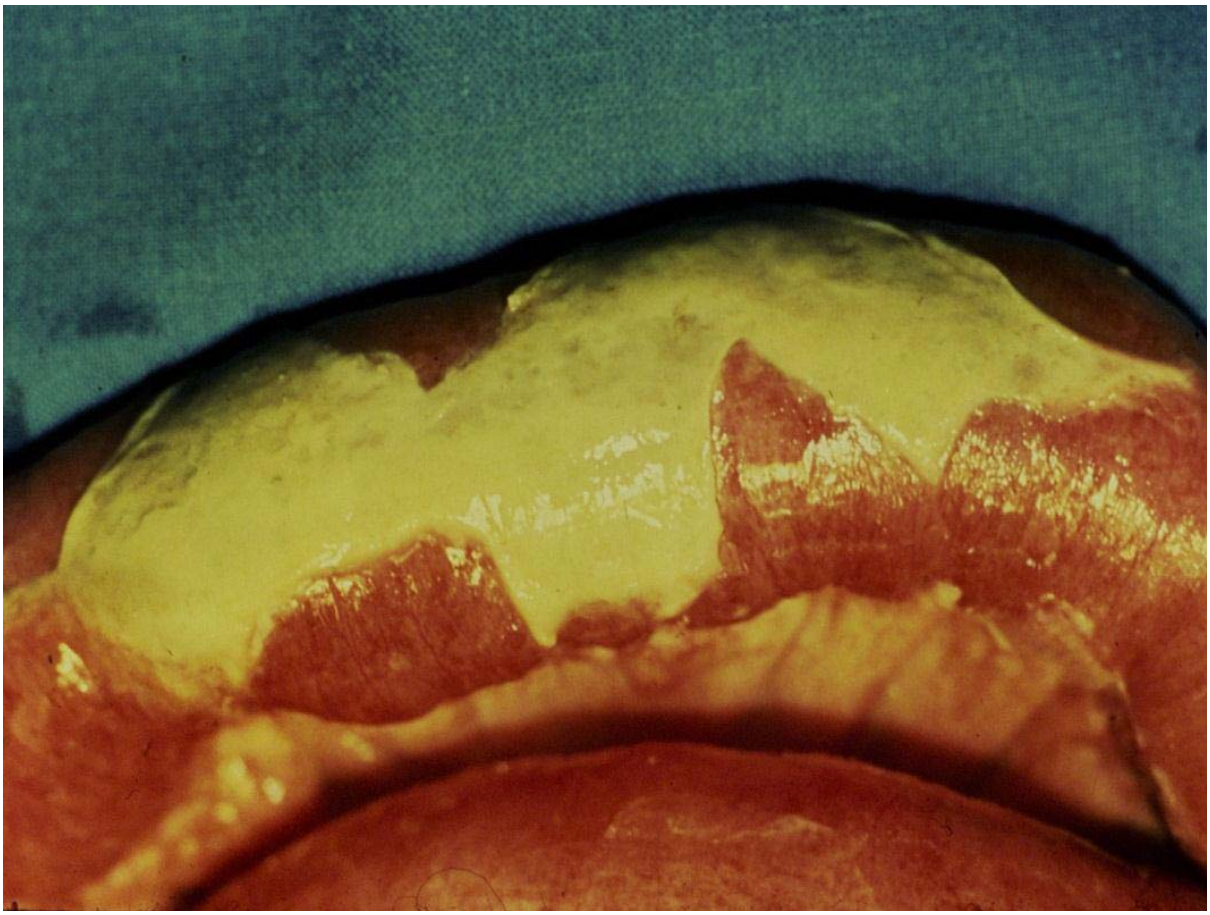
initial removal of bacteria before the appearance of PMNs. These observations and experimental studies of animal models of peritonitis support the concept that peritoneal macrophages function as the first line of cellular defence against bacterial invasion.

Phagocytic and Bactericidal Capacity of Peritoneal Macrophages

The efficient function of phagocytic cells generally requires opsonic molecules, which facilitate the ingestion of micro-organisms. Phagocytosis of most E. *coli* strains requires a heat-labile opsonin, which appears to be the C3 component of the complement cascade. Indirectly, there is evidence to suggest that phagocytosis of S. *epidermidis* proceeds predominantly through an Fc receptor mechanism while that of E. *coli* occurs predominantly via C3b receptors on the surface of peritoneal macrophages.

Relationship between opsonic activity and peritonitis

Deficiencies in opsonic molecules seriously compromise the bactericidal function of phagocytic cells.[25] Whether clinical infection occurs or not is decided by the relative concentrations of these factors at the site of bacterial invasion.[26,27] Studies have demonstrated two to five fold variability in heat-stable opsonic activity in peritoneal effluent.



FIBRIN FORMATION

Hence, it seems reasonable that a deficiency in these molecules in the peritoneal cavity would contribute to the development of peritonitis.

Therapeutic considerations

Although many factors operate in the pathogenesis of peritonitis of patients, the alterations in the host defences of the peritoneal cavity may play an important role. In particular, marked reductions in IgG with opsonic activity against E.coli appear to be critical. It is possible, therefore, that passive immunization of the peritoneal cavity would reduce the incidence of peritonitis. Once peritonitis is established, as indicated by increased numbers of PMNs, standard antimicrobial agents will still be required.

Fibrin deposition

Intact mesothelial cells maintain fibrinolytic activity within the peritoneal cavity by the secretion of tissue plasminogen activator (t-PA) under normal circumstances.[28] Local fibrinolytic activity is suppressed in the setting of mesothelial cell injury due to loss of plasminogen activator.[29] When high concentrations of fibrinogen are present, fibrin deposition occurs readily by activation of intrinsic pathway. This is enhanced further by the release of thromboplastin (factor 3) from injured

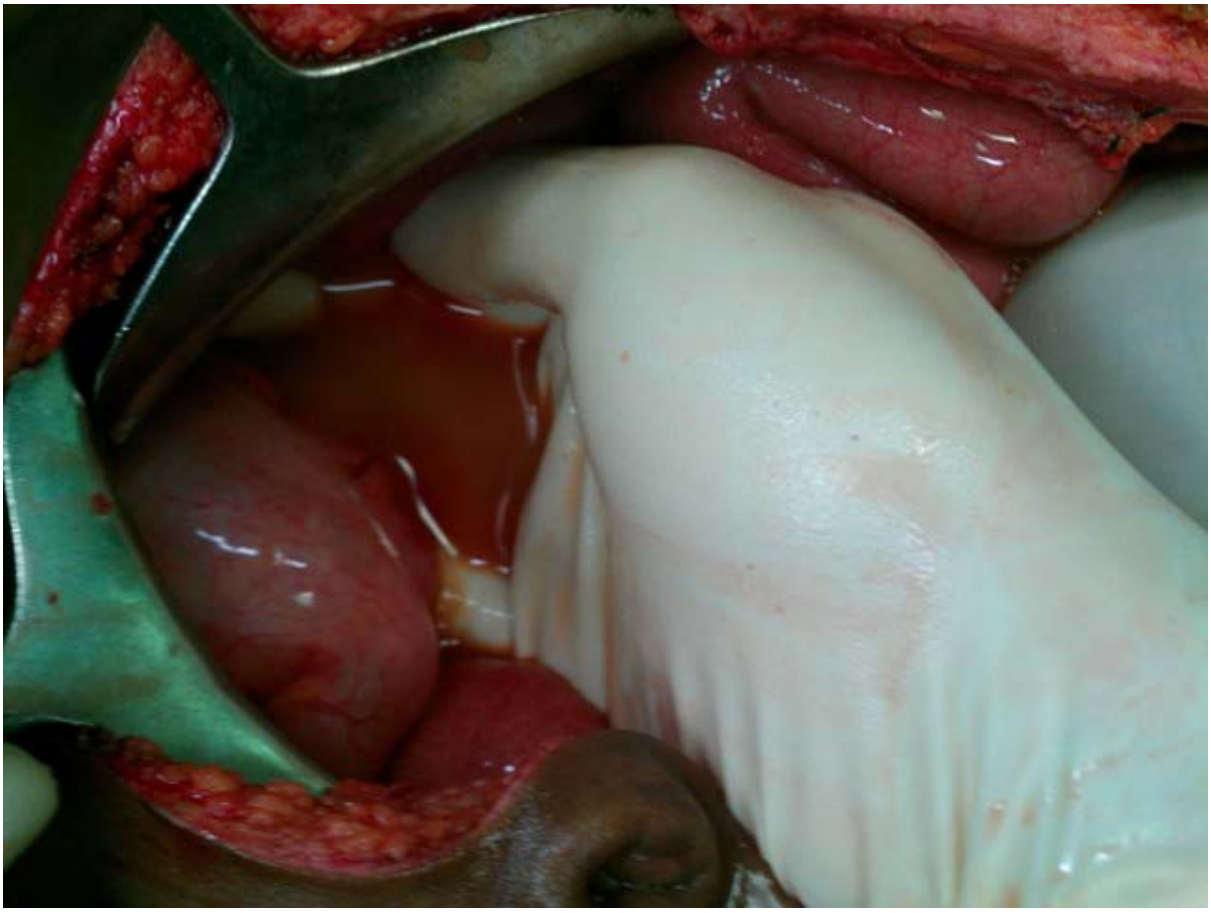


FIGURE 1: INTRA PERITONEAL ABSCESS

mesothelial cells, and surface procoagulant activity from stimulated peritoneal macrophages.[30]

Fibrin deposition plays a definite role in local inflammatory response. The most credible objective of this process is to isolate or contain contamination and thereby preventing widespread dissemination. This occurs by entrapment of bacteria in the fibrin matrices, and by causing adherence of loops of intestine and omentum to one another as well as to the parietal peritoneal surface, thus creating physical barriers against bacterial spread. However, fibrin entrapment is a double edged sword, as bacterial encasement also inhibits phagocytosis by isolating the microbes from the neutrophils.[31]

ABSCESS FORMATION

The development of abscess is the culmination of the sequestration process. The rate of fibrin deposition exceeds the fibrin degradation produced by bacterial enzymes. Within the adhered mass of viscera and omentum, fibrin and bacterial liquefaction develops from the release of proteolytic enzymes of leukocytes and bacterial exoenzymes. The osmolarity of the developing abscess cavity is high, which promotes an influx of water, and thereby increasing the hydrostatic pressure within the cavity.[6] The abscess capsule, composed of organizing fibrin and adherent

viscera, retards the diffusion of oxygen and nutrients into the abscess, thereby promoting anaerobic glycolysis. The hypoxic, hypercarbic, and acidic environment impairs both neutrophils and phagocyte function. The high concentrations of bacterial cell wall components and enzymes impair the phagocytic function and increase the local tissue damage.

The presence of necrotic debris depletes complement components, and contributes to neutrophil deactivation. Hypoxia and acidosis impair neutrophil migration as well as killing. Increased osmolarity inhibits neutrophil release of lysosomal granules and hypercarbia lowers the cytoplasmic pH, leading to neutrophil dysfunction.

FACTORS INFLUENCING PERITONEAL INFLAMMATION AND INFECTION

Bacterial virulence

Numerous organisms are well known for their innate ability to produce intra-abdominal infections in humans. Common faecal pathogens include aerobic coliform bacteria, anaerobic *Bacteroides* species, aerobic and anaerobic *Streptococci*, *Enterococci* and *Clostridia* species. In contrast, other organisms like *Propionibacteria* rarely produce disease. Despite the massive contamination that occurs with faecal peritonitis, **within 24 to 48 hours**, only a few isolates are identifiable in peritoneal

fluid culture. This indicates that only a few pathogenic species survive to predominate in the infection.

The predominance of a particular microorganism within the local inflammatory process has been found to be varied. *Weinstein et al,* in an animal model of peritonitis, demonstrated that *E. coli* and Enterococci were the predominant organisms during the peritonitis stage; while *B. fragilis* predominated during the abscess stage.[33] A unique pathogenicity observed with encapsulated anaerobic bacteria is abscess formation, a characteristic attributed to capsular polysaccharide components. The size of bacterial inoculums also influences virulence, as both, the ability to produce infection and the disease severity increases with increasing bacterial dose. Experimental studies indicate that some organisms are resistant to mechanical removal through peritoneal lavage, which increases the difficulty in clearing these microbes from peritoneum.

Bacterial synergism potentiates the virulence of a single organism. Polymicrobial infections, specifically combinations of aerobic and anaerobic species, exhibit greater lethality than single species of pathogenic bacteria. Aerobic bacteria may benefit anaerobic bacteria by lowering the redox potential of the microenvironment, while anaerobic

bacteria may provide the ability to inhibit neutrophil function and to develop antibiotic resistance.

Adjuvant factors

A number of substances found in conjunction with peritoneal infection may be detrimental to host defences and jeopardize the success of eradicating infection. Adjuvants enhance the virulence of bacteria by interference with host defence mechanisms, whether mechanical or cellular. Blood components, haemoglobin, and ferrous iron are the most studied, till date. The **lethality of E. coli** is enhanced by **haemoglobin**.[34] It may be due to either induction of leukocyte derived toxin injurious to neutrophils, or to the presence of iron, which enhances bacterial growth. **Fibrin** entrapment of bacteria may be helpful or detrimental to the overall host immune response. The presence of fibrin may inhibit neutrophilic killing of bacteria as well as cause premature degranulation of neutrophils as the PMNs attempt to cause phagocytosis of fibrin particles.[35,36] The presence of platelets within the peritoneal cavity may impair physical bacterial clearance by blocking the diaphragmatic lymphatics. The presence of necrotic tissue causes depletion of complement and neutrophil inactivation.

A number of substances originating from the gastrointestinal tract have also been shown to cause lethal peritonitis. Gastric juice, pancreatic

juice, urine and meconium induce sterile chemical peritonitis. Bile salts facilitate bacterial spreading and are toxic to neutrophils and peritoneal mesothelial cells. Even non-irritant fluid may impair bacterial elimination. Copious amounts of intra peritoneal fluid may diminish phagocytosis by diluting opsonins, or immersing bacteria in solution. This eliminates the surface phagocytic cells required for engulfing and ingesting bacteria.[37]

Foreign materials like barium sulphate, talc, suture material, and cellulose (gel foam) also impairs phagocytosis by causing premature activation of neutrophils and release of lysosomal enzymes.[38] From a clinical stand point, a rational approach in the surgical management of peritoneal infection should include attention to meticulous haemostasis, copious lavage to remove adjuvant material, thorough evacuation of intra peritoneal fluid, and minimizing the use of foreign materials like haemostatic agents and suture materials.

SYSTEMIC RESPONSE TO PERITONEAL INJURY

The systemic response to peritoneal infection emulates the response to any other injury, such as trauma or surgery. The development of hypovolemia probably results from the fluid influx occurring in the peritoneal cavity. The subsequent reduction in intravascular volume leads

to decreased venous return and cardiac output. Incomplete compensation is obtained with increases the heart rate. Systemic hypotension may also be due to secretion of TNF, IL-1, NO, and platelet activating factors, which all have vasodilatory effects and reduce systemic resistance.[39] In particular, a significant degree of precapillary shunting may occur in pulmonary and splanchnic circulation, which leads to a decrease in oxygen delivery and subsequent consumption. Diminished urine output results as a result of increased amounts of aldosterone and anti diuretic hormones, reduced cardiac output and intra renal shunting of blood. This is the setting of ***hyper dynamic*** *or* ***warm septic shock***, characterized by tachycardia, fever, oliguria, hypotension, and warm extremities. Hemodynamic and oxygen transport measurements will reveal an elevated cardiac output, low peripheral vascular resistance, low arterio-venous O_2 difference, and higher mixed venous oxygen content.

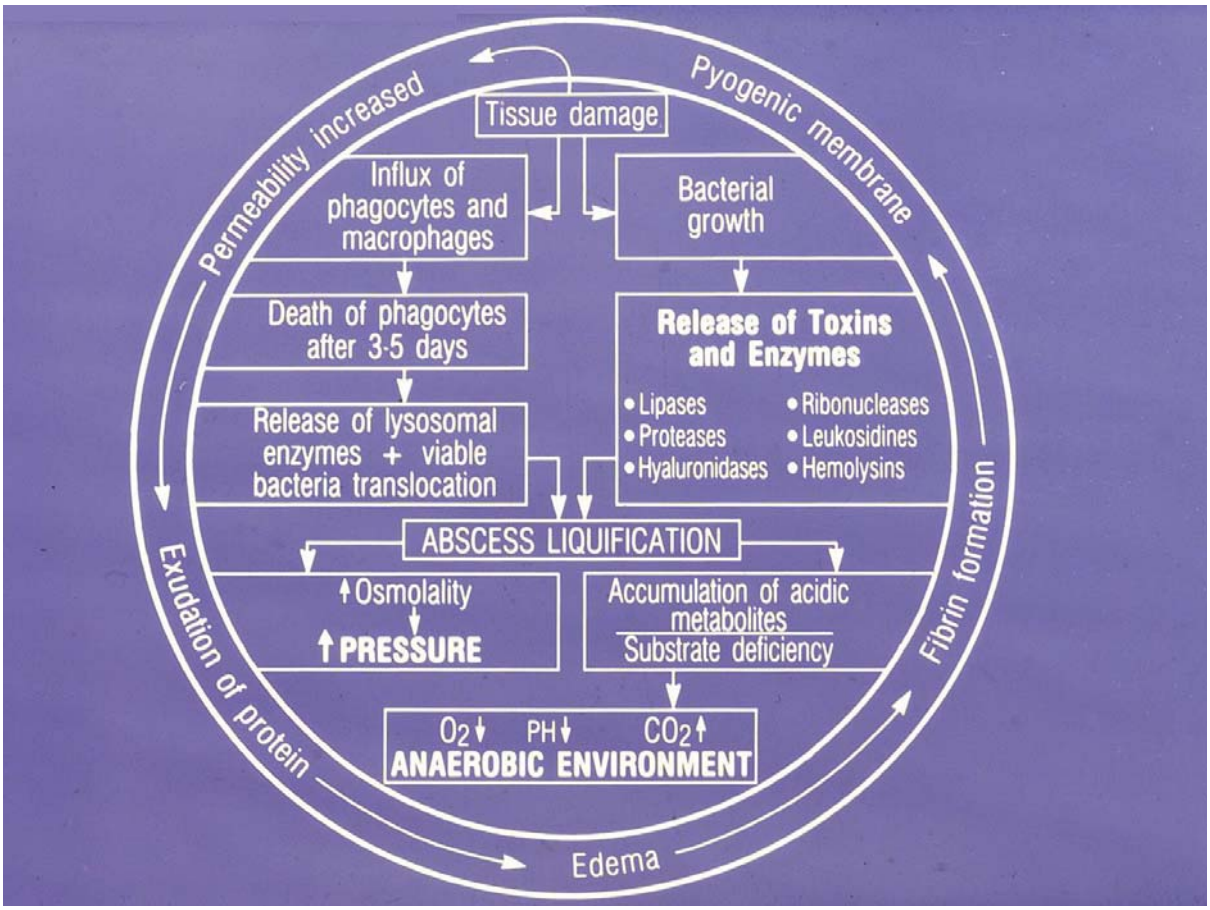
Abdominal distension secondary to accumulated fluid within the peritoneal cavity and bowel creates restriction of diaphragmatic mobility and decreases ventilatory volume, creating eventual atelectasis. Ventilation-perfusion mismatch develops from both atelectasis and intra-pulmonary shunting due to beta-adrenergic stimulation. Increases in pulmonary vascular permeability also develop as a result of inflammatory

mediators. The accumulation of fluid in the pulmonary interstitium and alveoli decreases the pulmonary compliance and increases the work of breathing. These manifest as hyperventilation and respiratory alkalosis. With worsening pulmonary oedema and atelectasis, severe hypoxemia will develop, creating **Adult Respiratory Distress Syndrome (ARDS**).

Tissue metabolism is altered during the response to peritonitis. The metabolic rate is increased due to increased levels of catecholamines and cortisol. Tissue hypoxia develops as a result of reduced oxygen delivery, owing to both decreased perfusion and shunting. Increasing anaerobic glycolysis produces increasing amounts of lactic acid and acid by-products. Renal and pulmonary clearance of this increasing acid load leads to metabolic acidosis.

Significant change in substrate metabolism also occurs in peritonitis. There occurs early depletion of hepatic glycogen stores, and protein catabolism is increased in the skeletal muscle to release branched chain amino acids for use by myocytes as an energy source. Other amino acids are released into the circulation to be utilized for gluconeogenesis as well as the production of acute phase proteins. Though the body lipolysis is also increased causing increased levels of free fatty acids in blood, they are not used as an energy source in the early septic phase. The

severe loss in lean body mass that occurs from net protein catabolism is rapid and only partly corrected with nutritional support.



PATHOGENESIS

PATHOGENESIS

Biphasic Infection

Bacteriology of secondary peritonitis encompasses two key processes, namely **bacterial simplification** and **synergism.** These have been confirmed with experimental rodent studies where the initial inoculums of contaminating bacteria is spontaneously reduced to only a few microorganisms that are capable to survive and thrive in the new *milieu*: the **acute peritonitis phase** with positive blood cultures are produced mainly by the facultative anaerobes, especially ***Escherichia coli***, and the **late abscess formation** stage is predominated by the obligate anaerobe, ***Bacteroides fragilis***. These bacteria act in series and not in parallel; both are necessary to produce an abscess, and the obligate anaerobic organism has the potential to enormously increase the lethality of an otherwise nonlethal inoculums of the facultative organism.

ROLE OF CYTOKINES

Many reviews suggest that many of the abdominal and systemic responses in peritonitis are cytokine mediated amidst others.[40] Tumour Necrosis Factor (TNF), Interleukin-1 (IL-1), Interleukin-6 (IL-6), Interferon-gamma (IFN-g), and others have been shown to be much more involved than others. Studies have established

their levels to increase in patients with peritonitis, both in the blood and to a much greater extent in the exudates.[41] The source of these cytokines is varied: macrophages and other host cells can produce them in response to bacterial endotoxins.[42] Other potential route is direct translocation of cytokines across the intestinal barrier. On the contrary, the levels could simply rise in the blood due to tissues traumatized during operative procedure.[43]

The cytokine responses have also given a new pharmacological dimension, wherein these responses are blocked using appropriate antibodies and their effects in reducing systemic response and peritoneal inflammation have been studied in the animal models of peritonitis, and in patients with severe secondary bacterial peritonitis undergoing planned re-laparotomy.[41] In animal studies of peritonitis, only antibodies to IFN-g afforded a protective effect on a consistent basis following experimental intravenous endotoxin injection. Anti-TNF antibodies did not protect against death and the serum levels of IL-1 and IL-6 continued to remain high in all experimental models. In contrast, anti-endotoxin antibodies are found to prevent death in the same model, and additionally to reduce the bacterial colony counts in peritoneal exudates.[43]

Tumour Necrosis factor (TNF) levels have shown a direct linear correlation with the occurrence of peritoneal adhesions. Further, neutralization of

TNF with antibodies prevented adhesion formation in "*Caecal ligation and puncture*", an experimental model of septic peritonitis. The results also showed an increased mortality as neutralization resulted in prevention of localization of the septic focus. Similar to experimental studies, adhesion formation following a surgery had a linear positive correlation with higher levels of TNF in patients.[42] Higher TNF concentrations are found to cause disseminated intravascular coagulation (DIC), as TNF is found to have anti-fibrinolytic and procoagulant effects. Hence, prevention of coagulation was found to protect against sepsis.[44]

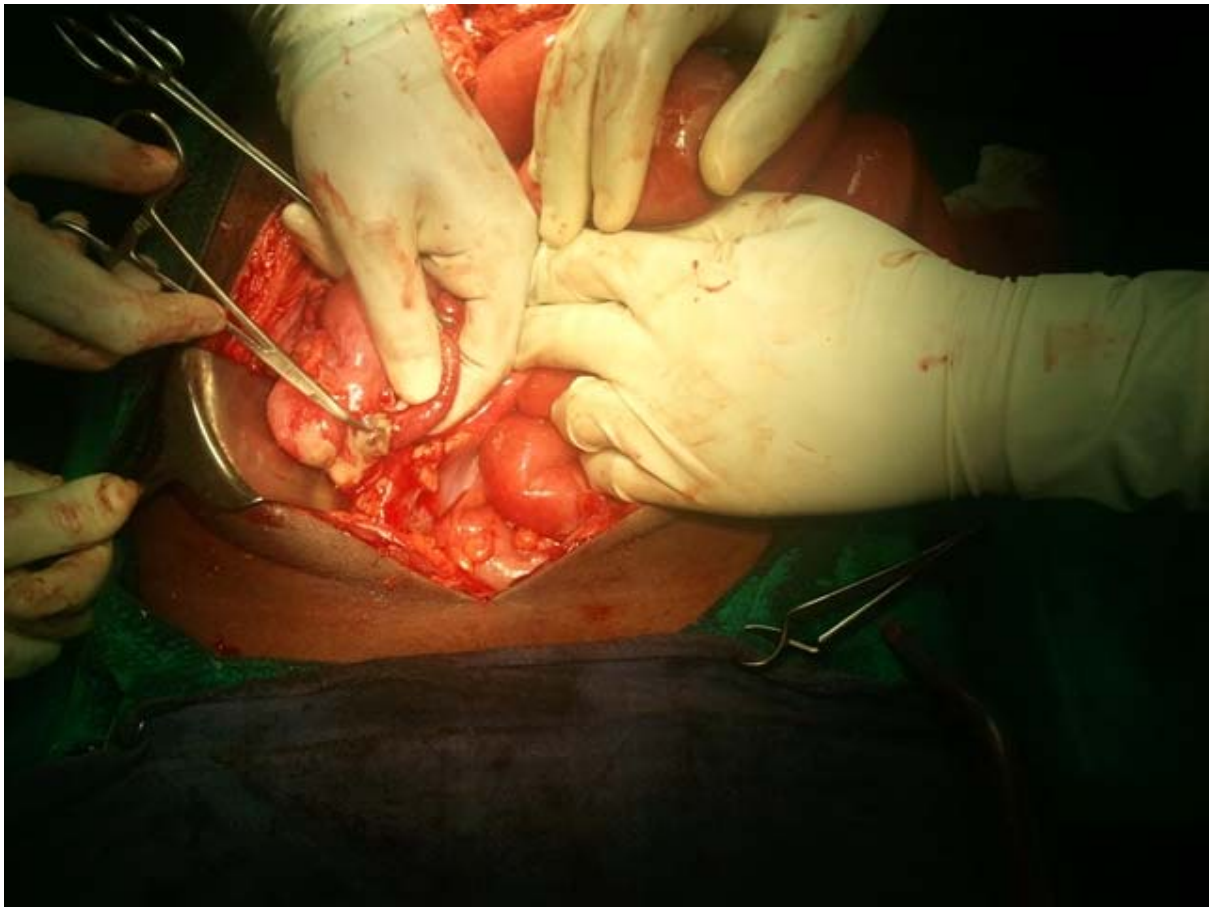
A study compared the effects of Anti-thrombin III or hirudin with antibiotics versus administration of heparin with antibiotics in prevention of DIC in rat models. The results showed anti-thrombin or hirudin group to suffer less DICs, improved survival rates and protected rats against intravenous administration of LPS or bacteria. On the other hand, heparin had no effect on survival. This might be because heparin acts indirectly by accelerating anti-thrombin III. The situation in bacterial peritonitis is different and much more complex. The potential risk of anticoagulant treatment is improper localization of the septic focus which enhances the spread of bacteria and exacerbation of the disease process. Whether abscess formation is favourable or detrimental are still debatable and conflicting opinions exist. Abscess formation secondary to containment of infection is beneficial to the

host, at least in initial stages. Besides TNF, **interleukin-12 (IL-12)** is another cytokine important for formation of protective abscesses.

MICROBIOLOGY

The type and the number of microorganisms isolated from the peritoneal cavity depend mainly on the level of perforation. In a fasting state, lactobacilli and Candida species which are relatively more acid resistant predominate in the stomach. Similar to the stomach, only a sparse micro flora exists in the fasting state in the duodenum and the small bowel. The colon contains the highest microbial density, majority of which are obligate anaerobes of the *B. fragilis* group. They are found at counts of **10^{12}/gm of faeces** in the colon. Other relatively less numerous organisms are the facultative anaerobes, namely *E. coli* which is found at counts of **$10^{5–8}$/gm**, and even less numerous are the Enterococci found at counts of **$10^{4–6}$/gm**.

The characteristic microflora, however, are not present at all times and any previous or present disease process or anti-microbial therapy can alter them. For example, in conditions of gastric outlet obstruction, the presence of a gastric ulcer or carcinoma or with use of acid reducing drugs may alter the naturally present flora. There are less number of lactobacilli and oral anaerobes, such as non-*fragilis Bacteroides* and *Fusobacterium* species predominate among the other



APPENDICULAR PERFORATION

oropharyngeal organisms such as microaerophilic streptococci, viridans streptococci and other *Candida* species.

Gastric perforation is associated with only sterile chemical peritonitis in the early stages when no chronic disease process exists in the stomach. Any underlying gastric disease can alter the flora and peritonitis is usually due to one of the above mentioned pathogens. The gastric disease or the small bowel ileus consequent to perforation can alter the sparse normal flora of the small intestine.

With colonic perforations, because of the high bacterial load of the contents, there occurs an initial spillage of hundreds of different species of the normal colonic microflora. A simplification process then occurs, so that only about **five pathogens** on an average remain with established peritoneal infection. Studies have shown even more specific pattern among the flora of peritoneal infection, wherein **three anaerobic** and **two aerobic species** exists. These obligate anaerobes have identifiable virulence factors and more oxygen tolerant than others. An exception to this is seen among patients with **gangrene** or **perforated appendicitis**, where higher numbers of organisms are seen on an average. The average numbers of organisms are found to be **9.8** with the former and **12.7** with the latter, and as expected for a colonic perforation, nearly 75 % of the flora are anaerobes. The most frequent anaerobe following colonic or appendicular

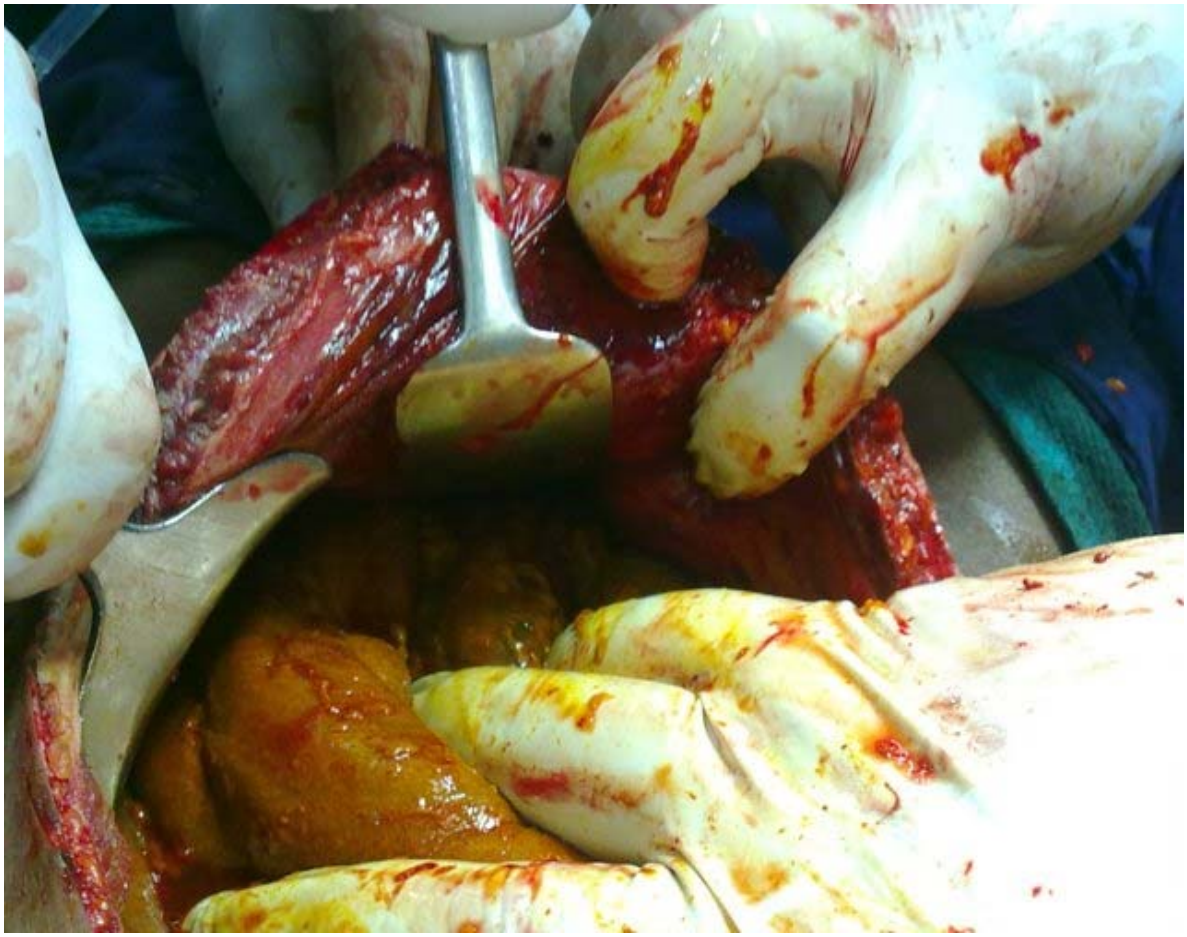
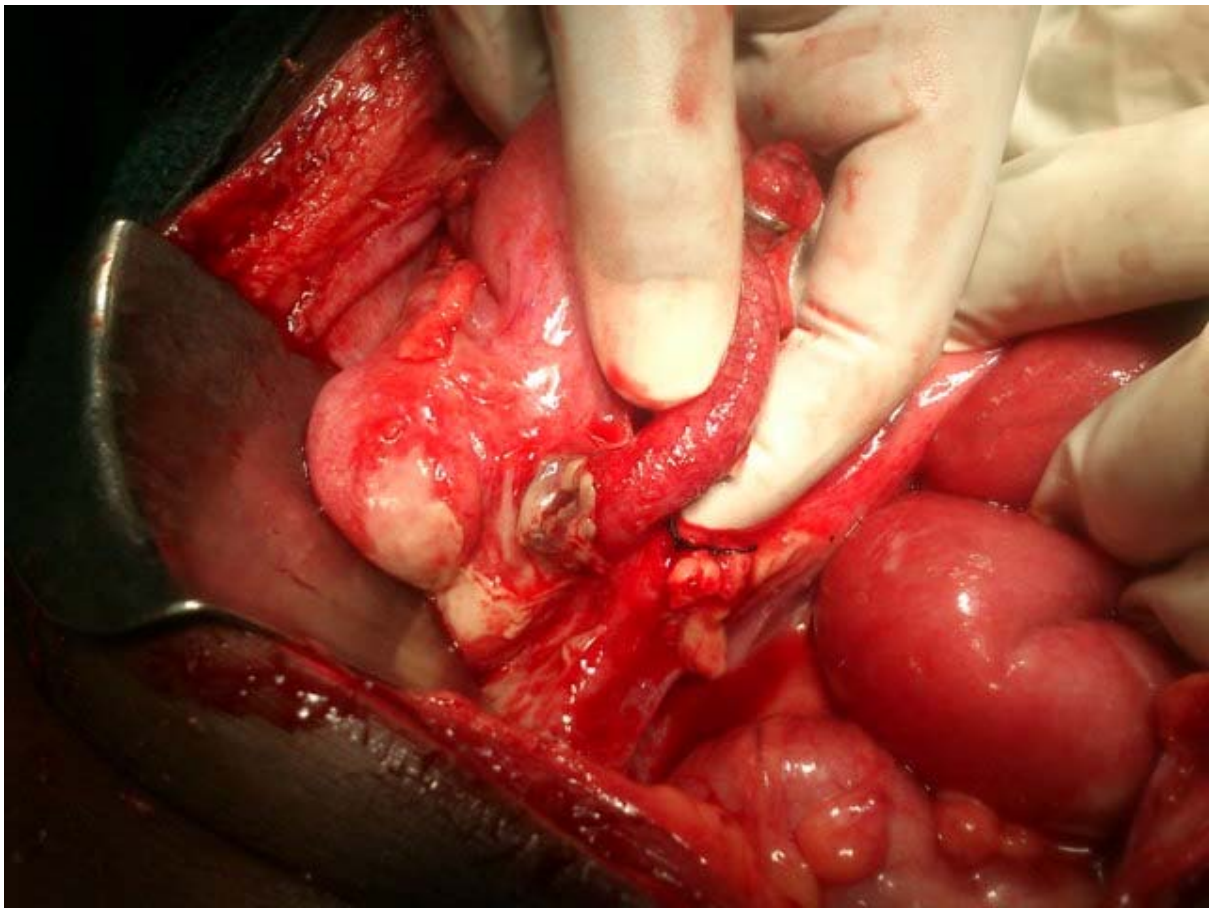


FIGURE 3: FECULENT PERITONITIS

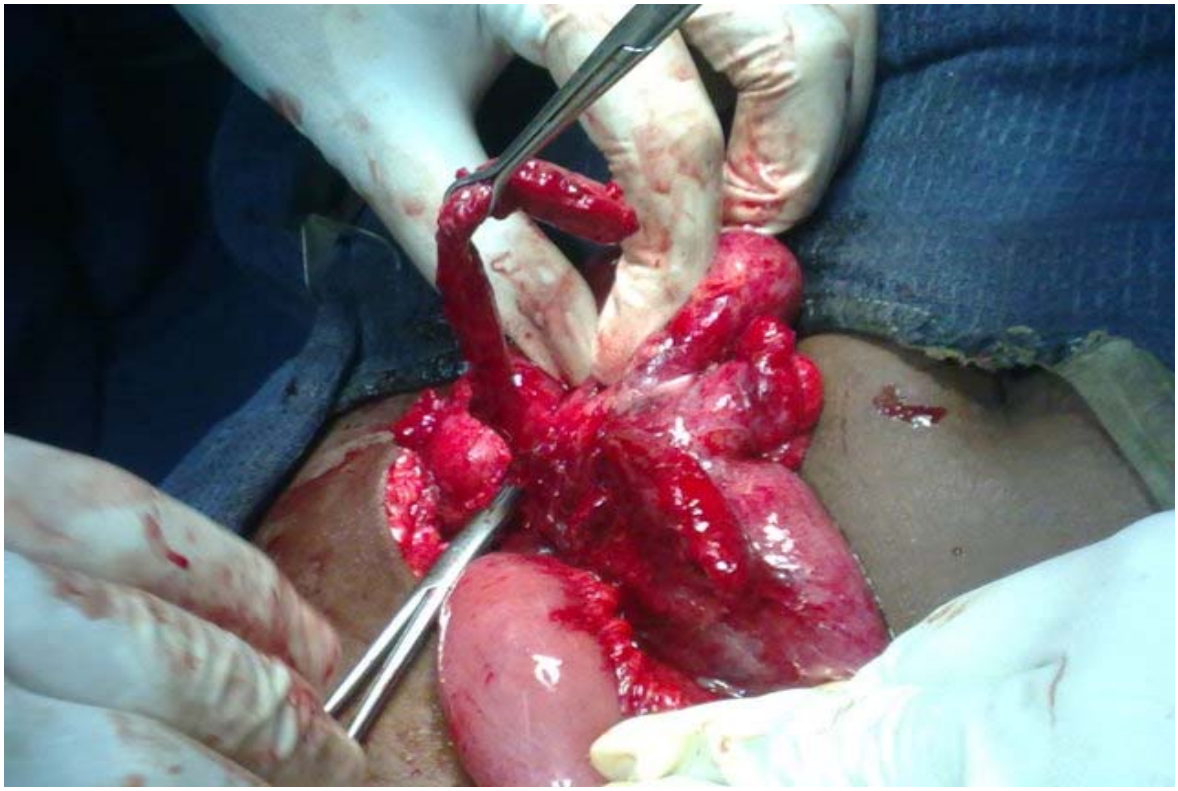


APPENDICULAR PERFORATION - BASE

perforation is Bacteroides fragilis and among the facultative anaerobes, E. coli predominates.[33]

Synergism between the different groups of micro organisms exists and is well recognized with mixed infections.[45] ***Weinstein et al*** have documented the sequence of events that occurs with faecal contamination of the peritoneum in experiments with rodents.[33] In this model, florid sepsis and its associated mortality seen with early peritonitis is attributed to *E. coli* and the late peritoneal abscess formation is due to B. fragilis species acting alongside with other organisms such as *Enterococci* and *E. coli*.

B. fragilis and *E. coli* are the primary perpetuators in all stages of peritoneal infection and other organisms isolated from this poly-microbial infection are hugely dependant on these two for their survival. The poly-microbial infection also did not require to be treated for all the organisms cultured. This is proven with studies where specific antimicrobial therapy directed against the two organisms resulted in simultaneous disappearance of these other microorganisms.[33] Over-zealous treatment resulted in appearance of the potentially dangerous multi-drug resistant organisms, not usually seen with community acquired infection. In the severely ill, hospitalized patient under the pressure of antibiotic usage, the colonic flora comprises of *Pseudomonas aeruginosa, Enterobacter* species, multi-drug



APPENDICULAR ABSCESS WITH CAECAL PERFORATION

resistant Enterococci, and *Candida* species. When a perforation occurs in this sitting, these organisms cause fulminant infection and subsequent sepsis.

These latter organisms have been isolated in the setting of tertiary peritonitis, in patients with impaired host defences and multi-organ dysfunction. In patients with severely impaired defences, even organisms of low pathogenicity such as Enterococci, coagulase negative staphylococci and even Candida species can cause mono-microbial infection and peritonitis. P. aeruginosa is usually a nosocomial pathogen arising under pressure of irrational antibiotic usage. Despite this, recent study revealed this organism to be present in 24 % community acquired perforated appendicitis.[45]

Although Enterococci are found in 20% of intra abdominal infections, their exact role in poly-microbial infection is unknown and the need for specific antimicrobial therapy against them remains controversial. Reduction of enterococcal counts is seen with anti microbial treatment directed against *E. coli* and *B. fragilis*. In another animal model of experimental polymicrobial intra abdominal infection, Enterococci have been found to promote weight loss, bacteraemia with *B. fragilis* and *E. coli*, subsequent abscess formation, and mortality.

Similarly, clinical reports have confirmed the occurrence of enterococcal abscesses and bacteraemia after treatment of intra abdominal sepsis with antimicrobial agents that lack significant in vitro enterococcal activity. In a recent multicenter study of intra abdominal infection, treatment failure with broad spectrum anti microbial regimens was found to be due to presence of Enterococci in the initial culture. In this study, APACHE II score also predicted treatment failure. The factors affecting the presence of Enterococci are age, length of pre-infection hospital stay, APACHE II score and the presence of Enterococci in post operative wound infections. It still remains to be seen if initial inclusion of anti-enterococcal therapy will improve outcome for these high-risk patients.

CLINICAL FEATURES AND DIAGNOSIS

The respected aphorism that "the diagnosis of peritonitis is made by clinical evaluation" remains true even today.

Abdominal pain is the predominant presenting symptom. The characteristics of the pain vary tremendously depending on the cause. In lesions of stomach, duodenum and jejunum (T5 to T8) the pain is felt in the epigastrium, in affections of ileum and appendix (T9 – T10) around the umbilicus, whereas in case of colon (T11-T12, L1, L2) in the hypogastrium.

Perforation of an anteriorly placed duodenal ulcer is initially sudden and sharp over the epigastrium. It can also present as a gradual low intensity right lower quadrant pain, the so called ***"right paracolic gutter phenomenon"*** due to tracking of leaking contents along the right paracolic gutter. The pain of fully established peritonitis is constant, burning and aggravated by motion or movement. The extent of pain is localized or diffuse, depending on the area of parietal peritoneum that is inflamed. The abdominal pain typically starts at the site of local peritoneal inflammation and becomes more diffuse later on with spreading infection. However, if the initial inflammation is effectively isolated from the

parietal peritoneum (e.g. covered by loops of intestine or omentum), the discomfort is only minimal and vague.

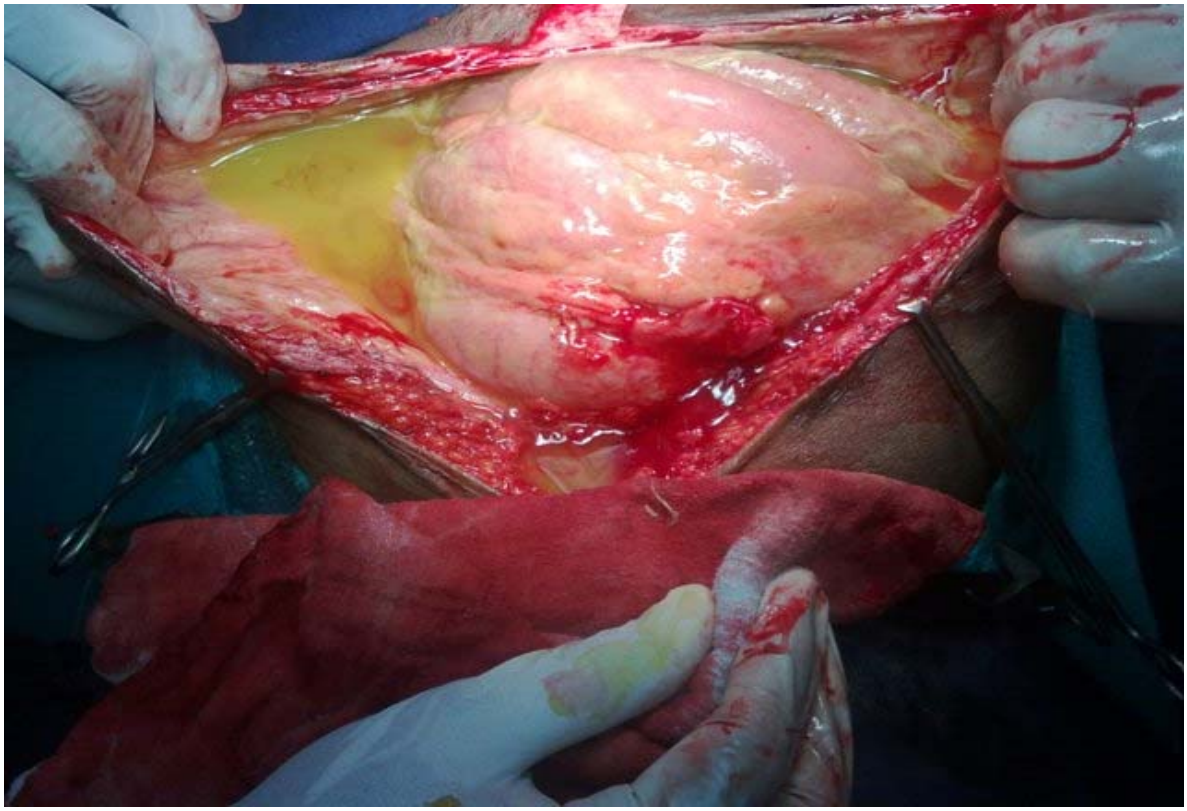


FIGURE 2: PYOPERITONEUM

Anorexia is almost always an accompanying symptom. Nausea and vomiting, as well as thirst and oliguria are mostly present.

Typical signs include **fever and diaphoresis**. Tachycardia resulting due to hypovolemia is prominent. Severe shock manifests as hypotension, hypothermia, and cool extremities. Septic shock is identified with warm and pinkish peripheries. The tenderness can be elicited by percussion followed by direct palpation, a process that yields better localization with minimal discomfort to the patient. ***Bapat test*** or the bed shaking test refers to pain at the site of inflammation when the bed is shaken. ***Blumberg sign*** refers to transient abdominal wall rebound tenderness that occurs due to peritoneal inflammation. Generalized peritonitis reveals diffuse tenderness, though it is maximal where it first originated. Bowel sounds are diminished or absent and distension owing to paralytic ileus is present. Abdominal wall rigidity due to voluntary guarding and reflex muscle spasm may be extensive.

A patient in peritonitis is said to pass through three stages

1. Stage of peritoneal irritation (up to 3 hours)

It is due to irritation of the peritoneum due to leaked intestinal contents.

2. Stage of Illusion (after 3 to 6 hours)

"Diminution of pain is not always a happy symptom". The irritant fluid is diluted with peritoneal exudates with a temporary remission of symptoms.

3. Stage of diffuse peritonitis(after 6 hours)

The classic board like rigidity is seen with chemical induced diffuse peritonitis. The other causes are accompanied by silent abdominal distension.

Routine laboratory investigations reveal leukocytosis with a shift to left i.e. immature neutrophils. Erythrocyte sedimentation rate is usually raised and elevated renal parameters may be present. Delayed presentation with profound sepsis causes raised liver enzymes and bilirubin values.

A **plain abdominal X-ray** is an important means of establishing a hollow viscus perforation in equivocal cases. It reveals loss of pro-peritoneal fat stripe and obliteration of psoas shadow indicating peritoneal edema. Pneumoperitoneum is

demonstrated on an erect chest X-ray in mid-inspiratory phase as *free air under the diaphragm*. Sufficient time must be given for air to migrate to the upper quadrant to reduce a false negative film. Lateral decubitus horizontal beam abdominal X-ray in expiratory phase is useful, especially in patients who cannot be placed in an upright posture.[46]

Plain radiography demonstrates a sensitivity of 80%, a specificity of 65%, and a positive predictive value of 95% for pneumoperitoneum. Air in right upper quadrant can manifest as ***parahepatic*** air i.e. air bubble lateral to right border of liver; a triangular collection of air in Morrison pouch known as ***Doge's cap sign***; as ***hyperlucent*** area over the liver; gas beneath the central tendon of diaphragm known as the ***cupola*** *sign.* In all other areas, extra luminal air is evident when it lines both sides of the bowel known as ***Rigler's sign***[47] or as ***Tell tale triangle sign***, which is collection of air between three loops of bowel. Besides this, air can outline any of the named ligaments: Ligamentum teres, Falciform ligament, or Urachus known by their corresponding names.

The role of other diagnostic studies is limited to patients with abdominal pain who have no compelling indication for abdominal exploration, unreliable physical examination and on suspicion of extra abdominal and non surgical causes of peritonitis.

Ultra sonogram diagnosis of a perforated hollow viscus is also made through the demonstration of pneumoperitoneum.[47] Air accumulates anteriorly and is evident as an echogenic area with reverberation artefacts. Ultrasonography demonstrated a sensitivity of 92%, a specificity of 65%, and a positive predictive value of 96% for the diagnosis of pneumoperitoneum. Reverberation artefacts always demonstrate a shifting phenomenon due to displacement of air seen with positional changes. It is also useful to identify the presence of free fluid which may helps in diagnosing the possible site of perforation.[48]

Computerised tomography is the most reliable imaging modality at present for detecting small amounts of intra peritoneal gas. Free air is seen very clearly especially on a lung window setting. *Extravasation of oral contrast is the gold standard sign of a perforation*, although its sensitivity is rather low (20–40%).[47] Discontinuous intestine wall, focal wall thickening with nearby air pockets and mesenteric fat stranding may indicate the possible site of perforation.

Differential diagnosis

A. Surgical conditions

1. Intestinal obstruction
2. Acute pancreatitis
3. Acute cholecystitis
4. Ruptured ectopic gestation
5. Ruptured aneurysm
6. Mesenteric ischaemia

B. Medical conditions

1. Basal pneumonia
2. Myocardial infarction
3. Pleurisy
4. Herpes zoster

MANAGEMENT OF PERITONITIS

I. SUPPORTIVE MEASURES

A. To correct hypovolemia and inadequate tissue oxygenation

In all cases of peritonitis, certain degree of hypovolemia is present owing to the "third spacing" of extracellular fluid within the peritoneal cavity. The rapidity of resuscitation is dependent on the degree of hypovolemia and the physiologic status of the patient. The acuity of the situation also determines the rate of fluid resuscitation. If immediate operation is required, for example, in a case of intestinal ischemia, preoperative fluid resuscitation may be curtailed short to avoid potentially fatal delays. In contrast, if condition permits, it is better to spend the initial 2 or 3 hours with adequate fluid resuscitation.

The deficit in volume is usually estimated with a combination of symptoms and signs of hydration status of the patient and an acute change in body weight. Crystalloids such as isotonic 0.9 % Sodium chloride or Ringer's lactate solutions are used. A bolus of 1500-2000 ml is given, followed by twice the maintenance dose requirement. Continuous clinical monitoring of vitals is required and fluids are adjusted accordingly.

Controversy surrounds the use of colloids as resuscitative fluids. Proponents argue that the volume of crystalloids used causes oedema which is deleterious to wound healing. On the other hand, colloids cause rapid expansion of intravascular volume at low pressures. One litre of dextran raises the intravascular volume by 800ml; Hetastarch by 750 ml; Five percent albumin by 500ml; whereas one litre of crystalloid causes a modest rise of only 180 ml.

The disadvantages with colloids are the risk of anaphylactic shock, inhibition of coagulation cascade and increased risk of acute renal shut down seen with hetastarch. Several meta-analyses have compared the potential benefits and disadvantages between the two. Results showed an increased mortality in patients resuscitated with colloids.

The fluid replacement is gauged by monitoring of pulse, blood pressure and improvement in mental status of the patient. Establishing a **minimum urine output of 30-50 ml/kg/ hour** is a reliable indicator of adequate fluid resuscitation. Invasive central venous pressure monitoring is a must in patients with septic shock and organ insufficiency. Supplemental oxygen is necessary, and hyperbaric oxygen or mechanical ventilation may be necessary to improve oxygenation.

Thus, crystalloids are still the fluid of choice for volume resuscitation, although patients with **profound volume deficits may benefit from colloids** in addition to crystalloids.

II. OPERATIVE MANAGEMENT

INCISIONS AND EXPOSURES

There are three main incision techniques for abdominal exploration..

- Midline
- Muscle cutting
- Muscle splitting

There is little doubt that a correct preoperative diagnosis, with the appropriate siting of the surgical incision, is the handmaid to a successful operation. If, on opening the abdomen, the expected findings are not present, a decision must be made whether to proceed with the original incision, extending it as necessary, or to close it up and proceed with one sited in a more appropriate position. If the preoperative diagnosis remains obscure but the decision to operate is clear, then the incision should be sited in a position that takes into account the most likely diagnosis. This will usually be a midline incision, which can be extended above or below the umbilicus as required.

This dilemma of whether to site the initial incision above or below the umbilicus is made more difficult by the fact that free perforation of colonic diverticulum may produce much gas under diaphragm and central or upper signs, mimicking a perforated ulcer. Similarly, the lateral gutters, particularly the right, may provide a path for liquids to descend from a perforated stomach or duodenum, resulting in the predominant symptoms and signs appearing below the umbilicus. If the spread of intra peritoneal exudates is kept in forefront of the mind, mistakes in choosing the upper or lower halves should be rare.

Thus a vertical midline incision is the outright choice in an emergency surgery, which can be extended up or down as circumstances dictate. True, this is the decision of indecision but, although it may end up larger than one designed for the problem ultimately found, the benefits clearly outweigh risks. ***To say that incisions heal from side to side, not from end to end, is too simple; nevertheless, it is a dictum worth remembering when an extra few centimeters of exposure are required.***

ABDOMINAL EXPLORATION

Upon opening the abdomen, a routine sequence of steps can avoid the unnecessary delay of diagnosis.

- When the peritoneum is opened, muffled ‘pop’ of escaping gas may be heard.

- Peptic ulcer perforation is sought after by systematically exploring along the greater and the lesser curvatures of the stomach and duodenum. The site of perforation is usually where the fluid is welling up most plentifully.

- If the perforation is elusive, palpating the lesser curve between the fingers and the thumb will reveal indurations around the edges of a perforated gastric ulcer.

- If the lesser sac is filled with gastric fluid, an opening is made in the omentum between the stomach and the colon to examine the posterior surface of the stomach.

When fluid is present and a systematic search does not reveal a peptic ulcer perforation, other organs are examined to rule out perforations as follows.

- Gall bladder is examined to rule out an empyema or a gangrenous cholecystitis.

- Possibility of acute pancreatitis is considered and the relevant examination made.

- Appendix is palpated to rule out a perforation.

- Pelvic colon is palpated: indurations or adherent omentum with pus or fecal matter will indicate acute diverticulitis with perforation.
- Small intestine is examined from end to end. Perforated primary jejunal ulcer, perforated Meckel's diverticulum, foreign body perforations of jejunum and ileum are possibilities.
- Finally, examination of the colon is completed from end to end by palpation.

The classical, single operation for intra-abdominal infection established during the earlier decades and reduced overall mortality from over 90% in earlier days to approximately 35%. Where ever be the site of perforation, treatment of peritonitis involves set standard principles.

Principle 1: (*repair*) *eliminating the source of infection*

This involves a definitive procedure for the source of all the abdominal sepsis. It could be an appendectomy for perforated appendicitis or an omentopexy for a perforated duodenal ulcer. Occasionally, resections are required to remove the infective focus, such as distal gastrectomy for a perforated gastric ulcer /carcinoma or a colectomy for perforated colonic diverticulitis. Usually, the source of infection, degree of peritoneal contamination and the patient's general condition dictates the choice of the procedure. Generally, in the presence of severe

peritonitis, it is recommended to avoid an anastomosis to minimize the risk of dehiscence and other complications.

Principle 2: ***Purging the infected abdominal cavity***

This involves thorough aspiration of all infectious fluids and removal of particulate matter by swabbing. Despite being widely followed, there is no evidence that intra operative lavage reduces mortality or the incidence of sepsis in patients receiving adequate systemic antibiotics. Intra operative irrigation with antibiotics is not advantageous because antibiotics need to act against the bacteria for some time to be effective. The addition of antiseptics may produce toxic effects. In a few experimental studies, intra peritoneal instillation of heparin has decreased mortality, but clinical trials are lacking. In a prospective randomized study, debridement of the peritoneal cavity was undertaken to see if this reduced the post operative septic complications. The results were not supportive and aggressive debridement endangered the integrity of the inflamed bowel and resulted in excessive bleeding from the denuded peritoneum. Postoperative peritoneal lavage is simply not useful53 because it is not possible to irrigate the entire abdominal cavity and carried the risk of introducing extraneous infection. Though drains are commonly used, it is impossible to drain the entire peritoneal cavity effectively. Besides providing a false sense of security and reassurance,

drains can rarely cause erosion of intestine or blood vessels and allow ascending infections. Thus, drains should be limited to the evacuation of an established abscess and to establish a controlled intestinal fistula sans exteriorization.

The following two techniques are to be instituted in specific well selected patients with selected indications.

Principle 3: ***To Decompress and relieve abdominal compartment syndrome***

Open management addresses principle 3 and facilitates frequent re-exploration. The principle is to decompress the increased intra-abdominal pressure resulting from ongoing infectious process and prevents the development of compartment syndrome.[49] Early results were promising mainly for infected pancreatic necrosis, but similar results could not be reproduced for peritonitis.[50] The initial problems of intestinal fistulas with laparostomies was almost eliminated with the introduction of temporary abdominal closure methods such as mesh-zipper techniques.[50]

Principle 4 (Control)

To verify both repair and purge

The principle is to explore a second time to remove persisting infection and debride until the resolution of disease process. Moreover, planned re-laparotomy

provides a chance to look at the integrity of anastomoses fashioned at the first operation. If the bowel has been exteriorized, then it provides a chance for restoration of intestinal continuity.

Nearly 10% to 15% of all patients undergoing surgery for peritonitis will need a planned re-laparotomy at a later stage. Few nonrandomized trials did not demonstrate an advantage of the open method. However, a prospective study comparing patients at equal operative risk showed the staged abdominal repair approach to be better to conventional operative therapy under certain circumstances. A potential disadvantage is escalation and precipitation of organ failure due to “second hit”. This has been proven beyond doubt in trauma cases where repetition of operation caused worsening of inflammation by adding second insult to the switched on inflammatory cascade. Bacteriological studies reveal the bacterial inoculum to regrow in a **24 hours** time period. Hence ***this 24-hour interval is mandatory between surgeries to avoid adding wood to the inflammatory fire.***

MANAGEMENT OF SPECIFIC DISEASE PROCESSES

PERFORATED PEPTIC ULCER

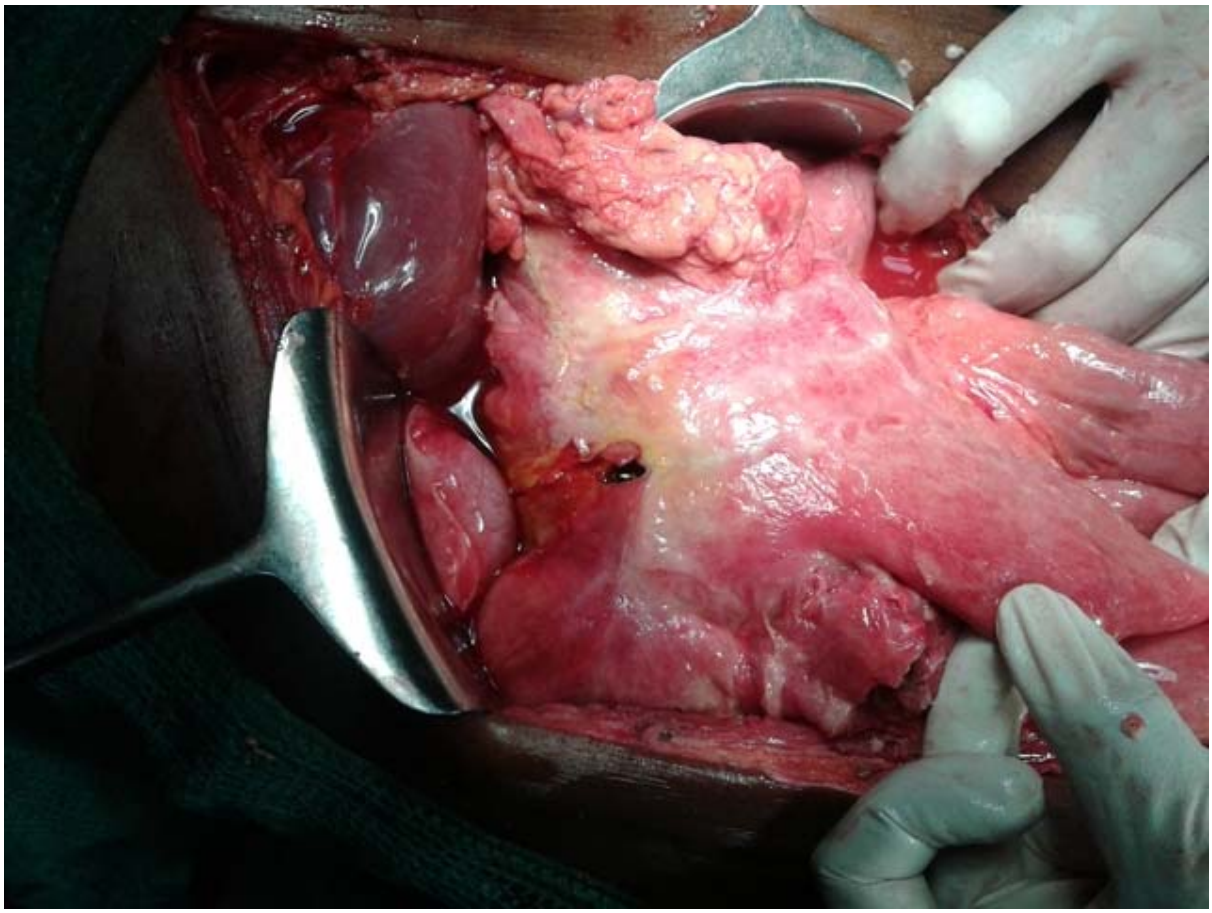
Perforated peptic ulcer operations are most often performed in the elderly and the sick. Patients may have associated bleeding, perforation, or obstruction. The objectives of surgery in these cases are:

- To deal with the complication that necessitated surgery
- To reduce the risk for future ulcer recurrence
- To perform a safe, quick, and effective operation
- To minimize long-term effects on the gastrointestinal tract
- To establish the H. pylori status of the patient

PERFORATED DUODENAL ULCER

An acute perforation is estimated to occur in 2% to 10% of patients with a duodenal ulcer.[51] Once a duodenal perforation has been confirmed, pads are placed around the perforation to contain further spillage, and 3-0 PDS or silk sutures are placed across the perforation. Usually, three to four sutures are needed. It is important to take bites of appropriate width (0.5 to 1 cm) to prevent the sutures

from cutting through the inflamed duodenal tissue. These sutures should not be tied to approximate the ulcer; rather, the adjacent omentum should be mobilized with



DUODENAL PERFORATION

an intact vascular pedicle and brought up. Sutures are tied over this omental pedicle to secure the omentum in place. These sutures should not be tied too tightly to avoid strangulation of the omental patch. Sewing the ulcer closed before placing the omental pedicle over the perforation is discouraged because it reduces surface contact of the omentum with the duodenal mucosa. If duodenal induration or edema precludes closure of the defect, then use of an omental or jejunal serosal patch can be helpful.

While many methods of **postoperative duodenal decompression** have been described, a transpyloric nasogastric sump tube is the simplest method. While some surgeons place a closed suction drain in the sub hepatic space, strong data do not exist to support this practice.[65] Insertion of a nasoenteric or jejunal feeding tube should be considered, especially in patients with evidence of chronic malnutrition or in whom a prolonged postoperative course is expected. A retrograde duodenal drain and jejunal feeding tube also can be placed in the proximal jejunum to decompress the duodenum if the closure appears tenuous.

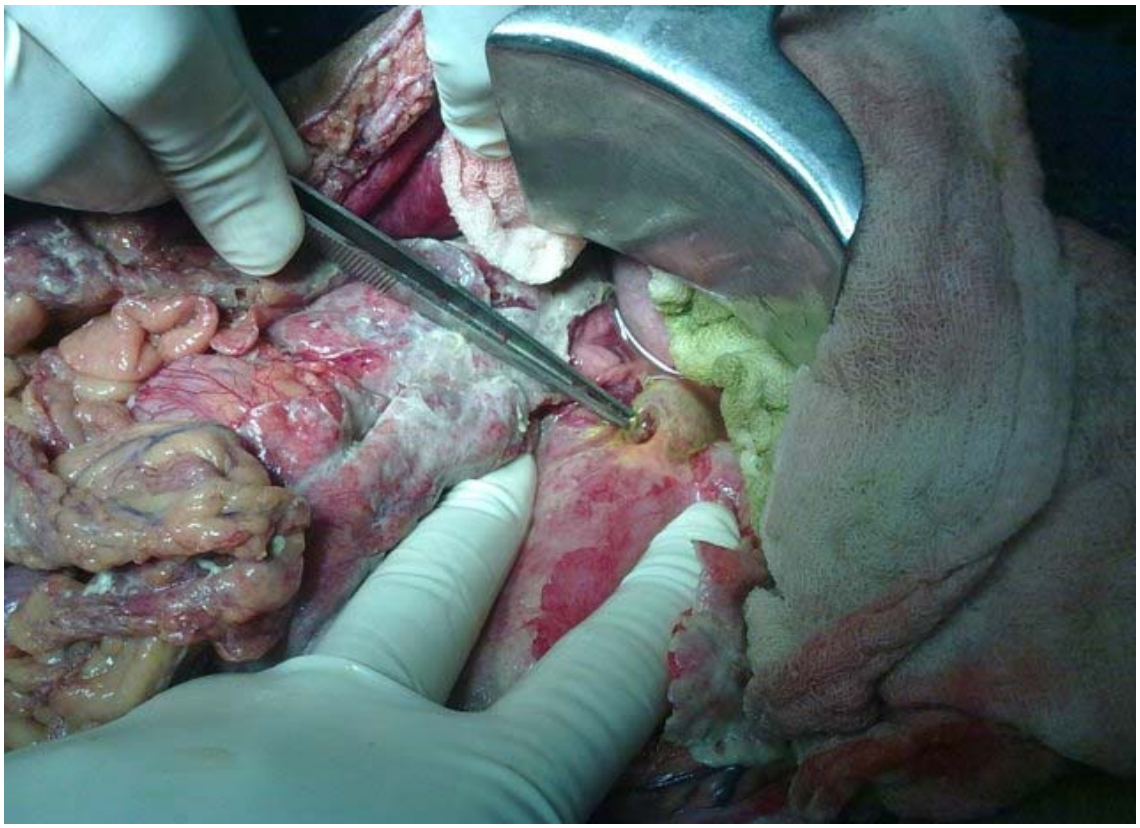
In the unusual circumstances of a **large ulcer** and significant inflammation, duodenal drainage and pyloric exclusion as described for use in the treatment of

traumatic duodenal injuries can be helpful.[51] A combination of gastrostomy, duodenostomy, and jejunostomy tubes then would be indicated. Alternatively, a lateral duodenal fistula can be prevented by a Roux-en-Y jejunal "patch" sutured over the defect with a transjejunal drain that extends from the duodenum through the jejunal "patch" and exits via a Witzel closure several centimeters downstream in the jejunal limb.

PERFORATED GASTRIC ULCER

A perforated gastric ulcer is associated with greater mortality that ranges from 10% to 35% and increases significantly with age (>65 years).[52] There has been debate in cases of **perforated type I ulcers** over whether to perform partial gastrectomy or proceed with simple patching of the perforation. Partial gastrectomy is the preferred approach unless the patient is at unacceptably high risk because of advanced age, comorbid illnesses, hemodynamic instability, or severe peritoneal contamination.[52] Even in the high-risk group who may initially be in shock, there is increasing evidence that definitive surgery can be tolerated as well as the quicker and simpler patching technique. It is therefore recommended that a patient with a perforated type I gastric ulcer undergoes partial gastrectomy unless contraindicated due to instability and significant comorbid conditions. If closure techniques are to be used, patch closure is preferred over simple suturing

and closure of the ulcer, which has a reported mortality of greater than 60%. Because the pathophysiology of such ulcers does not involve acid hypersecretion,



GASTRIC PERFORATION

an antacid procedure is not required. It is also important to perform an adequate four-quadrant biopsy of ulcers that are not excised.

For **type II ulcers,** the pathophysiology is very similar to that of perforated duodenal ulcers, and hence the treatment algorithm should be similar. This means that the ulcers should be adequately patched, the *H. pylori* status of the patient determined by intraoperative biopsy, and the patient treated appropriately. For these ulcers, it is important to obtain an intra operative biopsy to rule out malignancy, which can be associated with these gastric ulcers. Similar to a perforated duodenal ulcer, an acid-reducing procedure is not required unless the patient has a history of recurrent ulcer disease and has been previously treated for *H. pylori.* In circumstances in which a definitive antiulcer procedure is deemed necessary because of the chronicity of symptoms and lack of response to proton pump inhibitors, Highly Selective Vagotomy or truncal vagotomy and antrectomy should be considered.

Type III ulcers are thought to have a pathogenesis similar to that of perforated duodenal ulcers; however, their treatment in the event of acute

perforation deserves special mention. Patch repair of such pre pyloric ulcers is associated with a high incidence of gastric outlet obstruction, and HSV has been shown to be associated with a high recurrence rate. Therefore, antrectomy and vagotomy may be the best surgical approach.

Biopsy and patch closure may be an appropriate treatment for a high **type IV ulcer**, where more extensive resection may lead to total gastrectomy in a critically ill patient.

TRAUMATIC PERFORATIONS OF SMALL INTESTINE

In isolated small bowel injury, there is usually only a small amount of bile stained free fluid but, particularly in gunshot wounds, a multiplicity of perforations and associated mesenteric damage leads to extensive bleeding. The site of rupture is usually evident by itself or identified with flakes of coagulated lymph in the vicinity and by the presence of edema. After completion of a formal laparotomy to rule out associated injuries, bowel perforations are treated with simple closure or resection and anastomosis.

- Single short tears (not more than 4 cms) are repaired in the transverse axis of the gut.

- Longer single tears are repaired in the long axis with utmost tissue conservation.

Resection is needed when the following are present

- Associated mesenteric lesion has devitalized the damaged section.
- Presence of mangled intestine in high velocity injuries.
- Presence of several perforations grouped close together where the closure will be time consuming and result in a distorted loop of doubtful efficiency.

B. To treat persisting minor infection with antibiotics

Antimicrobial therapy should be initiated in a case of acute abdomen as soon as an infection is considered likely. Antibiotics may need to be initiated in the emergency department in the setting of septic shock. Blood levels of antibiotics must be in the therapeutic range through-out the intervention for it to be effective. Hence additional doses are repeated at the start of surgery.

Blood cultures are not routinely recommended for patients with community-acquired intra abdominal infection. It is neither necessary to perform a routine Gram staining of the aspirated materials. Knowledge of bacteraemia may be helpful in determining the choice and duration of antibiotic therapy in an

immunocompromised patient. Routine culture and antibiotic susceptibility studies are indicated when resistance is shown by more than 10%– 20% of *Escherichia coli* isolates in the community. Anaerobic cultures are routinely not necessary in community acquired infections.

Clinical Factors Predicting Failure of Source Control measures:[53]

- Advanced age
- Interventional delay of > 24 hrs
- High severity as indicated by APACHE II score greater than 15
- Presence of organ failure
- Low serum albumin levels
- Poor nutritional and general status
- Presence of diffuse peritonitis
- Failure to achieve adequate drainage
- Presence of malignancy

The following are the agents commonly used in the various settings mentioned herewith.[53,54]

1. For the Initial Empiric treatment of complicated Intra-abdominal Infection due to extra biliary causes

An empiric treatment of community acquired intra-abdominal infection should be active against aerobic gram negative bacilli and facultative anaerobes. Coverage for obligate anaerobes should be provided for distal gastrointestinal perforations of appendix/colon. It is also deemed necessary with delayed presentations of proximal gastrointestinal perforations especially in the setting of obstruction or ileus.

Community-acquired infection in adults of mild-to-moderate severity such as perforated or abscessed appendicitis are treated with *Cefoxitin, moxifloxacin, ticarcillin-clavulanic acid* and *tigecycline* as single agents, or combination regimen of a third generation cephalosporin such as *Cefazolin, ceftriaxone, cefotaxime,* or a fluoroquinolone such as *ciprofloxacin, or levofloxacin,* each in combination with *metronidazole*.

For high risk cases, such as those with severe physiologic disturbance, advanced age, or immunocompromised state *Imipenem-cilastatin, meropenem and*

piperacillin-tazobactam are used as single agents. Combination therapy with third generation cephalosporins such as *cefepime, ceftazidime, or* fluoroquinolones *such as ciprofloxacin, or levofloxacin,* each in combination with *metronidazole* is also successful.

2. Regimens that are used for Empiric Treatment of Biliary Infection in Adults

Imipenem-cilastatin, meropenem, piperacillin-tazobactam, ciprofloxacin,levofloxacin,or cefepime, each in combination with *metronidazole* is used. For community-acquired biliary infection, antimicrobial activity against enterococci is not required. For selected immune-suppressed patients, enterococcal infection may become significant and require specific treatment.

3. Empiric Therapy for Health care–associated Intra-abdominal Infection

Empiric antibiotic therapy for health care–associated intra-abdominal infection should be driven by the results of local microbiologic testing and reporting. Likely pathogens are *Pseudomonas aeruginosa,* Enterobacteriaceae, *Acinetobacter,* or other multi drug resistant Gram negative bacteria. Empirically, multidrug regimens against gram-negative aerobic bacilli and facultative bacilli may be needed. These agents *include meropenem, imipenem-cilastatin, piperacillin-tazobactam, or ceftazidime* in combination with *metronidazole*.

Aminoglycosides are used when greater than 20 % pseudomonas strains are resistant to ceftazidime.

Antimicrobial therapy for enterococci should be given when enterococci are recovered from patients with health care–associated infection. Antibiotics that are useful against Enterococci include ampicillin and piperacillin-tazobactam. Vancomycin is the drug of choice for treatment of Methicillin Resistant Staphylococcus Aureus strains.

Antifungal therapy is initiated if *Candida* is grown from intra-abdominal cultures. Fluconazole is the initial drug of choice. For fluconazole-resistant *Candida* species, one of the echinocandins (caspofungin, micafungin, or anidulafungin) is appropriate.

Duration of Therapy in Adults

Antimicrobial therapy of an established infection should be for 4–7 days, unless the infection persists.[55] Longer durations of therapy have not been associated with improved outcome. Even twenty four hours of antibiotic was found sufficient for gastric/ jejunal perforations without any evidence of malignancy. Treatment with acid reducing drugs and delayed presentations necessitate the antimicrobial spectrum to include mixed flora as that for colonic perforations.

Similar principle hold true for traumatic bowel injuries that are repaired within 12 hours. The degree of contamination is however relevant as even small contamination requires treatment for 3 to 5 days.

SCORING SYSTEMS

APACHE SYSTEM

In 1981, Knaus and others proposed a scoring system to be used for classifying patients admitted to intensive care units.[57] It consists of two parts:

1. Acute physiology score reflecting the degree of severity of acute illness
2. A preadmission health status indicating the health of the patient prior to the acute illness.

The APS was proposed by an expert panel of multi-disciplinary physicians who selected laboratory and clinical measurements important in predicting mortality. Only those physiological variables that were available or shortly obtainable on admission to ICU were used. The initial score had a list of 34 variables and a health questionnaire that assessed health status before admission.

APACHE II SYSTEM

In 1985, a revised version of the original APACHE was published in which the number of physiologic measurements was reduced from 34 to 12. This followed many studies which suggested that the smallest number of variables that reflected physiologic derangements and maintained statistical precision as well to be 12.[56,57]

Special consideration was given to age of the patient and severe chronic health problems as they indicate decreased physiologic reserve of the patient. They are included in the APACHE II scoring. Age was found to be an independent risk factor as well.

When age and acute physiologic derangements were controlled during validation, it was found that chronic health classifications were associated with higher death rates. But only the most severe organ inefficiency or immunocompromised state affected outcome. It was also discovered that non-operative and emergency surgery admissions had a higher risk for death from their organ system inefficiency than elective surgical admissions. This was because patients with most severe chronic conditions were not fit enough to undergo the planned elective procedure. Therefore, it was decided to add five points in cases of

emergency operative admissions with a severe chronic organ dysfunction, whereas only two points was added in similar elective admissions. The maximum possible APACHE II score is 71.[58]

MANHEIM'S PERITONITIS INDEX

Wacha and co-workers developed this index which encompasses information regarding age, gender, organ failure, presence of a cancer, duration of peritonitis, origin of sepsis, the extent of spread within the peritoneum and the character of the peritoneal fluid, to define risk.

The possible scores range from 0 to 47, and patients with score above 26 are defined as having peritonitis.[57] *Billing et al* evaluated the effectiveness of this system in a study involving 2000 patients. The overall mortality was 19.5 %. Patients with a score of more than 26 had a mortality rate of 55 % which was significantly greater than the 7 % mortality observed with patients who had score of less than 26.[59]

USES OF PROGNOSTIC SCORING SYSTEMS

Prognostic scoring systems have proved to be useful in risk stratification of patients for clinical trials and in the assessment of quality of care delivered in ICUs. It is likely that they will assist the decision process regarding the need for

ICU admission. The role they will ultimately have in individual patient care decisions still remains to be determined.

1. Clinical studies

A central problem encountered in a clinical trial with acutely ill patients is that the control and treatment groups are at an unequal baseline risk of death or another important outcome. These risks can be minimized with randomization between the two patient groups. But, randomization only ensures that patients and not their risk factors are randomly distributed. For example, in the evaluation of peritonitis, patients could range from a 20 year old with rupture appendix to a 70 year old with perforated colon. Appropriate conclusions regarding the efficacy of a new treatment could be reached only if the patients and their risks are evenly distributed between the control and treatment groups. A prognostic scoring system ensures even distribution by allowing investigators to stratify according to different risk categories.[60]

Schein et al, in their study in emergency operations for perforated ulcers, divided their patients based on APACHE II score into two groups – those with low score (< 10) and high score (> 10). They found the mortality rate in the low risk group was only 8 % whereas it was 33.3 % in the patients with high scores.[61] Similar stratification was done in numerous other studies.[61,62]

2. Quality of care measurement

At the costs of medical care, quality assessment has become a major priority for ICUs, government hospitals, and third party payers. A suburban shock and trauma unit will have a different patient population from an inner city ICU. A prognostic scoring system that establishes a predicted mortality rate before treatment for an ICU on the basis of patient-by-patient measurement of risk will permit the ICUs to compare the predicted outcome to its observed outcome. The difference between predicted and actual death rate is a direct measure of quality of care and can also provide unique insights regarding the usefulness of specific treatments.

Michael Marsh et al in 1990, in a study conducted to assess prediction of mortality with APACHE II scores in ICUs, observed that the predicted risk for hospital death among non-operative patients in Rochester Methodist Hospital was significantly higher than the risk predicted at St. Mary's Hospital. Further evaluation revealed that the mean ages were similar in both groups. When APACHE II scores were used, they observed that the mean acute physiology score of the patients in the former was significantly higher than the score observed in the latter[63].

In 1982, Knauss and co-workers compared the outcome of acutely ill patients treated in French and American ICUs. In patients with severe gastro-intestinal disorders, the French hospital death rate was significantly higher than the one predicted in American hospitals. Investigations into this discrepancy led to the conclusion that the disparity may have been due in part to a more aggressive surgical approach for acute pancreatitis in France.[64]

3. Allocation of Resource

An important issue for every ICU is in deciding the need for admission. An objective method to identify relative risk of patients might be useful in supporting clinical judgement and in establishing priorities for ICU admission during the periods of limited bed availability. Another important issue is to identify patients who have 100 % mortality as further aggressive therapy is futile.

Borlase et al in their study conducted in 1990, suggested that an APS > 25, a Glasgow coma scale < 7 and a creatinine > 4.5 mg/dL were good predictors of mortality on the first day of ICU admission. But, this study did not reveal an enhanced predictive power with sequential APACHE scoring as shown by trend analysis. When daily cost of predicted SICU non-survivors were considered, if treatment had been stopped after 10 days of aggressive therapy with no

improvement, the potential saving would have reached an amount equivalent to 4 % of the total cost[64].

4. Statistical versus clinical judgement

An interesting use of scoring systems is a comparison of the expectations that physician and patients have regarding their prognosis and how the clinical and personal assessments compare to probabilities produced by the application of prognostic scoring systems.

Kruse and associates found that there were no significant difference in accuracy between the APACHE II scores at ICU admission and the assessment made by physicians and nurses. But, there was a substantial disagreement regarding the outcome of 40 % of the admissions between the physicians and nurses[65].

Meyer and associates observed that clinical assessment is superior to APACHE II in predicting outcome in critically ill patients. A similar observation was made by Marks et al with a combined medical and surgical patient population[66].

5. Individual patient care decisions

The most important question regarding the scoring system is how they can help with individual patient care decisions. Prognostic scoring system will never be able to predict outcome with 100 % specificity, but risk estimates of death or complications at the 90 to 99 % level could be useful. Before integrating such risk estimates into practice, however, compatibility of group statistics to individual patients should be considered. Individual patients do have unique features, they also share many features with previous patients and consideration of these common characteristics permits us to anticipate their response and predict their outcome. If probabilities did not have a role in clinical decision making, then we would never be able to use past experience to guide future decisions.[67]

Prognostic scoring systems can help in ensuring that clinical predictions are well calibrated and accurate for a patient. Since they estimate a patient's potential benefit from therapy, they also estimate an individual's comparative entitlement to medical care in an unbiased manner.

Singh and associates used APACHE II scoring system to identify the need for zipper laparotomy in management of abdominal sepsis. They defined the patient group as those with an APACHE II score between 27 and 30[68].

Schein and his associates used the APACHE II score in choosing the type of surgery to be performed in patients with perforated ulcers. In duodenal ulcer

patients, definitive surgery was done in the group of patients who had a score of less than 11. Likewise in stomach perforations, antrectomy or gastrectomy was undertaken in patients whose score was less than 11, while in patients with score of more than 11, wedge resection / patch closure was done.

LIMITATIONS OF PROGNOSTIC SCORING SYSTEMS

The use of prognostic scoring systems for clinical decision making raises many ethical and practical issues. The most important requirements are that its predictions must be infallible and reproducible. The inadequacies in predicting individual response are the following.

1. It does not reflect the dynamic changes that occur during the patient's stay in ICU.

2. Although APACHE II score is based on objective data, derivation of risk of death is based on a subjective choice of a single specific diagnostic category or major organ system as the primary cause of ICU admission. The correct choice can be difficult to make, especially among patients with multiple organ system failure and high mortality rates, the group of patients in whom a correct prediction is very important. An incorrect choice can lead to wrong computation of risk of death and hence a wrong prediction.

Therefore, it seems unwise on part of clinicians, and injustice to patients and relatives to make major clinical decisions on just one assessment.

Aims and Objectives

AIMS AND OBJECTIVES

1. To stratify the patients with peritonitis based on their scores at admission. Acute Physiology and Chronic Health Evaluation (APACHE) II and Mannheim Peritonitis Index (MPI) scoring systems are used in this study.

2. To correlate the mortality rates observed with the scores obtained.

3. To compare the various postoperative outcomes and complications of bacterial peritonitis due to gastro intestinal perforations.

Materials and Methods

MATERIALS AND METHODS

This study was conducted in The Department of General Surgery of The Government Stanley Medical College Hospital. Fifty cases of acute bacterial peritonitis secondary to gastro intestinal tract perforations were encountered during the study period of one year from 01.01.2012 to 31.12.2012. Nature of the study was prospective study and cases were included into the study by application of following criteria.

INCLUSION CRITERIA

1. Peritonitis secondary to hollow viscus perforation.
2. Age group between 15 to 75 yrs.
3. Both males and females were included in the study.

EXCLUSION CRITERIA

1. Spontaneous bacterial peritonitis.
2. Post operative peritonitis due to anastomotic leak, etc.
3. Pancreatitis induced peritonitis.
4. Ruptured liver abscess induced peritonitis

5. Age group less than 15 yrs

6. Select sealed perforations managed conservatively.

All the necessary preoperative data were recorded. Blood sample was taken and relevant basic investigations were carried out. The patient was resuscitated with fluids and electrolytes brought and maintained within the normal range. Urethral catheter was inserted to monitor hourly urine output and nasogastric tube inserted to decompress the stomach. The parameters of modified APACHE II score and Manheim's Peritonitis Index were recorded at the time of admission.

APACHE II scoring

The following acute physiological parameters of APACHE II were included – temperature, mean arterial pressure, heart rate, respiratory rate, serum sodium, serum potassium, creatinine, serum bicarbonate, haematocrit, white blood cell count and GCS of the patient.

The scores ranged from 0 to 4 on each side of the normal value. Zero score represents normal values, an increase to 4 indicating the extreme end of high or low abnormal values. The sum of all the individual score values were obtained which denotes the acute physiology score.

APACHE II SCORING SYSTEM

ACUTE PHYSIOLOGIC ASSESSMENT AND CHRONIC HEALTH EVALUATION (APACHE) II SCORING SYSTEM*

PHYSIOLOGIC VARIABLE†		POINT SCORE								
		+4	+3	+2	+1	0	+1	+2	+3	+4
1	Temperature, core (°C)	≥ 41°	39–40.9°	—	38.5–38.9°	36–38.4°	34–35.9°	32–33.9°	30–31.9°	≤ 29.9°
2	Mean arterial pressure (mm Hg)	≥ 160	130–159	110–129	—	70–109	—	50–69	—	≤ 49
3	Heart rate	≥ 180	140–179	110–139	—	70–109	—	55–69	40–54	≤ 39
4	Respiratory rate (nonventilated or ventilated)	≥ 50	35–49	—	25–34	12–24	10–11	6–9	—	≤ 5
5	Oxygenation:									
	a) $FIO_2 \geq 0.5$: use A-aDO_2	≥ 500	350–499	200–349	—	< 200	—	—	—	—
	b) $FIO_2 < 0.5$: use PAO_2 (mm Hg)	—	—	—	—	> 70	61–70	—	55–60	< 55
6	Arterial pH	≥ 7.7	7.6–7.69	—	7.5–7.59	7.33–7.49	—	7.25–7.32	7.15–7.24	< 7.15
7	Serum Na (mmol/L)	≥ 180	160–179	155–159	150–154	130–149	—	120–129	111–119	≤ 110
8	Serum K (mmol/L)	≥ 7	6–6.9	—	5.5–5.9	3.5–5.4	3–3.4	2.5–2.9	—	< 2.5
9	Serum creatinine (mg/dL); double point score for **acute** renal failure	≥ 3.5	2–3.4	1.5–1.9	—	0.6–1.4	—	< 0.6	—	—
10	Hct (%)	≥ 60	—	50–59.9	46–49.9	30–45.9	—	20–29.9	—	< 20
11	WBC (in 1000s)	≥ 40	—	20–39.9	15–19.9	3–14.9	—	1–2.9	—	< 1
12	Glasgow coma score (GCS)	Score = 15 minus actual GCS								

Acute physiology score is the sum of the 12 individual variable points.

Add 0 points for age <44; 2 points, 45–54 yr; 3 points, 55–64 yr; 5 points, 65–74 yr; 6 points ≥ 75 yr.

Add chronic health status points: 2 points if elective postoperative patient with immunocompromise or history of severe organ insufficiency; 5 points for nonoperative patient or emergency postoperative patient with immunocompromise or severe organ insufficiency.‡

(13)§	Serum HCO_3 (venous–mmol/L)	≥ 52	41–51.9	—	32–40.9	22–31.9	—	18–21.9	15–17.9	< 15

*APACHE II score = acute physiology score + age points + chronic health points. Minimum score = 0; maximum score = 71. Increasing score is associated with increasing risk of hospital death.

†Choose worst value in the past 24 h.

‡Chronic health status: Organ insufficiency (eg, hepatic, cardiovascular, renal, pulmonary) or immunocompromised state must have preceded current admission.

§Optional variable; use only if no ABGs.

A-a DO_2 = Alveolar–arterial oxygen gradient; FIO_2 = fractional inspired O_2.

Organ insufficiency or immune-compromised state must have been evident prior to hospital admission and conform to following criteria:

- **Liver**: Documented portal hypertension and biopsy proven cirrhosis and/ or prior episodes of hepatic failure, encephalopathy or coma.
- **CVS**: Class IV New York Heart Association ailment
- **RS:** chronic restrictive, obstructive or vascular disease resulting in severe exercise restriction, chronic hypoxia, hypercapnoea, severe polycythemia,
- **RENAL**: patient on chronic dialysis
- **IMMUNOCOMPROMISED**: the patient has received therapy that suppresses resistance to infection. E.g.: immunosuppression, chemotherapy, radiation, long term or recent steroids, or has a disease that is sufficiently advanced to suppress to infection. E.g.: leukaemia, lymphoma, AIDS.

Manheim's peritonitis index includes

MANNHEIM PERITONITIS INDEX

RISK FACTOR	WEIGHTING IF PRESENT
AGE>50 years	5
Female sex	5
Organ failure	7
Malignancy	4
Origin of sepsis not colonic	4
Diffuse generalised peritonitis	6
Preoperative duration of peritonitis >24h	4
Intra peritoneal Exudates	
Clear	0
Cloudy, purulent	6
Faecal	12

Organ failure criteria

Creatinine level>177 micro mol per litre

Urea level >167 mmol per litre

Oliguria <20 ml per hour

PaO2<50 mm of hg

PaCO2>50mm of hg

Shock: systolic blood pressure<90mm of hg, MAP<60mm of hg

Intestinal obstruction only if profound with paralytic ileus>24h, complete mechanical

After adequate resuscitation and assessment, patients underwent exploratory laparotomy. At surgery, the pathology was identified and treated accordingly. Thorough and copious irrigation of the cavity was given and insertion of drains was decided on case to case basis. Abdomen was closed with non-absorbable suture material in a continuous fashion. All patients received appropriate broad spectrum antibiotics for a minimum period of 5 to 7 days.

Analysis

Demographic, clinical, preoperative, and/or post operative complications on each patient were entered into a standard proforma. Each patient's postoperative outcome/ mortality were compared to determine the significance of illness on postoperative complications and mortality.

Observation and Results

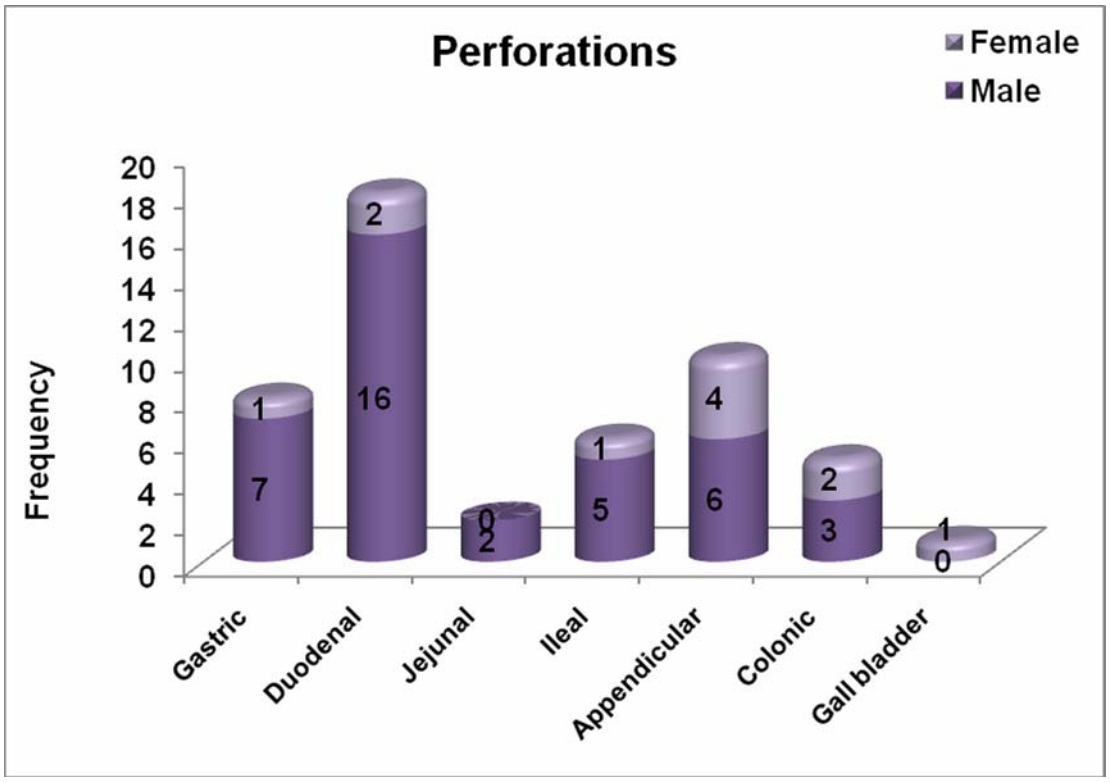
OBSERVATION AND RESULTS

TABLE 1: PERCENTAGE OF CASES

TOTAL NUMBER OF CASES				
	MALE	**FEMALE**	**TOTAL**	
GASTRIC	7	1	8	16%
DUODENAL	16	2	18	36%
JEJUNAL	2	0	2	4%
ILEAL	5	1	6	12%
APPENDICULAR	6	4	10	20%
COLONIC	3	2	5	10%
GALL BLADDER	0	1	1	2%
	39	11	50	

Upper Gastro-intestinal perforations, namely **peptic ulcer** perforations constituted the most common perforation in our study. They accounted for 52 % of the total cases, with **duodenal ulcer** constituting **36 % (18 cases)** and **gastric ulcer** forming the rest **16 % (8 cases)**. 10 cases of appendicular perforation were included in the study (20 %).

FIG 1: DISTRIBUTION OF CASES OBSERVED IN OUR STUDY

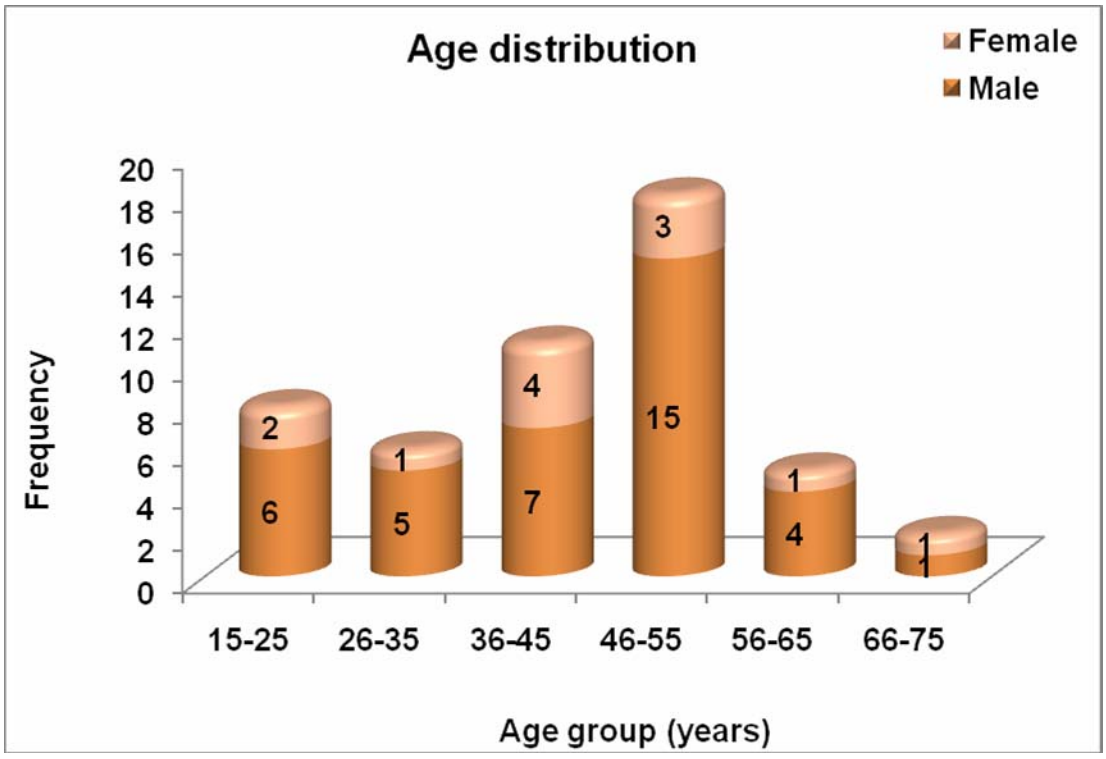


Duodenal perforation was the most common perforation among the **male patients** (16/39 patients) and most of them had a binge of alcohol within a day or two of presentation. Among **females**, **appendicular perforations** were identified commonly (4/11 patients).

TABLE 2: AGE DISTRIBUTION

AGE DISTRIBUTION						
	15-25	**26-35**	**36-45**	**46-55**	**56-65**	**66-75**
MALE	6	5	7	15	4	1
FEMALE	2	1	4	3	1	1
TOTAL	8	6	11	18	5	2

FIG. 2: AGE DISTRIBUTION IN OUR STUDY



The patients included in this study had a mean age of **42.7 years** with a range between **20 – 68 years**. The male: female ratio was **3.1: 1** with 38 male

patients and 12 female patients. The mean age of males was **42.6 yrs (20- 68 years)** and females were **42.9 years (21-68 years).**

TABLE 3: PERCENTAGE OF NON-SURVIVORS IN VARIOUS PERFORATIONS

NON SURVIVORS				
	MALE	**FEMALE**	**TOTAL**	
GASTRIC	1	1	2	25%
DUODENAL	2	0	2	11.10%
JEJUNAL	1	0	1	50%
ILEAL	0	0	0	0
APPENDICULAR	1	0	1	10%
COLONIC	2	1	3	60%
GALL BLADDER	0	0	0	0

Majority of cases were diffuse generalised peritonitis. 11 cases of **localized** peritonitis **(22 %)** were encountered and most of them were **appendicular (7/11 cases)** in origin. Few early cases of duodenal perforation were also limited in nature.

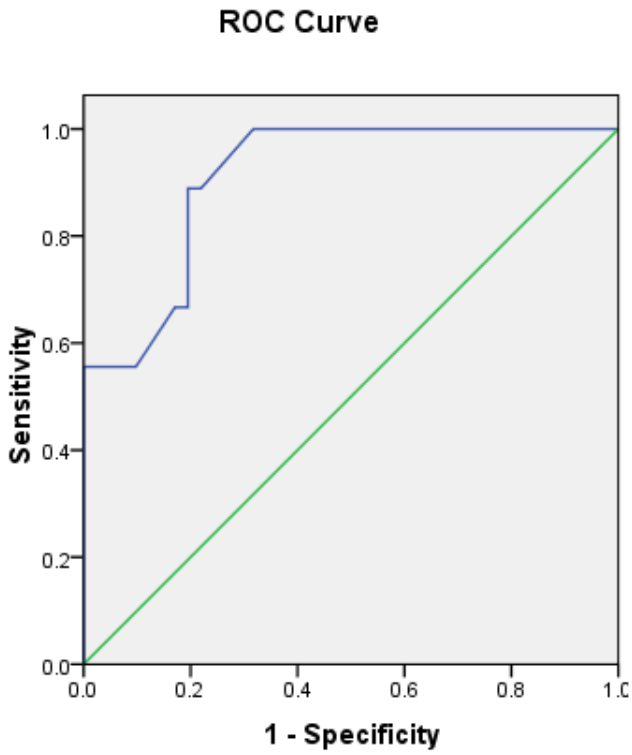
TABLE 4: MORTALITY RATES IN VARIOUS PERFORATIONS ACCORDING TO MANHEIMM'S SCORE GROUPS

SCORE	≤ 15			15 - 25			≥ 26		
	n	NS	%	n	NS	%	n	NS	%
GASTRIC	4	0	0	1	0	0	3	2	66.7
DUODENAL	11	0	0	5	1	20	2	1	50
JEJUNAL	1	0	0	0	0	0	1	1	100
ILEAL	1	0	0	5	0	0	0	0	0
APPENDICULAR	6	0	0	3	1	33.3	1	0	0
COLONIC	1	0	0	0	0	0	4	3	75
GALL BLADDER	0	0	0	1	0	0	0	0	0

The mortality rates among patients who had score of less than 15 are found to be zero. Only **two deaths** were observed with a score between 15-25 group and mortality rose to high levels among patients with higher values. **7 deaths** were recorded out of the 11 patients who had a **score of more than 25**.

To find the appropriate cut off point above which the mortality can be predicted requires the construction of an ROC curve.

FIG. 3: ROC CURVE ANALYSIS TO FIND THE BEST CUT-OFF POINT FOR MANHEIM'S SCORE TO PREDICT MORTALITY



AREA UNDER THE CURVE = 0.912

The ROC curve analysis predicted that the MANHEIM'S score of 22 or more will predict the non-survival status.

TABLE 5: SENSITIVITY AND SPECIFICITY OF MANHEIM'S INDEX

		MORTALITY		Total
		Yes	No	
MANHEIM'S SCORE	≥22	8	8	16
	<22	1	33	34
Total		9	41	50

Parameter	Estimate	Lower - Upper 95% CIs
Sensitivity	88.89%	56.50, 98.01
Specificity	80.49%	65.99, 89.77
Positive Predictive Value	50.00%	28.00, 72.00
Negative Predictive Value	97.06%	85.08, 99.48
Diagnostic Accuracy	82.00%	69.20, 90.23

ROC curve analysis predicted the AUC (Area Under the Curve) to be **0.972** for a Manheim's score of 22. Of the total 9 mortality observed in this study, 8 cases had a score of 22 and above. Only one case with a score of less than 22 expired during the study. This gives the score a **sensitivity of 88.89 %** (56.5 –

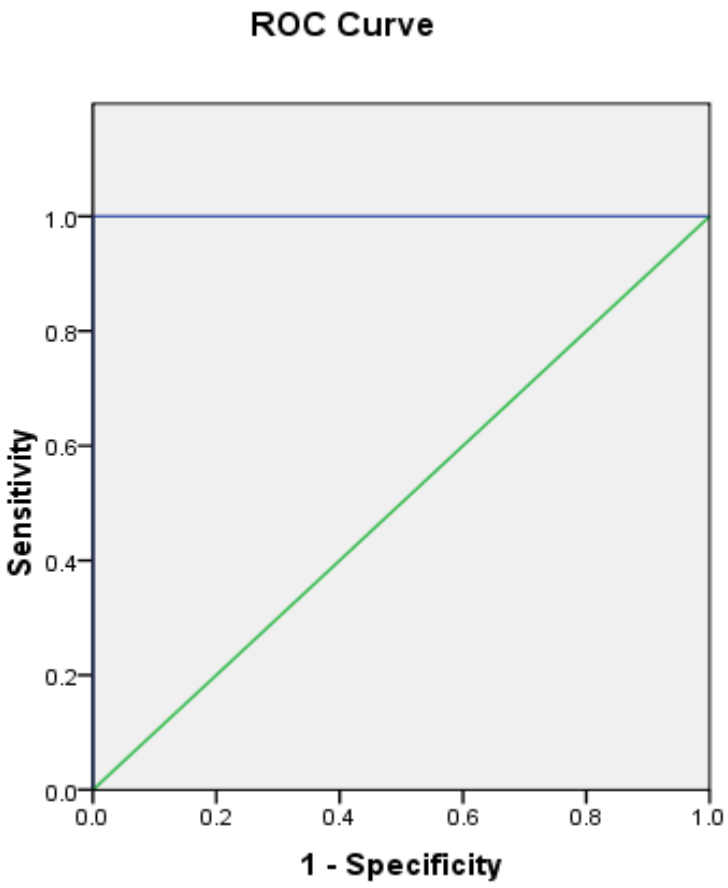
98.01) and a **specificity of 80.49 %** (65.99-89.77). The overall diagnostic **accuracy of this score is 82 %** in our study.

TABLE 6: MORTALITY RATES IN VARIOUS PERFORATIONS ACCORDING TO APACHE II SCORE GROUPS

	≤ 5			6 to 15			≥ 16		
	N	**NS**	**%**	**n**	**NS**	**%**	**n**	**NS**	**%**
GASTRIC	4	0	0	3	0	0	2	2	100
DUODENAL	12	0	0	3	0	0	2	2	100
JEJUNAL	1	0	0	0	0	0	1	1	100
ILEAL	1	0	0	4	0	0	0	0	0
APPENDICULAR	9	0	0	0	0	0	1	1	100
COLONIC	1	0	0	3	1	33.3	2	2	100
GALL BLADDER	0	0	0	1	0	0	0	0	0
TOTAL	**28**	**0**		**14**	**1**		**8**	**8**	**0**

The mortality was found to rise as the score rises. Below a score of 5, no deaths were observed. There was only one death among the group with scores between 6 and 15. The expired case had a value of 15. The last group had 8 patients and all 8 expired. The mortality rate was 2.3% below the score of 15 and rose proportionately beyond it. The timing of death was varied in different cases, but most cases expired on the second post operative day.

FIG. 4: ROC CURVE ANALYSIS TO FIND THE BEST CUT-OFF POINT FOR APACHE II SCORE TO PREDICT MORTALITY



AREA UNDER THE CURVE = 1

The ROC curve analysis predicted that the APACHE II score of 15 or more will predict the non-survival status.

TABLE 7: SENSITIVITY AND SPECIFICITY OF APACHE II INDEX

		MORTALITY		Total
		Yes	No	
APACHE II SCORE	≥ 15	9	0	29
	< 15	0	41	41
Total		9	41	50

Parameter	Estimate	Lower - Upper 95% CIs
Sensitivity	100%	70.08, 100.00
Specificity	100%	91.43, 100.00
Positive Predictive Value	100%	70.08, 100.00
Negative Predictive Value	100%	91.43, 100.00
Diagnostic Accuracy	100%	92.86, 100.00

Of the total 9 deaths observed in this study, all cases had a score of 15 and above. Hence an **APACHE II score of 15** predicts mortality with a **sensitivity** of 100 % and a **specificity** of **100 %.** The overall diagnostic accuracy of mortality with this score is found to be 100 %. An ROC curve plotted for a score of 15 gives an area under curve to be **1.0.**

Discussion

DISCUSSION

Temperatures of patients presenting with perforation peritonitis were recorded. Rectal temperature measurements are ideal, but for practical reasons, axillary temperatures were recorded and used in the study. Rectal temperatures are found to be 0.5 – 0.7 ^{o} C higher than the recorded axillary readings. Mean Temperature in our study was found to be **38.6 ^{o} C** with a range between **37 ^{o} C- 41 ^{o} C**. Higher temperatures were observed with appendicular and ileal perforations, especially when there was a delay of more than 3 days before presentation to the hospital. Mean temperature for ileal perforations was 39.1 ^{o} C and for appendicular perforations was 38.9 ^{o} C. Subnormal temperatures were found in two cases who presented with features of shock.

Mean arterial pressures (MAP) were calculated by systolic and diastolic blood pressure measurements using a sphygmomanometer. It is computed using the formula "Diastolic pressure + 1/3(Pulse pressure)". Pulse pressure is the difference between systolic and diastolic blood pressures. The MAP ranges in our study is between *65- 155 mm Hg,* with a *mean value of 98.5 mm Hg*. Low values were observed with a case of shock consequent to stab injury to the abdomen (65

mm Hg) and with another case of colonic malignant perforation with septic shock (68 mm Hg).

Of the total 50 cases, **7** patients had **malignancy**. A gastric malignancy presented as perforation in a 64 years old male patient. He was treated with subtotal gastrectomy and gastro jejunostomy. Biopsy report turned out to be moderately differentiated adenocarcinoma and the resected margins were free from tumour invasion. Two of the resected 7 nodes showed metastatic deposits. Patient was followed up with chemotherapy using 5 FU based regimens.

One case of incidentally diagnosed well **differentiated hepatocellular carcinoma** in a duodenal ulcer perforation was encountered. Post operatively it was treated with chemotherapy as the patient was not amenable to liver resection.

Two cases of colonic perforations with malignancy were encountered. Due to hemodynamic instability, one case was treated with diversion colostomy alone and the other with Hartmann's procedure. Both the patients were in fulminant sepsis and needed post operative ventilator and hemodynamic support. These patients had a downhill course and expired on second and third post operative days.

Two cases of **rectal malignancy**, one at the extra peritoneal site and the other in intra peritoneal location. The former had posterior fixity and was not resectable, and hence a diversion loop transverse colostomy was done. The patient was started with chemotherapy. The second case had a cancer at the recto-sigmoid junction with hugely dilated proximal descending colon with a contained perforation in it. The patient underwent primary Hartmann's procedure with adjuvant chemotherapy and a colo-rectal anastomosis in a secondary sitting with a covering loop colostomy which was closed under local anaesthesia. Both the patients are on regular follow up.

Another case of jejunal Gastro Intestinal tumour perforation was seen. The malignancy had invaded adjacent loops of ileum, and hence an *en bloc* resection was done. The patient deteriorated with sepsis and succumbed on second post operative day.

The mortality rate in our study is found to be **18%**. Various trials have estimated the mortality rate to be between 10-60% and the average mortality is **19.5%** which is close to the value noted with our study. The mortality rates are influenced by disease specific as well as patient related factors. In a prospective study was conducted by ***Carlos*** over a period of 10 years 1994-2004 (n=267), overall mortality was 20% and mean hospital stay was 20 days.

In our study, a **Manheim's score of 22** was found to predict mortality which was statistically significant. This is in accordance with previous studies where a score of 21 was found to predict mortality. In a study by ***Billing et al***, mortality rate in patients with a score of less than 21 was found to be 2.3% and above this score a mortality rate of 60-80% was observed. The mortality rate was found to rise proportionately beyond this score.[69]

Demmel et al evaluated the usage of MPI in acute peritonitis (n=438). Analysis revealed the MPI to have a sensitivity of 87% and a specificity of 77% for a score of 26. In our study, the cut off score of 22 had a sensitivity of 89% and a specificity of 80%.[70]

An **APACHE II score of 15** was found to predict mortality with significant difference between the two groups. Below this score, the mortality rate was **2.3%** and above this value, the mortality rose to **90-100%.** This is in accordance to ***Schein et al*** where the APACHE II score was found to predict mortality very well between a score of 11-20.[61]

Kulkarni et al evaluated the APACHE II score among patients with perforation peritonitis. A score between 11 and 20 was found to predict mortality with greater accuracy than a score of less than 10 or more than 20. Our study is in

accordance with this study, as the best cut off score was found to be 15 with a diagnostic accuracy and positive predictive value of 98% and 100% respectively.[71]

The comparison as to which score is the best is varied among different studies. ***Bosscha et al*** evaluated the various scoring systems in a study sample of 50 patients. A multivariate analysis revealed both APACHE II and MPI to predict the outcome independently.[72] ***Malik et al*** also arrived at similar conclusions but favoured APACHE II score as it better identified the physiological reserve of the patient under study.[73]

Ohmann et al found that the APACHE II score was better a predictor of mortality than MPI score. It was also useful to decide on the treatment formulations and repetitive monitoring in ICU set up.[74,75]

In our present study, both scoring systems are useful to predict mortality beyond their respective cut off scores. Though the Manheim's peritonitis index is accurate and easy to apply, it does not consider the **underlying physiological derangements** in the patient. An MPI also required intra operative details without which the score cannot be computed. Hence an APACHE II score, which is more physiological, is useful for risk stratification in acute settings. Despite its relative demerits of being cumbersome to calculate and not including the aetiology of the

underlying process, it is widely being followed for prediction of mortality and outcome.

Conclusions

CONCLUSION AND SUMMARY

- APACHE II score is the **current gold standard** for assessing the severity of acute perforation peritonitis.
- The mortality rate in our study of 50 patients was found to be **18 %**
- An **APACHE II score** of **15 and above** predicted mortality in our study population with a **positive predictive value** of **100%.**
- The overall **accuracy** of this score was found to be **100**%
- APACHE II score is **more physiological** in emergency settings compared to Manheim's score.
- Compared to the MPI score, APACHE II score could be **used serially** to monitor the patient in the immediate post operative period.
- Patient treatment can be optimized by appropriate **intensive supportive care** when it is determined to be needed.
- APACHE II score can **triage the patients** with the treatment directed to the most effective patient.

- Scoring patients into groups based on risk could help **future clinical research** by comparing therapeutic interventions in similar patients. Of the two scoring systems evaluated, the APACHE II seems to be better suited to achieve these goals.

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Annexure

ANNEXURE I

MANNHEIM PERITONITIS INDEX

RISK FACTOR	WEIGHTING IF PRESENT
AGE>50 years	5
Female sex	5
Organ failure	7
Malignancy	4
Origin of sepsis not colonic	4
Diffuse generalised peritonitis	6
Preoperative duration of peritonitis >24h	4
Intra peritoneal Exudates	
Clear	0
Cloudy, purulent	6
Faecal	12

Organ failure criteria

Creatinine level>177 micro mol per litre

Urea level >167 mmol per litre

Oliguria <20 ml per hour

PaO2<50 mm of hg

PaCO2>50mm of hg

Shock: systolic blood pressure<90mm of hg, MAP<60mm of hg

Intestinal obstruction only if profound with paralytic ileus>24h, complete mechanical

ANNEXURE II

ACUTE PHYSIOLOGIC ASSESSMENT AND CHRONIC HEALTH EVALUATION (APACHE) II SCORING SYSTEM*

PHYSIOLOGIC VARIABLE†		POINT SCORE								
		+4	+3	+2	+1	0	+1	+2	+3	+4
1	Temperature, core (°C)	≥ 41°	39–40.9°	—	38.5–38.9°	36–38.4°	34–35.9°	32–33.9°	30–31.9°	≤ 29.9°
2	Mean arterial pressure (mm Hg)	≥ 160	130–159	110–129	—	70–109	—	50–69	—	≤ 49
3	Heart rate	≥ 180	140–179	110–139	—	70–109	—	55–69	40–54	≤ 39
4	Respiratory rate (nonventilated or ventilated)	≥ 50	35–49	—	25–34	12–24	10–11	6–9	—	≤ 5
5	Oxygenation:									
	a) FIO_2 ≥ 0.5: use A-aDO_2	≥ 500	350–499	200–349	—	< 200	—	—	—	—
	b) FIO_2 < 0.5: use PAO_2 (mm Hg)	—	—	—	—	> 70	61–70	—	55–60	< 55
6	Arterial pH	≥ 7.7	7.6–7.69	—	7.5–7.59	7.33–7.49	—	7.25–7.32	7.15–7.24	< 7.15
7	Serum Na (mmol/L)	≥ 180	160–179	155–159	150–154	130–149	—	120–129	111–119	≤ 110
8	Serum K (mmol/L)	≥ 7	6–6.9	—	5.5–5.9	3.5–5.4	3–3.4	2.5–2.9	—	< 2.5
9	Serum creatinine (mg/dL); double point score for **acute** renal failure	≥ 3.5	2–3.4	1.5–1.9	—	0.6–1.4	—	< 0.6	—	—
10	Hct (%)	≥ 60	—	50–59.9	46–49.9	30–45.9	—	20–29.9	—	< 20
11	WBC (in 1000s)	≥ 40	—	20–39.9	15–19.9	3–14.9	—	1–2.9	—	< 1
12	Glasgow coma score (GCS)	Score = 15 minus actual GCS								

Acute physiology score is the sum of the 12 individual variable points.

Add 0 points for age <44; 2 points, 45–54 yr; 3 points, 55–64 yr; 5 points, 65–74 yr; 6 points ≥ 75 yr.

Add chronic health status points: 2 points if elective postoperative patient with immunocompromise or history of severe organ insufficiency; 5 points for nonoperative patient or emergency postoperative patient with immunocompromise or severe organ insufficiency.‡

(13)§	Serum HCO_3 (venous–mmol/L)	≥ 52	41–51.9	—	32–40.9	22–31.9	—	18–21.9	15–17.9	< 15

*APACHE II score = acute physiology score + age points + chronic health points. Minimum score = 0; maximum score = 71. Increasing score is associated with increasing risk of hospital death.

†Choose worst value in the past 24 h.

‡Chronic health status: Organ insufficiency (eg, hepatic, cardiovascular, renal, pulmonary) or immunocompromised state must have preceded current admission.

§Optional variable; use only if no ABGs.

A-a DO_2 = Alveolar–arterial oxygen gradient; FIO_2 = fractional inspired O_2.

INSTITUTIONAL ETHICAL COMMITTEE, STANLEY MEDICAL COLLEGE, CHENNAI-1

Title of the Work : Comparative study between APACHE-II and MPI scoring Systems in assessing the severity and final outcome in Cases of perforation peritonitis

Principal Investigator : Dr.S.B. Sudharsan

Designation : PG in M.S (Gen.Sur)

Department : Department of Gen.Sur
Government Stanley Medical College,
Chennai-1

The request for an approval from the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 06.03.2012 at the Council Hall, Stanley Medical College, Chen

.

nai-1 at 2PM

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

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

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

30/10/12

MEMBER SECRETARY,
IEC, SMC, CHENNAI

PROFORMA

GENERAL DATA

1. Patient name : 2. Age : 3. Gender :
4. Hospital number: 5. Address:
6. Date of admission:
7. Date of surgery:
8. Date of death/ discharge:

PATIENT DATA

- Clinical history
  - Duration
  - Nature of onset
  - Progression
- Basic laboratory investigations
  - Haematocrit:
  - Total leukocyte count:
  - Blood urea, creatinine:
  - Serum electrolytes:

OPERATION DATA

1. Operation date and time:
2. Antibiotic prophylaxis:
3. ASA class:
4. Anaesthesia:
5. Diffuse / generalized peritonitis/ Nature of exudates:
6. Origin of sepsis:
7. Presence of malignancy:

MANHEIM'S PERITONITIS INDEX SCORING

RISK FACTOR	**PATIENT SCORE**
Age > 50 years	
Female sex	
Organ failure*	
Malignancy	
Preoperative duration of peritonitis > 24 hrs	
Origin of sepsis not colonic	
Diffuse generalised peritonitis	
Exudate	
Clear	
Cloudy, purulent	
Fecal	

TOTAL SCORE =

ACUTE PHYSIOLOGY AND CHRONIC HEALTH EVALUATION II SCORE

PHYSIOLOGIC VARIABLES	**PATIENT'S SCORE**
Temperature (C)	
Mean arterial pressure(mm of Hg)	
Heart beat	
Respiration rate	
Oxygenation PaO_2 (mm Hg)	
Arterial Ph	
Serum HCO_3 (mmol/L) (used if ABG is not available)	
Serum Na (mmol/L)	
Serum K (mmol/L)	
Serum creatinine (mg/dL)	
Haematocrit %	
White blood count (total / mm^3)	
Serum urea (mmol /L)	

TOTAL SCORE =

FOLLOW UP

1. Need for ventilatory support? If yes, duration?
2. Development of organ failure?
3. Development of complications:
    - Wound infection
    - Wound dehiscence
    - Leaks / reperforations
    - Abscess/collections
    - Pulmonary complications
4. Date of discharge / death:

சுய ஒப்புதல் படிவம்

ஆய்வு செய்யப்படும் தலைப்பு

குடலில் ஓட்டை ஏற்படுவதால் வரும் பெரிடோனைடிஸ் நோயின் தீவரத்தையும் இறுதி வெளிப்பாட்டையும் முன்கூட்டி அறிவதற்கு அபாக்கி-2 மற்றும் மண்ஹீம்ஸ் மதிப்பீட்டு முறையின் ஒப்பீட்டாய்வு

ஆராய்ச்சி நிலையம் : அரசு ஸ்டான்லி மருத்துவமனை
சென்னை - 600 001.

பங்கு பெறும் நோயாளியின் பெயர் : வயது :

பங்கு பெறும் நோயாளியின் எண் : பாலினம் : ஆண் ☐ பெண் ☐

நோயாளியின் விலாசம் :

நோயாளி இதனை (✓) குறிக்கவும்.

மேலே குறிப்பிடப்பட்டுள்ள மருத்துவ ஆய்வின் விவரங்கள் எனக்கு விளக்கப்பட்டது. என்னுடைய சந்தேகங்களை கேட்கவும். அதற்கான தகுந்த விளக்கங்களை பெறவும் வாய்ப்பளிக்கப்பட்டது. ☐

நான் என்னை இவ்வாய்வில் தன்னிச்சையாகதான் பங்கேற்க அனுமதிக்கிறேன். எந்த காரணத்தினாலோ எந்த கட்டத்திலும் எந்த சட்ட சிக்கலுக்கும் உட்படாமல் என்னை இவ்வாய்வில் இருந்து விலக்கி கொள்ளலாம் என்றும் அறிந்து கொண்டேன். ☐

இந்த ஆய்வு சம்பந்தமாகவோ. இதை சார்ந்த மேலும் ஆய்வு மேற்கொள்ளும் போதும் இந்த ஆய்வில் பங்குபெறும் மருத்துவர் என்னுடைய மருத்துவ அறிக்கைகளை பார்ப்பதற்கு என் அனுமதி தேவையில்லை என அறிந்து கொள்கிறேன். என்னை ஆய்வில் இருந்து விலகிக் கொண்டாலும் இது பொருந்தும் என அறிகிறேன். ☐

இந்த ஆய்வின் மூலம் கிடைக்கும் தகவல்களையும், பரிசோதனை முடிவுகளையும் மற்றும் சிகிச்சை தொடர்பான தகவல்களையும் மருத்துவர் மேற்கொள்ளும் ஆய்வில் பயன்படுத்திக் கொள்ளவும் அதை பிரசுரிக்கவும் என் முழு மனதுடன் சம்மதிக்கிறேன். ☐

இந்த ஆய்வில் என்னை ஈடுபடுத்த முழுமனதுடன் ஒப்புக் கொள்கிறேன். இந்த மயக்க மருந்துகள் மற்றும் மயக்க முறையினால் ஏற்படக்கூடிய பின் விளைவுகள் மற்றும் எதிர்பாராத விளைவுகள் பற்றி எனக்கு விளக்கமாக தெரிவிக்கப்பட்டது. ☐

பெரிடோனைடிஸின் கடுமைத் தன்மையை அறிந்து கொள்ள மண்ஹீம்ஸ் பெரிடோனைடிஸ் எண்ணிக்கையும், அபாக்கி - இரண்டு எனப்படும் எண்ணிக்கையும் முதன்மையாக விளங்குகின்றன. இவை இரண்டில் நோயின் கடுமை மற்றும் உயிர்போக்கு தன்மையை எது துல்லியமாக கணிக்கிறது என்பதை அறிந்த கொள்வதே இந்த ஆய்வின் நோக்கம் என்பதை அறிவேன். இந்த ஆய்வில் எனக்கு செய்யப்பட்டும் சிகிச்சை மற்றும் பரிசோதனைகளுக்கு முழுமனதுடன் சம்மதிக்கிறேன். இந்த ஆய்வில் எந்தவித புதிய சிகிச்சை முறையும் பயன்படுத்தப்படவில்லை என்பதை மருத்துவர் விளக்கி கூறினார். ☐

நோயாளியின் கையொப்பம் ..... இடம் ..... தேதி

கட்டைவிரல் ரேகை (இந்த படிவம் படித்து காட்டப்பட்டு புரிந்து கைரேகை அளிக்கின்றேன்)

ஆய்வாளரின் கையொப்பம் ..... இடம் ..... தேதி

ஆய்வாளரின் பெயர் .....

நோயாளி தகவல் தாள்

குடலில் ஓட்டை ஏற்படுவதால் வரும் பெரிடோனைடிஸ் நோயின் தீவரத்தையும் இறுதி வெளிப்பாட்டையும் முன்கூட்டி அறிவதற்கு அபாக்கி-2 மற்றும் மண்ஹீம்ஸ் மதிப்பீட்டு முறையின் ஒப்பீட்டாய்வு

நோயாளிக்கான தகவல்கள் :
ஆராய்ச்சின் நோக்கமும், ஆதாரங்களும் :

தாங்கள் இந்த ஆய்வில் கலந்து கொள்ள அழைக்கப்படுகிறீர்கள் கலந்து கொள்வதற்கு முன் என்ன செய்யப்படுகிறது என்பதை அறிந்து கொள்ளுங்கள்.

பெரிடோனைடிஸ் என்றால் என்ன?

பெரிடோனைடிஸ் என்பது வயிற்றின் உட்பகுதியில் உள்ள சவ்வின் சுழற்சி.

பெரிடோனைடிஸ் ஏன் வருகிறது?

பெரிடோனைடிஸ் பெரும்பாலும் குடலில் ஏதாவது ஒரு பகுதியில் பொத்தல் ஏற்பட்டு அதன் பின்விளைவாக ஏற்படுகிறது. அன்றாட வாழ்க்கையில் அறுவை சிகிச்சை நிபுணர்களே பார்க்கக்கூடிய பிரச்சினையாக விளங்குகிறது.

பெரிடோனைடிஸிற்கு சிகிச்சை முறை யாவை?

பெரிடோனைடிஸ் ஏற்பட்டால் நமது உடலில் உள்ள மற்ற உறுப்புக்களின் சீரான செயல்பாட்டையும் குலைத்துவிடுகிறது. இதனால் உயிருக்கே ஆபத்து ஏற்படுகிறது. இத்தகைய கொடிய நோய்க்கு உடனடியாக அறுவை சிகிச்சை செய்வதே சிறந்த வைத்தியம் ஆகும்.

இந்த ஆய்வின் நோக்கம் என்ன?

பெரிடோனைடிஸின் கடுமைத் தன்மையை அறிந்து கொள்ள பல்வேறு ஆபத்து காரணிகள் உள்ளன. அவைகளில் மண்ஹீம்ஸ் பெரிடோனைடிஸ் எண்ணிக்கையும், அபாக்கி - இரண்டு எனப்படும் எண்ணிக்கையும் முதன்மையாக விளங்குகின்றன. இவை இரண்டில் நோயின் கடுமை மற்றும் உயிர்போக்கு தன்மையை எது துல்லியமாக கணிக்கிறது என்பதை அறிந்த கொள்வதே இந்த ஆய்வின் நோக்கம்.

இந்த ஆய்வின் பெறப்படும் தகவல்கள் யாவை?

இந்த ஆய்வில் தங்கள் நோய் வரலாறு, தங்கள் இதயத்துடிப்பு மற்றும் இரத்த அழுத்தம், அறுவை சிகிச்சையின் போது கண்டெடுக்கப்படும் தகவல்கள், அன்றாடம் செய்யப்படும் இரத்தப்பரிசோதனைகள் இவை அனைத்தும் தங்களிடமிருந்தோ தங்கள் உறவினர்களிடமிருந்தோ குறிப்பு எடுத்துக் கொள்ளப்படும்.

இதனால் தங்களுக்கு சிகிச்சை மாறுபடுகிறதா?

இந்த இரண்டு எண்ணிக்கை பட்டியல்கள் பொருத்துவதால் நோயாளிகளுக்கு எந்தவித பாதிப்போ, சிகிச்சை பெறுவதில் தாமதமோ, சிகிச்சை முறையில் மாறுதலோ ஏற்படாது.

இந்த ஆய்வில் பெறப்படும் தகவல்கள் அனைத்தும் மருத்துவ ஆராய்ச்சிக்கே பயன்படுத்தப்படும். தங்களுடைய பெயரோ, முகவரியோ எக்காரணத்திலும் வெளியிடப்படமாட்டாது. இந்த இரண்டு மதிப்பீட்டுகளில் எந்த மதிப்பீடு தங்களின் நோயின் தன்மையையும், தீவிரத்தையும், இறுதி வெளிப்பாட்டையும் துல்லியமாக கணிக்கிறது என்பது ஆய்வு செய்து கண்டறியப்படும்.

நாள் :

நோயாளி (அல்லது) உறவினர்
கையொப்பம் /
இடது பெருவிரல் ரேவை
(மருத்துவரால் படித்துகாட்டப்பட்டது)

EVALUATION OF PROGNOSTIC SCORING SYSTEMS IN PERFORATION PERITONITIS - COMPARITIVE STUDY BETWEEN APACHE II AND MPI SCORING SYSTEMS

1. I confirm that I have read and understood the information sheet for the above study.
2. I understand that my participation is voluntary and can withdraw at any time.
3. I agree to take part in the above study.
4. I, _____, being a person of sound mind, hereby give my permission to use a photograph(s) of me taken en face or intra operatively in the dissertation titled " *Evaluation of prognostic scoring systems in perforation peritonitis- comparative study between APACHE II and Manheim's peritonitis index scoring systems"*.

I declare, in consequence of granting this permission, that I have no claim on ground of breach of confidence or on any ground in any legal system against the researcher in respect of the publication of the photograph(s) and also the data collected during the process of the study.

Name of the patient	Date	Signature of the patient or blood relative
Name of the person taking consent (if different from researcher)	Date	Signature
Researcher	Date	Signature

When completed, 1 for patient, 1 for researcher site file, 1 (original) to be kept in medical notes

PATIENT INFORMATION SHEET

EVALUATION OF PROGNOSTIC SCORING SYSTEMS IN PERFORATION PERITONITIS - COMPARATIVE STUDY BETWEEN APACHE II AND MANNHEIM'S INDEX SCORING SYSTEMS

You are being invited to take part in a research study. Before you decide, it is important why the research is being done and what it will involve. Please take time to read the following information carefully. This leaflet informs you about hollow viscus perforation peritonitis, the mortality for which has been high. Taking part in this study is entirely voluntary. It is up to you to decide whether or not to take part.

There will be no monetary benefits for participating in the study.

What is peritonitis?

Peritonitis is the inflammation of the peritoneum,the thin tissue that lines the inner wall of the abdomen and covers most of the abdominal organs.

What are its causes?

Peritonitis occurs most often as a result of a perforation of any part of the gastro-intestinal tract. It is a common condition which a surgeon encounters on a day to day basis.

What are the treatment options?

Peritonitis has the potential to cause multiple organ dysfunctions and can endanger the life of the diseased. Hence it should be treated with urgent laparotomy after appropriate resuscitative measures.

What are the objectives of this study?

To assess the severity of peritonitis, there are various scoring systems. Among them, Mannheim's peritonitis index (MPI) and Acute Physiology and Chronic Health Evaluation (APACHE II) are very useful. The purpose of this study is to compare the two scoring systems and determine which of the two systems predicts the mortality accurately.

What information will be obtained?

In this research details regarding the nature and duration of your symptoms such as abdomen pain will be obtained only from you or your accompanying blood relative. Vital parameters such as heart rate, respiratory rate, temperature and blood pressure will be recorded at the time of hospital admission by the duty doctors. Blood samples will be drawn and necessary

basic investigations will be performed. Trained technicians will be used to withdraw blood samples. The timing of surgery will be decided by the duty assistant surgeon on duty for the day who will be performing the surgery. All the intra operative findings will be recorded precisely.

Will this research study alter your treatment?

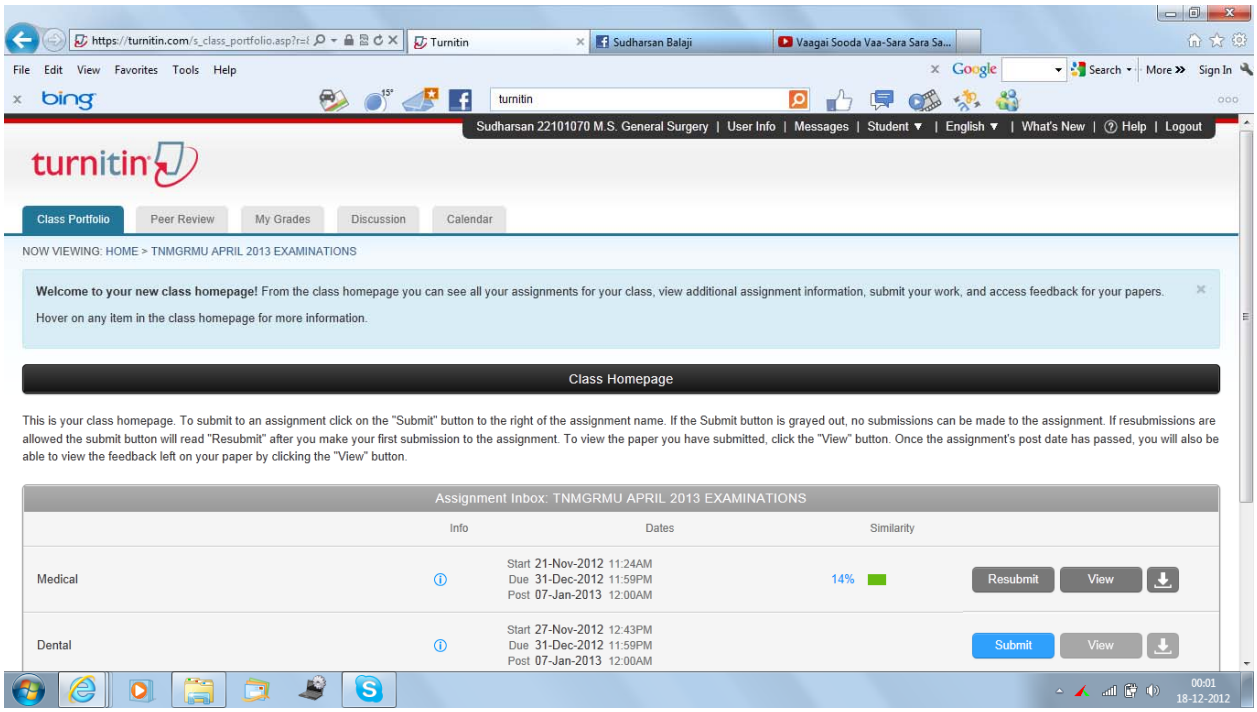
The two scoring systems are used only to predict the severity of the illness. This does not form a basis for any delay or change in the treatment offered to you. Following the operation, thorough post operative care will be given and your well being and complications, if any, will be recorded.

All the information obtained about you will be compiled and used solely for research purposes. Your identity will not be revealed on any circumstances. If the data are sent out of the hospital, your name and address will be untagged from it so that you cannot be identified out of it.

The ultimate outcome of the disease will be found out in each case and be compared with the pre- operative score. In doing so, which of the two scoring systems predicts the outcome better can be found.

Thank you for taking part in this study.

TURNITIN – PLAGIARISM CHECK TOOL



Name	IP no	MANHEIM'S PERITONITIS INDEX PARAMETERS									APACHE II PARAMETERS												
		Age	sex	rgan failur	malignancy	duration	origin	nature	exudate	SCORE	Temp	MAP	heartrate	resp rate	oxyg	HCO3	Sodium	potassium	Creatinine	hematocrit	WBC	GCS	AGE SCORE	SCORE
Hamanullah	12918	43	male	nil	no	< 24 hrs	gastric	diffuse	clear	10	38.5	102	102	15	70	24	136	3.8	1.2	42	11000	15	0	1
Bavani	13084	38	female	yes	no	5 days	appendicular	diffuse	purulent	26	38.5	100	108	18	70	22	140	3.9	2.1	38	13000	15	0	4
Gnanamuthu	20764	62	male	nil	no	3 days	ileal	diffuse	purulent	25	39	100	110	16	70	26	138	3.5	1.6	40	11000	15	3	10
Hemanthkumar	25730	21	male	nil	no	2 days	appendicular	localised	purulent	10	39	100	105	15	70	24	138	3.7	0.9	38	9000	15	0	3
Devendiran	27744	52	male	nil	no	< 24 hrs	gastric	diffuse	clear	15	37.4	98	110	18	70	26	134	3.4	1	32	9000	15	2	4
Senguttuvan	28713	38	male	yes	no	3 days	duodenal	diffuse	purulent	**32**	41	102	115	18	70	20	129	2.9	2.1	44	15000	15	0	**16**
Kumaresan	28317	53	male	nil	no	< 24 hrs	ileal	diffuse	clear	15	38.4	103	110	18	70	23	134	3.3	1.4	45	9000	15	2	5
Soosai	27170	55	male	nil	no	< 24 hrs	duodenal	localised	clear	4	38.5	100	100	18	70	24	131	3.5	1.1	39	8000	15	3	4
Munusamy	28727	55	male	yes	no	2 days	gastric	diffuse	purulent	**32**	39.1	98	120	30	70	20	131	2.4	3.1	42	11000	15	3	**18**
Gopal	29690	30	male	nil	no	5 days	appendicular	localised	purulent	10	38	95	100	16	70	22	131	3.8	0.8	38	11000	15	0	0
Arjunan	29958	56	male	nil	yes	< 24 hrs	gastric	diffuse	clear	19	38	156	100	22	80	24	147	3.7	0.9	55	7800	15	3	11
Boopathy	27041	50	male	yes	no	< 24 hrs	duodenal	diffuse	purulent	19	39	90	110	25	75	22	136	3.8	2	33	5300	15	2	11
Kumari	28910	42	female	nil	no	4 days	appendicular	diffuse	purulent	21	39.5	100	100	18	80	20	134	3.5	0.9	33	8900	15	0	5
Chandrakanth	30365	20	male	nil	no	< 24 hrs	duodenal	diffuse	clear	10	38	102	110	18	70	24	135	3.4	1.3	44	10300	15	0	3
Prabhu	29008	25	male	nil	no	1 day	duodenal	diffuse	clear	14	38	102	120	20	70	23	130	3.3	1.4	40	8900	15	0	3
Govindasamy	31840	51	male	nil	no	3 days	gastric	diffuse	purulent	25	38.6	85	96	16	70	21	133	3.5	1.2	37	3400	15	2	5
Chelladurai	32026	50	male	yes	no	2 days	colonic	diffuse	purulent	24	39	105	120	25	85	14	135	3.6	1.6	44	4000	15	2	14
Murugesan	32616	55	male	nil	no	3 days	duodenal	localised	clear	13	37.2	105	110	20	80	24	128	3.1	1	39	6400	15	3	8
Syed	32028	23	male	nil	no	< 24 hrs	colonic	diffuse	bloody	0	37.5	70	130	30	80	25	134	3.8	0.8	27	10800	15	0	5
Pandian	33954	39	male	nil	no	> 24 hrs	gastric	diffuse	clear	14	39.1	100	102	18	70	24	131	3.5	1.5	42	8900	15	0	6
Chakrapani	23698	68	male	yes	yes	2 days	colonic	diffuse	faeculent	**38**	36.5	92	115	20	75	18	128	2.9	2.4	30	9000	15	5	15
Krishnamma	35689	30	male	nil	no	> 24 hrs	ileal	diffuse	purulent	25	37	96	120	12	70	20	130	3.1	1.4	32	8800	15	0	8
Lakshmi	38790	50	female	nil	yes	2 days	colonic	diffuse	faeculent	31	38.5	140	115	18	75	20	134	3.5	1.6	32	13000	15	2	12
Marimuthu	29767	54	male	yes	yes	< 24 hrs	jejunal	localised	clear	20	38	110	130	22	75	20	129	2.9	2.3	46	7900	13	2	**20**
Sensiraman	35679	47	male	nil	no	2 days	gastric	diffuse	clear	14	38.5	100	104	14	70	25	142	3.5	1.2	40	8900	15	2	3
Bhuvaneswari	39602	38	female	nil	no	4 days	appendicular	localised	purulent	15	38.8	102	100	18	70	25	134	3.5	1.2	38	10800	15	0	1
Arumugam	39717	58	male	nil	no	< 24 hrs	duodenal	diffuse	clear	11	38.5	100	105	20	70	22	135	3.4	1.2	38	8800	15	3	5
Purushothaman	38697	20	male	nil	no	2 days	duodenal	diffuse	clear	14	38	88	96	14	70	24	135	3.5	0.8	32	10400	15	0	0
Kala	39258	38	female	nil	no	> 24 hrs	appendicular	localised	purulent	15	38.5	100	104	18	70	24	132	3.3	0.8	33	10300	15	0	2
Sasikumar	40025	49	male	yes	no	5 days	duodenal	diffuse	purulent	25	41	105	110	20	70	21	129	2.9	1.8	46	15000	15	2	**18**
Lakshmi	41079	60	female	yes	yes	3 days	colonic	diffuse	purulent	**37**	40	68	130	25	65	20	130	2.9	2.2	30	8900	13	3	**21**
kaliappan	43771	43	male	nil	no	< 24 hrs	duodenal	diffuse	clear	10	38.5	100	110	18	70	24	132	3.9	1.3	32	13000	15	0	3
annamalai	43569	25	male	nil	no	4 days	appendicular	localised	purulent	10	39	100	110	16	80	26	138	3.2	1.1	32	11000	15	0	3
Rose	48872	43	male	yes	no	6 d	appendicular	diffuse	purulent	23	39	90	120	25	75	21	129	2.8	3.1	44	15000	15	0	**16**
Gangadaran	50791	31	male	nil	no	4 days	ileal	diffuse	purulent	20	40	95	110	20	75	24	134	3.3	1.1	39	11600	15	0	6
Hanumanaiah	52395	64	male	yes	yes	1 day	gastric	diffuse	clear	26	38.5	110	120	24	75	21	135	3.4	1.8	30	11000	15	3	13
Abdul rahim	53271	26	male	nil	no	3 days	ileal	diffuse	purulent	20	40	95	110	21	75	21	133	3.4	1.2	34	8900	15	0	8
Satyavathi	54178	38	female	nil	no	< 24 hrs	duodenal	diffuse	clear	15	38.5	90	110	18	75	22	129	3.1	1.2	29	8900	15	0	8
Siluvai	54767	52	male	yes	yes	1 day	colonic	localised	purulent	**22**	37	65	140	30	75	22	130	2.7	2.1	30	9800	13	2	**18**
chellathayee	58714	46	female	nil	no	2 days	gall bladder	localised	purulent	19	40	90	104	18	75	24	135	2.9	1.3	31	11000	15	2	7
Pooshanam	53196	48	male	nil	no	2 days	duodenal	diffuse	clear	14	38.5	90	100	20	75	24	135	3.4	1.3	34	11000	15	2	4
anjaneyalu	54511	55	male	nil	no	< 24 hrs	duodenal	localised	clear	9	38	95	100	16	75	23	138	3.2	1.2	33	8500	15	3	4
saraswati	55082	68	female	yes	no	3 days	gastric	diffuse	purulent	**37**	39	110	115	20	80	21	129	2.8	2	29	10500	15	5	21
Satish	55865	28	male	nil	no	2 days	duodenal	diffuse	cloudy	20	38.5	100	105	18	80	26	131	3.3	1.3	46	15000	15	0	3
Saranya	56997	21	female	nil	no	6 days	appendicular	localised	purulent	15	38	100	110	16	80	21	136	3.6	1.2	33	12000	15	0	5
Lakshmiamma	56653	52	female	nil	no	2 days	duodenal	diffuse	cloudy	30	38.5	110	105	16	80	22	134	3.3	1.3	34	8900	15	2	5
Abdul shafiq	57019	34	male	nil	no	< 24 hrs	jejunal	diffuse	bilious	10	38	80	125	18	80	26	136	3.5	1.4	31	8900	15	0	2
sakthiseelan	57083	38	male	nil	no	3 days	duodenal	diffuse	purulent	20	38	100	130	16	75	22	131	3.3	1.2	33	8800	15	0	4
Mercy	57904	24	female	nil	no	4 days	appendicular	localised	purulent	19	38.5	95	110	14	75	24	134	3.5	1	31	13400	15	0	3
Ilayaraja	59903	29	male	nil	no	2 days	duodenal	diffuse	clear	14	38.5	100	100	16	75	24	135	3.4	1.2	36	13900	15	0	2
		2135								936														