"A COMPARATIVE STUDY BETWEEN POVIDINE IODINE AND METRONIDAZOLE FOR PERITONEAL LAVAGE IN CASES OF PERITONITIS"

By

Dr. ASADULLA BAIG

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MASTER OF SURGERY
IN
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Under the guidance of

Prof. MOHAN KUMAR.K M.S.

DEPARTMENT OF GENERAL SURGERY
SRI DEVARAJ URS MEDICAL COLLEGE,
TAMAKA, KOLAR
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Date:

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Dr. ASADULLA BAIG

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Date:
Place: **Kolar**

Signature of the Guide
DR. MOHAN KUMAR.K. M.S.,
PROFESSOR
DEPARTMENT OF GENERAL SURGERY
SRI DEVARAJ URS MEDICAL COLLEGE
TAMAKA, KOLAR

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH CENTRE, TAMAKA, KOLAR

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Date:
Place: **Kolar**

Signature of the Co- Guide
DR. S.R.Prasad, M.D
PROFESSOR
DEPARTMENT OF MICROBIOLOGY
SRI DEVARAJ URS MEDICAL COLLEGE
TAMAKA, KOLAR

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH CENTRE, TAMAKA, KOLAR

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Date:
Place: **Kolar**

Signature of the HOD
Dr. Bhaskaran. A, M.S
PROFESSOR AND HOD
DEPARTMENT OF GENERALSURGERY
SRI DEVARAJ URS MEDICAL COLLEGE,
TAMAKA, KOLAR

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH CENTRE, TAMAKA, KOLAR

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Dr. A. Bhaskaran
Professor and HOD

Dr. M.B.Sanikop
Principal

Date:

Place: S D U M C Kolar

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Date: Signature of the Candidate

Place: Kolar. Dr. Asadulla Baig

ABSTRACT

Background;

Peritonitis secondary to hollow viscus perforation is one of the commonest emergency encountered in General surgical practice. The rate of secondary infection is higher as majority of patients being from rural areas, present late to the hospital due to low awareness, local beliefs and faith in native medicine. This study aims to compare the efficacy of povidine iodine and metronidazole in peritoneal lavage and to ensure adequate control of infection and to decrease the chances of post operative wound infection thereby preventing prolonged hospital stay.

Objectives;

To evaluate the frequency of post operative wound infection in patients with hollow viscus perforation who have received peritoneal lavage with Povidine iodine / metronidazole intra-operatively.

To compare the efficacy of Povidine iodine and metronidazole in case of peritonitis secondary to hollow viscus perforation by quantitative analysis of bacterial colony count in patients who have received peritoneal lavage with the same.

Methods;

The material for the study consisted of 100 patients who presented to Department of General surgery of R. L. J. H., KOLAR, between FEB-2010 – FEB-2011 with features of peritonitis secondary to hollow viscus perforation. This was a randomized study; the patients were divided in two groups.

GROUP 1: Patients with all odd serial numbers were included in this group and given peritoneal lavage using Povidine iodine (**1%** wt/volume of Povidine iodine in 2 liter of normal saline).

GROUP 2: Patients with all even serial numbers were included in this group and given peritoneal lavage using metronidazole (**200** ml of metronidazole in 2 liter of normal saline).

Pre-operative history of all cases has been taken to arrive at a diagnosis. In all the cases, operative findings and post-operative diagnosis were recorded.

Results;

The difference in bacterial colony count was studied using povidine iodine and metronidazole both pre and post peritoneal lavage in cases of peritonitis which were proved surgically. There was decrease in bacterial colony count with the use of both the agents.

The p- value was significant in cases who received povidine iodine < 0.05 (0.007).

There were 4 deaths, all of them who had severe form of peritonitis and delayed presentation to the hospital. This study also revealed that men are commonly affected and duodenal ulcer perforation is the commonest site of perforation.

E.coli is the most common organism isolated.

Conclusion;

In our study Peritonitis is more common in men compared to women. The common age group is in between 21 – 40 in cases of peritonitis with mean age of 33years .Duodenal Ulcer Perforation is the commonest site of perforation. E.coli is the commonest organism isolated from the peritoneal contamination. Povidine iodine lavage (< 0.05) significantly decreases the bacterial load when compared to metronidazole lavage. As per as clinical outcome is concerned there is no significant differences in both the groups.

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INTRODUCTION

Peritonitis secondary to hollow viscus perforation is one of the commonest emergency encountered in General surgical practice. The rate of secondary infection is higher as majority of patients being from rural areas, present late to the hospital due to low awareness, local beliefs and faith in native medicine. This study aims to compare the efficacy of povidine iodine and metronidazole in peritoneal lavage and to ensure adequate control of infection and to decrease the chances of post operative wound infection thereby preventing prolonged hospital stay.[1s]

Peritonitis has been a well known entity from the days of Hippocrates i.e., 400 BC. He described Hippocrates facies, which is also well known. He said coldness of extremities in peritonitis is a very bad sign, which is true even today. Since then the pendulum has swung between the non-operative management to the most aggressive approach. In spite of the best ICU care facilities, sepsis remains a clinical challenge to the modern day surgeon. Severe diffuse peritonitis is still a "Giant Killer".

Goldstein cites description of early gastric ulcer in 1581. Rawlison in the year 1727 was the first to give a clear description of the symptoms and signs of gastric ulcer and peritonitis. Later on Smith, Travers and Elliston in the early 19th century gave a clear description of peritonitis independently.[2] With the advent of general anesthesia, and the advent of asepsis and antisepsis techniques, laparotomy as a part of management of peritonitis was gradually established. In the 19th century, treatment of peritonitis consisted of absolute rest, purgation, abstinence from food intake, application of cold to the abdomen, opium administration, etc.

Treves in 1885 stressed the importance of drainage of infected area mainly in relation to appendicitis, then known as perityphilitis.

Irrigation of the peritoneum with water was also followed in the late 19th century. Some workers condemned irrigation and excessive manipulation in the early part of 20th century and insisted on gentle handling of inflamed viscera. Murphy was the first to attempt counteraction of the fluid loss in peritonitis, and suggested rectal route for administration of fluids.

The use of peritoneal irrigation was introduced by Dr. Pierce in 1905. The purpose of this study was to device a relatively low - cost irrigant that could be used for peritoneal lavage, following surgical treatment for generalized bacterial peritonitis secondary to hollow viscus perforation.[3]Recent studies indicate management of diffuse peritonitis by prolonged post operative peritoneal lavage may enhance confidence in the treatment of intraperitoneal abscess ,and it enhances a quick recovery.[4]

Perforation of an intra-abdominal hollow organ with spillage of contents into the peritoneal cavity always leads to severe pain, shock, sepsis, and a high risk of death. However in posterior perforation, the features are less dramatic.[5]

Hoffmann in 1988 suggested peritoneal lavage as an aid in the diagnosis of peritonitis of non-traumatic origin.[6] The pathogenesis of peritonitis due to hollow viscus perforation is currently accepted as being mainly based on the local as well as the systemic release of pro and anti inflammatory mediators triggered by the presence of bacteria and bacterial products in the abdominal cavity. Therefore, treatment consists of focal restoration, intraoperative debridement and lavage. In order to evaluate the pathophysiological relevance of the bacterial load of the peritoneal exudate, the peritoneal fluid from patients presenting with peritonitis secondary to hollow viscus perforation will be obtained for bacterial colony count prior to as well as after peritoneal lavage.

Simple closure of perforation, with a thorough peritoneal wash has been the important step in managing peritonitis, which is practiced now-a-days. In cases of small intestinal perforation, resection and anastamosis can be performed.

OBJECTIVES OF STUDY

To evaluate the frequency of post operative wound infection in patients with hollow viscus perforation who have received peritoneal lavage with Povidine iodine / metronidazole intra-operatively.

To compare the efficacy of Povidine iodine and metronidazole in case of peritonitis secondary to hollow viscus perforation by quantitative analysis of bacterial colony count in patients who have received peritoneal lavage with the same.

REVIEW OF LITERATURE

Nearly all surgical intervention into the abdominal cavity had ended up with peritonitis and death till the 19th century. Introduction of antiseptic surgery by Lister led to a dramatic decline in the mortality following surgical exploration of the peritoneal cavity.[7]

An experimental study in rats conducted that survival in established peritonitis depends on the adequate antibiotic therapy as well topical antibiotic peritoneal lavage.[1]

Another study recommened that peritoneal lavage to be an effective, safe and simple alternative to the surgical treatment of generalized peritonitis wherein peritoneal lavage was combined intraoperatively with debridement of any peritoneal or any viscus exudates and there was post operative intraperitoneal sepsis in 4 patients out of 722 patients who eventually underwent reoperation.[8]

Recent advances in the field of research have shown that the peritoneal mesothelial cells have a remarkable capacity to respond to peritoneal insult. They generate an intense biological response and play an important role in the formation of adhesion. Yao V and associates studied this mesothelial cascading process that has evolved to protect life in the absence of surgery. They suggested a modification in the activity of the mesothelial cells by molecular strategies, could help in more advancement in managing peritonitis.[9] Researchers have also shown that the mesothelial cell is a critical component of the peritoneal membrane. The intraperitoneal use of several antibiotics, which is been done nowadays, can lead to apoptosis of the peritoneal mesothelial cells.[10]

Prolonged antibiotic peritoneal lavage appears to be beneficial for patients with gross and diffuse peritoneal soiling after the causative lesion has been repaired. [11]

Primary Peritonitis;

Inflammation of the peritoneum from suspected extraperitoneal source often via Haematogenous spread constitute primary peritonitis.[12] Spontaneous peritonitis occurs in children and in adults. Children with Nephrotic syndrome and S.L.E. are affected disproportionately.[13] Spontaneous peritonitis in adults is commonly seen in patients with ascitis and is monomicrobial infection.[14]

Route of Spread:

The route of spread of organisms into peritoneal cavity in primary peritonitis is unclear but the mechanisms are haematogenous from distant sites supported in studies by Gross (1953); Extension from female genital tract supported by Reports of Benson and Weinstokin (1940). The hypothesis of Transmural Migration of bacteria from the intestine is supported by the presence of endotoxin in ascitic fluid and blood in cirrhotic patients.[15]

Organisms:

Haemolytic Streptococci and Penumococci were the commonest pathogenic organisms reported. In 2/3 of 158 patients reported by Gross in 1953 the cause was Haemolytic streptococci while in 2/3 of patients reported by Fowler in 1957 the cause was Penumococci, similar findings were seen by Friedrich.[16] Acute suppurative diffuse peritonitis was treated by early drainage of peritoneal cavity and supportive treatment.[17]

Secondary Peritonitis;

The earliest recognized cases of secondary peritonitis were those of perforated appendix and perforated peptic ulcer. According to Lister, earliest case of acute perforation of an ulcer producing peritonitis was recognized in 1070.

In 1727, Rawlinson gave accurate descriptions of peritonitis following perforated gastric ulcer. Smith in 1790, Treves in 1870 and Ellison in 1827 described accurately most of signs and symptoms of peritonitis following perforated peptic ulcer.

By 19th Century, clinical manifestations of peritonitis following perforated peptic ulcer was well known to medical man. A detailed description of the clinical features of the disease can be seen in Cruvelteir's published in 1983.

Peritonitis following perforation of colon has been recognized since the time of Hippocrates. Appendicular perforation was first described by Jeen Fernal in 1554.

Intraperitoneal infections in secondary peritonitis are covered by members of GIT Flora viz. E coli, Enterococci, Klebsiella, Enterobacter, Proteus, Bacteroids.[18]The organisms are directly absorbed into the lymphatic via the stoma of the diaphragmatic peritoneum which is increased during post inflammatory period.[19] Blockade of their trans diaphragmatic absorption of bacteria increases survival.[20] Bile , Gastric juice, blood and necrotic tissue play an adjuvant role in the spread of peritonitis. Fibrin is responsible for the inhibition of phagocytic killing by neutrophils.[21] The organisms in peritonitis are locally destroyed through phagocytosis by resident macrophages or polymorphonuclear granulocytes attracted to the peritoneal cavity.[22] The Gram negative aerobic bacteria exert their potential mainly through endotoxin acting via mediators causing systemic and local septic response while anaerobic bacterial exert their potential through exotoxin. [23]

Investigations:

Laboratory findings in secondary peritonitis due to acute perforated gastro duodenal ulcers were reviewed by Kozoll and Meyer who demonstrated electrolyte imbalances.[24]Radiological investigations to determine the spread of intraperitoneal infections was done by Autio V. with Roentgen contrast medium.[25] Ultrasonographic diagnosis of post operative intra abdominal abscess was studied by Maklad N.G. and colleagues. Bhansali studied 96 cases of peritonitis and reported that X-ray evidence of pneumo peritoneum was seen in 92% of peptic ulcer perforations. 74% of typhoid perforations and 43% of tuberculosis ulcer perforations. The role of CT in confirming the diagnosis of intra abdominal infection after initial laparotomy is high as compared to ultrasonography,[26] and it is not limited by overlying bowel gas.[27] The sensitivity and specificity of abdominal CT for diagnosing an intra abdominal focus of infection was 97.5% and 61.5% respectively. [28]

Treatment:

In the early 19th century treatment of peritonitis was considered to be absolute bed rest, abstinence from food, various types of enemas including irritable vegetable juices and application of cold to the abdomen. In 1823 Dupetryn condemned a direct surgical approach on peritonitis. With the advent of general anaesthesia in 1846 and following the teaching of Pasteur and Lister the place of laparotomy and cleaning the peritoneal cavity in the management of peritonitis was considered.

Trait in 1890 advocated irrigation with tap water and post operative enemas.

Mickulicz with a few German surgeons successfully located and sutured a perforated gastric ulcer at laparotomy but the patient did not survive following this. Early closure and peritoneal lavage were attempted and published by Simon and Barling in 1982, Gilford in 1893 Anson in 1894.

Boward and Dickson in the same year, Burnet and Page in 1894 did laparotomy and peritoneal lavage in an early case of peptic ulcer perforation and the patient survived. Following this, early surgical intervention was recognized as the treatment of choice. The prognosis of late cases continued to be fatal till in 1909 when Bound condemned excessive irrigation and rough handling of viscera and enemas. He also advocated the necessity for the correction of dehydration.

A.D. Lembert in 1826 did a closure of a small intestinal perforation by suturing all layers excluding the mucosa. Mickulicz was the first to treat typhoid ulcer perforation and peritonitis by suture in 1884. It was Oschner in 1934 who put forward clear visions in the treatment of perforation peritonitis which is followed today without much changes.

Use of Antimicrobials:

Antibiotic trials in peritonitis have revealed that despite marked differences in antibacterial spectra; substantial differences in treatment results have not been documented. Overall success rate was 84% for Aminoglycoside + Clindamycin, 89% for aminoglycoside + Metronidazole and 93% for cephalosporin regimens.[29]

A surgical infection society policy statement on anti infective agents for intra abdominal infections was provided by Bohnen J. M.A. and Colleagues.[30] Lau W.Y. and colleagues did a randomized prospective trial on prophylaxis of post appendicectomy sepsis and concluded the combination of Metronidazole and Cefotaxime to be superior.[31]

Tertiary Peritonitis;

It can occur after any surgery for secondary bacterial peritonitis. Risk factors include malnutrition presence of organisms resistant to antimicrobials and organ system failure. Early recognition and effective intervention are critical to achieve success.[32]

Role of Laparoscopy:

Laparoscopy is nowadays suggested to be feasible and safe in peritonitis. It is found to be effective in appendicular and gastroduodenal perforation. But experiences with colonic perforation are at present limited, the conversion rates remaining still on higher side.[33]In a study conducted in PGI Chandigarh over a period of one year, on patients with non-traumatic perforation and peritonitis, post laparotomy group underwent feeding jejunostomy and received enteral feeding from 12 hours post operative.

AIDS Patient:

Early diagnosis and prompt intervention is essential. Morbidity and mortality during surgical procedures depends on stage of HIV disease. Surgical interventions should not be denied because of risk of occupational transmission .Earlier studies showed high mortality and morbidity more recent studies contradict these conclusions.[34] Factors increasing mortality and morbidity are stage of disease; presence of opportunistic infection; presence of concurrent organ failure.[35]

ANATOMY OF PERITONEAL CAVITY

Embryology of Peritoneal Cavity:

Peritoneal cavity is derived from the two limbs of the horseshoe shaped intraembryonic coelom. The two parts are first separated but fuse to form one cavity, as a result of lateral folding of the embryonic disc.

The splanchnic mesoderm then encloses the primitive gut. This layer of mesoderm will form the lining of the peritoneal cavity, the peritoneum. Double layers of peritoneum form mesentries and ligament which connect the viscera to the body wall and to other viscera. Organs covered by peritoneum and suspended in the abdominal cavity are intraperitoneal, those attached to the posterior body wall and covered by peritoneum on their anterior surface only are retroperitoneal.[36]

The peritoneal cavity, therefore, comes to be subdivided into a number of pockets that are partially separated by folds of peritoneum.[37]

Surgical Anatomy: [38]

The peritoneal cavity is divided by the shape of its posterior wall into upper abdominal and lower pelvic portions. The abdominal part is sub divided into an upper and lower compartment by the transverse colon and mesocolon. The supracolic space is between the diaphragm and transverse colon and mesocolon. In this space lies the stomach, liver, gall bladder, spleen and first part of duodenum. This space is again divided by the liver and its ligaments into subhepatic and subsplenic spaces.

The infracolic space is divided into right and left infracolic spaces by the mesentery of the small intestine. The right infracolic space lies below the transverse colon and to the right of the mesentery of the small intestine. The ascending colon divides this space into external and internal paracolic gutters. Left infracolic space lies

below the transverse colon and to the left of the mesentery of the small intestine. The descending colon divides this space into external and internal paracolic gutters.

The pelvic cavity is subdivided by the pelvic mesocolon into secondary pouches which differ in male and female. In the female the free ends of the uterine tubes ones into the peritoneal cavity.

The complicated arrangement of the peritoneum results in the formation of four intraperitoneal and three extraperitoneal spaces, in which pus may collect.

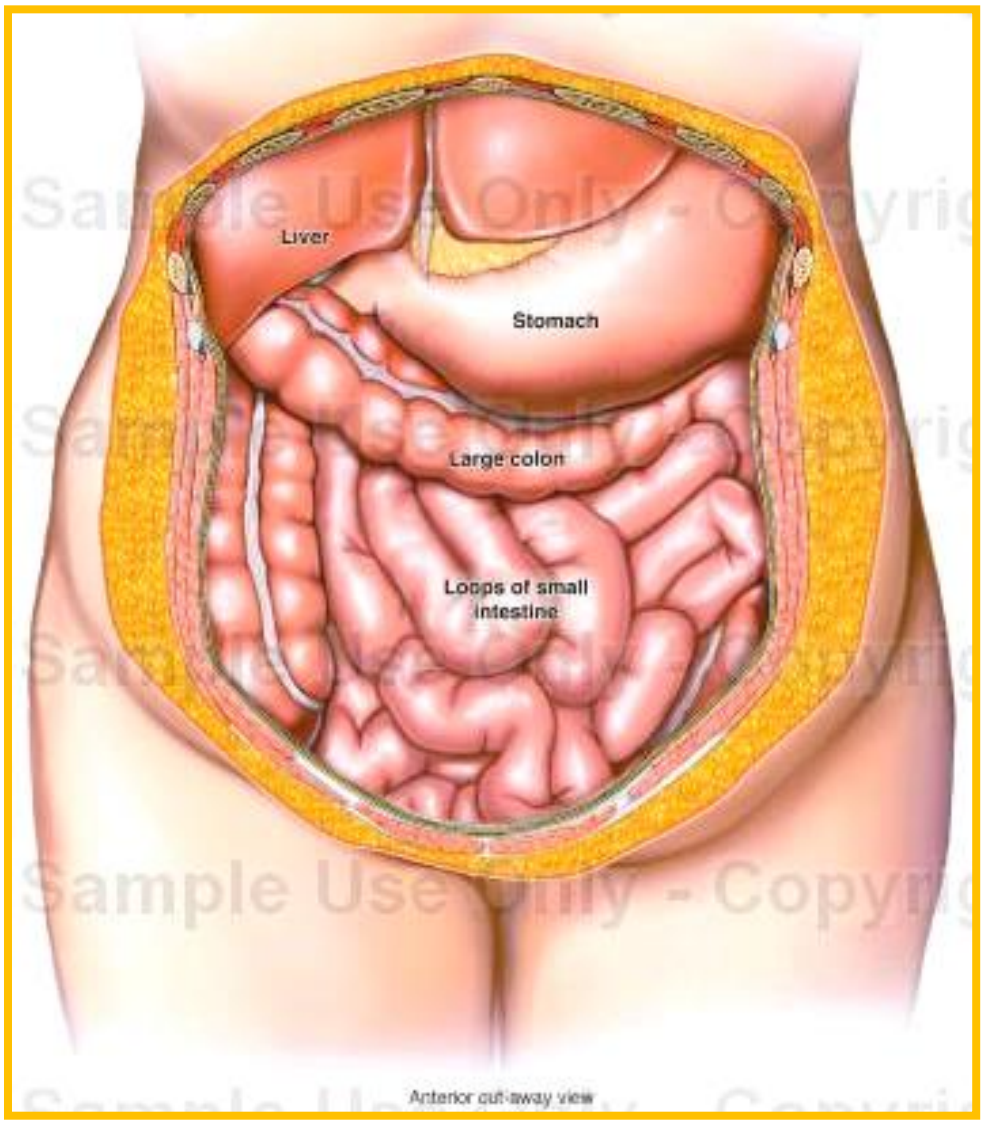


Fig 1 .Anatomy of abdominal cavity

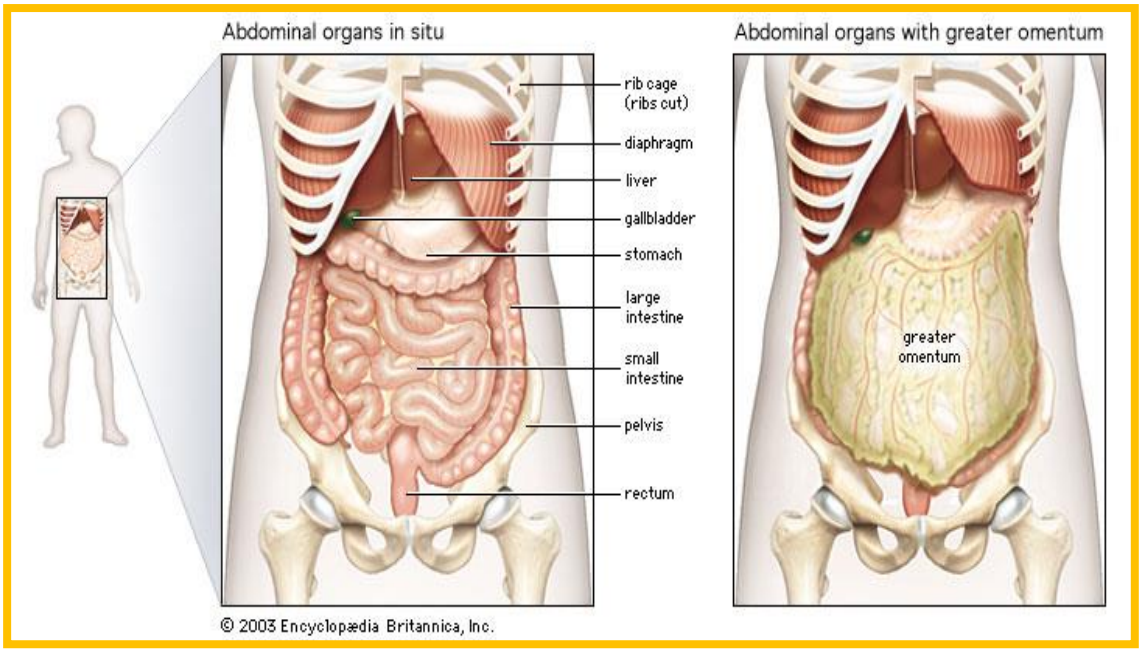


Fig 2.Abdominal organs

Intraperitoneal Spaces;

1. Left anterior Intraperitoneal Space:

Bounded above the diaphragm behind by the left triangular ligament and left lobe of the liver, the gastrohepatic omentum and anterior surface of the stomach, to the right by falciform ligament and to the left by the spleen, gastrosplenic omentum and diaphragm.

2. Left posterior intraperitoneal space (Lesser sac) Boundaries:

a) Anterior wall: Stomach, Lesser omentum, anterior two layers of the greater omentum, caudate lobe of the liver.

b) Posterior wall: Structures forming the stomach bed (diaphragm, left supra renal gland, left kidney, splenic artery, pancreas, transverse mesocolon, splenic flexure of the colon), posterior two layers of the greater omentum.

On the right side lesser sac communicates with the greater sac through the epiploic foramen and left side is bounded by gastrophrenic gastrosplenic and linorenal ligament.

3. Right anterior intraperitoneal space:

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

4. Right posterior intraperitoneal space (Rutherford Morrison's Kidney pouch):

It lies transversely beneath the right lobe of the fiver.

Boundaries:

Anteriorly: Inferior surface of the right lobe of the liver and gall bladder.

Posteriorly: Inferior layer of the coronary ligament, and

Inferiorly: Opens into the general peritoneal cavity.

It is the deepest space of the four and the commonest site of subphrenic abscess, which usually arises from appendicitis, cholecystitis, perforated duodenal ulcer or following upper abdominal surgery.

Extra Peritoneal Spaces:

Mid line extraperitoneal space (Bare area of liver): lies between superior and inferior layer of the coronary ligament, where liver is in direct contact with diaphragm. This is the commonest site for amoebic liver abscess.

Right and Left extra peritoneal spaces:

Lie around the right and left supra renal glands and upper poles of the kidneys. These are the sites for the perinephric abscess.

Peritoneal Fluid:

The peritoneum contains normally about 100 ml of fluid. The fluid layer which covers the peritoneal surfaces contains water, electrolytes and other solutes derived

from the intestinal fluid of neighbouring tissue and from the plasma of adjacent blood vessels.

It also contains proteins and variety of cell types. Normally the cells consist of desquamated fat mesothelial elements derived from the peritoneal surfaces and of wandering macrophages, mast cells, fibroblasts, lymphocytes and small number of other leucocytes. Lymphocytes in the fluid provide both cellular and humoral immunological defence mechanisms.

Classification of Peritonitis:

I. Primary Peritonitis:

A. Spontaneous peritonitis in children

B. Spontaneous peritonitis in adults.

C. Peritonitis in patients with CAPD.

D. Tuberculosis and other granulomatous peritonitis.

E. Other forms

II. Secondary Peritonitis:

A. Acute perforation peritonitis (acute suppurative peritonitis)

1. Gastrointestinal tract perforation.
2. Bowel wall necrosis (intestinal ischemia)
3. Pelvic peritonitis
4. Other forms

B. Postoperative peritonitis

1. Anastomotic leak
2. Leak of a simple suture.
3. Blind loop leak
4. Other iatrogenic leaks.

C. Posttraumatic peritonitis.

1. Peritonitis after blunt abdominal trauma
2. Peritonitis after penetrating abdominal trauma
3. Other forms

III. Tertiary peritonitis

A. Peritonitis without evidence for pathogens.

B. Peritonitis with fungi

C. Peritonitis with low grade pathogenic bacteria.

IV. Other forms of peritonitis

A. Aseptic /Sterile peritonitis.

B. Granulomatous peritonitis.

C. Drug related peritonitis

D. Periodic peritonitis

E. Lead Peritonitis

F. Hyperlipidemic peritonitis

G. Porphyric peritonitis

H. Foreign body peritonitis

I. Talc peritonitis

V. Intrabdominal Abscess

A. Associated with primary peritonitis

B. Associated with secondary peritonitis

PHYSIOLOGY OF THE PERITONEUM

Mesothelial Cells:[39]

Mesothelial cells are organized into two discrete population: cuboidal cells and flattened cells. Gaps (stomata) between neighbouring cells of the peritoneal mesothelium are found only among cuboidal cells. Peritonitis increases the width of these stomata. Beneath the mesothelial cells is a basement membrane of loose collagen fibers. The basement membrane overlies a complex connective tissue layer that includes collagen and other connective tissue proteins, elastic fibers, fibroblasts, adipose cells, endothelial cells, mast cells, eosinophils, macrophages and lymphocytes. The capillaries branch and ramify within the peritoneal lining layer. In addition there is a rich lymphatic network.

The diaphragmatic surface was found to be covered by a single layer of mesothelial cells, which exhibited regional variation. Dome shaped cells often appeared in discrete bands that extended from the musculotendinous junction towards the rib cage. Adjacent cells margins were separated to form mesothelial pores, which were found over lymphatic vessels. The second type of cell covering the diaphragm appeared very flattened and gave the appearance of a continuous pavement layer. The cell margins of the flattened cells were closely opposed to each other without the indication of pores.[40]

Peritoneal Fluid Exchange:[39]

The mesothelial lining cells of the peritoneum secrete serous fluid that circulates within the peritoneal cavity. The peritoneal cavity normally contains 50-100 ml of fluid with solute concentration nearly identical to that of plasma. The protein content of peritoneal fluid is somewhat less than that of plasma, about 3 g/dl. Fluid is absorbed by the peritoneal mesothelial lining cells and sub-diaphragmatic lymphatic. Mesothelial cells also absorb solutes by the continuous process of endocytosis. Exchange of solutes across the peritoneal lining is the basis for peritoneal dialysis.

The normal peritoneal fluid of man contain about 2300 white cells/ per cubic millimeter. Of these about 45 percent are histiocytes, very few are neutrophils, a few are eosinophils and basophils, and many are lymphocytes.[41]

Two primary forces govern the movement of fluid within the peritoneal cavity: gravity and the negative pressure created beneath the diaphragm with each normal respiratory cycle. Subphrenic purulent collection occur because a relatively negative pressure is created beneath the diaphragm with each exhalation.[39]

Defense Mechanisms:[42]

Microbes invading the peritoneal cavity encounter several different host defense mechanism that:

i) Directly remove bacteria from the peritoneal cavity (translymphatic absorption).

ii) Kill the bacteria within the peritoneal cavity (complement bacteriolysis, phagocytosis and killing).

iii) Sequester bacteria and prevent this export into the systemic circulation (fibrin trapping, ileus and omental containment).

Peritoneal Response to Injury:

Any inflammation in the peritoneal cavity results in local peritoneal irritation with loss of regional mesothelial cells. A large peritoneal defect heals in the same amount of time as a small defect, usually 3-5 days.[39]Experimentally, it has been shown that 3 days after peritoneal injury the wound surface is covered by a layer of connective tissue cells resembling new mesothelium. At day 5, the new surface layer closely resembles adjacent normal mesothelium. By day 8, mesothelial regeneration is complete. The exact origin of cells responsible for mesothelial regeneration remains unclear. It has been suggested that regeneration mechanism include submesothelial stem cells producing new mesothelial cells, surviving mesothelial cells floating free or attached at the wound edges migrating into the

wound, and the peritoneal fluid, monocytes and macrophages differentiating into mesothelial cells.[43]

The following suggestions are put forward as possible explanations for the origin of the new mesothelium:[44]

1. It arises directly from primitive mesenchymal cells present in the perivascular connective tissue.
2. It arises indirectly from primitive mesenchymal cells via fibroblasts.
3. It arises from the subperitoneal fibroblast which in turn arises from differentiated, but resting, fibroblasts in the perivascular connective tissue.

The visceral peritoneum appears to differ little in its healing properties from the parental peritoneum.[44]

Normal peritoneal wound heals smoothly without adhesion formation. Adhesion develops in response to factors other than simple peritoneal wounding. Local tissue hypoxia and ischemia of the wound appear to be important factor in stimulating adhesion formation, other causes are mechanical injury to the subperitoneal surface, intraabdominal infection and contamination of the peritoneal cavity by foreign material.[43]

The peritonal cavity has an enzyme system that function to lyses fibrin deposits, plasminogen is concentrated in the endothelium of the submesothelial blood vessels and to a lesser degree in the peritoneal mesothelin itself. The plasminogen, when converted to plasmin by a plasminogen activator system, is capable of lysing fibrin deposits. Mechanical abrasion of the peritoneal surface completely abolishes this activity, which returns only over a period of days.[45]

Intraperitoneal adhesions form in the majority of patients following abdominal surgery and also occur after other causes of peritoneal inflammation.[46] They account for 25-33 percent of all cases of intestinal obstruction in developed countries, and are the commonest cause of small bowel obstruction.[47] Any inflammatory exudate forms following peritoneal injury and leads to the deposition of fibrin in the peritoneal cavity.[48]

The finding of decreased PAA (Plasminogen activating activity) in patients with peritoneal inflammation and ischemia, conditions which produce adhesions, supports the unifying hypothesis that a reduction in mesothelial PAA leads to fibrous adhesion formation.[46]

In essence, the final resolution of the infecting focus would seem to depend on the critical interaction between the phagocyte and the bacterium within a fibrin laden microenvironment.[22]

PATHOPHYSIOLOGY OF PERITONITIS:

The peritoneal cavity has a single response to trauma of all kinds - the acute inflammatory response.[49]

Primary responses in peritonitis:

Membrane inflammation :[43]

Following injury, histamine and other permeability factors are released from peritoneal mast cells, increasing vascular permeability of the peritoneum with exudation of protein rich plasma containing fibrinogen into the peritoneal cavity. The inflammatory cells and cytokines, autocoids and other chemical moieties participate in this process.

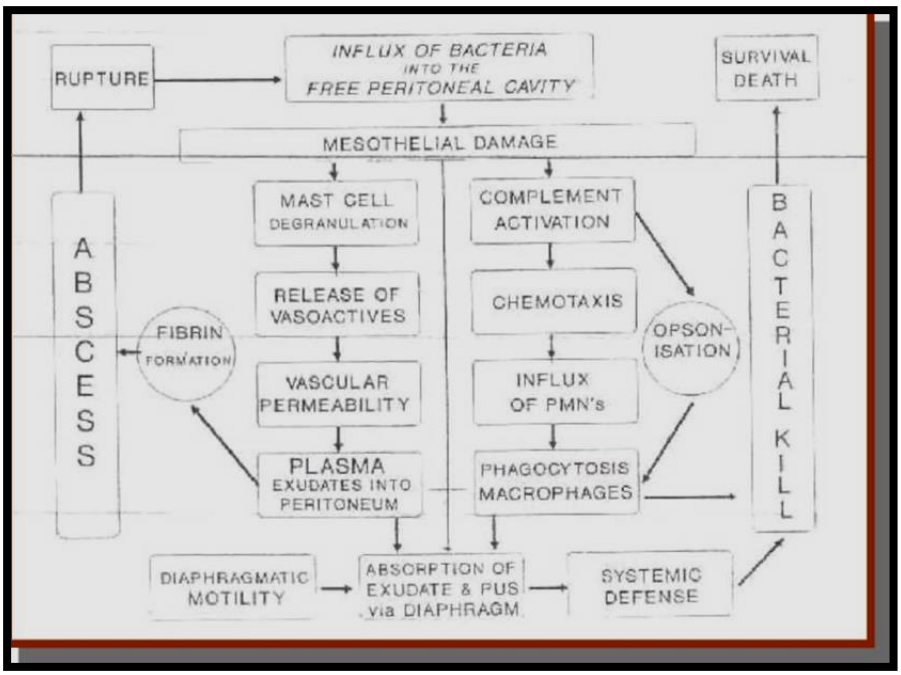


Fig 3. Physiologic Events and Responses in Peritonitis

Transduation of fluid with low protein content from the extracellular interstitial compartment into the abdominal cavity is accompanied by diapedesis of large number of polymorphonuclear (PMN) leukocytes. During the early vascular and transudative phase, the peritoneum acts as a "two-way street" so that toxins and other materials that may be present in peritoneal fluids are readily absorbed, enter the lymphatic's and bloodstream and can lead to systemic symptoms.

Transudation of interstitial fluid into the peritoneal cavity across the inflamed peritoneum is shortly followed by exudation of protein rich fluid. The fluid exudate in the peritoneal cavity contains large amounts of fibrin and other plasma protein in concentrations sufficient to bring about clotting, so that, in turn, adherence of loops of bowel, other viscera occurs in the area of peritoneal inflammation. There is increased synthesis of membrane glycoproteins and proteoglycans. The concentration of uric acid also increases, probably reflecting exudation of plasma protein in the early stages of peritonitis and in later stages, increased synthesis of glycoaminoglycans due to the activation of fibroblasts and mesothelial cells.

Changes in non-collagen protein synthesis and in collagen concentration are two other metabolic events that occur in the inflamed peritoneum during peritonitis.

In early peritonitis, non-collagen protein synthesis is increased, while collagen concentration is decreased. Later collagen concentration increases owing to increased total protein synthesis. The RNA-DNA ratio, an index of the protein synthesizing capability of tissues, increases during the first week of peritonitis.

Experimental studies of energy metabolism of the peritoneum in peritonitis have shown increased oxygen and glucose consumption and increased lactate production. There is also increased anaerobic metabolism mainly due to glycolysis. Coupled with a decreased partial pressure and increased consumption of oxygen, these changes lead to hypoxic environment in the peritoneal cavity that favours adhesion formation and growth of anaerobic bacteria.

Bowel Response:

The initial response of the bowel to peritoneal irritation is transient hypermotility. After a short interval, motility becomes depressed and nearly complete adynamic ileus soon follows. The bowel distends and both air and fluid accumulate in the lumen.

Hypovolemia:

Upon perforation and influx of irritating fluid and bacteria into the peritoneal cavity, a massive inflammatory response involving vasodilatation and fluid exudation can shift up to 10 L of fluid into the peritoneal cavity and into its loose subendothelial connective tissue. The atonic dilated bowel also accumulates fluid in its lumen. Depending on the magnitude of the insult, the formation of inflammatory peritoneal edema might happen so quickly as to be manifest clinically or hypovolemic shock.

Secondary Responses in Peritonitis

Metabolic Response:

The metabolic rate generally is increased, with a corresponding increase in peripheral oxygen demand. Simultaneously, the capacity of the lungs and the heart to deliver oxygen is diminished. Poor circulation leads to a shift from aerobic to anaerobic metabolism in muscle and other peripheral tissue, and as a result, anaerobic end products of carbohydrate metabolism accumulate and lactic acidosis begins to develop.

The appearance, in some cases of peritonitis, of lactic acidosis before systemic hypotension or hypoxemia suggests the possible existence of a primary cellular insult that makes cell unable to use available oxygen and substrate.

Protein catabolism begins early in peritonitis and becomes progressively more severe. Only a part of the amino acids made available by catabolism are readily used as energy sources because muscle cell can use only branched chain amino acids directly. Some data indicate that the rate of synthesis of plasma protein is increased in terms of both concentration and turn over.

Endocrine Response:

Peritonitis acts as a stimulus to many endocrine organs. There is almost immediate adrenal medullary response, without pouring of epinephrine and nor epinephrine, producing systemic vasoconstriction, tachycardia and sweating. The adrenal cortex is stimulated to increase secretion of cortical hormones during the first 2 or 3 days after peritoneal injury. Secretions of both aldosterone and antidiuretic hormone also are increased as a response to hypovolemia of peritonitis, resulting in enhanced renal conservation of sodium and water.

Cardiac Response:

The effects of peritonitis on cardiac function are a reflection both of the decrease in extracellular fluid volume and of progressing acidosis. The volume deficit results in decreased venous return and diminished cardiac output. The heart rate increases in attempt to maintain cardiac output, but compensation is usually is incomplete.

Respiratory Response:

Abdominal distension caused by inflammatory peritoneal edema and, later, by adynamic ileus, coupled with restriction of both diaphragmatic and intercostal respiratory movements in response to pain, results in a decrease in ventilatory volume and the early appearance of basilar atelectasis.

Renal Response:

Hypovolemia, reduced cardiac output, and increased secretion of antidiuretic hormone and aldosterone in peritonitis all act synergistically on the kidney. Renal blood flow in diminished, resulting in a decrease in the volume of glomerular filtrate and in tubular flow. Reabsorption of both sodium and water is increased, potassium is wasted. Urine volume output is diminished, and renal capacity to handle an excess of solute is impaired.

MICROBIOLOGY

The causative organisms in primary peritonitis as in pus culture studies are notably pneumococcus, Beta - haemolytic streptococcus or gonococcus.

While most examples of secondary peritonitis represent mixed flora of intestinal tract and its adnexa bacteriological examination of pus show E. Coli., Streptococcus faecalis, Pseudomonas aeruginosa, Staphylococcus, Klebsiella, and Proteus. Anaerobic culture shows strains of Bacteroides especially B. Fragilis and other anaerobes like Clostridia and anaerobic Streptococci. [38]

1. Bacteria from the GIT:

E. coli aerobic and anaerobic Streptococcus and Bacteroids are the most frequent invaders. Less frequently Clostridium or Klebsiella pneumoniae are found. Many strains of E. coli, Bacteroids and Welchii produce toxins which cause severe illness or death when they invade a large absorptive area. This is termed as "Endotoxic Shock".

The characteristic foul smell associated with pus of peritonitis of G. I. origin is due to production of free fatty acids and their esters as the result of anaerobic bacterial action, mainly Beta fragilis.[50]

2. Bacteria from outside the GIT:

Peritonitis due to gonococcus, Beta - haemolytic streptococcus, pneumococcus and mycobacterium tuberculosis occurs.

Routes of bacterial invasion.

A. Direct Infection:

1. Through a perforation of some part of alimentary tract.
2. Through a penetrating wound of abdominal wall.
3. Post operative.

B. Local extension:

1. From an inflammed organ eg. Appendicitis; cholecystitis.
2. Migration through the gut wall eg. Strangulated Hernia.
3. From the fallopian tubes.

C. Blood Stream:

As a part of general septicemia in cases of non bacterial peritonitis eg. intraperitoneal rupture of urinary bladder, even though chemical peritonitis sets in at the onset, in a matter of hours bacterial peritonitis supervenes by transmigration of organisms from the bowel.

MANIFESTATIONS OF PERITONITIS

Primary Peritonitis:

Primary peritonitis refers to inflammation of the peritoneum from a suspected extraperitoneal source, often via haematogenous spread. It occurs in children and in adults, and can be a life threatening illness, particularly in patients with cirrhosis.

Spontaneous peritonitis of adults is seen almost commonly in patients with ascites. This form of peritonitis is a monomicrobial infection, i.e. there is only a single species of bacteria present, in contrast to the polymicrobial infection of typical supportive (secondary) peritonitis. E. coli being the most common pathogen in adults, accounting for 70% 10 to 20% of cases are gram positive cocci and anaerobes are seen in 6 to 10%.

In children, the bacteria responsible for primary peritonitis are haemolytic streptococci and pneumococci. Two peaks of incidence are characteristic, one in the neonatal period and the other at age four to five. There may be history of previous respiratory infection.

Clinical Manifestations:

Clinical symptoms usually are of short duration in children; the onset of symptom is more insidious in ascitic adults. Most patients complain of some abdominal pain and distension; vomiting, lethargy, and fever are more prominent in children, Diarrhea is usual in neonates but seldom seen in adults. Bowel sounds are variable, Leucocytosis is usually present. Free air usually is not seen on abdominal radiographs.

Diagnosis: A peritoneal tap is the most useful diagnostic test. Fluid is examined for PMN cell count, pH; a gram stain should be done and a specimen sent for culture. A PMN cell count greater than 250 / mm^3 is considered positive. If the gram stain shows only gram +ve cocci, the presence of spontaneous peritonitis is strongly suggested.

Treatment: When the spontaneous bacterial peritonitis is confirmed, antibiotic therapy should be started and the patient initially managed non operatively. Usually, cephalosporin or ampicillin -sulbactam is appropriate since 90% of the organisms causing spontaneous peritonitis are sensitive to these antibiotics.

Tuberculosis Peritonitis:

The tubercle bacillus presumbly gains entry to the peritoneal cavity transmurally from diseases, bowel, from tuberculous salphingitis, or form nephritis or other haematogenous distant source. Clinically they present with fever, anorexia, weakness, weight loss, ascites in almost always present and more than half of affected patients have dull, diffuse abdominal pain. On examination, the abdomen is modestly tender, but the classically described "Doughy abdomen" is rarely found.

Diagnosis: A peritoneal fluid tap will show mostly lymphocytes ascitic fluid culture for tubercle bacilli. The ascitic fluid has an increased protein concentration,

and a glucose concentration below 30 mg /dl. Biopsy of the peritoneum can be done if the above measures fail to establish the diagnosis.

The laparoscopic appearance of tuberculous peritonitis is characteristic. Satellite like fibrinous masses during from the parietal peritoneum in the lower part of the abdomen.

Treatment is administration of antituberculous drugs. Prednisolone can be used to reduce the incidence of adhesion formation.

Secondary Peritonitis:

Perforation Peritonitis:

Perforation peritonitis is the most common form of acute intraabdominal infection. Perforation of the stomach and duodenum: Infection after peptic ulcer perforation presents acutely; the patient is commonly able to give the exact time at which the perforation occurred. This form of peritonitis is initially chemical in nature, but with the passage of time becomes infected. The patient most often seeks help early because of the severe pain, and operative repair often is possible before significant bacterial over growth has occurred. The proper management is simple closure of the perforation; some authors have recommended definitive treatment of the ulcer disease in addition if the perforation is less than 12 hour old.

Perforated peptic ulcer;

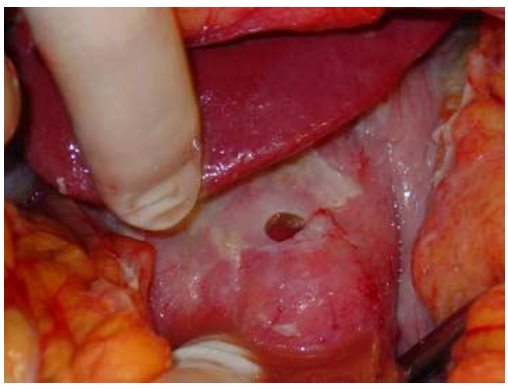


Fig 4.Duodenal ulcer perforation

Epidemiology:

Peptic ulceration, with its major complications such as perforation, seems to be a relatively modern disease, Rawlinson was credited with the first published report in 1726, of a perforated ulcer with happened to be gastric. The first published report of a perforated duodenal ulcer was by Hambergeri in 1746. According to Bonnevie (1958) most of the ulcers were gastric and occurred predominantly in female patients. Striking changes in the sex and age distribution of perforation have also taken place in recent years.

a) Sex: Perforated ulcer has been more common in men and than in women during the century. The male female ration for perforated duodenal ulcer fell in England. Wales and Scotland from approximately 6:1 in 1958 to 1962 to 2:1 in 1978 to 1982. In the same period the male - female ratio for perforated gastric ulcer fell from 3:11 tol: l.

However, the sex ratios for perforated peptic ulcer, with preponderance in males, were reported to the steady in Hong-Kong by Koo and colleagues in 1983 and in Finland by Negre in 1985. A proportion of females with perforated ulcer was noted by Hugh et al in Australia in 1969.

b) Age: Perforated ulcer was most common in the third to fifth decades of life during the first half of the twentieth century. By contrast in the Hospital Inpatient Enquiry (HIPE) study in United Kingdom reported by Walt and Colleagues in 1986, perforation rates for duodenal ulcer and gastric ulcer were highest in patients more than 65 years of age.

Peptic ulcer occurs rarely in infancy and childhood. Acute peptic ulcers in infants are usually proceeded by major systemic illness. Perforation in infants may also be associated with ulcerogenic medication such an indomethacin (used to

encourage closure of a patient ductus arteriosus) as reported by Nagaraj and colleagues in 1981 on tolazoline (used to treat pulmonary hypotension) as reported by Butt et al in 1986.

c) Occupation: Langman noted in 1979 that, since 1950 gastric and duodenal ulcers have been more frequent in lower socio economic groups in the United Kingdom and the United States of America. The association of peptic ulcer frequency with low educational achievement and independent of smoking habits.

d) Season: Several studies have examined possible seasonal variations in the incidence of perforation. This question was reviewed by Kurata and Haile in 1985, who outlined the reasons for the conflicting reports of seasonal variations in peptic ulcer disease and in perforation Confounding factors are geographic variations is seasonal length and severity, lack of agreement in the temporal limits of the seasons and the effect of non climatic seasonal events such as holidays, there seems to be non worldwide constituency in seasonal patterns of perforation.

Peritonitis following appendicitis:

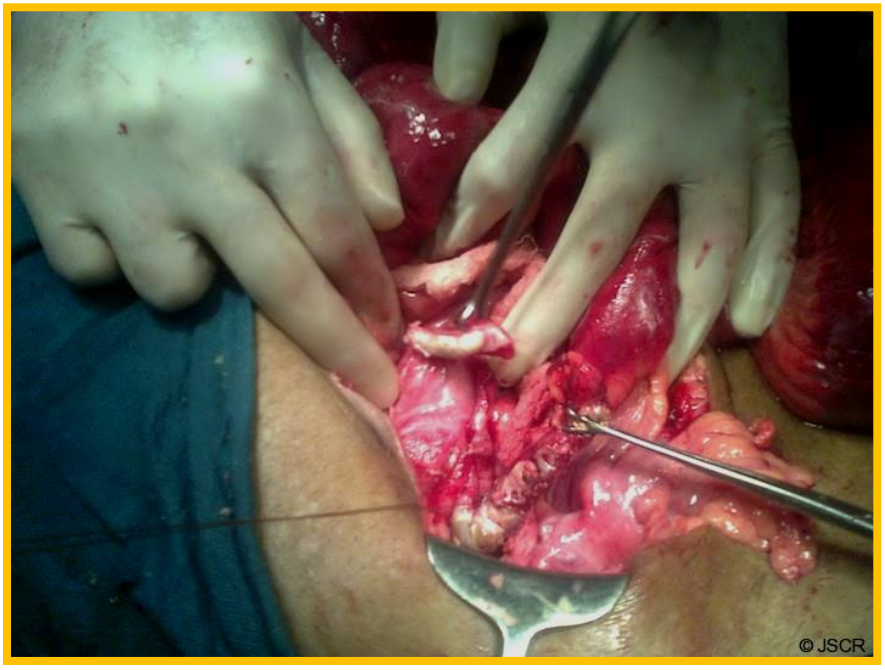


Fig 5.Perforated appendix.

Disseminated intraabdominal infection from appendicitis is not seen as often today as in the first decades of this country when vital statistics showed that appendicitis was the major cause of peritonitis seen in hospitals. Treatment is more likely to be successful once the appendix is removed and the source of infection is thus controlled.

Small bowel perforation:

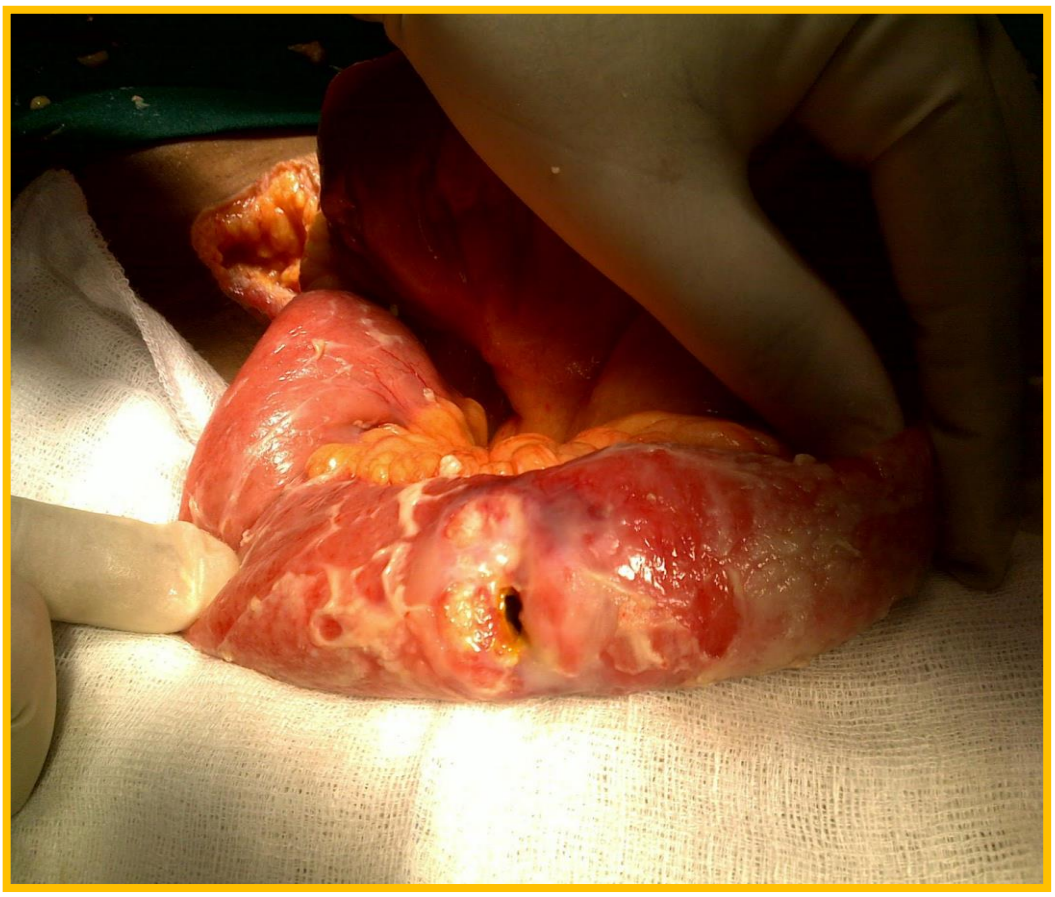


Fig 6. Ileal perforation

Intraabdominal infection after small bowel perforation falls into two major categories. In the first, bowel obstruction is present, so that ileus precedes peritonitis. In the second form, bowel wall necrosis due to inflammation or inadequate vascular supply leads to perforation.

Typhoid perforation:

Typhoid perforation is usually seen in third week of infection with salmonella typhi. Infection occurs from patients with acute disease, Typhoid bacilli are thought to penetrate the peyer's patches of the intestinal wall, mainly in the distal ielum. These collections of lymphoid cells hypertrophy, leading to hemorrhage and then perforation.

Incidence and diagnosis:

The incidence of perforation varies widely in the literature ranging from 0.5 to 5.6%. However, these figures merely indicate the frequency of detectable cases of perforation proved by operation. They do not by means indicate the actual frequency of perforation in typhoid patients.

Clinical features remain the main stay of diagnosis. A few days history of fever followed by severe abdominal pain in the most important feature in the diagnosis. Cases present with peritonitis, degree of guarding and rigidity varies. Pneumoperitoneum is demonstrate on X-ray in 60-80% of cases, Peristaltic sounds are heard in 40% of cases.

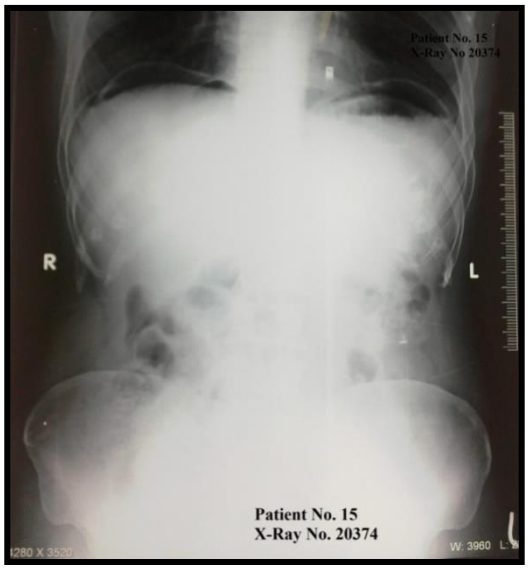


Fig 7.X ray erect abdomen showing Pneumoperitoneum

Treatment consists of closure of the punched out lesion and peritoneal lavage. Trimethoprim sulfamethoxazole is the treatment of choice of uncomplicated typhoid, but in patients with peritonitis, ceftriazone plus metronidazole is a better treatment regimen.

Colon perforation:

About 22 per cent of cases of peritonitis have their anatomic origin in the colon. More than half of these are due to inflammatory disease such as diverticulitis and colitis. The remaining are due to malignancy or external bowel obstruction.

Amoebic perforation:

Entamoeba histolytica infection of the intestine usually causes a dysentery like illness, but sometimes liver abscess or perforation of the large bowel occurs. Liver abscesses also can perforate secondarily and cause diffuse peritonitis. The clinical picture is that of bacterial peritonitis. Treatment consists of resection of the diseased bowel segment with anastamosis, application of the general principles of treating peritonitis and the administration of metronidazole in combination with a third generation cephalosporin.

Post operative peritonitis:

Post operative peritonitis is usually a leak from a suture line and is typically discovered between the fifth and seventh post operative day.

Peritonitis following pancreatitis: Translocation of bacteria is possibly the mechanism of progress from the initial chemical inflammation of pancreatitis. In severe acute pancreatitis, the corrosive activity of pancreatic digestive enzymes causes, extensive destruction of tissues in and around the pancreas may result in secondary infection.

Other forms of peritonitis;

Aseptic /Sterile peritonitis:

This form of peritonitis develops whenever irritant materials gain entry into the peritoneal cavity, usually because of rupture of solid or upper abdominal viscous. The soilage is sterile. Secondary bacterial invasion occurs usually after 12 hours of initial peritoneal insult.

Foreign body peritonitis:

Foreign bodies may be deposited in the peritoneal cavity during operations or might result from penetrating injuries or perforation of the intestines following ingestion.

The relative amounts of acute exudation and later fibrosis vary greatly depending on the nature of foreign body and degree of bacterial contamination.

Periodic Peritonitis:

Recurrent episodes of abdominal pain, fever and Leucocytosis occur in certain population groups in notably in Americans, Jews and Arabs. The disease appears to be familial. Colchicine is effective in preventing recurrent attacks, and infact, a favorable response to chronic administration of chochicine is a definitive diagnostic test.

Differential Diagnosis:

In early stages, acute peritonitis may be confused with the following conditions.

1. Thoracic diseases.
    a. Pleurisy and pneumonia
    b. Pericarditis and coronary disease.
2. Intestinal obstruction
3. Intraperitoneal hemorrhage

4. Acute pyelonephritis
5. Spinal cord ; Tabes dorsalis, spinal tumour, herpes, zoster, spinal carries and psoas abscess
6. Torsion of the solid organ like ovarian cyst, subserous fibroid
7. Diabetes mellitus with hyperglycemia.

CLINICAL FEATURES OF PERITONITIS

The onset is sudden is followed by a distinct intermediate latent interval, which in turn gives place to the classic signs and symptoms of diffuse peritonitis.

Symptoms of early peritonitis:

Of all symptoms, pain is the most important and constant, It varies considerably in intensity. It is severe and unremitting, but at times it may be no more than a discomfort. In postoperative peritonitis it is nothing more than a discomfort. It is a rule, most intense in that part of the abdominal wall which lies immediately over the spreading edge of the peritoneal inflammation. When the peritoneal infection subsides or localizes, pain diminishes in severity and becomes limited to one area of the abdomen.

Collapse, which is so often after a viscus perforation, is due in the initial stages to peritoneal shock, in late stages it is caused by toxemia and septicemia.

Vomiting may be slight at the start, but as the peritonitis advances, it becomes persistent. At first only stomach contents are voided; later the fluid that is brought up is bile stained and brownish, finally when the obstruction becomes complete, it becomes faeculent. Vomiting may be absent or infrequent if fluids by mouth are withheld. In early stages of peritonitis, vomiting is reflex in origin. Later probably it is toxic but in final stages it is caused by paralytic ileus. In acute peritonitis pain frequently precedes vomiting.

The temperature is often subnormal or normal in cases in which onset is sudden (eg. perforated duodenal ulcer) but it tends to rise gradually as true peritonitis supervenes.

A rising pulse rate and falling temperature are of the gravest significance. As the disease process advances, the pulse steadily rises and will be bounding, later

becoming weaker and more rapid. It is important, therefore, to take half hourly records of the pulse rate, because this will often indicate the true course of the disease.

The respiration from the start is quick, shallow and thoracic in character because of the immobility of the diaphragm.

The tongue is furred or moist in the early stages, later becoming coated and dry.

The bowels are usually constipated, although in some cases of pelvic peritonitis there may be diarrhea.

Signs of early peritonitis:

Inspection: There is absence or marked diminution of abdominal respiratory movements. The position of the patient in the bed is often characteristic. He lies still with the legs drawn up in an effort to relieve tension on abdominal muscles. It will be noticed that, in the early cases there is a marked retraction of the lower half of the abdomen.

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

There are two other signs that are constantly present - rebound tenderness and rigidity of the abdominal muscles: which is the most reliable and important. It may be localized, as in some early cases in which the peritoneal inflammation has involved only a limited area, or it may be generalized when the diffusion is extensive. The muscles are board like in their rigidity.

Percussion: Shifting dullness is positive. In certain instances, for example following the perforated peptic ulcer, obliteration of liver dullness may be detected, but this examination is by no means always reliable.

Auscultation: Intestinal sounds are diminished from the onset, they may be absent over the area of greatest mischief, and in a well established case of peritonitis with ileus, there is often a sinister illness.

Picture of the last few hours:

A late stage is reached when the peritonitis has progressed for some days, when the intestines have become paralyzed and intensely inflammed and blown out with gas and when the patient shows signs of peritonitis, toxemia, ileus and oligaemia. It is the stage of despair and of lost hope.

The pain now changes in character and becomes continuous and exhausting to a marked degree. Vomiting is no longer terrible but is of regurgitate type, profuse and effortless. The vomitus is dark and faeculent. The pulse, which is full and bounding and rapid, becomes quicker and weaker. The temperature may rise, but more frequently falls precipitously. Constipation is absolute, no flatus or faeces are passed. The face - The Hippocrates facies - tells at a glance that the end is near; the eyes are hollow, inquisitive and bright; the face is mask like, pale, pinched and blotchy, the brow and head are covered with cold perspiration. The lips are blue indicating circulatory failure and the tongue is dry, brown and fissured. The whole body is icy cold and clammy and the abdomen is distended, tympanic, tender and rigid. On auscultation nothing can be heard except perhaps the transmitted feeble quickened heart beats. It is said that the mind remains remarkably clear till the end. When renal and circulatory failures are evident, the battle for life is over.

MANAGEMENT OF PERITONITIS

Pre-operative Management :[6]

It is a mandatory that a short time be spent on resuscitation of the patient before operation. Precipitate surgery often leads to death, whereas preliminary attention to restoration of fluids, electrolytes and blood and control of septicemia makes surgery much safer and prevents many of the possible complications in the post operative period. The following regimen is suggested.

1. As soon as the presumptive diagnosis of perforation is made pain should be relieved by an appropriate dose of narcotic analgesic, if pain is severe or shock is present and if intramuscular absorption of drugs is slow, intravenous administration of narcotics should be slowly and carefully titrated. It is not necessary to obtain a firm diagnosis before relieving pain and any masking of abdominal signs will be transient if intravenously administered analgesia is used.
2. Gastric aspiration through the large bore nasogastric tube should be done as a first aid measure.
3. Nothing is allowed by mouth, although thirst may be relieved by sucking ice.
4. Blood should be drawn for grouping. Rh typing and across matching, cell count, serum amylase and other biochemical profile. An intravenous infusion should be commenced with appropriate electrolytes and plasma expanders. Pharmacological support in the form of drugs such as dopamine may be necessary to control the hypotension, and to ensure adequate cerebral, renal and coronary blood flow.
5. Urinary catheter should be inserted and urine output is monitored closely.

6.Electrocardiogram (ECG) should be taken to monitor the cardiac function.

7. Antibiotics are used in appropriate spectrum for the aerobic and anaerobic organisms commonly found in the gastro intestinal tract.
8. If the patient's physical conditions permits, the consent should be obtained for a definitive ulcer operation in case this step is found to be necessary.
9. Fever: A temperature above 38.5°c can be associated with difficulty in administration of anaesthesia. Administration of salicylates often will be effective in reducing fever.

Operative Management :[6]

The primary therapy in management of generalized peritonitis is surgical. An operation is usually required since the fundamental objective of treatment is to control the source of peritoneal contamination. This may require closure of a perforation, or resection of bowel with primary anastamosis or exteriorization. The septic procedure performed depends upon the findings at operation.

In adult patients with diffuse supportive peritonitis, operative approach is made through a paramedian as midline incision is likely to give away it can be used only in selective cases. In infants less than two years of age, transverse incision is safer.

As soon as the abdomen is opened, odour of the peritoneal fluid is of significance. In case of lower small intestinal perforation the odour is faeculous whereas in case of upper G.I. trait perforation above the duodenojejunal flexure, the peritoneal fluid is almost odourless. The general rule is that aerobic infections do not produce marked odour, whereas anaerobes do.

Peritoneal toilet :[6]

There are various approaches to cleaning the abdominal cavity, including suction, swabbing, lavage, radical debridement, and post operative irrigation. All

peritoneal pus, pseudo membranes, loosely adherent fribrins and other exudates should be completely removed; contents of any localized collections or abscesses should be completely evacuated. This is called peritoneal debridement.

Intraoperative high volume lavage :[6]

This treatment involves extensive lavage of the entire abdominal cavity. Initially 1 to 1.5 L of Ringer's solution is placed into the abdominal cavity, the viscera are manipulated so that the fluid reaches all parts of the abdominal cavity to wash out pus, feces and necrotic material, and then the fluid is suctioned away. This procedure is repeated until the suctioned fluid, runs respectively clear. It is repeated until the suctioned fluid, runs respectively clear. The total volume of lavage should be at least equal to the estimated volume of empty peritoneal cavity. Usually, a total of 8 to 12 Its. is used in adults. Sometimes antimicrobial agents are added to the lavage fluid.

Closure: In case of sutures the linea alba are sutured as a single layer with polypropylene or nylon talking generous bites of tissues on either side. In case of paramedian or transverse incision, the closure is performed in the usual fashion temporarily using new device such as Zipper with larger seams (Ethizip) Marlex mesh with a Zipper. This new concept is termed as staged abdominal repair.

Relaparotomies are performed at 24 hours intervals in the operating room. Once all problems are solved, the abdominal cavity is closed definetly.

Treatment of perforated Duodenal Ulcer:

Perforated duodenal ulcer may be treated surgically either by:

1. Simple closure of the perforation with an omental patch for
2. A definitive ulcer operation.

Simple closure of the perforation: Technique:

1. The technique of laparascopic surgery: The patient is operated upon in the supine position with a slight head up tilt. The peritoneal cavity is entered with the telescope. If the perforation is small (> 4mm) is closed by three full thickness interrupted sutures using the Dundes Ski needle preferably mounted with silk.
2. Sutureless Laparascopic closure of perforated duodenal ulcer in this technique the gelatin sponge 20x15 mm size was cut into cone shape and it was plugged to the perforation with its base facing towards serosa and then 2 ml of fibrin sealant is used to seal. Nasogastric aspiration was continued for 2 days.
3. Endoscopic closure of perforation: Peritoneal cavity is viewed through a 10 mm diameter access port. By using a needle holder inserted through a second port and forceps via a third, the perforation may be closed. The peritoneal cavity may then be washed out and the patient spared on abdominal incision.
4. Other methods: When the perforation is large other techniques like extended pyloroplasty may be employed. For the small perforations without much surrounding fibrosis, simple approximation of the edges of perforation with several sutures with avoidance of inversion or eversion is sufficient.

Non absorbable full thickness sutures should not be used, because they may cause silk ulcers, which many bleed or produce pain, it is preferable to use simple synthetic monofilament sutures, such as polydioxone (PDS). The absorption characterstics of this material are more predictable than those of catgut.

Seromuscular inverting sutures once popular are best avoided because they tend to work free from edematous tissues around the perforation. A purse string suture is not recommended because it may cause narrowing and has tendency to work free.

If simple opposition is not possible because of indurations and edema, then a omental patch technique may be used. Cellan Jones in 1929 described the most widely used method, often wrongly credited to Roscoe Graham, in which strand of omentum is drawn under an arch of full thickness sutures placed on either side of the perforation. Graham described the identical technique, but suggested using a free graft of omentum, a variation that is seldom used. Peritoneal cleaning should be done meticulously after the closure in all the cases.

Definitive ulcer operation:

In deciding which procedure is best it is important to establish the relative safety of each procedure and also the necessary for a definitive operation in each patient.

Definitive surgical treatment is strongly advocated for the patients with

1. No risk factor.
2. The perforated duodenal ulcer is associated with bleeding or gastric outlet obstruction.
3. Previous radiological or endoscopic evidence of chronic ulcer disease where there has been a poor response to full adequate medical treatment.
4. Past history of hemorrhage or perforation. A further group who may be benefited are those who perforate on NSAID's and are likely to require long-term treatment with these agents.

Points of controversy:

For patients who give a definitive history of dyspepsia before the perforation of their chronic ulcer, some will support definitive surgery while others argue that the combination of simple closure and effective medical treatment is a better option. No definitive opinion can yet be given until better studies including endoscopic

assessment are available for medical treatment. It should be remembered that the majority of these operations will be performed by relatively inexperienced surgeons; therefore the less invasive procedure is the correct choice at present.

Treatment of perforated Gastric Ulcer:

- The most suitable operation for gastric ulcer perforation is
- Primary gastric resection with Billroth I (gastro duodenal) Anastamosis. Simple closure of perforation is advised if the perforation is near the cardis. Contaminated fluid form the peritoneal cavity is aspirated and peritoneal wash is given.

Postoperative Management:

Intravenous fluids, nasogastric aspiration and antibiotics are continued, urine output, pulse, blood pressure, temperature, blood gases, serum creatinine, coagulation factors and ventilatory volume are monitored frequently. When the results of antibiotic sensitivity of the peritoneal pus are available, due consideration should be given to change the antibiotic therapy to the most specific and least toxic of the sensitive drugs.

Complications of Peritonitis:

Complications of peritonitis may be early and late. Early implication are shock high output respiratory failure. ARDS, Acute renal tubular necrosis leading to renal failure and hepatic failure associated with liver abscesses and pyelonephritis, paralytic ileus. Late complications include residual abscess and intestinal obstruction.

Prevention of complication is by early recognition and treatment.

MATERIALS AND METHODS

SOURCE OF DATA

The material for the study consisted of 100 patients who presented to Department of General surgery of R. L. J. H., KOLAR, between FEB-2010 – FEB-2011 with features of peritonitis secondary to hollow viscus perforation.

This was a randomized study; the patients were divided in two groups.

GROUP 1: Patients with all odd serial numbers were included in this group and given peritoneal lavage using Povidine iodine (**1%** wt/volume of Povidine iodine in 2 liter of normal saline).

GROUP 2: Patients with all even serial numbers were included in this group and given peritoneal lavage using metronidazole (**200** ml of metronidazole in 2 liter of normal saline).

Methods of collection of data

All patients who presented with features of peritonitis secondary to hollow viscus perforation and confirmed by laparotomy findings of a detectable perforation of a hollow viscus organ and by the presence of free fluid in the peritoneal cavity.

Data was collected after devising a clinical proforma in which the following factors were included.

The name, age, sex, address of each patient was noted. Complaints regarding pain abdomen, vomiting, constipation, fever were documented chronologically.

Past history regarding pain abdomen was noted.

History suggestive of co – existing conditions like diabetes mellitus, hypertension, cardiovascular, respiratory and renal disease were recorded.

Detailed physical examination for presence of pre – operative shock and co – existing associated diseases was done.

Investigations Done : Pre – operative – relevant hematological ; biochemical and serological investigations ; plain X-ray erect abdomen ; ultrasonography of the abdomen (if X- ray was inconclusive) X –ray chest pa view and four quadrant diagnostic peritoneal tapping was done and recorded.

Pre-operative preparation

The pre-operative preparation of each case consisted of correction of shock, electrolyte imbalance, dehydration, gastric aspiration, parental broad spectrum antibiotic coverage and tetanus prophylaxis.

Operative details

Operative details such as date of surgery, hospital no, were noted.

Operative findings such as the site of perforation, degree of peritoneal contamination were noted.

After opening the peritoneal cavity 2ml of the peritoneal contaminated fluid is collected and the appropriate surgery was done and thorough wash was given to the peritoneal cavity either with povidine iodine or metronidazole and again 2ml of peritoneal fluid was collected. Both the samples are labeled and sent immediately for isolation of the organism and Bacterial colony count. Semi quantitative bacterial count, of the peritoneal fluid collected before wash and the peritoneal fluid collected after was performed by platting on blood agar, meconic agar. Sometimes to facilitate the counting specially in cases which are heavily contaminated, fluid dilutions of the peritoneal fluid were done and then plated and the resulting colonies were counted. The colonies thus grown were identified by using routine bacteriological methods.

Post operative progress is assessed by comparing the development of surgical site infection (SSI), duration of hospital stay, pre wash and post wash bacterial colony count in both the groups.

The treatment to be adopted in each case was decided by the attending surgeon .Post operative fluid and electrolyte balance was maintained by input and output charts and adequacy of replacement was judged mainly on the basis of clinical features. In most of the cases empirical broad spectrum antibiotics were started pre-operatively. The drainage tubes were removed between 3^{rd} to 5^{th} post operative day and gastric aspiration was discontinued as soon as the patient passes flatus and appearances of normal bowel sounds. The post operative complications, especially SSI were studied in the immediate follow up period.

Method of Statistical Analysis;

The following methods of statistical analysis have been used in this study.

Independent student‘t’ test.

Paired sample‘t’ test.

Chi square test.

Proportions were compared using Chi-square test of significance. A”P” value of less then 0.05 was accepted as indicating statistical significance. Data analysis was carried out using SPSS package.

Inclusion criteria

All the patients with peritonitis secondary to hollow viscus perforation

Exclusion criteria

Patients with peritonitis secondary to blunt injury abdomen.

Patients with peritonitis secondary to penetrating injury to abdomen.

OBSERVATIONS AND RESULTS

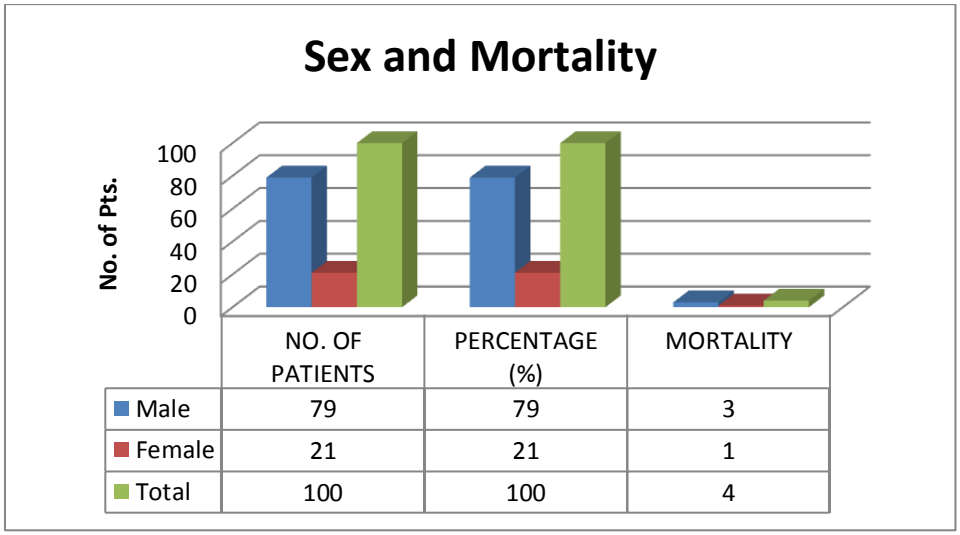
100 patients who presented with peritonitis secondary to hollow viscus perforation admitted and treated in R.L. Jalappa Hospital and Research Centre attached to Sri. Devaraj Urs Medical College, Kolar were studied during the period of Feb- 2010 to Feb- 2011.

Incidence of perforation in males and females

In our study of 100 patients of peritonitis secondary to hollow viscus perforation males showed higher incidence of hollow viscus perforation accounting for 79% in comparison with females who accounted for about 21% . There were 4 deaths in the study, 3 were male patients and 1 was a female patient. All of them had severe form of peritonitis.

Table – 1 Incidence of perforation in males and females

SEX	NO. OF PATIENTS	PERCENTAGE (%)	MORTALITY
Male	**79**	**79**	**3**
Female	**21**	**21**	**1**
Total	**100**	**100**	**4**

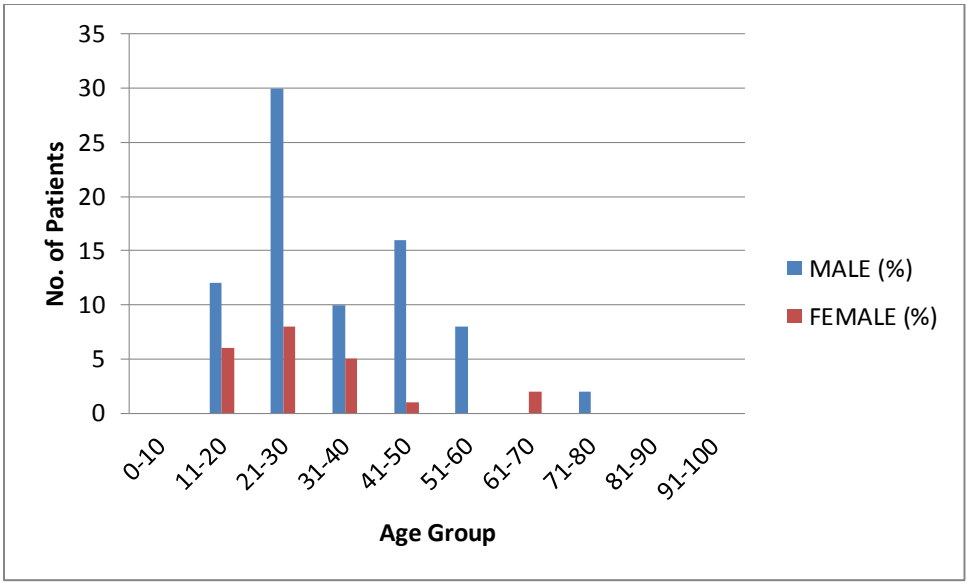


Incidence of perforation in different age groups

Patients of all the age groups were included in the study and the youngest patient in this study is a 12 year old boy who was diagnosed to have duodenal perforation, and the oldest patient in this study is an old man of 75 year who was diagnosed and treated for duodenal perforation. 33 years is the mean age of perforation in this study. More then half of the patients fall between the age group of 21 – 40 , out of which 38 patients were in the age group of 21 – 30 and 15 patients in the age group of 31 – 40 .This shows that more then half of the perforation is seen in young adults .

Table – 2 Incidence of perforation in different age groups

Age	MALE (%)	FEMALE (%)	NO. OF PATIENTS
0-10	0(0%)	0(0%)	0
11-20	12(67%)	6(33%)	18
21-30	30(79%)	8(21%)	38
31-40	10(67%)	5(33%)	15
41-50	16(94%)	1(6%)	17
51-60	8(100%)	0(0%)	8
61-70	0(0%)	2(100%)	2
71-80	2(100%)	0(0%)	2
81-90	0(0%)	0(0%)	0
91-100	0(0%)	0(0%)	0
Total	**100**	**100**	**100**

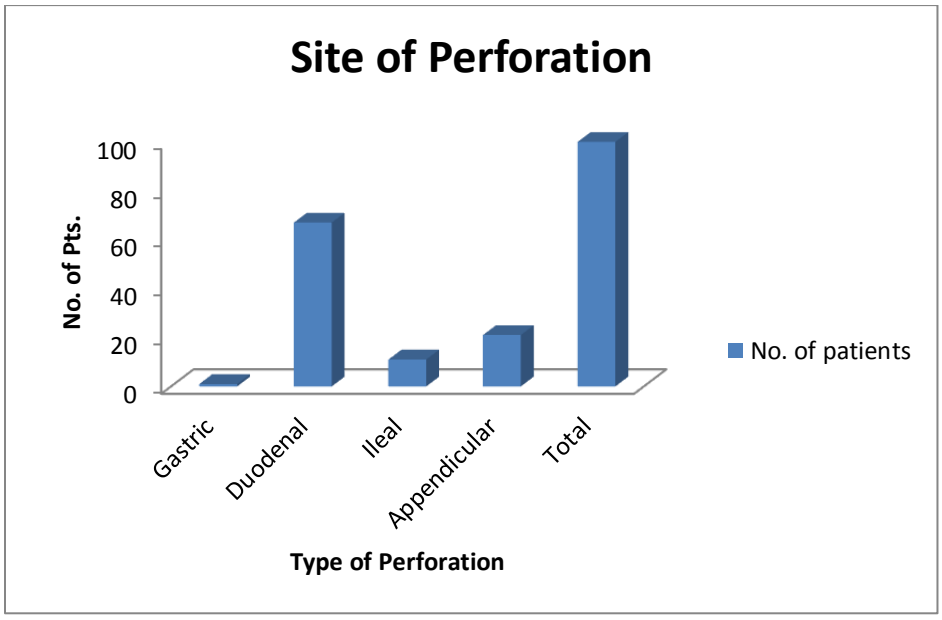


Type of Perforation

Depending on different sites of perforation and the data collected from our study revealed that duodenal ulcer perforation is the commonest being 67%, Appendicular perforation was the next commonest accounting for 21%, followed by Ileal and Gastric perforation. Out of 11 cases of ileal perforation 3 cases had Widal positive.

Table – 3 Incidence of type of Perforation

Site of Perforation	No. of patients	Percentage
Gastric	1	1
Duodenal	67	67
Ileal	11	11
Appendicular	21	21
Total	**100**	**100**

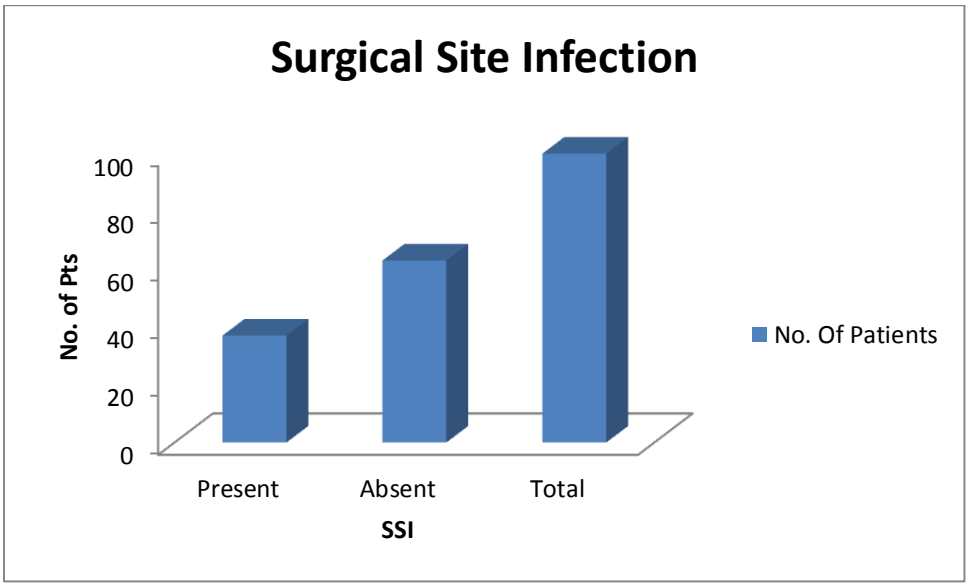


Surgical Site Infections in patients with perforation

With regard to surgical site infection, 37 patients had wound infection, such as wound gaping, stitch abscess, subphrenic abscess. In our study of peritonitis secondary to hollow viscus perforation we came across 3 cases of fecal fistula which was treated conservatively. There was no case of burst abdomen and no infection was observed in 63 patients.

Table – 4 Incidence of Surgical Site Infection

SSI	No. Of Patients	Percentage
Present	37	37
Absent	63	63
Total	**100**	**100**

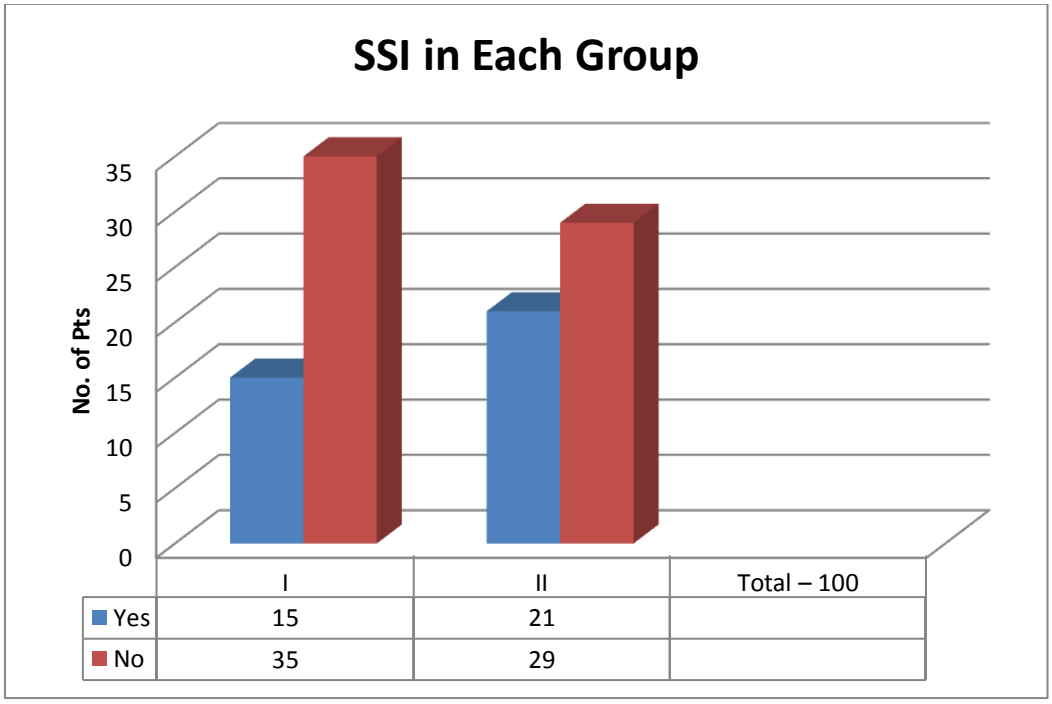


Surgical site infection in each group

Incidence of surgical site infection in patients who received lavage with povidine iodine is 15 were as patients who received lavage with metronidazole is 21.Hence surgical site infection is less in Group 1 compared to Group 2.

Table – 5 Incidence of Surgical site Infection in each group

Group	Present (%)	Absent (%)	Total
Povidine iodine	15 (30%)	35 (70%)	**50**
Metronidazole	21(42%)	29 (58%)	**50**
Total – 100			**100**

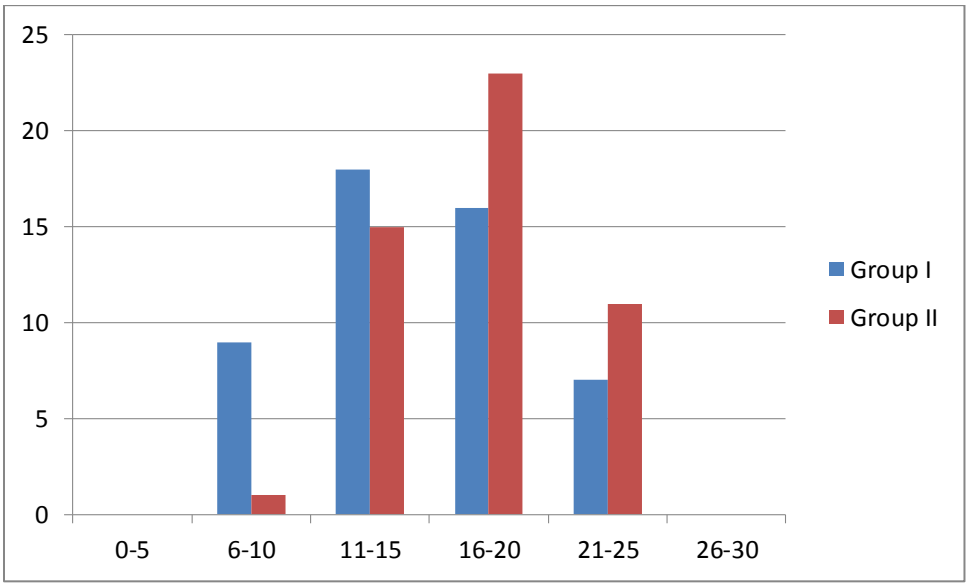


No. Of days of hospital stay

Duration of hospital stay extended from 10 to 25 days. The maximum number of days of hospital stay is between 11 - 20 days were 39 patients stayed between 16 – 20 days followed by 11 – 15 which accounted for 33 patients. The mean stay in hospital was 16 days for patients who received peritoneal lavage by povidine iodine were as it was 17 days for pts who received lavage with metronidazole. The maximum number of days a patient stayed in hospital is for 25 days who was operated for duodenal perforation and minimal stay of a patient is for 8 days who underwent surgery for appendicular perforation. In our study we found that patients who received lavage with povidine iodine group had shorter stay in the hospital.

Table – 6 No. of days of hospital stay

No of Days	Total	Group I	Group II
0-5	0	0	0
6-10	10	9	1
11-15	33	18	15
16-20	39	16	23
21-25	18	7	11
26-30	0	0	0
Total	**100**	**50**	**50**



Organisms isolated in peritoneal lavage

The most common organism isolated in Pre – Lavage is E.coli in 65 cases followed by Enterococci in 14 cases, where as in post – Lavage E.coli is present in 33 cases followed by Enterococci. There was no growth of any organism in 5 patients. There was significant decrease of organism in Post – Lavage compared to Pre – Lavage.

Table – 7 Incidence of organism isolated in peritoneal lavage

Organism isolated	Pre- lavage	Post-lavage
E. coli	65	33
Enterococci	14	6
Klebsiella	10	7
Candida albicans	9	0
Acinetobacter	4	0
No organism	5	5

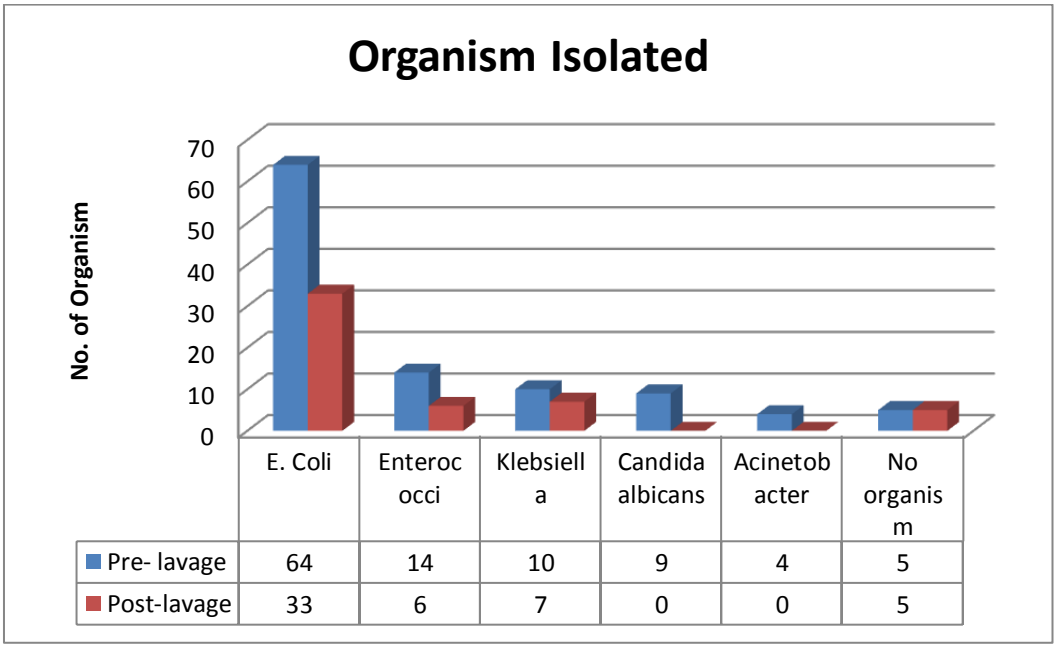


Table No. 8 shows the bacterial counts in the two groups of the patients , one receiving povidine iodine lavage and another receiving metronidazole lavage. The bacterial counts have been done before lavage was given [pre – lavage] and after the lavage was given [post-lavage]. No growth in both the pre and post lavage was seen in 5% of the patients [1 belongs to povidine iodine group, 4 belongs to metronidazole group].

The organisms grown were E.coli, Enterococci, Candida albicans, Klebsiella and Acinetobacter. However E.coli was the major organism grown accounting for growth in 65 of the patients. There were 31 patients in povidine iodine group[62%] who yielded E.coli in the pre – lavage sample , the colony count varied from 8 – 21000 organisms/ml with an average of 3645 organisms/ml , and in the post – lavage sample no organism could be isolated from 15[50%] of the patients. The colony counts in the post – lavage sample with povidine iodine had drastically dropped with the range becoming 0 -7200 organisms/ml and average organism becoming 554 organisms/ml.

In the metronidazole lavage group, there were 34[68%] patients who had E.coli in the pre – lavage sample. The colony count ranged in between them is 3 – 60,000 organisms/ml and the average was 7,725 organisms/ml. After lavage with metronidazole, 17 of them did not yield any organism. In the rest, the post – lavage sample showed that colony count varied from 2 – 25,600 organism/ml and average was 3006 organisms/ml. The drastic reduction seen with povidine iodine lavage was not thus seen in the metronidazole group among those who yielded the organism even after the lavage. The proportion of those who did not grow any organism in post – lavage sample was almost same in either of the group.

Enterococci and Klebsiella were found in pre – lavage sample of 7 and 5 respectively in either of the groups receiving povidine iodine lavage or metronidazole. The reduction in those who yielded no organism after the lavage was also similar [out of 7, 4 did not grow any organism in the sample in either of the groups]. However the ranges and average numbers varied widely between the two groups. The reduction in the post – lavage sample was more drastic in the metronidazole group than in povidine iodine group, which could be related to the initial number of organisms. Similarly in those patients who yielded Klebsiella in pre – lavage sample, the clearing of the organism in post – lavage sample was marginally better with metronidazole group lavage [2 out of 5 cleared with povidine iodine and 3 out of 5 cleared with metronidazole lavage].In those yielding organism in the post – lavage sample there was a drastic reduction in the range and the average seen with metronidazole group , this could be related to low initial count seen in the pre – lavage sample of that group compared to the average number of organism and the range seen with povidine iodine group.

There were 13 patients with Candida in the pre – lavage sample, but in all of them after lavage no Candida can be grown. Similarly 4 patients who underwent laparoscopic duodenal closure, Acinetobacter was grown in pre – lavage and was not found in post – lavage sample.

These observations indicate that Candida are easily cleared by either type of lavage. The Acinetobacter could have been a contaminant carried from the environment to the peritoneal cavity through the instrument used for laparoscopic closure of perforation.

Organism Isolated	Povidine iodine lavage group (n=50)		Metronidazole lavage group (n=50)	
	Pre lavage No. of sample yielding (average colony count , range)	**Post lavage No. Of sample yielding (average colony count , range)**	**Pre lavage No. of sample yielding (average colony count , range)**	**Post lavage No. Of sample yielding (average colony count , range)**
E.coli	31 (3645; 8 - 21000	16 (554 ; 0 – 7200)	34 (7725 ; 3 – 60000)	17 (3006 ; 2 – 256000)
Enterococci	7 (2942 ; 30 – 18200)	3 (2405 ; 0 – 7200)	7 (662 ; 4 – 4000)	3 (7 ; 0 – 10)
Candida albicans	7 (105 ; 1 –180)	0	6 (6 ; 0 – 6)	0
Klebsiella	5 (3451 ; 27 – 7200)	3 (1450 ; 150 – 2200)	5 (413 ; 10 – 2000)	2 (6 ; 4 – 8)
Acinetobacter	2 (21 ; 20 – 22)	0	2 (79 ; 60 – 98)	0

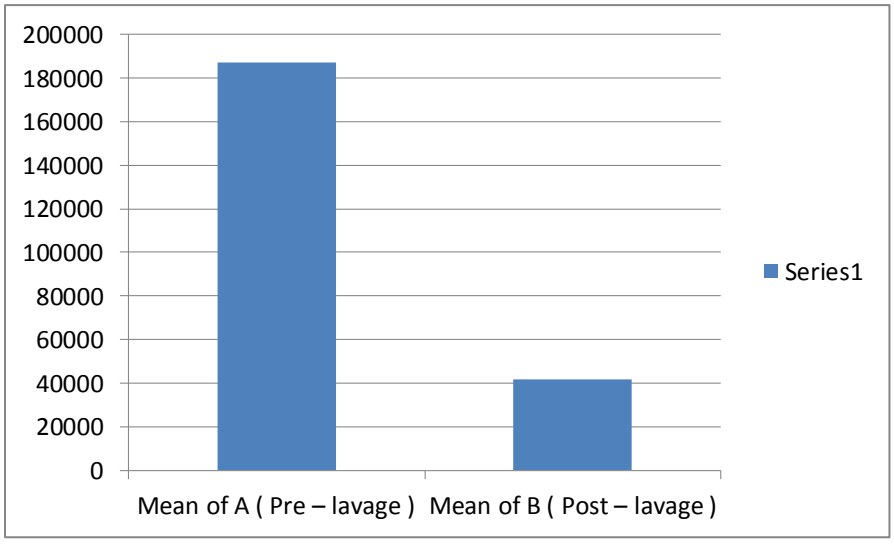
Table - 8

Comparison of mean values of pre and post lavage with povidine iodine

Random 50 patients were selected for peritoneal lavage with povidine iodine and the mean of pre – lavage (A) and post lavage (B) colony count values were derived. There was an obvious reduction in the colony count prior to and after the lavage.

Table -9 Comparison of mean values of pre and post lavage with povidine iodine

Mean of A (Pre – lavage)	187368.5
Mean of B (Post – lavage)	41525.93

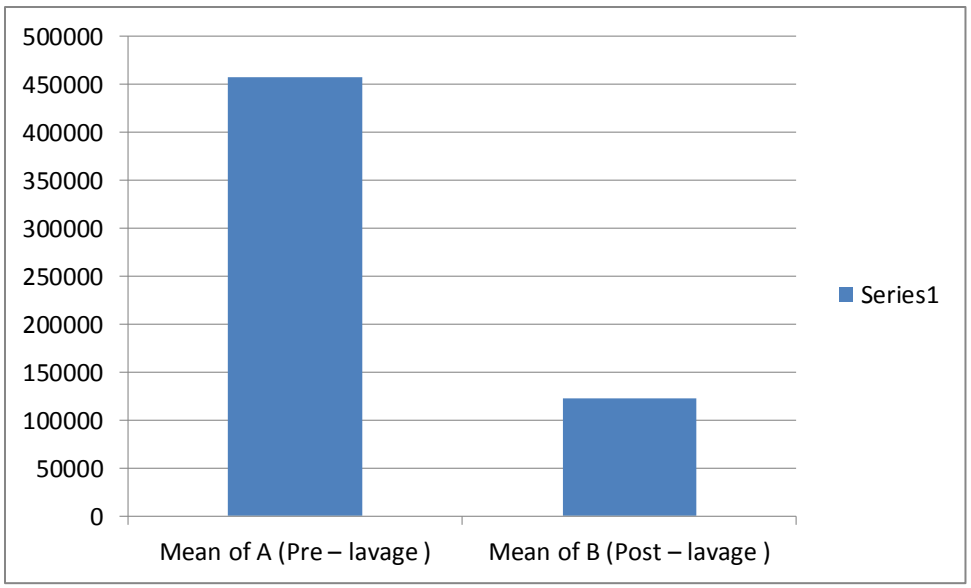


Comparison of mean values of pre and post lavage with metronidazole

Random 50 patients were selected for peritoneal lavage with metronidazole and the mean of pre – lavage (A) and post – lavage (B) colony count were derived. There was a decrease in the colony count prior to and after the lavage but was less compared to povidine iodine lavage.

Table – 10 Comparison of mean values of pre and post lavage with metronidazole

Mean of A (Pre – lavage)	457583
Mean of B (Post – lavage)	122417.4



Comparison of p- values with povidine iodine and metronidazole

The p – value was derived using the formula mentioned initially in materials and methods. Reduction in bacterial colony count was seen in both the groups of patients however, the p – value was significant in the patients who received lavage with povidine iodine. i.e. $p = 0.007 (< 0.05)$ when compared to the p – value in patients who received lavage with metronidazole i.e. $p = 0.06 (> 0.05)$.

Table – 11 Comparison of p- values with povidine iodine and metronidazole

Agent Used	P – value
Povidine iodine	0.007
Metronidazole	0.06

Table – 12 Comparison of pre –lavage and post – lavage p values with povidine iodine and metronidazole

	Bacterialcolonycounts		**'t' test**
	Pre –lavage	Post -lavage	P
Lavage with Povidine iodine	187369	41526	0.007 (significant)
Lavage with Metronidazole	457583	122417	0.06 (not significant)

DISCUSSION

In this study we have studied 100 patients who presented with features of peritonitis secondary to hollow viscus perforation to R.L.Jalappa Hospital and Research Centre , Tamaka ,Kolar , from Feb – 2010 to Feb – 2011.

This was a randomized study; the patients were divided in two groups.

In Group 1patients with all odd serial numbers were taken and received peritoneal lavage with Povidine Iodine and in Group 2 Patients with all even serial numbers were taken and received peritoneal lavage using Metronidazole.

This clinical study was intended to determine the efficacy of Povidine Iodine and Metronidazole and the incidence of surgical site infection and number of days of hospital stay in these patients.

In general this study revealed duodenal ulcer perforation to be the commonest followed by appendicular, Ileal and the rest.

Male population was affected more than female population, the maximum number of patients were in the age group of 21 – 40 with mean age of 33years.

37 patients had surgical site infection ranging from wound gaping, stitch abscess, paralytic ileus to fecal fistula. 3 patients had fecal fistulas which were treated conservatively and successfully.

Peritoneal Lavage seemed to be therapeutically effective in this study which was similar to the study conducted by Nathens and Colleagues[51] where in peritoneal toilet was one of the therapeutic options in peritonitis which proved beneficial. There was reduction in bacterial colony count following peritoneal lavage in this study. The study done by Hunt and colleagues[52] whether to irrigate or not to irrigate the abdominal cavity also reveal reduction in bacterial contamination.

Another experimental study in rats done by Hau and colleagues who investigated on a group of rats with fecal peritonitis revealed that all rats died and that peritoneal irrigation along with systemic antibiotics were effective.

We have used povidine iodine lavage in one group of patients and metronidazole in another group of patients. Iodine is known to be a bactericidal agent against both aerobic and anaerobic organisms; however metronidazole is effective only against anaerobic organisms. In our study we found that both the types of lavage were equally effective in terms of clinical outcome. There was no significant differences as per as mortality of patients, SSI, number of days of hospital stay.

E.coli was the most common organism isolated from the pre and post lavage samples. The number of patients who cleared the organism as evidences by no growth in the post – lavage sample were similar in both the groups. However there is drastic reduction in the E.coli count in 16 patients of the povidine iodine group. No such drastic reductions in the E.coli count were seen in metronidazole lavage group. When the bacterial count of all the pre and post lavage samples were taken together for comparison there was a significant reduction[p -0.007] in the post lavage sample of povidine – iodine group. Thus this study suggests that povidine iodine to be better lavage agent as far as reduction in the colony count are concerned.

In this study there were 4 deaths which were due to severity of illness due to delay in seeking medical treatment; all of them were seen by us on 4^{th} – 5^{th} day of onset of severe abdominal pain. We could not relate to the bacterial contamination as evidenced by counts in pre – lavage and post – lavage samples. We think the complications due to unhindered pathological process and irreversible damage might have been responsible for death in this patients. This is in contrast to the observation

by Atkins and colleagues[12] who attributed mortality in there series to gross and diffuse peritoneal soiling.

The incidence of surgical site infection in this study is less in patients who received lavage with Povidine Iodine however it is not statically significant when compared to metronidazole group.

The incidence of hospital stay was more in patients who received lavage using metronidazole compared to patients using povidine iodine group is similar. This is also not significant statically.

SUMMARY

The difference in bacterial colony count was studied using povidine iodine and metronidazole both pre and post peritoneal lavage in cases of peritonitis which were proved surgically.

There was decrease in bacterial colony count with the use of both the agents.

The p- value was significant in cases who received povidine iodine < 0.05 (0.007).

The p- value was insignificant in cases who received metronidazole > 0.05 (0.06).

There were 4 deaths, all of them who had severe form of peritonitis and delayed presentation to the hospital

This study also revealed that men are commonly affected and duodenal ulcer perforation is the commonest site of perforation.

E.coli is the most common organism isolated.

CONCLUSION

This clinical and bacteriological study has demonstrated the following;

- ✓ Peritonitis is more common in men compared to women.
- ✓ The common age group is in between 21 – 40 in cases of peritonitis with mean age of 33 years .
- ✓ Duodenal Ulcer perforation is the commonest site of perforation.
- ✓ E.coli is the commonest organism isolated from the peritoneal contamination.
- ✓ Povidine iodine lavage (< 0.05) significantly decreases the bacterial load when compared to metronidazole lavage.
- ✓ As per as clinical outcome is concerned there is no significant differences in both the groups.

Keys to Master Chart

Absent	A
Agent Used	A.U.
Appendicectomy	App.
Appendicular perforation	A.P
Colony forming unit	Cfu
Days of hospital stay	D.
Death	D
Duodenal perforation	D.P.
Estericia coli	E.Coli
Female	F
Gastric perforation	G.P.
Ileal perforation	I.P.
Male	M
Metronidazole	MET
No growth	N.G.
No.	N
Number	NO.
Omental Patch	O.P.
Organism isolated	O.I.
Post-Lavage bacterial colony count	Po. L.B.C.C.
Povidine Iodine	P.I
Pre-Lavage bacterial colony count	Pre L.B.C.C.
Present	P
Primary closure	P.C.
Procedure	PRO.
Recovered	R
Surgical site infection	SSI
Type of perforation	T.O.P.
Yes	Y

PROFORMA OF THE CASE SHEET

A COMPARATIVE STUDY BETWEENPOVIDINE IODINE AND METRONIDAZOLE FOR PERITONEAL LAVAGE IN CASES OF PERITONITIS

Case No.

Particulars of the Patients

Name;

Age;

Sex;

Occupation;

Address;

Ward;

IP No.;

Unit;

Date of Admission;

Date of Discharge;

Complaints

1) **Pain**

-Time of onset

-Mode of onset

-Site of pain

-Migration of pain

-Character of pain

-Relation to vomiting

-Relation to food intake

2) Vomiting

-Onset

-Duration

-Frequency

-Amount

-Character of onset

-Content

3) Bowels

-Last evacuation

-Constipation / Normal

-History of passing worms

4) Distension

-Duration

-Location upper / lower abdomen

-Relation to pain

5) Fever

-Duration

-Nature; Continuous / Intermittent /Remittent

-Relation to pain

-Whether associated with Chills and Rigors

Previous history

- Of similar complaints

-Haematemesis

-Treatment of peptic ulcer

-Ingestion of drugs

Personal History

-Diet

-Appetite

-Smoking

-Alcohol

-Bowel Habits

-Menstrual History

Family History

-Peptic Ulcer

-Diabetes

-Hyper tension

General Examination

-Appearance

-Attitude

-Build and nourishment

-Level of Consciousness

-Dehydration

-Temperature

-Pulse

-Blood Pressure

-Respiration

Local Examination

Inspection

- Contours of the abdomen
    - Normal
    - Distension
        - Uniform
        - Upper
        -Lower
- Visible peristalsis

- Skin

- Umbilicus

- Operation Scars

- Hernial Orifices

- Genitalia

Palpation

-Temperature

-Tenderness

-Localized

-Diffuse

-Rebound

-Muscular rigidity

-Localized

-Generalized

-Mass

-Liver

-Spleen

-Abdominal girth

Percussion

-obliteration of liver dullness

-shifting dullness

Auscultation

-Bowel sounds

-Present

-Frequency

-Character

-Absent

Other relevant examinations

-Per rectal

-Empty

-Loaded

-Bleeding

-Mass felt

-Per vaginal

Other systems

-Respiratory system

-Cardiovascular system

-Central Nervous system

INVESTIGATIONS

Hematological

-Complete blood count

-Blood grouping

-Random blood sugar

-Renal function test

-Serum electrolytes

-Widal

Urine Routine

Diagnostic tapping of peritoneal fluid

Pre operative treatment

-Antibiotics

-Other drugs

-Intravenous Fluids

-Gastric aspiration

Pre medication and Anaesthesia

Operative details

Organism isolated

Bacterial colony count

- Pre lavage

-Post lavage

Complications

-Stitch abscess

-Intestinal obstruction

-Burst abdomen

-Paralytic ileus

-Faecal fistula

Condition at the time of discharge

Follow up

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