"EVALUATION OF MANNHEIM PERITONITIS INDEX IN PATIENTS WITH SECONDARY PERITONITIS"

BY

Dr. SHARVANI L. NAIK

Dissertation submitted to the

B .L.D.E UNIVERSITY, BIJAPUR, KARNATAKA.

In partial fulfillment

of the requirements for the degree of

M. S.

in

General Surgery

Under the guidance of

Dr. B. P. KATTIMANI M.S

ASSOCIATE PROFESSOR, DEPARTMENT OF SURGERY

B. L. D. E. U 'S

SHRI B. M. PATIL MEDICAL COLLEGE HOSPITAL &

RESEARCH CENTRE, BIJAPUR.

2011

DECLARATION BY THE CANDIDATE

I hereby declare that this dissertation entitled **"EVALUATION OF MANNHEIM PERITONITIS INDEX IN PATIENTS WITH SECONDARY PERITONITIS**" is a bonafide and genuine research work carried out by me under the guidance of **Dr. B. P. KATTIMANI** M.S**,** Associate Professor, Department of Surgery at B. L. D. E. U 's Shri. B. M. Patil Medical College Hospital and Research Centre, Bijapur.

Date: **Dr . SHARVANI L. NAIK**

Place: Bijapur

B. L. D. E. U 'S

SHRI B. M. PATIL MEDICAL COLLEGE HOSPITAL & RESEARCH CENTRE, BIJAPUR,KARNATAKA.

CERTIFICATE BY THE GUIDE

This is to certify that the dissertation entitled **"EVALUATION OF MANNHEIM PERITONITIS INDEX IN PATIENTS WITH SECONDARY PERITONITIS"** is a bonafide research work done by **Dr. SHARVANI L. NAIK** under my overall supervision and guidance, in partial fulfillment of the requirement for the degree of **M. S. in General Surgery**.

DR. B. P. KATTIMANI M.S
ASSOCIATE PROFESSOR,
DEPARTMENT OF SURGERY,
B. L. D. E. U 's Shri. B. M. PATIL
MEDICAL COLLEGE HOSPITAL &
RESEARCH CENTRE, BIJAPUR.

Date:

Place: Bijapur

B. L. D. E. U 'S

SHRI B. M. PATIL MEDICAL COLLEGE HOSPITAL &

RESEARCH CENTRE, BIJAPUR.

ENDORSEMENT BY THE HOD / PRINCIPAL

This is to certify that the dissertation entitled **"EVALUATION OF MANNHEIM PERITONITIS INDEX IN PATIENTS WITH SECONDARY PERITONITIS"** is a bonafide research work done by **Dr. SHARVANI L. NAIK** under the guidance of **Dr. B. P. KATTIMANI** M.S**,** Associate Professor, Department of Surgery at B. L. D. E. U's Shri. B. M. Patil Medical College Hospital and Research Centre, Bijapur.

Dr.TEJASWINI U M.S.
PROFESSOR AND HOD
DEPARTMENT OF SURGERY
B. L. D. E. U 's Shri. B. M. PATIL
MEDICAL COLLEGE HOSPITAL
& RESEARCH CENTRE,BIJAPUR

Date:

Place: Bijapur

Dr. R. C. BIDRI M.D
PRINCIPAL,
B. L. D. E. U 's Shri. B. M. PATIL
MEDICAL COLLEGE HOSPITAL
& RESEARCH CENTRE,BIJAPUR

Date:

Place: Bijapur

COPYRIGHT

Declaration by the candidate

I here by declare that the B L D E University, Karnataka shall have the rights to preserve, use and disseminate this dissertation / thesis in print or electronic format for academic/ research purpose.

Date: **Dr. SHARVANI L. NAIK**

Place: Bijapur

ACKNOWLEDGEMENT

*First and foremost, My salutations to God the **ALMIGHTY** , A tangent between zero & infinity - for his divine grace bestowed when needed. With proud privilege and deep sense of respect I express my gratitude and indebtedness to my teacher and guide **Dr. B .P . KATTIMANI** **M.S,** Associate Professor and Prof **Dr. P. L. KARIHOLU** (former guide and former Head), Department of Surgery, B.L.D.E.U'S Shri B. M. Patil Medical College, for their constant inspiration, patience, encouragement and support, which they rendered in preparing this dissertation and in pursuit of my post graduate studies. I sincerely thank my Professor and Head,Department of Surgery **Dr. TEJASWINI .U**.*

*I am forever grateful to my teachers and Professors **Dr. D.C.Patil**, **Dr.B.B.Metan, Dr.M.B.Patil**, D**r.S.B.Patil,** Associate Professors of Surgery; **Dr Manjunath S. Kottenavar, Dr.Vijaya Patil, Dr.Babugouda Nyamannavar,** and also Assistant Professors of Surgery ; **Dr.Vinay Kundargi**, **Dr Prasad Sasnur, Dr. M.S.Kadeangadi, Dr. Ashok Biradar , Dr. Deepak C, Dr. Ramakant B, Dr. Hemanth Kumar , Dr. Vikram** for their valuable help, support and guidance when required during my study.*

*I am grateful to **Dr. R. C. Bidri,** Principal of B.L.D.E.U 'S Shri. B. M. Patil Medical College Hospital and Research Centre, Bijapur, for permitting me to utilize hospital resources during my study period.*

*I am thankful to my seniors **Dr Nataraj.B, Dr. Pavan Patil, Dr. Praveen chandra, , Dr. Sapna, Dr. Digvijay** , my colleagues **Dr Manjunath, Dr Mandar , Dr Rushab, Dr Siddu** and my juniors **Dr. Rashmi Patil, Dr. Uday Karjol, Dr.JayKarthik ,Dr. Amith***

M, Dr.Dayanad *for their co-operation. I also thank* ***Dr. Vijaya B. Wali*** *for her needful help in statistics in my study.*

I express my thanks to one and all in Department of Surgery, medical records section, library staff and all hospital staff for their kind co-operation in my study.

No amount of words can measure up to deep sense of gratitude and fullness that I feel towards my parents ***Dr L.B Naik and Meenakshi Naik,*** *for their cherished blessings and persuasion. I also thank my brother* ***Srinivas Naik,*** *brother-in-laws* ***Dr.Srinivas & Dr. Sandeep Kanakaraddi,*** *Co-sister* ***Dr.Veena*** *whose encouragement and support are behind whatever success I have achieved in my life.*

Last but not the least I thank my husband ***Dr. Ajit Kanakaraddi*** *for all the love and support he has shown. I am indebted to almighty for having given me my son* ***Venkatesh*** *whose smiles were always energizing.*

From the bottom of my heart I convey my heart felt gratitude to ***all my patients*** *without whose co-operation this study would have been incomplete.*

I also thank Mr Kalyan Kumar ,Preethi Net Zone,Bijapur for computerizing my dissertation patiently.

Date: **Dr. SHARVANI L. NAIK**

Place: Bijapur

LIST OF ABBREVIATIONS USED

BP	-	Blood pressure.
BT	-	Bleeding time.
CT	-	Clotting time.
DC	-	Differential count.
ESR	-	Erythrocyte sedimentation rate.
Hb	-	Haemoglobin.
IP	-	In patient number.
LFT	-	Liver function test.
N	-	No
PA	-	Per abdomen.
PR	-	Pulse rate.
PR	-	Per rectum.
PO2	-	Partial Pressure of Oxygen
RBS	-	Random blood sugar.
TC	-	Total count.
Y	-	Yes

ABSTRACT

Background and Objectives :

Peritonitis is still one of the most important infectious problems that a surgeon has to face. Despite the progress in the anti-microbial agents and ICUs, present mortality due to peritonitis continues to be unacceptably high.

Reproducible scoring systems that allows a surgeon to determine the severity of the intra abdominal infections are essential to:

a. Rarify the effectiveness of different treatment regimens,

b. Scientifically compare surgical ICUs,

c. Help indicate individual risk to select patients who may require a more aggressive surgical approach,

d. To be able to inform patients' relatives with greater objectivity.

Mannheim peritonitis index (MPI) is one of the most simple scoring systems that allows a surgeon to easily determine the outcome of risk during initial surgery.

The present study is planned to study the role of MPI scoring systems in patients of secondary peritonitis in our hospital and to correlate the final outcome of surgical treatment with score.

Materials And Methods:

The prospective study was undertaken at BLDEU's Shri B. M. Patil Medical College, Bijapur from October 2008 to May 2010. A total of 45 patients being operated for Secondary Peritonitis were included in the study. Depending on symptoms, investigations and intra operative findings each patient was given an MPI score. The MPI scores were used to predict the prognosis and surgical outcome of the patients.

Results:

In this study increasing MPI scores were associated with increased rate of mortality. Organ failure was the only independent risk factor to have an adverse effect on the prognosis.

Conclusion:

MPI provides simple and objective means to predict outcome of patients with peritonitis. The simplicity of MPI makes it ideal for hospital with various shortages to assess the prognosis of the patients.

Key words: Secondary peritonitis, MPI score and prognosis.

LIST OF CONTENTS

S. No	Topic	Page No
1	INTRODUCTION	1
2	AIMS AND OBJECTIVES	2
3	REVIEW OF LITERATURE	3
4	METHODOLOGY	35
5	RESULTS	36
6	DISCUSSION	51
7	SUMMARY	58
8	CONCLUSION	59
9	BIBLIOGRAPHY	60
10	ANNEXURES ➢ PROFORMA ➢ CONSENT FORM ➢ KEY TO MASTER CHART ➢ MASTER CHART	 63 67 71 72

LIST OF TABLES

Table No.	Description	Page No
1.	Age distribution of patients studied.	36
2.	Sex distribution of patients studied.	37
3.	Duration of presenting symptoms.	38
4.	Presence of organ failure in the patients studied.	39
5.	Presence of diffuse peritonitis during surgery.	40
6.	Types of exudates during surgery.	41
7.	Site of perforation.	42
8.	Findings of Malignancy during surgery .	43
9.	Mean hospital stay.	44
10	Local complications seen in patients.	45
11	Outcome of study.	46
12	Cause of death.	47
13	Mortality in severity group.	48
14	Chi-square test.	48
15	Association of Risk factors with mortality.	50

LIST OF GRAPHS

Graph. No.	Description	Page No
1.	Age distribution of patients studied.	36
2.	Sex distribution of patients studied.	37
3.	Duration of presenting symptoms.	38
4.	Presence of organ failure in the patients studied.	39
5.	Presence of diffuse peritonitis during surgery.	40
6.	Type of exudates during surgery.	41
7.	Site of perforation.	42
8.	Findings of Malignancy during surgery .	43
9.	Mean hospital stay.	44
10	Local complications seen in patients.	45
11	Total mortality rate.	46
12	Mortality in severity group.	49

LIST OF FIGURES

Figures No.	Description	Page No
1.	Anatomy of the peritoneal cavity.	9
2.	Spaces in the peritoneal cavity.	11
3.	Arterial supply of duodenum.	17
4.	Arterial supply of jejunum and ileum.	17
5.	Arterial supply of large bowel.	18
6.	Perforation over second part of duodenum.	54
7.	Stomach perforation with multiple ulcers below it.	55
8.	Closure of stomach perforation.	55
9.	Perforation of ileum.	56

INTRODUCTION

Peritonitis is associated with a high mortality rate despite its surgical treatment, sophisticated intensive care units, good antibiotics and a better understanding of peritonitis and pathophysiology.

The outcome of an abdominal infection depends on the complex interaction of many different factors and the success obtained with the early onset of specific therapeutic procedures. It may also depend upon exact recognition of the seriousness of the disease, an accurate assessment and classification of the patient's risks.

The symptoms are often non-specific and are influenced by the age of the patient, medications and co-existing diseases. Early prognostic evaluation of abdominal sepsis is desirable to select high risk patients for more aggressive therapeutic procedures such as radical debridement, lavage systems, open management, and planned reoperations.

The reaction of the closed peritoneal cavity, divided into various stages is a sincere effort on the part of the body to maintain as close an internal milieu as possible and the stage of a neglected perforation is culmination of the victory of fear over this hope[1].

Many score systems have been created for assessing patient risks of death during an event of peritonitis, equal results have been achieved with the Mannheim peritonitis index (MPI).

AIMS AND OBJECTIVES

To evaluate the reliability of the Mannheim Peritonitis Index (MPI) in predicting the outcome of patients with peritonitis and to assess each risk factor independently regarding its contribution towards final outcome in 45 cases of secondary peritonitis.

REVIEW OF LITERATURE

HISTORY

Perforation of hollow viscera with concomitant peritonitis was treated with application of warm oil and starvation in the Egyptian era. Needless to say that the mummies examined with these features are proof enough of failure of this therapy[2].

"The patient looks sick and wasted. The nose is pointed, the temples sunken, the eyes lay deep, are rimmed and dull. The face expresses fear, the tongue is furred, the skin shiny. The patient avoids all movements and breathes shallowly. The abdominal wall is rigid with muscular guarding, no bowel sounds can be heard. The pulse is quick and small. A hard, tender mass in the hypochondrium is a bad prognostic sign if it involves the whole area. This presence of such a mass at the beginning of the fever indicates that death is imminent this keen clinical observation and the emphasis on prognosis, resulted in the first description of the peritonitis, during the era of Koic medicine. If one reads this account closely one can distinguish between the description of the local signs of diffuse peritonitis and "Systemic Inflammatory Response Syndrome" (SIRS). Unfortunately, this description was not intended to treat the disease, but to prognosticate the certain death[3].

In England the pioneering work of Howard & Dickenson, Bennet & Page in 1890 brought about standardization in the procedure for closure of specific perforations and the usage of huge quantities of warm water to lavage the peritoneal cavity.

In 1926, the fundamental role of operative therapy in the treatment of peritonitis was documented. Kirschner (1926) reported that the mortality rate from

intra-abdominal infections decreased from more than 90% to less than 40% during the period from 1890 - 1924 with the introduction of operative management as an effective therapeutic modality.

DEVELOPMENTAL ANATOMY

Development of the Peritoneum and the Peritoneal Cavity :

Once the lateral mesoderm has split into somatic and splanchnic layers, a cavity is formed between the two called as the intraembryonic coelom .The peritoneal cavity is derived from that part of the embryonic coelom situated caudal to the septum transversum.

In its earliest stage, the peritoneal cavity is in free communication with the extraembryonic coelom on each side. Later with the development of the head, tail, and lateral folds of the embryo, this wide area of communication becomes restricted to a small area within the umbilical cord.

Early in development, the peritoneal cavity is divided into right and left halves by a central partition formed by the dorsal mesentery, the gut, and the small ventral mesentery. However, the ventral mesentery extends only for a short distance along the gut so that below this level the right and left halves of the peritoneal cavity are in free communication. As a result of the enormous growth of the liver and the enlargement of the developing kidneys, the capacity of the abdominal cavity becomes greatly reduced at about the sixth week of development. It is at this time that the small remaining communication between the peritoneal cavity and extraembryonic coelom becomes important. An intestinal loop is forced out of the abdominal cavity through the umbilicus into the umbilical cord. This physiologic herniation of the midgut takes place during the sixth week of development[4].

Formation of the Peritoneal Ligaments and Mesenteries:

The peritoneal ligaments are developed from the ventral and dorsal mesenteries. The ventral mesentery is formed from the mesoderm of the septum transversum.. It forms the falciform ligament, the lesser omentum, the coronary and triangular ligaments of the liver.

The dorsal mesentery is formed from the fusion of the splanchnopleuric mesoderm on the two sides of the embryo, it extends from the posterior abdominal wall to the posterior border of the abdominal part of the gut. The dorsal mesentery forms the gastrophrenic ligament, the gastrosplenic ligament, the splenicorenal ligament, the greater omentum, and the mesenteries of the small and large intestines.

Formation of the Lesser and Greater Peritoneal Sacs:

The extensive growth of the right lobe of the liver pulls the ventral mesentery to the right and causes rotation of the stomach and duodenum. By this means, the upper right part of the peritoneal cavity becomes incorporated into the lesser sac. The right free border of the ventral mesentery becomes the right border of the lesser omentum and the anterior boundary of the entrance into the lesser sac.

The remaining part of the peritoneal cavity which is not included in the lesser sac is called the greater sac and the two sacs are in communication through the epiploic foramen[4].

Formation of the Greater Omentum:

The greater omentum is formed as a result of the rapid and extensive growth of the dorsal mesentery caudal to the spleen. To begin with, the greater omentum extends from the greater curvature of the stomach to the posterior abdominal wall superior to the transverse mesocolon. With continued growth, it reaches inferiorly as an apron like double layer of peritoneurn anterior to the transverse colon.

Later the posterior layer of the omentum fuses with the transverse mesocolon; as a result, the greater omenturn becomes attached to the anterior surface of the transverse colon[4].

PERITONEUM

General Arrangement:

The peritoneum is a thin serous membrane that lines the walls of the abdominal and pelvic cavities and covers the viscera. The parietal peritoneum lines the walls of the abdominal and pelvic cavities and the visceral peritoneum covers the organs. The potential space between the these layers is called the peritoneal cavity.

In males this is a closed cavity but in females there is a communication with the exterior through the uterine tubes, the uterus, and the vagina.

Between the parietal peritoneum and the fascial lining of the abdominal and pelvic walls is a layer of Connective tissue called the extraperitoneal tissue, in the area of the kidneys this tissue contains a large amount of fat, which supports the kidneys.

The peritoneum secretes a small amount of serous fluid called the peritoneal fluid, which lubricates the surfaces of the peritoneum and allows a free movement between the viscera[5].

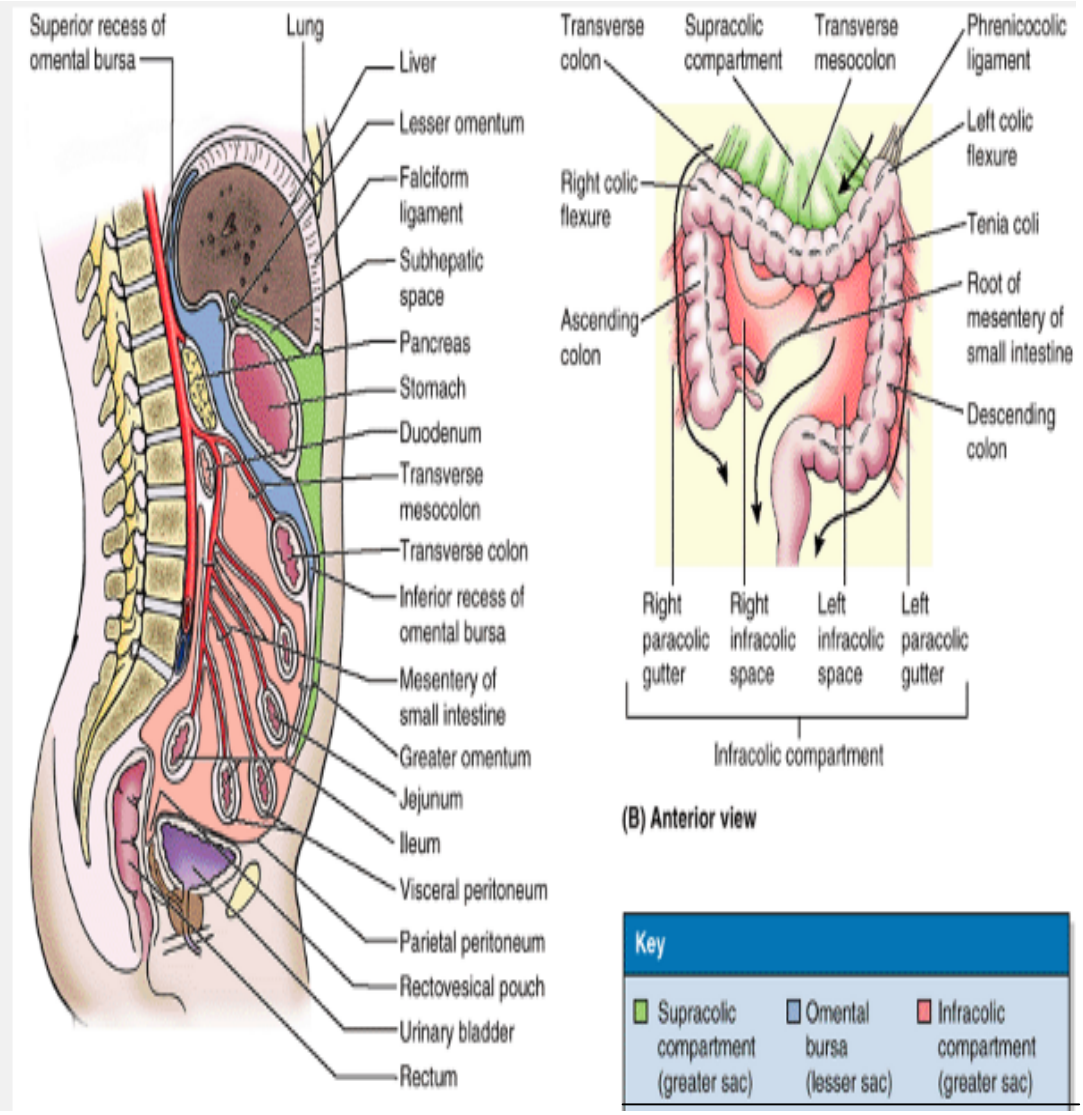


Figure 1 : Anatomy of the peritoneal cavity.

Special Regions Of The Peritoneal Cavity :

Subphrenic Spaces : A. The intraperitonal Spaces are (1) The left anterior space; (2) the left posterior space; (3) the right anterior space; and (4) the right posterior space.

B. The extraperitoneal space includes: (1) the right extra peritoneal space; and (2) the left exraperitoneal space.

Subhepatic Space (Morison's Pouch) : Boundaries (A) Anteriorly: (1) the inferior surface of the right lobe of the liver; and (2) the gall bladder ;(B) Posteriorly: (1) the right suprarenal gland;(2) the upper part of the right kidney; (3) the second part of the duodenum; (4) he hepatic flexure of the colon; (5) the transverse mescolon;(6) a part of the head of the pancreas; (C) Superiorly: the inferior layer of the coronary ligament ;(D) Inferiorly it opens into the general peritoneal cavity.

This space is of importance as it is the most dependent part of the abdominal cavity when the body is supine. Fluids tend to collect here. This is the commonest site of subpherenic abscess, which may be caused by spread of infection from the gall bladder, appendix or other organs in the region.

Infra Colic Compartment : The right one lies between the ascending colon and mesentery below the transverse mesocolon ,the left one lies between the ascending colon and mesentery.

Paracolic Gutter: The right one opens freely into the hepatorenal pouch at the upper end. The left one opens freely into the pelvis at the lower end[6].

Pelvic Cavity : Being the most dependent part of the peritoneal cavity pus tends to collect here.

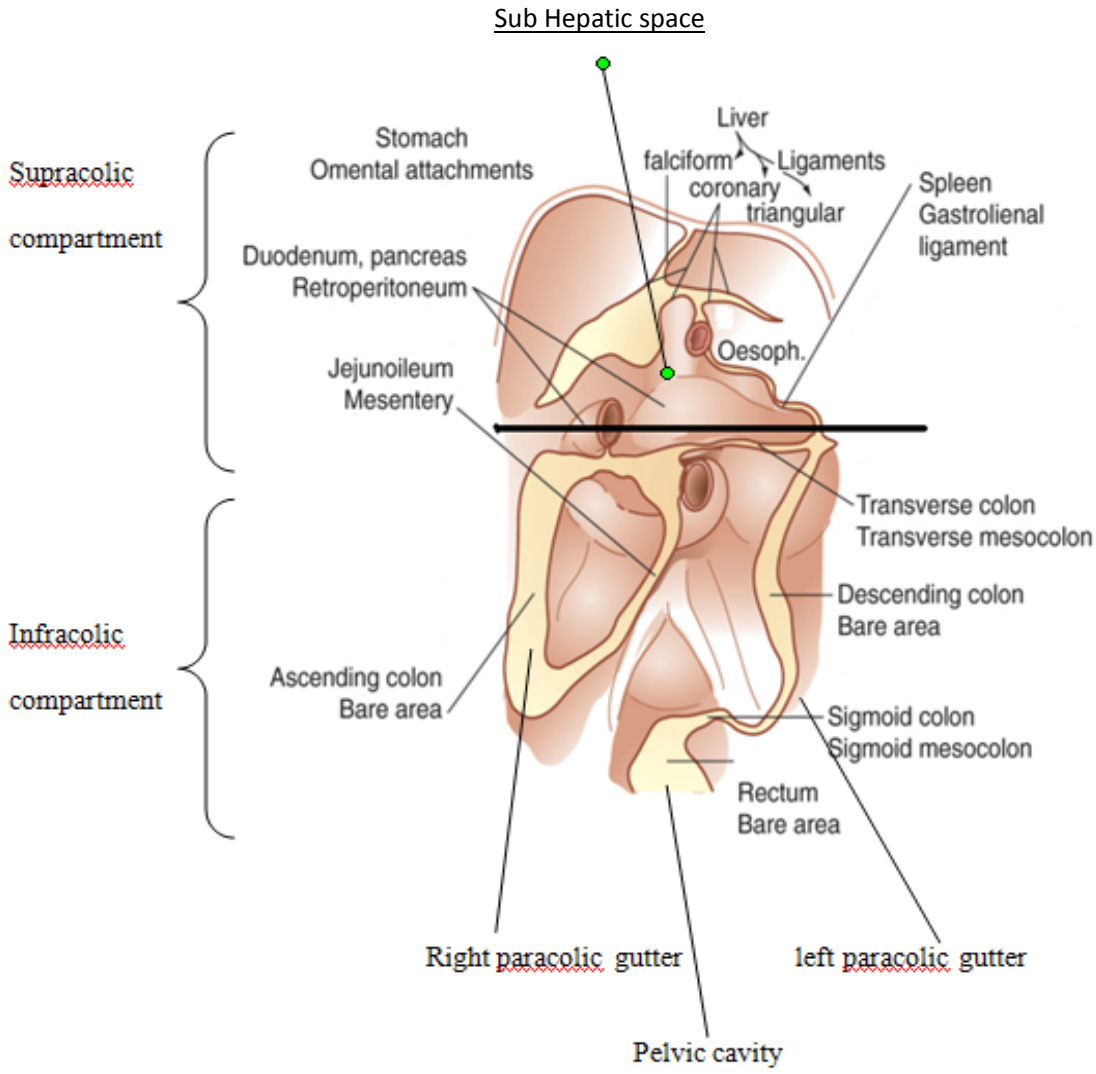


Figure 2 : Spaces in the peritoneal cavity.

Intraperitoneal and Retroperitoneal Relationship :

An organ is said to be intraperitoneal when it is almost totally covered with visceral peritoneum. The stomach, jejunum, ileum, and spleen are examples of intraperitoneal organs. Retroperitoneal organs lie behind the peritoneum and are only partially covered with the visceral peritoneum. The pancreas, the ascending and descending parts of the colon are examples of retroperitoneal organs. No organ however is actually within the peritoneal cavity.

Peritoneal Ligament:

Peritoneal ligaments are two-layered folds of peritoneum that connect solid viscera to the abdominal walls. For example liver is connected to the diaphragm by the falciform ligament, the coronary ligament, the right and left triangular ligaments[5].

Omentum :

Omenta are two-layered folds of peritoneurn that connect the stomach to another viscus. The greater ornentum connects the greater curvature of the stomach to the transverse colon. It hangs down like an apron in front of the coils of the small intestine and is folded back on itself to be attached to the transverse colon. The lesser omentum suspends the lesser curvature of the stomach from the fissure of the ligamentum venosum and the porta hepatis on the undersurface of the liver[5].

The gastrosplenic omentum connects the stomach to the hilum of the spleen.

Nerve Supply of the Peritoneum :

The parietal peritoneum is sensitive to pain, temperature, touch, and pressure. Its lining on the anterior abdominal wall is supplied by the lower six thoracic and first lumbar nerve, the nerves that innervate the overlying muscles and skin. The parietal peritoneum in the pelvis is mainly supplied by the obturator nerve, a branch of the lumbar plexus.

The visceral peritoneum is sensitive only to stretch and tearing and is not sensitive to touch, pressure, or temperature. It is supplied by autonomic afferent nerves that supply the viscera or are traveling in the mesenteries. Overdistention of a viscus may lead to the sensation of pain. The mesenteries of the small and large intestines are sensitive to mechanical stretching.

The central part of the diaphragmatic peritoneum is supplied by the phrenic nerves. Peripherally the diaphragmatic peritoneum is supplied by the lower six thoracic nerves[5].

ANATOMY OF THE ALIMENTARY CANAL

The oesophagus projects through the diaphragm at the level of the seventh costal cartilage, a thumb breadth below and to the left of the sternum. The right and left gastric nerves (vagi) lie on its surface. It is invested by peritoneum which passes to the right as lesser omentum and to the left as upper part of the greater omentum. It enters the stomach at the cardiac orifice.

The stomach is a muscular bag fixed at both ends and mobile in between. It consists of fundus, body, pyloric antrum and pylorus. The fundus is that part which projects upwards and is in contact with the diaphragm above the level of the cardiac orifice. The body extends from the fundus to the level of the incisura angularis. The pyloric antrum extends from this level & narrows towards the pylorus.

The stomach has an outer longitudinal muscle coat and an inner circular coat and in between lies the oblique muscle coat. The stomach is covered by peritoneum all around except for a small area posteriorly called the bare area.

The duodenum (12 fingers) has a first part, which is 2 inches long and is peritonealised. It runs upwards and towards the right. The second part is 3 inches long and runs downwards forming a “C” with the third part of duodenum and containing within it the head of pancreas.

The superior mesenteric artery crosses the third part anteriorly. The fourth part crosses the aorta and turns left lying on the left psoas muscle and the left lumbar sympathetic chain.

It breaks free being covered by peritoneum and leads to the duodeno- jejunal flexure.

The small intestine (other than duodenum) is about 6 meters long. It is made up of jejunum (upper two fifths) and ileum (lower three fifths).

The jejunum is thicker than the ileum and occupies the upper part of the infracolic compartment, the ileum occupying the pelvis and mid- abdomen.

The mucosa is thrown into folds, the valves of Kerkring. These along with the villi increase the absorptive surface of the jejunum. They are finger like projections in the jejunum and club shaped and sparse in the ileum.

The caecum is a blind proximal pouch of the large intestine with the appendix attached to its infero-medial aspect.

The three longitudinal muscle coats converge (as taenia) at the base of the appendix.

The ascending colon is about 6 inches in length extending from the ileo-caecal junction to the right colic flexure .It lies on the iliac fascia being connected and fixed to it by the connective tissue of the extra peritoneal fascial envelope. Bulbous pouches of fat in the peritoneum project in places as ‘appendices epiploicae’.

The transverse colon is about l8 inches long and extends from the hepatic to the splenic flexure. It is quite mobile and is in contact with anterior abdominal wall. The greater omentum hangs down from its lower convexity.

The descending colon, which is about 12 inches long, extends from the splenic flexure to the pelvic brim and is retroperitoneal.

The sigmoid colon too is peritonealised and extends form the pelvic brim to the rectum and is about 18 inches in length.

The apex of the sigmoid mesocolon lies on an inverted 'V' at the bifurcation of the common iliac artery, over the sacro-iliac joint at the pelvic brim.

The word rectum, which means 'straight' is a misnomer. As the mesentery of the sigmoid ends, the rectum begins. The three taenia of the colon - taenia libera, taenia omentalis, taenia mesocolica, come together and form a complete outer layer of longitudinal muscle. It starts at the level of the 3rd piece of coccyx and passes through the pelvic floor into the anal canal behind the perineal body[5].

Blood Supply:

The left gastric artery (branch of celiac) and the right gastric artery (branch of the common hepatic artery) supply the stomach. Along the greater curvature is the left gastroepiploic artery a branch from the splenic artery and right gastroepiploic artery a branch from the gastro duodenal artery.

The short gastric vessels 5-7 in number supply the cardiac end and are branches from the splenic artery.

The superior and inferior pancreatico-duodenal arteries, branches of the gastroduodenal and superior mesenteric arteries respectively supply the duodenum and pancreas.

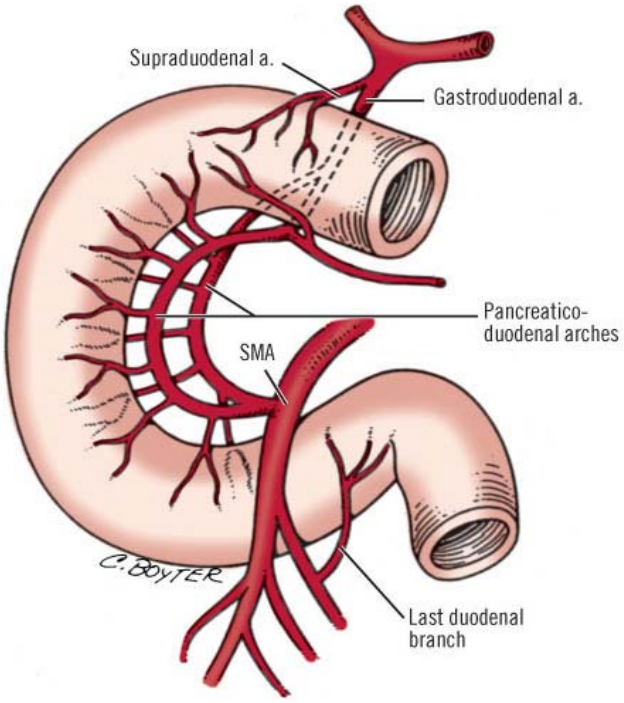


Figure 3 : Arterial supply of duodenum.

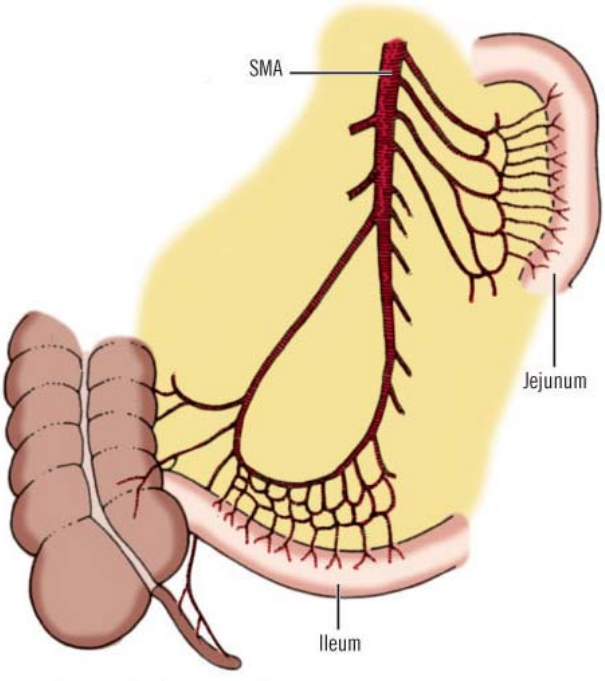


Figure 4 : Arterial supply of jejunum and ileum.

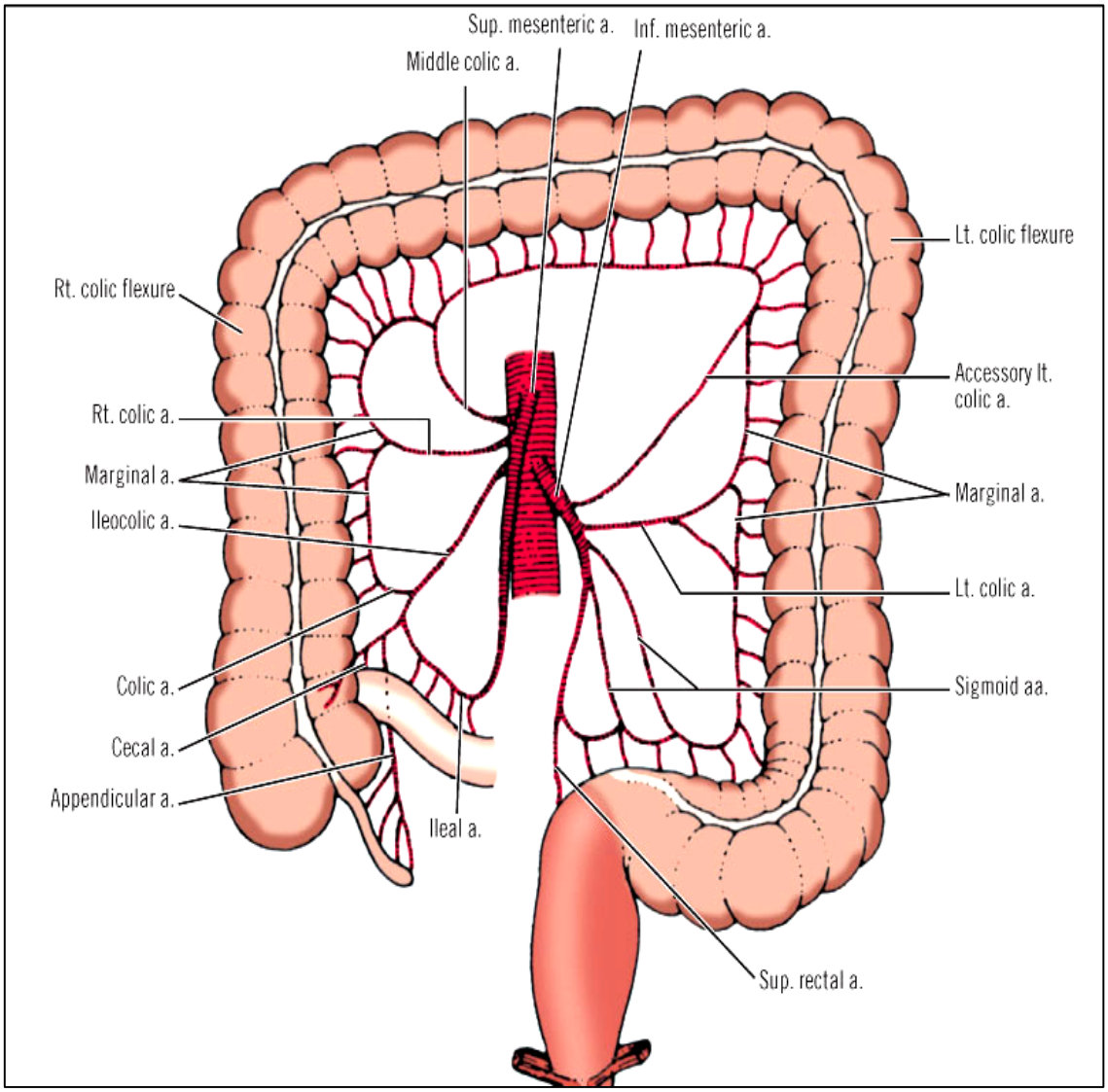


Figure 5 : Arterial supply of large bowel.

The venous drainage of the stomach follows the arteries with the exception that there is no gastro duodenal vein. They drain into the portal system.

The jejunal and ileal branches of the superior mesenteric artery supply the jejunum and ileum. These branches form arcades “vasa Recta” which are long and far apart in the jejunum and become narrow and short towards the ileurn.

The ilio-colic artery is a branch of the superior mesenteric artery, which gives an ileal branch that anastomoses with the terminal branch of the superior mesenteric artery. The other branch of ascending colic artery supplies the caecum, appendix and a part of the ascending colon.

The right colic artery, a branch of the superior mesenteric artery divides into an ascending and a descending branch that anastomose with the ileocolic artery and middle colic artery respectively. It supplies the ascending colon and the right third of the transverse colon. The middle colic artery, a branch of the superior mesenteric artery supplies the transverse colon up to the splenic flexure.

The descending colon is supplied by the left colic artery, a branch of the inferior mesenteric artery that later gives a sigmoid artery branch which supplies the sigmoid colon. The inferior mesenteric artery continues as the superior rectal artery supplying the rectum. The remainder of the lower rectum and the anal canal are supplied by the internal iliac system via the pudendal system through the via media of the middle and inferior rectal arteries[7].

The middle rectal artery, a branch of the internal iliac artery supplies the lower rectum and anal canal.

Venous drainage follows corresponding arteries and drains into the portal system.

Histology :

The stomach consists of a serosal layer, a muscular layer, submucosa and mucosa. The muscle coat consists of an outer longitudinal, middle circular and an inner oblique layer. The circular layer forms the pyloric sphincter. The longitudinal layer is continuous with that of the oesophagus and duodenum. The mucosa is thrown into folds forming a 'MAGENSTRASSE' along the lesser curvature, a gutter for fluid drainage. The mucosa has chief cells that secrete pepsinogen and oxyntic cells which secrete intrinsic factor and hydrochloric acid.

The small intestine has three layers :mucosa, muscularis propria and serosa. The mucosa is lined by columnar cells, intestinal glands which secrete digestive enzymes and goblet cells which secrete mucous. The crypts of Leiberkuhn have simple tubular cells secreting digestive enzymes. Argentaffin cells are neuroendocrine cells regulating secretion in a paracrine fashion.

The mucosa is thrown into villi, finger like projections that increase the surface area of absorption. Lymphatic follicles are arranged in the mucosa singly or in aggregates as Peyer's patches. They are circular or oval in shape containing 100-250 follicles and are about 2-10 cm in length ;more numerous towards the ileum.

The large intestine has a serosal and a muscle coat whose 6 longitudinal layers are arranged as three taenia. The serosa has outpouches of fat called appendices epiploicae. The mucosa is predominantly mucous secreting and has a limited absorptive role[8].

Functions of the Peritoneum :

The peritoneal fluid is a pale, viscid fluid containing leukocytes. It is secreted by the peritoneum and ensures that the mobile viscera glide easily on one another. As a result of the movements of the diaphragm and the abdominal muscles, together with the peristaltic movements of the intestinal tract, the peritoneal fluid is not static. It seems that intraperitoneal movement of fluid toward the diaphragm is continuous and there it is quickly absorbed into the subperitoneal lymphatic capillaries.

This can be explained on the basis that the area of peritoneum is extensive in the region of the diaphragm and the respiratory movements of the diaphragm aid lymph flow in the lymph vessels[8].

The peritoneal coverings of the intestine tend to stick together in the presence of infection. The greater omentum which is kept constantly on the move by the peristalsis of the neighbouring intestinal tract, it may adhere to a peritoneal surfaces around the focus of infection. In this manner, many of the intraperitoneal infections are sealed off and remain localized[9].

The peritoneal folds play an important part in suspending the various organs within the peritoneal cavity and serve as a means of conveying the blood vessels, lymphatics, and nerves to these organs.

It is also a storehouse of fat.

MODE OF INTRA ABDOMINAL SEPSIS

Peritonitis causes a reduction in the intra-abdominal fibrinolytic activity (increased plasminogen activator inhibitor activity) and fibrin sequestration with subsequent adhesion formation. The production of fibrinous exudates is considered an important part of the host defense, but large number of bacteria may be sequestered within this fibrin matrix. This may lead to retardation of spread and systemic dissemination and may decrease early mortality rates from sepsis, but it also is integral to the development of residual infection and abscess formation. As the fibrin matrix matures, the bacteria within are protected from host clearance mechanisms.

The ultimate effect (containment vs persistent infection) of fibrin may be related to the degree of peritoneal bacterial contamination.

Abscess formation has been viewed as a host defense strategy to contain the spread of infection; however, this process can lead to persistent infection and life-threatening sepsis.

The initiation of abscess formation involves the release of bacteria and an abscess potentiating agent into a normally sterile environment. The host defence is unable to eliminate the infecting agent and attempts to control the spread by compartmentalization. This process is aided by a combination of factors that share a common feature i.e. impairment of phagocytic killing[10].

Transient bacterial peritoneal contamination (caused by primary visceral disease and intentional or unintentional violation of the gut) is common. The resultant exposure to bacterial antigens has been shown to alter subsequent immune responses

to recurrent peritoneal inoculation. This may lead to an increased incidence of abscess formation, alteration of the bacterial content and increased late mortality rates[9].

MICROBIOLOGY

Bacterial virulence factors that interfere with phagocytosis and neutrophil-mediated bacterial killing are important mediators leading to persistence of infections and abscess formation. Among these factors are capsule formation, facultative anaerobic growth, adhesion capabilities, and succinic acid production. Synergy between certain bacterial and fungal organisms may also play an important role in impairing the host's defense. One such synergy may exist between *B fragilis* and gram-negative bacteria, particularly *E coli* where co-inoculation significantly increases bacterial proliferation and abscess formation.

Enterococci may be important in enhancing the severity and persistence of peritoneal infections. Abdominal infections, particularly with Candida species are becoming increasingly common in critically ill patients. Additional common peritoneal organisms in this patient population are Enterococcus, Enterobacter species and Staphylococcus epidermidis.

Existing data suggest that bacterial peritonitis is associated with an immense intraperitoneal compartmentalized cytokine8 response. Higher levels of certain cytokines (i.e tumor necrosis factor-alpha [TNF-alpha], interleukin [IL]-6) have been associated with worse outcomes, as well as secondary (uncontrolled) activation of the systemic inflammatory cascade[9,10].

CLINICAL FEATURES AND STAGES OF PERITONITIS

1. Primary Stage (Stage of peritonism) : This is stage of irritation. It occurs due to the sudden leak of fluid into the peritoneal cavity. The pain may be in the right hypochondrium and epigastrium due to perforation of a duodenal ulcer or in the left hypochondrium due to gastric ulcer perforation. There may be a syncopal attack. The pain may radiate to the back in case of a perforation into the lesser sac. There may be shoulder tip pain with the pain later becoming generalised. There may be associated nausea and vomiting[11].

On examination, the patient lies still with sweating , tachypnoea and tachycardia, abdominal guarding and rigidity may be evident, but may be absent in cases of pelvic peritonitis. Liver dullness obliteration is based on the amount of gaseous escape[12].

Abdomen may be lax in multipara and elderly. A leaking duodenal ulcer can give rise to drainage along the right paracolic gutter 'Moynihan's Gutter Sign' or 'Valentino's Appendicitis'.

2. Secondary Stage (Stage of reaction): It lasts for six hours. The escaping fluid is neutralized by peritoneal reaction and gives the doctor a sense of false security as the signs and symptoms abate. Liver dullness may be obliterated; muscles may be soft. Shifting dullness and paralytic ileus set in. Per-rectal examination elicits tenderness and erect chest x-ray shows pneumoperitoneum.

3. Stage of Bacterial Peritonitis: The leaking fluid carries with it bacteria from the oesophagus, stomach, small and large intestines. Transudation ensues. The fluid

becomes purulent.The bowels become oedematous. Patient has fever with chills, hypoxia and renal failure passing into a stage of paralytic ileus if untreated.

4. Stage of Peritonitis: Patient is in septic shock with paralytic ileus. He has a characteristic appearance, the 'facies hippocratica', with an anxious look, wide eyes, rising pulse rate and falling blood pressure.

"FORME FRUSTE" occurs in a sub-acute perforation that becomes walled off immediately, tenderness and rigidity are localised. The ulcer may perforate again and give rise to signs of frank peritonitis.

A chronic, perforation will not provide any specific signs of peritonitis as it gets walled off.

Origin of peritonitis due to different anatomic sites and different etiology:

Perforated ulcer/carcinoma of stomach

Small/large bowel perforation

Appendicitis

Acute pancreatitis

Acute Cholecystitis

Uterus and adnexal pathology: ruptured ectopic pregnancy, follicle/ovarian cysts (hemorrhage/ cyst rupture), salphingitis, pelvic inflammatory disease Complicated hernias, strangulated inguinal hernia/incarcerated umbilical hernia[11].

INVESTIGATIONS

1. A complete blood picture with blood grouping; urine examination.

2. Radiography: Ideally an erect chest x-ray. One cc of air can be detected on x-ray in 80% of cases especially in left lateral decubitus position (in case the patient cannot stand erect). On an erect chest x-ray other causes of gas under the diaphragm should be considered, viz, liver abscess caused by gas forming organisms, recent pneumoperitoneum due to laparoscopy or open surgery. "Chiladiti" sign (interposition of colon between the liver and diaphragm.).

3. Ultrasound/CT scan in case of sub-acute or chronic perforation

4. Instillation of gastrograffin through a Ryle's tube in case of a suspected perforation when no gas under the diaphragm is present.

Conventional radiography still remains the method of choice for a suspected gastro-duodenal perforation. A CT scan can be helpful in detection of free gas in the abdomen, if at least a 6 hours delay after radiographic examination has been allowed. Ultrasound can mainly demonstrate intraperitoneal fluid and intestinal paresis[11].

TREATMENT

Successful treatment ultimately depends upon effective early surgery together with appropriate intensive supporting care.

Preoperative Preparation:

Resuscitation:

The plasma volume must be restored and plasma electrolyte concentration corrected. All patients should undergo urinary catheterization and the restoration of a urine output (30-50ml/hr) is the best indicator of adequate initial resuscitation.

Gastrointestinal Decompression:

A nasogastic tube is passed into the stomach and aspirated. Intermittent aspiration is maintained.

Antibiotic Therapy:

Parenteral broad spectrum antibiotics active against both aerobic and anaerobic should be given.

Ventilation:

In any patient with severe generalized peritonitis there is poor diaphragmatic movement and some impairment of tissue perfusion and oxygen should be given via a face mask.

Vital System Support :

Particularly in older patients a close watch must be kept for pulmonary edema and atrial fibrillation. Renal function needs to be monitored carefully[11].

Surgical Therapy:

The operative approach is directed by the underlying disease process and the type and severity of the intra-abdominal infection.

A vertical midline incision is the incision of choice in most patients with generalized peritonitis for easy access. In patients with localized peritonitis (e.g., acute appendicitis[16]), an incision directly over the site of pathology (e.g., right lower quadrant) is usually adequate. The inflammed organs are often very friable, and the surgeon must exercise great caution when exploring the patient with peritoneal infection. Careful dissection and meticulous haemostasis are of utmost importance.

When faced with extensive abdominal inflammatory disease and septic shock, draining the infection temporarily and controlling the visceral leak quickly (e.g., over sewing,primary closure,resection and anastomosis)is the principle line of management. Definitive repair is deferred in case of unstable patients,grossly contaminated peritoneal cavity ,suspicion about the viability of the bowel[13,14,15]. Abdomen is best suited for closure by mass closure technique[15].

Peritoneal Lavage :

After the cause for peritonitis has been dealt with, the entire peritoneal cavity is explored with sucker and mopped dry. Use of 1 – 2 liters of saline to wash the peritoneum has been found to be very effective.

Laparoscopy :

Laparoscopy is gaining wider acceptance in the diagnosis and treatment of abdominal infections. No definitive guidelines have been established regarding the optimal selection of patients for successful laparoscopic repair.

PROGNOSIS AND COMPLICATIONS

With the modern treatment, diffuse peritonitis due to hollow viscus perforation carries a mortality of about 10 % .

1. Systemic complications :

- Bacteraemic or endotoxic shock
- Broncho pneumonia or respiratory failure
- Renal failure
- Bone marrow suppression
- Multi system failure

2. Abdominal complications :

- Adhesional small bowel obstruction
- Paralytic ileus
- Residual or recurrent abscess
- Portal pyaemia / liver abscess.
- Surgical site infection
- Wound dehiscence/burst abdomen
- Entero cutaneous fistula [11]

SCORING SYSTEMS IN ABDOMINAL SEPSIS

In the last two decades, several prognostic scores for abdominal sepsis have been proposed for prediction of final outcome. These systems measure derangement in various physiological factors representing functions of major organ systems. Numerical points are given for the severity of deviation from normal, and outcome of the disease is predicted by the sum of the points of all factors. Some of them are[17]:

Multi Organ Failure Score (MOF) :

Because organ dysfunction and failure evolve in patients with sepsis, organ function is monitored routinely in intensive care patients. In 1985, Goris et al published the Multiple Organ Failure (MOF) score that grades patients on a three-point scale. The MOF score takes into consideration dysfunction of the pulmonary, cardiovascular, hepatic, renal, nervous, hematological and gastrointestinal (GI) systems; however, in a recent revision, GI and nervous systems have been excluded. This multiple organ dysfunction score, constructed using simple physiologic measures of dysfunction in six organ systems, and correlates strongly with the ultimate risk of mortality due to peritonitis in ICU[18].

Acute Physiological and Chronic Health Evaluation Score (APACHE) II :

In 1982, Knaus and others, proposed a scoring system to be used for patients admitted to I C U . In consultation with a large number of intensive care specialists, they derived a two part scale which includes a physiological portion, the acute physiological assessment which examines abnormalities among possible physiological measurements, obtained during the first day of admission to the I C U. The second part of score is a chronic health evaluation. The doctor examines the

patient's pre admission health by reviewing the medical history for details concerning functional status, productivity and medical attention during the six months before admission. The combination of two is the APACHE.

The APACHE score provides a pre treatment estimate of risk which is appropriate for the stratification of patients in scientific studies. Sequential scoring is used primarily to monitor the patients course. It is assumed that patients, who respond to the treatment will decrease their score while non responders continue to have higher or increasing scores. This system can be adopted for risk stratification of patient with intra abdominal infections. This provides an initial stratification of risk factors and a predictive equation to estimate patient outcome. They are, however, both complex and time consuming. So use of APACHE II score in under - staffed and under equipped circumstances is not practical[19,20].

Simplified Acute Physiology Score(SAPS):

Le Gall et al developed this score.It is composed of 14 easily measured physiological variables and the score ranges from 0-56[21,22].

Sepsis Severity Score (SSS) :

It was published by Elebute and Stoner in 1983 consisting of 4 components grading various effects of sepsis. There is multiple scoring of certain components as both underlying cause and secondary effects are included in the scoring[23].

Mannheim Peritonitis Index (MPI) :

It is a scoring system with prognostic value usually applied to patients with peritonitis.

The Mannheim peritonitis index is based on data from 1253 patients with peritonitis treated between 1963 and 1979 and was developed by discriminate analysis of 17 possible risk factors. Eight (shown below in the table) of these were of prognostic relevance and were entered into the current index, with a weighting according to the predictive power.

MPI was published in 1986 by Wacha H and Linder based on analysis of risk factors in patients with peritonitis[24].

The MPI score were calculated at admission or during management[25]. It is calculated for each patient on a pre-designed proforma and the patients were followed-up till death or discharged from the hospital.

Prognosis index is based on patient state at discharge.

The MPI is a specific score, which has a good accuracy and provides an easy way to handle with clinical parameters, allowing the prediction of the individual prognosis of patients with peritonitis. Statistical validation showed the MPI to be an accurate and reliable predictor of surgical mortality, the inclusion of a pathophysiological variable may raise its accuracy[26].

Score considers clinic risk factors routinely found in pre operative registers.

Evaluation of severity of illness using MPI allows us to know probability of patient survival.

It is a simple scoring system that allows the surgeon to easily determine outcome of risk during surgery.

Recollection from retrospective data is possible and valid, because it requires data routinely found in surgical registers.

It can be used in governmental, non governmental and private setup.

MANNHEIM PERITONITIS INDEX: SCORING SYSTEM [27]

RISK FACTOR	WEIGHTING IF PRESENT
Age > 50 years	5
Female Sex	5
Organ failure	7
Malignancy	4
Preoperative duration of peritonitis > 24 h	4
Origin of sepsis not colonic	4
Diffuse generalized peritonitis	6
Exudates	
Clear	0
Cloudy	6
Faecal	12

*Definitions of organ failure

Kidney = Creatinine level > or = 177 umol/l

Urea level > or = 167 mmol/l

Oliguria < 20ml/h

Lung = Po2 < 50 mmHg

Shock = Hypodynamic or Hyperdynamic

(Definition according to Shoemaker)

Intestinal obstruction = Paralysis > or = 24h or complete mechanical ileus (Only if profound).

METHODOLOGY

MATERIALS AND METHODS

Type of Study: Prospective analytical study

The study is conducted over a total of 45 patients, admitted in Shri B.M.Patil Medical College Hospital for secondary peritonitis between 1 October 2008 to May 2010.

Place of Study: Department of general surgery, Shri B.M.Patil Medical College.

Methodology: Based on history of illness, clinical examination, investigations and intra - operative findings a Proforma was prepared.

Exclusion criteria were : primary peritonitis, peritonitis due to pancreatitis, tuberculosis and peritoneal dialysis.

MPI Score was calculated for each patient immediately after surgery depending on data collected on pre-designed proforma. Patients were followed up till death or discharged from the hospital. The prognosis of each patient was judged by the scores obtained. Scores were analyzed under 3 categories – i) Scores<21, ii) Scores between 21 – 29, iii) Scores > 29.

STASTISTICAL METHODOLOGY :

The above mentioned variables were tabulated and analyzed by different types of graphs.

Data was analyzed on software SPSS.

Chi-square test was used to assess any significant association between scores and outcome.

Chi-square test was calculated for individual risk factor.

Mortality rate was calculated.

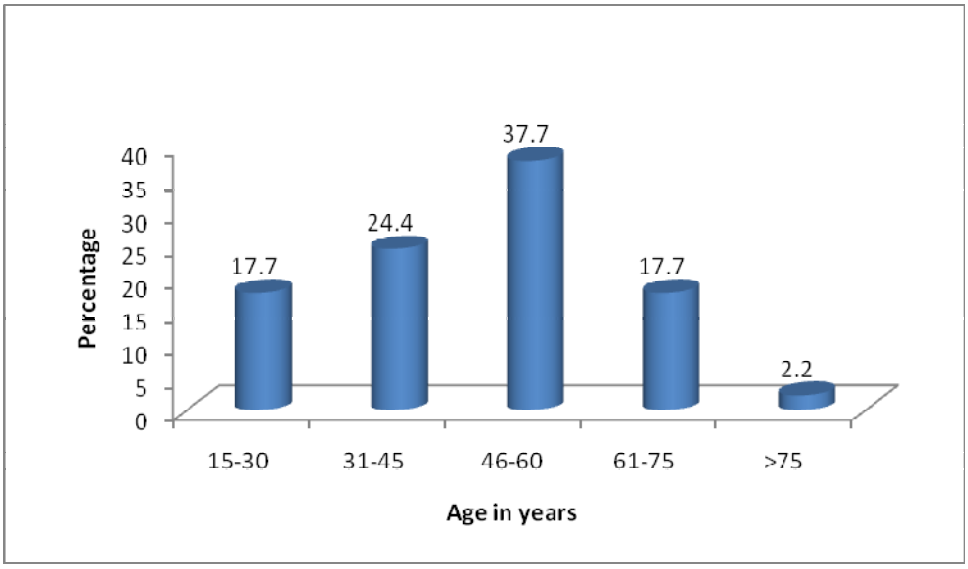
Tabulation and analysis of the data available was done.

OBSERVATION AND RESULTS

Table 1 : Age distribution of patients studied.

Age	Number	Percentage
15-30	8	17.7
31-45	11	24.4
46-60	17	37.7
61-75	8	17.7
>75	1	2.2
Total	**45**	**100**

Graph 1: Age distribution of patients studied.

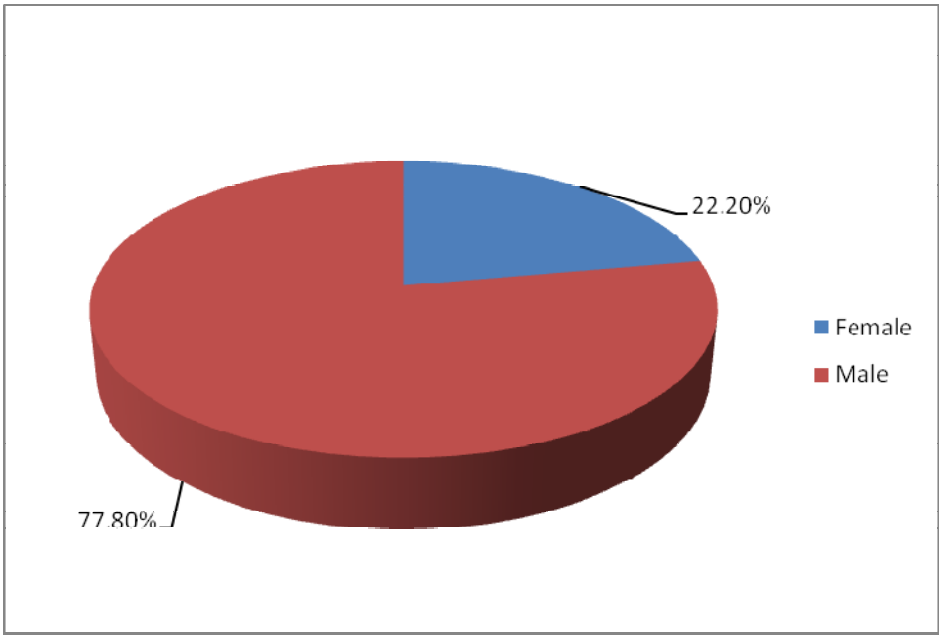


Maximum number of patients belonged to 46 to 60 years of age group. Patients more than 75 years were least in number.

Table 2: Sex distribution of patients studied.

Sex	Number	Percentage
Male	35	77.8%
Female	10	22.2%
Total	**45**	**100**

Graph 2: Sex distribution of patients studied.

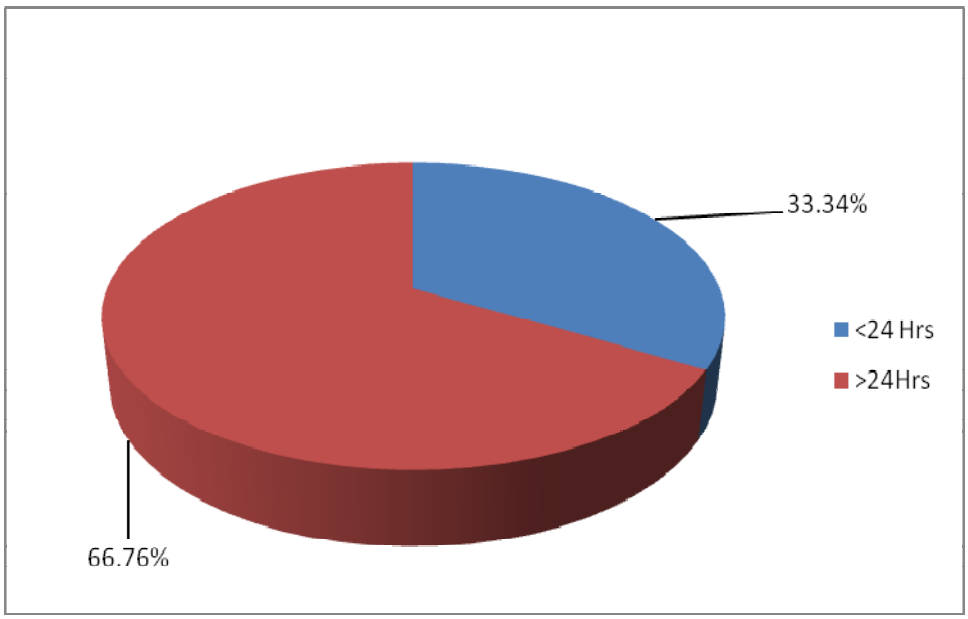


Males (77.8%) formed predominant population of patients.

Table 3: Duration of presenting symptoms.

Duration>24hrs	Number	Percentage
yes	30	66.7%
No	15	33.3%
Total	**45**	**100**

Graph 3: Duration of presenting symptoms.

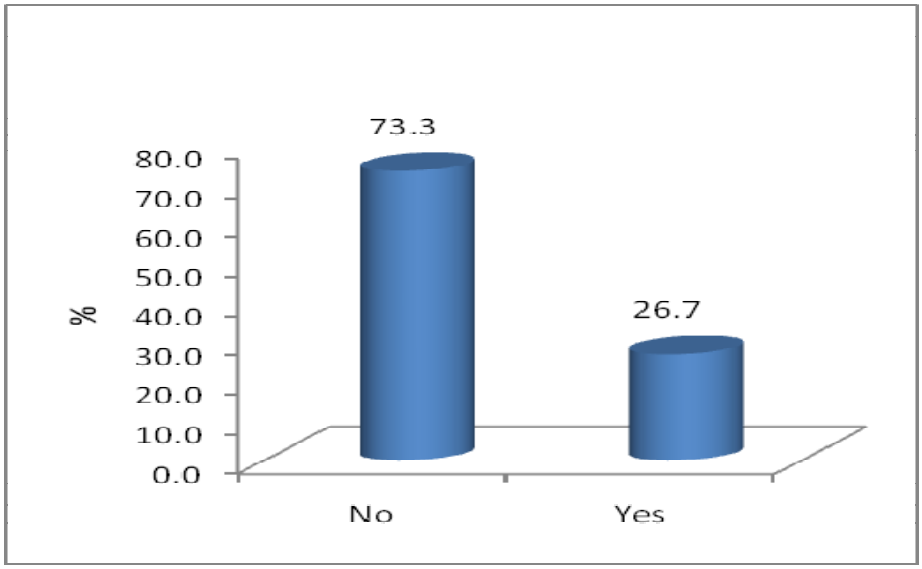


66.76% presented more than 24 hrs of onset of symptoms.

Table 4: Presence of organ failure in the patients studied.

Organ failure	Number	Percentage
Yes	12	73.3%
No	33	26.7%
Total	**45**	**100**

Graph 4: Presence of organ failure in the patients studied.

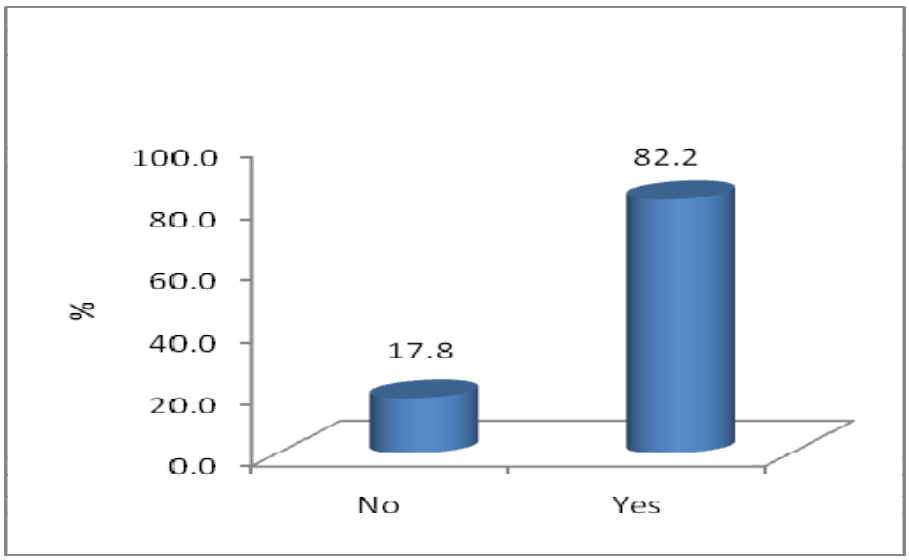


Only 26.7% of the patients had organ failure before surgery.

Table 5: Presence of diffuse peritonitis during surgery.

Organ failure	Number	Percentage
Yes	37	82.2%
No	8	17.8%
Total	**45**	**100**

Graph 5: Presence of diffuse peritonitis during surgery.

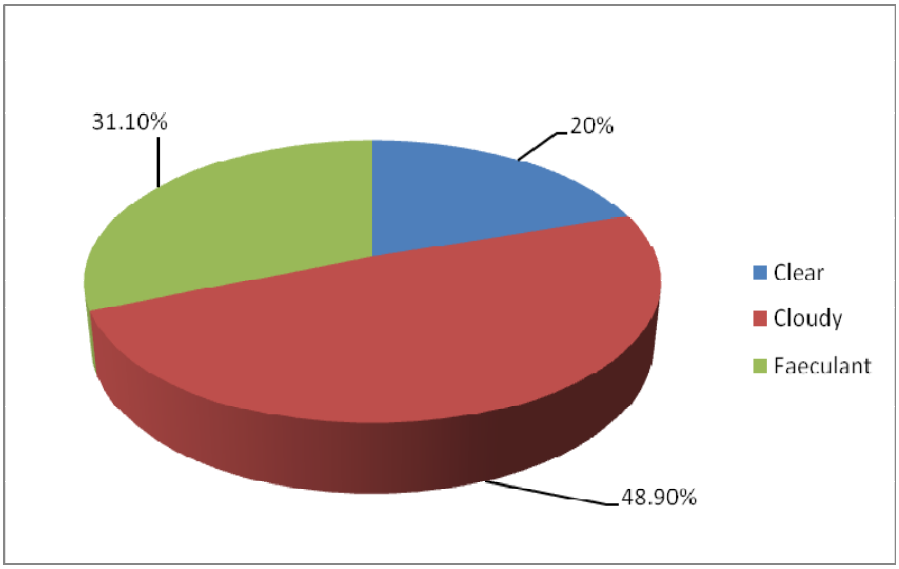


Majority of the patients (82.2%) had diffuse peritonitis intra operatively.

Table 6: Types of exudates during surgery.

Types of exudates	Number	Percentage
Clear	9	20
Cloudy	22	48.9
Faeculant	14	31.1
Total	**45**	**100**

Graph 6: Types of exudates during surgery**.**

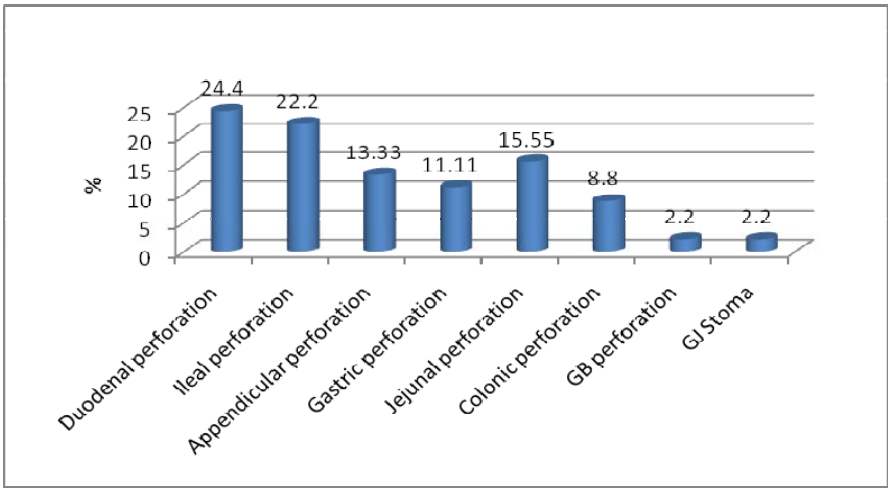


Half of the patients had cloudy exudates during surgery.

Table 7 : Site of perforation .

Site of perforation	Number	Percentage	Origin of sepsis not colonic
1.Duodenal perforation	11	24.4	Y
2.Ileal perforation	10	22.2	Y
3.Appendicular perforation	6	13.33	Y
4.Gastric perforation	5	11.11	Y
5.Jejunal perforation	7	15.55	Y
6.Colonic perforation	4	8.8	N
7.GB perforation	1	2.2	Y
8. GJ Stoma	1	2.2	Y
Total	**45**	**100**	

Graph 7 : Site of perforation.

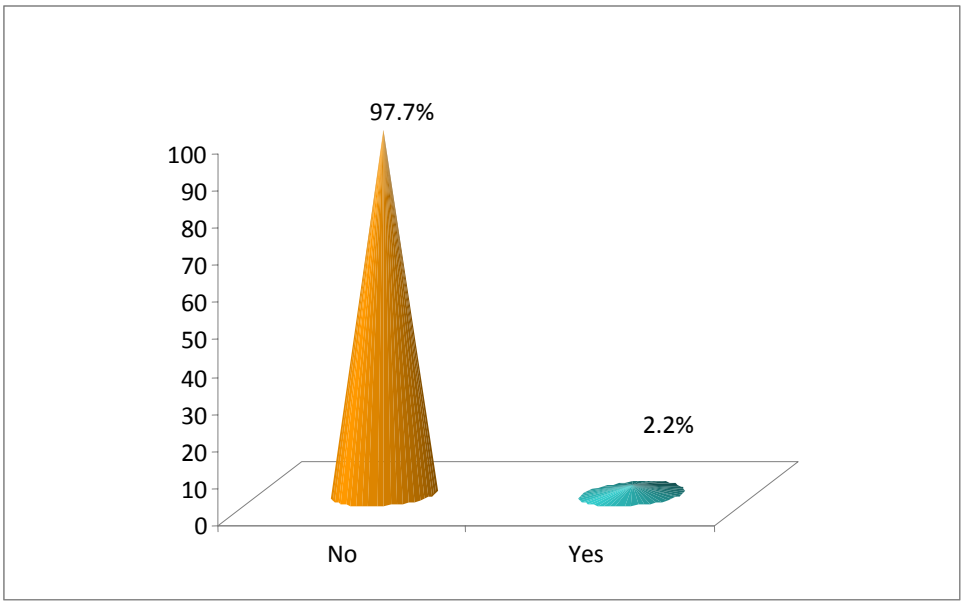


Duodenum was most common organ to be perforated. Gall bladder formed the least.

Table 8 : Findings of Malignancy during surgery.

Malignancy	Number	Percentage
Yes	1	2.2
No	44	97.8
Total	**45**	**100**

Graph 8 : Findings of Malignancy during surgery.

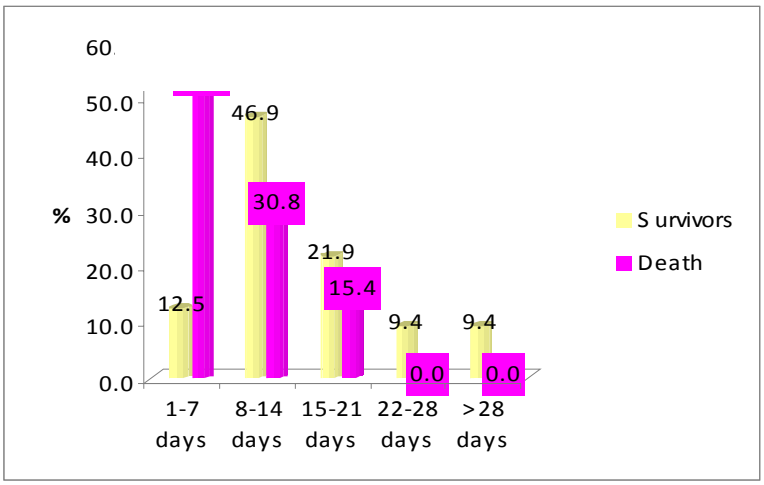


Only 1 patient had findings of malignancy intra operatively.

Table 9 : Mean hospital stay.

No of days of Hospital stay	**Patients**	**Survivors (%)**	**Non survivors (%)**
1-7 days	4	12.5%	53 %
8-14 days	15	46.9%	30.8%
15-21 days	7	21.9%	15.4%
22-28 days	3	9.4%	0
>28 days	3	9.4%	0
Total	**45**	**100%**	**100%**

Graph 9: Mean hospital stay.

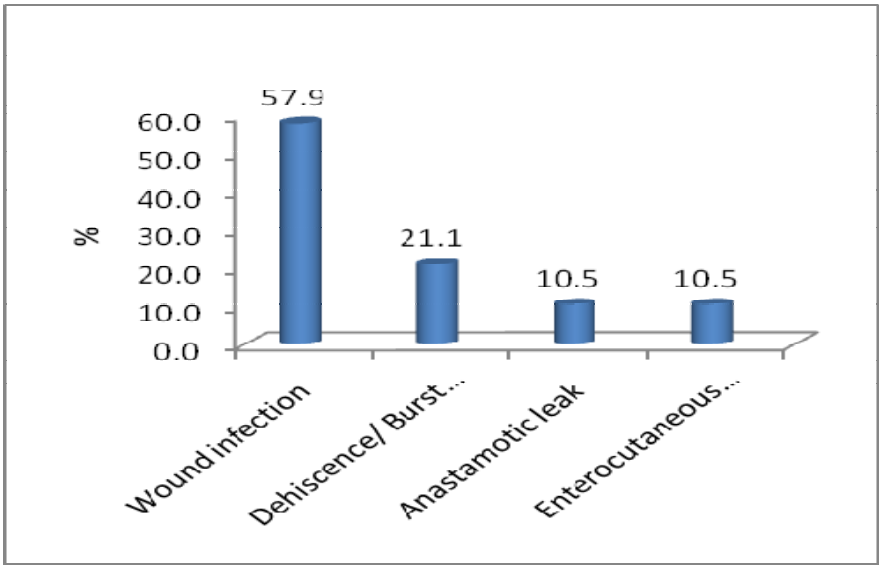


Maximum number of deaths (38.8%) took place between 1 to 7 days of admission and the maximum length of hospital stay among the survivors was 28 days.

Table 10 : Local complications seen in patients.

Local complications	No. of cases	Percentage
Wound Infection	11	57.8
Intra abdominal Abscess	4	21.05
Wound Dehiscence / Burst abdomen	2	10.5
Enterocutaneous fistula	2	10.5
Total	**19**	**100**

Graph 10 : Local complications seen in patients.

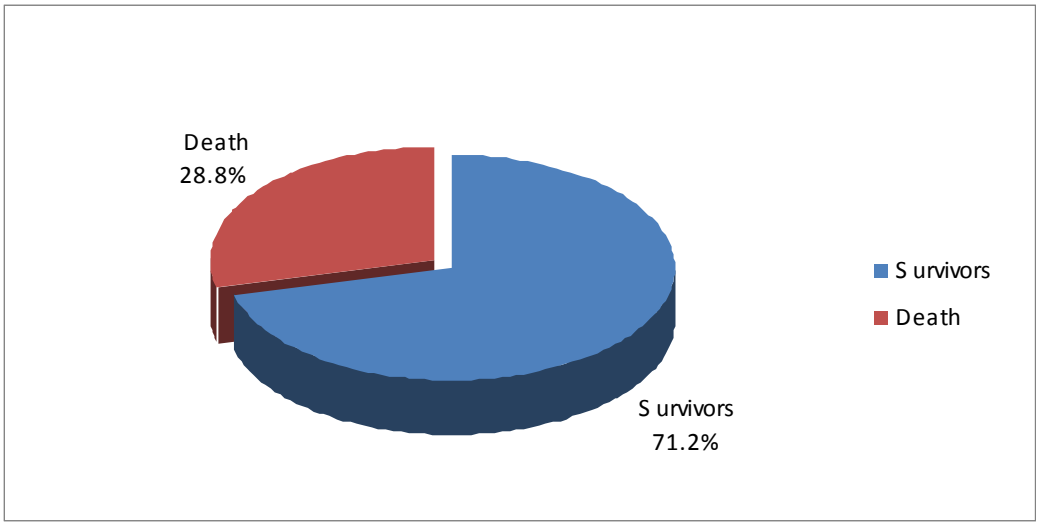


Surgical site infection was the most common local complication faced by the patients.

Table 11 : Outcome of study.

Outcome	Number	Percentage
Survivors	32	71.2
Non-survivors	13	28.8
Total	**45**	**100**

Graph 11 : Total mortality rate.



The total mortality rate in this study group was 28.8%.

Table 12: Cause of death.

CAUSES	Number	Percentage
Respiratory failure	4	30.76
Myocardial infarction	3	23.07
Multi organ failure	6	46.15
Total	**13**	**100**

The most common cause of death in patients was due to the development of multiorgan failure.

OUTCOME OF THE STUDY

Table 13: Mortality in severity groups.

MPI * DEATH Crosstabulation

			DEATH		
			Discharged	Death	Total
MPI	<21	Count	16	3	19
		% within MPI	84.2%	15.8%	100.0%
	21-29	Count	13	3	16
		% within MPI	81.3%	18.8%	100.0%
	>29	Count	3	7	10
		% within MPI	30.0%	70.0%	100.0%
Total		Count	32	13	45
		% within MPI	71.1%	28.9%	100.0%

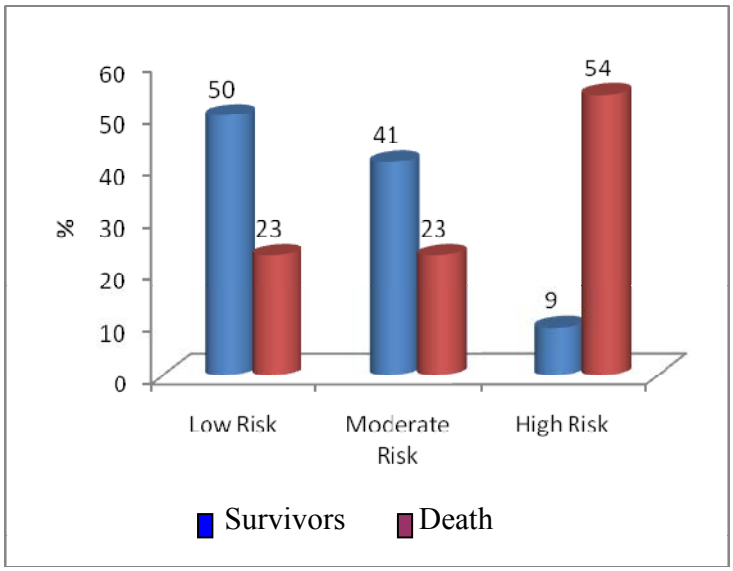
Table 14: Chi-Square test.

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	10.615[a]	2	.005
Likelihood Ratio	9.870	2	.007
Linear-by-Linear Association	7.622	1	.006
N of Valid Cases	45		

a. 2 cells (33.3%) have expected count less than 5. The minimum expected count is 2.89.

Graph 12 : Mortality in severity groups.



The mortality rate in low risk group was 15.8%, in moderate risk group it was 18.8% & in high risk group it was 70 %.

By using Chi-Square tests, p value was 0.005, showing association between MPI and mortality i.e mortality increased with increasing MPI score.

Table 15: Association of Risk factors with mortality.

Risk factors	Total number of patients (n=45)	Outcome		Chi-square	P value
		Survivors (n=32	Non-survivors (n=13)		
1.Age > 50 years	17(38%)	10(31%)	7(54%)	2.108	0.156
2.Female sex	10(22%)	6(19%)	4(31%)	0.773	0.379
3.Organ failure	12(27%)	5(16%)	7(54%)	**6.906**	**0.009****
4.Malignancy	01(2%)	1(3%)	0	0.415	0.519
5.Pre-op duration of peritonitis >24 hrs	30(67%)	21(66%)	9(69%)	0.054	0.81
6.Origin of sepsis not colonic	42(93%)	31(97%)	11(85%)	2.233	0.l35
7.Diffuse gen. Peritonitis	37(82%)	25(78%)	12(92%)	1.272	0.259
8.Clear exudates	9(20%)	7(22%)	2(15%)	1.193	0.381
9.Cloudy exudates	22(49%)	17(53%)	5(39%)		
10.Faecal exudates	14(31%)	8(25%)	6(46%)		
Total	**45**(100%)	**32**(71%)	**13**(29%)	-	-

By using Chi-Square tests, organ failure was the only independent risk factor to have an effect on the mortality rate.

DISCUSSION

Secondary peritonitis is one of the most common problems faced in general surgical practice. When severe and generalized it is associated with increased mortality.

Despite advances in intensive care medicine and introduction of aggressive surgical technique, prognosis of peritonitis and intra abdominal sepsis remains poor. Early objective grading of severity of peritonitis may help change surgical and medical management.

Among the most widely known prognostic scoring indices used for classifying patients with abdominal sepsis is Acute physiology and chronic health evaluation (APACHE). It is the best score used in intensive care units when done within first 24 hrs of admission. It has 12 variables ; Total score ranging from 0-17. It did not predict development of multi organ failure or death.

The MPI is a special score which has good accuracy and provides an easy way to handle with clinical parameters allowing the prediction of the prognosis of patients with peritonitis. Statistical validation showed MPI to be accurate and reliable predictor of surgical mortality, the inclusion of pathophysiological variable may increase it accuracy.

In our study MPI scores were given to all 45 patients. Data was analyzed on software SPSS. Chi - square test was used to assess any significant association between scores and outcome.

Patients were divided into categories of severity as described by fugger et al. Low risk score being <21, moderate risk score being 21-29 and high risk score being

more than 29. The biggest score which can be assigned to a case is 47 and the lowest being 0.

In our study highest score was 38 and lowest was 10. In accordance with the life table, when MPI score increased, mortality also increased. There was a mortality rate of 15.8 % in low risk group, 18.8 % in moderate risk group and 70 % in high risk group.

Chi-square test showed significant association between mortality and increasing MPI score (p value was 0.005).

In national cancer institute of Brazil study, MPI score varied from 5 to 47 with mean value of 31.7. The mortality rate increased proportionally according to MPI score. Wittman DH and et al showed in his study, high mortality rate (50%) when diagnosis of peritonitis was made after 48 hours. A billing, D Frohlich, F.W Schildberge et al peritonitis study group is 2003, showed that mean index score from 7 different centers ranged between 14 to 26 and mean mortality from 11 to 42%.

In our study maximum patients belonged to age group of between 31-45 years of age. No significant relation was established between age as an individual risk factor and mortality.

There is an obvious predominance of male patients (78%). However this did not influence mortality.

About 66% of total patients underwent surgery more than 24 hrs of their presentation. There was no association found between duration of symptoms and mortality in this study.

82.2% of patients were found to have diffuse peritonitis during surgery. But this had no effect on the outcome.

5 out of 7 patients having multi organ failure died. In our study organ failure was found to be the only independent risk factor to have an impact over mortality.

Intra - operatively duodenum was the most common site of perforation followed by ileum.

All gastric perforations were closed by interrupted simple vicryl sutures and omental patch closure wad done. Except for two duodenal perforations all the other perforations were closed by pedicled omental patch also called as the roscoe graham procedure. Omental plugging was done for remaining two duodenal perforation. 8 out of 10 ileal perforations were treated by simple 2 layer interrupted vicryl sutures in a plane perpedicular to the lumen. 2 patients underwent ileal segmental resection and end to end ileal anastomosis.

Jejunal perforations were treated by simple 2 layer interrupted vicryl sutures in a plane perpedicular to the lumen.

Caecostomy was done for one case of colonic perforation following bullgore injury. Two cases of colonic perforations were closed primarily in two layers. Right extended hemicolectomy was done for multiple colonic perforations in the ascending and proximal part of transverse colon following trauma.

One case of G.J stoma perforation was treated by sub total gastrectomy and roux-en-y gastrojejunostomy.

Gall bladder and appendicular perforations were treated by cholecystectomy and appendicectomy respectively.

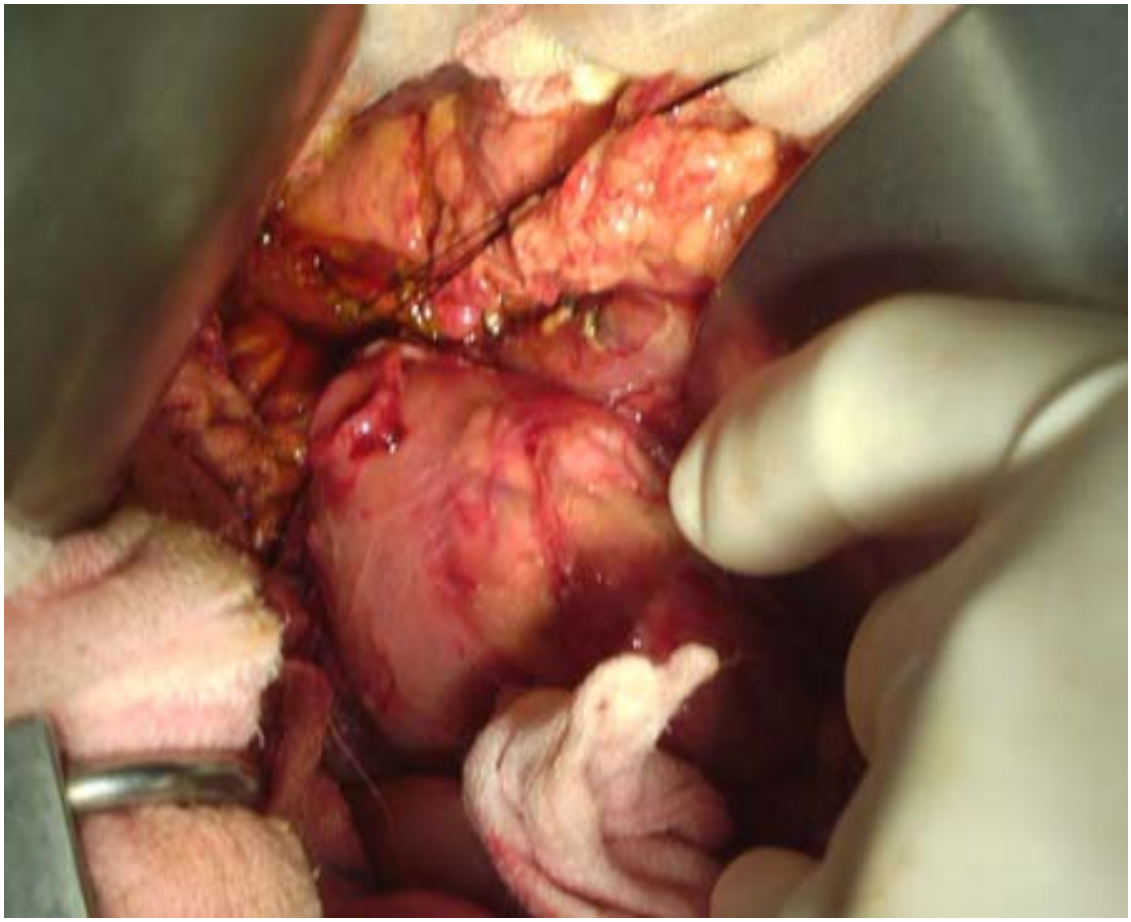


Figure 6 : Perforation over second part of duodenum.

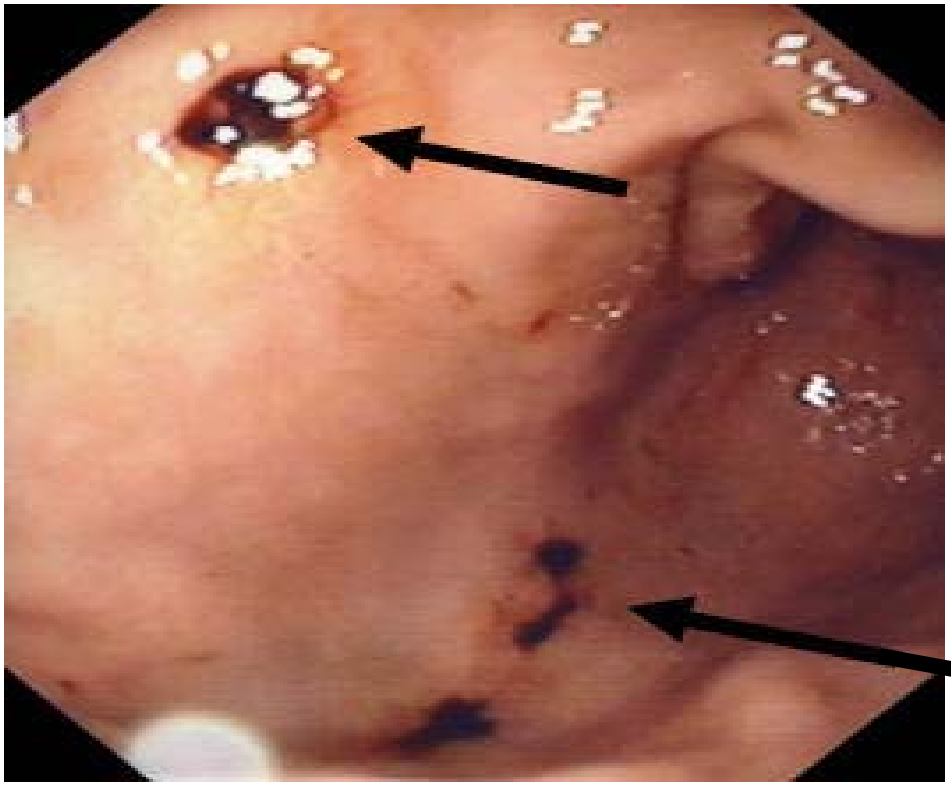


Figure 7 : Stomach perforation with multiple ulcers below it.

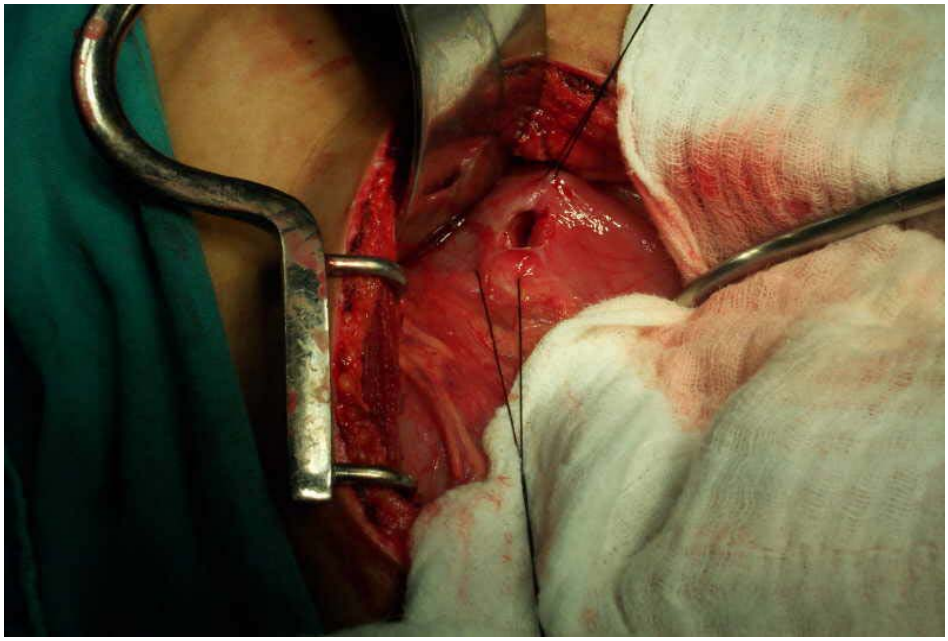


Figure 8 : Closure of stomach perforation.

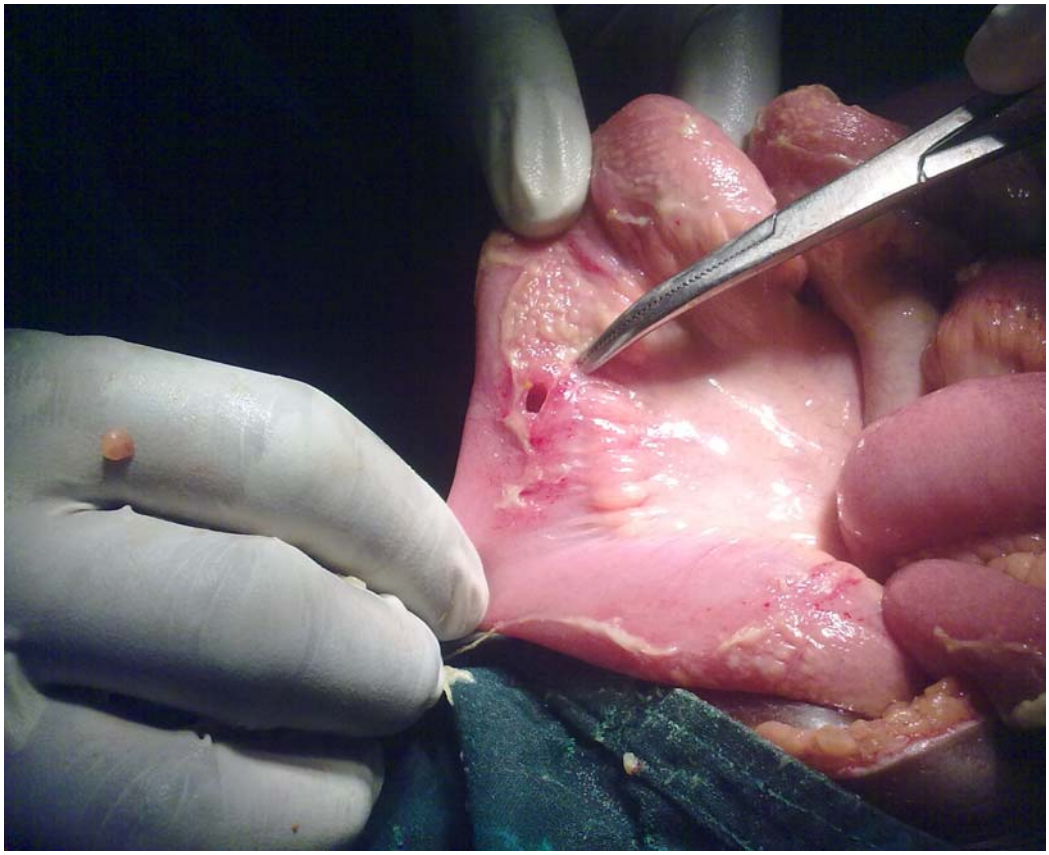


Figure 9 : Perforation of Ileum.

Only one patient in the study group had findings of malignancy intra operatively, therefore association between mortality and presence of malignancy could not be established.

The type of exudates found during surgery had no impact on mortality.

Wound infections, abdominal dehiscence, anastomotic leak, enterocutaneous fistula were some of the local complications faced by the patients. Re-laparotomy was performed in 2 patients of enterocutaneous fistula which ultimately resulted in their deaths. 2 out of 3 patients with burst abdomen were taken for secondary suturing who later recovered. 60% of all the complications were that of surgical site infections[28].

The mortality rate in this study was 28.8%. The most common cause of death in these patients was due to multi organ failure.

SUMMARY

This study was prospective descriptive analytical study conducted on 45 patients over a period of one and half years in Department of general surgery, Shri B. M. Patil Medical College. It was done only for causes involving secondary peritonitis.

The MPI index score was calculated for all 45 patients. 37.7% of total patients belonged to 31-45 years of age group. Males were predominant (77.8%). Most of the patients (66.7%) presented more than 24 hrs of onset of symptoms. 82.2% had diffuse peritonitis as an intra operative findings. Half of the patients had cloudy exudates intra abdominally during surgery. Duodenum is the most common site of perforation. Only 1 patient had findings of malignancy intra operatively. 5 out of 7 patients had multi organ failure pre operatively.

Surgical site infection was the most common local complication. Most common cause of death was multi organ failure.

The death rate in our study was 28.8%. The mortality rate in low risk group was 15.8%, in moderate risk group it was 18.8% and in high risk group it was 70%.

Chi-square test value was 0.005, showing association between MPI and mortality i.e. mortality rate increased with increasing MPI score. Organ failure was the only independent risk factor to have an effect on mortality rate.

CONCLUSION

Given the high accuracy, simplicity and lack of similar studies in India we conducted the study at our institution. The MPI is a specific score, which has a good accuracy and provides an easy way to handle with clinical parameters, allowing the prediction of the individual prognosis of patients with peritonitis very early in the disease.

Our statistical validation showed the MPI to be an accurate and reliable predictor of surgical mortality. We recommend that the MPI critical score should be adjusted for each hospital. Any patient with a MPI score above the critical score is to be regarded as predicted non survivors and treated aggressively. Obviously our present results can only be applied to hospitals with very similar characteristics, in order to support the prediction power of the index.

Based on our results we conclude that Mannheim Peritonitis Index is accurate, cost effective, reliable and simple reference for estimating risk of death in patients with perforative peritonitis.

BIBLIOGRAPHY

1. B.A Taylor. "Spontaneous perforation of the gastrointestinal tract in emergencies". 1992 ; 1st edn. 359 – 379.
2. Acta Chir. Austriaca 2000 ; 32 : 152 – 161.
3. Zimmerman L. M . "Great ideas in the history of surgery". 1st edn. 1961 ; 3-74.
4. Clinical anatomy of regions. Snell's anatomy. 8th edn. 201-221.
5. RJ Last. "Regional and Applied Anatomy". 5th edn. 413-466.
6. Chaursia B.D. Abdominal Cavity and Peritoneum. Human Anatomy Regional Applied Dissection and Clinical. 4th edn. 221 – 226.
7. Richard H.T. Sabiston Text book of surgery. 18th edn. 1129 – 1154.
8. Inderbir Singh. "Text book of Human Histology". 2nd edn. 212-216.
9. Micheal M et al. "Biology of Peritonitis and implications on treatment". Apirl ; 68 : 431-443.
10. John G. Barlett. "Intra abdominal Sepsis". Medical Clinics of North America. May 1995 ; 79 : 599-612.
11. Jeremy Thompson et al. "The peritoneum, Omentum, mesentery and retroperitoneal Space". Short practice of Surgery by Bailey & Love. 23rd edn. 1008-25.
12. Hamilton Bailey's Acute abdomen and demonstration of physical signs in clinical surgery. 18th edn. 1997 ; 299-529.
13. Ladha. Gastrointerstinal perforations. Indian journal of Surgery 1988 ; 50 : 500-505.
14. Farquharson's Text book of Operative surgery. 9th edn. 233 – 250.
15. Darryl T, Hiyama et al. Mainogot's Abdominal Operations. 10th edn. 633-655.

16. Somen Das. "Operation on the Appendix". Practical guide to Operative Surgery 2002 ; 4th edn. 369-376.

17. Bosscha K, Algra A et al. Prognostic scoring systems to predict outcome in peritonitis and intra abdominal sepsis. British Journal of Surgery 1997 ; 84 : 1532-34.

18. Goris RJ et al. "Multi organ failure. Generalised auto destructive inflammation. Annals of surgery 1985 ; 120 : 1109-15.

19. Knaus WA, Wagner DP et al. "APACHE – Acute Physiology and Chronic health Evaluation : A. Physiologically based classification system. Critical Care Medicine 1981 ; 9 : 591-59.

20. Knaus WA, Draper Eaet al. "Prognosis in acute organ System failure". Annals of Surgery 1985 ; 13 : 818 – 829.

21. Le Gall J-R, Loirat P, Alperovitch A. Simplified acute physiological score for intensive patients. Lancet 1983; ii : 741 (Letter).

22. Le Gall J-R, Loirat P, Alperovitch A et al. A simplified acute physiology score for ICU patients. Crit Care Med 1984; 12: 975-7.

23. Elebute EA, Stoner HB. The grading of sepsis. Br. J Surg 1983; 70: 29-31.

24. Wacha H, Linder M et al. Predication of risk of death from peritonitis. Theoretical Surg 1987; 1 : 169-277.

25. Evaluation of Manheim peritonitis index and multiple organ failure score in patients with peritonitis. Indian Journal of Gastroenterology Sep-Oct 2005; 24 (5): 197-210.

26. Brracho – Riquelme R et al. Peritonitis index validation study at the hospital General de Durango (Mexico). Cir Ciruj 2002 ; 70 : 217-225.

27. Billing A, Frohlich D, Schildbery FW. "Prediction of outcome using the Manheim peritonitis index in 2003 patients". Br J Surg 1994 ; 81 : 209-13.

28. Alicia J , Mangram MD , Teresa C, Horan, MPH, CIC et al. Guidelines for prevention of surgical site infection 1999 ; 20 (4) : 247 – 270.

PROFORMA

SCHEME OF CASE TAKING:

Name	:	Case No	:
Age	:	IP No	:
Sex	:	Date of Admission	:
Occupation	:	Date of Surgery	:
Residence	:	Date of Death/ Discharge	:
Chief Complaints	:	Duration < 24 hrs/> 24 hrs	
Provisional Diagnosis	:		
Past History	:		
Personal History	:		
Family History	:		

General Physical Examination :

Pallor	Present / Absent
Icterus	Present / Absent
Clubbing	Present / Absent
Generalized Lymphadenopathy	Present / Absent
Anasarca	Present / Absent
Built	Poorly built / Moderately built/ well built

Nourishment : well nourished/ poorly nourished

Vitals

Pulse Rate :

BP :

Respiratory Rate :

Temperature :

Weight :

PA Examination

Inspection :

Palpation :

Percussion :

Auscultation :

PR Examination :

PV Examination :

Other Systemic Examination

i) Respiratory system :

ii) Cardiovascular system :

iii) Central nervous system :

Investigations

Blood :

Hb :

TC :

DC :

ESR :

BT :

CT :

Total Serum Protein :

Serum Albumin :

RBS :

Serum Creatinine :

Serum Electrolyte : Chest X-ray view :

LFT : X-ray erect abdomen :

PO_2 : USG abdomen :

Final Diagnosis :

Details of Surgery :

- Incision
- Intra Operative Findings

1. Origin of Spesis

   Colonic – Y/N

   Noncolonic – Y/N

2. Malignancy – Y/N

3. Diffuse generalized peritonitis – Y/N

4. Type of exudate -

5. Presence of organ failure – Y/N

   Final MPI score -

SAMPLE INFORMED CONSENT FORM

TITLE OF THE PROJECT	:	EVALUATION OF MANNHEIM PERITONITIS INDEX IN PATIENTS WITH SECONDARY PERITONITIS
GUIDE	:	Dr. B.P. KATTIMANI (ASSOCIATE PROFFESOR OF SURGERY)
P.G. STUDENT	:	Dr. SHARVANI L. NAIK

PURPOSE OF RESEARCH:

I have been informed that this study is conducted to know about my prognosis following my surgery done for peritonitis. I have also been given free choice of participation in this study.

PROCEDURE:

I am aware that in addition to routine care received I will be asked series of questions by the investigator. I have been asked to undergo the necessary investigations and treatment, which will help the investigator in this study.

RISK AND DISCOMFORTS:

I understand that I may experience some pain and discomforts during the examination or during my treatment. This is mainly the result of my condition and the procedures of this study are not expected to exaggerate these feelings which are associated with the usual course of treatment.

BENEFITS:

I understand that my participation in the study will help to know the prognosis of secondary peritonitis following surgery.

CONFIDENTIALITY:

I understand that the medical information produced by this study will become a part of hospital records and will be subject to the confidentiality. Information of sensitive personal nature will not be part of the medical record, but will be stored in the investigations research file.

If the data are used for publication in the medical literature or for teaching purpose, no name will be used and other identifiers such as photographs will be used only with special written permission. I understand that I may see the photograph before giving the permission.

REQUEST FOR MORE INFORMATION:

I understand that I may ask more questions about the study at anytime Dr. Sharvani L. Naik. at the department of surgery who will be available to answer my questions or concerns. I understand that I will be informed of any significant new findings discovered during the course of the study, which might influence my continued participation. A copy of this consent form will be given to me to keep for careful reading.

REFUSAL FOR WITHDRAWAL OF PARTICIPATION:

I understand that my participation is voluntary and that I may refuse to participate or may withdraw consent and discontinue participation in the study at

any time without prejudice. I also understand that Dr. Sharvani L. Naik. may terminate my participation in the study after he has explained the reasons for doing so.

INJURY STATEMENT:

I understand that in the unlikely event of injury to me resulting directly from my participation in this study, if such injury were reported promptly, the appropriate treatment would be available to me. But, no further compensation would be provided by the hospital. I understand that by my agreements to participate in this study and not waiving any of my legal rights.

I have explained to _____the purpose of the research, the procedures required and the possible risks to the best of my ability.

_____ _____

Dr. Sharvani L. Naik. Date

(Investigator)

STUDY SUBJECT CONSENT STATEMENT:

I confirm that Dr. Sharvani L. Naik has explained to me the purpose of research, the study procedure, that I will undergo and the possible discomforts as well as benefits that I may experience in my own language. I have been explained all the above in detail in my own language and I understand the same. Therefore I agree to give consent to participate as a subject in this research project.

_____ _____

(Participant) Date

_____ _____

(Witness to signature) Date

KEY TO MASTER CHART

1	-	Clear
2	-	Cloudy
3	-	Faeculant
F	-	Female
M	-	Male
MI	-	Myocardial infarction
MOF	-	Multi organ failure
N	-	No
RF	-	Respiratory failure
Y	-	Yes

MASTER CHART

SL NO	NAME	IP NO	AGE (>45 YRS)	SEX	DURATION (>24HRS)	ORGAN FAILURE	DIFFUSE PERITONITIS	EXUDATE	MALIGNANCY	ORIGIN NOT COLONIC	SITE OF PERFORATION	MPI	DEATH	HOSPITAL STAY	CAUSE OF DEATH
1	IRAMMA	1138	25	F	Y	N	N	2	N	Y	APPENDIX	19	N	25	
2	AZHARUDDIN	1397	25	M	Y	Y	Y	3	N	Y	ILEUM	33	N	18	
3	GULAPPA	1989	55	M	N	N	N	3	N	N	COLON	17	Y	7	RF
4	ROOPSINGH	2056	50	M	N	N	Y	2	N	Y	STOMACH	21	N	10	
5	LAKSHMIBAI	2144	100	F	Y	N	Y	2	N	Y	GALL BLADDER	30	Y	12	RF
6	VITTAL	2166	50	M	Y	Y	Y	2	N	Y	APPENDIX	32	Y	6	MOF
7	MALLIKARJUN	2865	65	M	Y	N	Y	1	N	Y	JEJUNUM	19	N	16	
8	NEELAWWA	2871	65	F	N	N	N	2	N	Y	DUODENUM	20	N	7	
9	SUJATHA	3058	20	F	Y	N	Y	2	N	Y	APPENDIX	24	N	15	
10	SHARANAPPA	3586	65	M	Y	Y	Y	3	N	Y	ILEUM	38	Y	18	MI
11	PARSAPPA	3974	69	M	Y	N	Y	2	N	Y	GJ STOMA	25	Y	13	MOF
12	AMEENSAB	6542	40	M	N	N	Y	2	N	Y	DUODENUM	16	N	14	
13	RAJESAAB	7255	50	M	N	N	N	2	N	Y	STOMACH	15	N	45	
14	PANDU	7611	34	M	N	N	Y	3	N	N	COLON	18	Y	1	RF
15	SUBHAS	7879	35	M	Y	N	Y	3	N	Y	ILEUM	26	N	18	
16	MALLINATH	8137	35	M	Y	N	Y	2	N	Y	DUODENUM	20	N	18	
17	NINGANNA	8536	60	M	Y	N	Y	2	N	Y	ILEUM	19	N	14	
18	KALLAPPA	9847	70	M	Y	N	Y	1	N	Y	DUODENUM	19	N	11	
19	BOGAPPA	10414	50	M	Y	Y	Y	3	N	Y	JEJUNUM	33	N	1	
20	RAMU	10428	30	M	Y	N	Y	2	N	Y	DUODENUM	20	N	45	
21	MADIVALAPPA	10799	60	M	N	N	Y	1	N	Y	DUDONEUM	25	N	11	

22	JUMBO	11633	50	M	Y	Y	Y	3	N	Y	ILEUM	38	Y	11	MOF
23	SOMANNA	12981	35	M	N	N	Y	3	N	Y	JEJUNUM	22	N	13	
24	HANIF	13101	25	M	Y	N	Y	3	N	Y	ILEUM	26	N	33	
25	SHIVAPPA	13641	60	M	Y	N	Y	2	N	Y	APPENDIX	25	N	13	
26	RAVI	13877	20	M	Y	Y	Y	3	N	Y	JEJUNUM	33	Y	1	MOF
27	MALKANGOUDA	14583	40	M	Y	N	Y	1	N	Y	STOMACH	14	N	11	
28	JAGDISH	14615	37	M	N	N	N	2	N	Y	DUODENUM	10	N	9	
29	YELLAWWA	16206	60	F	Y	Y	Y	2	N	Y	JEJUNUM	38	Y	6	MOF
30	CHANDRASHEKAR	17122	43	M	N	Y	Y	3	Y	Y	ILEUM	33	N	24	
31	RACHAPPA	17365	65	M	Y	N	Y	2	N	Y	DUODENUM	25	Y	12	MOF
32	CHANAPPA	17386	65	M	Y	N	Y	2	N	Y	DUDONEUM	25	N	10	
33	BASAMMA	17928	55	F	N	Y	Y	1	N	Y	STOMACH	27	Y	1	RF
34	PARSAPPA	18153	65	M	Y	Y	Y	1	N	Y	DUODENAL	26	N	18	
35	GURULINGAPPA	18935	55	M	N	N	Y	1	N	Y	STOMACH	15	N	12	
36	SIDANNA	19175	60	M	Y	N	N	2	N	Y	APPENDIX	19	N	12	
37	RUDRAYYA	19542	50	M	N	N	Y	3	N	Y	ILEUM	27	N	7	
38	SHANKARAPPA	19580	50	M	Y	Y	Y	1	N	Y	JEJUNUM	28	N	24	
39	SABAWWA	20531	50	F	N	N	Y	3	N	N	COLON	28	N	20	
40	GURUPADAPPA	20532	35	M	Y	N	Y	2	N	Y	DUODENUM	22	N	10	
41	MAHADEVAPPA	20734	35	M	Y	N	N	2	N	Y	APPENDIX	14	N	10	
42	CHANDBEE	21840	40	F	Y	N	Y	2	N	Y	ILEUM	25	N	20	
43	RAJKUMAR	22880	15	M	Y	Y	Y	1	N	Y	ILEUM	21	Y	1	MI
44	DEEPIKA	23881	15	F	Y	N	N	2	N	Y	APPENDIX	19	N	12	
45	LAXMIBAI	26771	50	F	N	N	Y	3	N	Y	JEJUNUM	32	Y	17	MI