
"A ONE YEAR RANDOMISED CONTROLLED TRIAL TO
COMPARE LAPAROSCOPIC REPAIR VS OPEN REPAIR
FOR THE TREATMENT OF HOLLOW VISCUS
PERFORATION AT KLES PRABHAKAR KORE HOSPITAL
BELAGAVI"

REG NO. BH0114012

Dissertation

Submitted to the
KLE University, Belagavi, Karnataka

In Partial Fulfillment
of the requirements for the degree of

MASTER OF SURGERY (M.S.)
in
GENERAL SURGERY

**DEPARTMENT OF SURGERY,
JAWAHARLAL NEHRU MEDICAL COLLEGE,
BELAGAVI, KARNATAKA**

APRIL - 2017

**KLE UNIVERSITY, BELAGAVI,
KARNATAKA**

ENDORSEMENT

This is to certify that the dissertation entitled “**A ONE YEAR RANDOMISED CONTROLLED TRIAL TO COMPARE LAPAROSCOPIC REPAIR VS OPEN REPAIR FOR THE TREATMENT OF HOLLOW VISCUS PERFORATION AT KLES PRABHAKAR KORE HOSPITAL BELAGAVI**” is a bonafide research work done by **CANDIDATE REG NO. BH0114012.**

Dr. S. S. Shimikore MS
Professor and Head,
Department of Surgery,
J. N. Medical College,
Nehru Nagar, Belagavi – 10

Date:
Place: Belagavi

Dr. N. S. Mahantshetti MD
Principal,
J. N. Medical College,
Nehru Nagar, Belagavi – 10

Date:
Place: Belagavi

LIST OF ABBREVIATIONS USED

/Cumm	-	Per cubic millimeter
APACHE	-	Acute physiology and chronic health evaluation
B.T	-	Blood type
BC	-	Before Christ
BHVI	-	Blunt hollow viscus injury
CI	-	Confidence interval
cm	-	Centimeter
CT	-	Computed tomography
DBP	-	Diastolic blood pressure
DLC	-	Differential leukocyte count
ECL	-	Enterochromaffin-like
GE	-	Gastroesophageal
gm	-	Gram
H/O	-	History of
Hb	-	Haemoglobin
mg/dL	-	Milligrams per deciliter
min	-	Minute
mm Hg	-	Millimeters of mercury
mm	-	Millimeter
MPI	-	Mannheim peritonitis index
n	-	Total number
NSAIDS	-	Non steroidal anti-inflammatory drugs
p	-	Probability
PPU	-	Perforated peptic ulcer

RBS	-	Random blood sugar
RCTs	-	Randomised controlled trials
RLQ	-	Right lower quadrant
S. Creatinine	-	Serum creatinine
SAPS	-	Simplified acute physiology score
SBP	-	Systolic blood pressure
SD	-	Standard deviation
SMA	-	Superior mesenteric artery
SSS	-	Sepsis severity score
TLC	-	Total leukocyte count
U.K.	-	United Kingdom
vs.	-	Versus

ABSTRACT

Background and objectives

In patients presenting with hollow viscus perforation open surgery is considered as the standard approach, but the use of laparoscopy for diagnostic purposes and treatment appears to be a safe alternative with a number of advantages. The present study was compared results of laparoscopic repair versus open repair.

Methodology

This one year hospital based randomized controlled trial was conducted in the Department of General Surgery, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi. A total of 60 Patients admitted with hollow viscus perforation undergoing either laparoscopic or open repair from January 2015 to December 2015 were studied. These patients were sequentially randomized into two groups of 30 each as Group A (laparoscopic repair) and Group B (open repair).

Results

Most of the patients were males in Group A (63.33%) as well as group B (73.33%) ($p=0.405$). The mean age was almost similar in group A (48.30 ± 18.23 years) and Group B (49.30 ± 15.27 years) ($p=0.819$). Other characteristics including clinical presentation, medical and personal history, systemic examination findings and diagnosis were comparable between two groups, ($p>0.050$). The mean Manheim's peritonitis index Score was comparable in group A and group B (22.07 ± 4.65 vs. 21.47 ± 5.39 ; $p=0.646$). The mean

duration of surgery was significantly low in group A (105.13 ± 9.57 minutes) compared to group B (141.67 ± 20.19 minutes) ($p < 0.001$). The mean duration of resumption of daily activities was significantly low in group A (4.53 ± 0.73 Days) compared to group B (11.87 ± 2.93 Days) ($p < 0.001$).

Conclusion and interpretation

Laparoscopic repair is beneficial procedure for the management of hollow viscous perforation in terms of lower surgical time, and early resumption of daily activities.

Keywords

Hollow viscous perforation; Laparoscopic repair; Open repair;

CONTENTS

SL. NO.	TOPIC	PAGE NO.
1.	INTRODUCTION	1
2.	OBJECTIVES	4
3.	REVIEW OF LITERATURE	5
4.	METHODOLOGY	32
5.	RESULTS	43
6.	DISCUSSION	62
7.	CONCLUSION	69
8.	SUMMARY	70
9.	BIBLIOGRAPHY	72
10.	ANNEXURES	
	ANNEXURE I – CONSENT FORM	84
	ANNEXURE II – PROFORMA	88
	ANNEXURE III – MASTER CHART	93

LIST OF TABLES

TABLE NO.	DESCRIPTION	PAGE NO.
1	Sex distribution	44
2	Age distribution	45
3	Mean age	46
4	Abdominal distension	47
5	Vomitus	48
6	Comparison of Fever	49
7	History of drug intake	50
8	History of alcohol intake	51
9	History of smoking	52
10	History of tobacco consumption	53
11	Respiratory system	54
12	Per abdomen	55
13	Diagnosis	56
14	Surgical time	57
15	Mean Duration of surgery	58
16	Time for resumption of daily activities	59
17	Mean Duration for resumption of daily activities	60
18	Manhiem's peritonitis index	60
19	Clinical characteristics	61

LIST OF GRAPHS

GRAPH NO.	DESCRIPTION	PAGE NO.
1	Sex distribution	44
2	Age distribution	45
3	Abdominal distension	47
4	Vomitus	48
5	Comparison of Fever	49
6	History of drug intake	50
7	History of alcohol intake	51
8	History of smoking	52
9	History of tobacco consumption	53
10	Respiratory system	54
11	Per abdomen	55
12	Diagnosis	56
13	Surgical time	57
14	Time for resumption of daily activities	59

LIST OF FIGURES

FIGURE NO.	DESCRIPTION	PAGE NO.
1	Anatomy of stomach	6
2	Histology of stomach	6
3	Anatomy of small intestine	10
4	Layers of small intestine	11
5	Chest X-ray and erect abdomen X-ray showing free gas under diaphragm	19
6	CT Scan showing pneumoperitoneum	19

LIST OF PHOTOGRAPHS

PHOTO NO.	DESCRIPTION	PAGE NO.
1	Patient in Lloyd Davis position with Port placement	37
2	Laparoscopic view of hollow viscous perforation in duodenum	37
3	Laparoscopic repair of hollow viscous perforation – Primary closure	38
4	Laparoscopic knotting	38
5	Laparoscopic repair of hollow viscous perforation after knotting	39
6	Application of Omental patch	39
7	Open repair of hollow viscus perforation	40
8	Open primary closure of hollow viscus perforation	40

INTRODUCTION

Acute abdomen refers to the complaints of an acute attack of abdominal pain that may occur suddenly or gradually over a period of several hours and presents a symptom complex which suggests a disease that possibly threatens life and demands an immediate or urgent diagnosis for early treatment.¹ In 1921 Cope in wrote that ‘the majority of severe abdominal pain which ensure in patients who have been previously fairly well and which lasts as long as 6 hours are caused by conditions of surgical importance.’²

The challenging and most fascinating subject in surgery is study of acute abdomen. It constitutes about 5 to 10% of all emergency department visits. The syndrome of acute abdominal pain generates a large number of hospital visits and may affect the very young, the very old, either sex and all socio economic group.^{1,3}

Gastro intestinal perforation is a common abdominal emergency faced by general surgeon.^{4,6} It is a common dictum that abdomen is a Pandora’s Box and gastrointestinal perforation is one such condition to prove it. Perforation of a hollow viscus from wide variety of causes comprises the major portion of emergency surgical admissions and emergency laparotomies.^{7,8} The diagnosis and treatment of gastro intestinal perforation remains main problem in our country.^{9,10} This problem has been reduced owing to improved medical and diagnostic facilities in North America and the U.K., where vascular lesions and malignancies are major cause of perforations, whereas in our country, peptic disease, typhoid, tuberculosis are still common.¹¹

The first clinical description of perforated peptic ulcer was made by Crisp in 1843. Smoking and use of non-steroidal anti-inflammatory drugs are important risk factors for perforation. Easy availability and the frequent use of NSAIDS and other over the counter analgesics is a common risk factor.^{12,13}

Perforation of the stomach, duodenum and small bowel are common than colonic perforation. Perforation of the large intestine are a surgical challenge to the surgeon, not only because the technical aspects of the operation may be difficult but more importantly because the situation is commonly fatal, in the type of compromising patients in whom the condition usually presents.^{14,15} Perforation of the large intestine is a rapidly fatal condition, death being caused by sepsis from peritoneal contamination with various enteric pathogens both aerobic and anaerobic. Majority of patients present with sudden onset of abdominal pain.¹⁶⁻¹⁸

Because the treatment of choice in hollow visus perforation is emergency surgery, a workup is necessary to establish a diagnosis. The workup is done in the usual manner of history, physical examination, laboratory tests, and imaging studies. Although accuracy with which a correct diagnosis can be made is increased owing to advancement in imaging studies, a thorough history and careful physical examination still remain important.¹ Laboratory and imaging studies are usually needed, but are directed by the findings on history and physical examination.^{1,19} Early surgery is always advantageous than late. The diagnostic procedure involved should be giving a definite diagnosis in a short-time. And after a diagnosis is made, the method of management of the case holds prime importance.^{1,20} Successful treatment not only depends on timely surgery but competent post-operative care. Sir Henle's aphorism is that 'In acute abdominal emergencies, the difference between

the best and worst surgery is infinitely less, than between early and late surgery and greatest sacrifice is sacrifice of time'.¹

Hollow viscus perforation is an emergency which should be managed by a rapid and effective treatment. Open surgery has been considered the standard approach, but the use of laparoscopy for diagnostic purposes and treatment appears to be a safe alternative with a number of advantages.²¹

Laparoscopy has been used for gastrointestinal diseases like perforated peptic ulcer as early as 1990's. Reports have shown that peritoneal toilet can be performed and perforations can be closed safely with laparoscopy. But whether repair of the perforation by laparoscopic approach is better than by conventional open repair is undetermined. Royal College of England²² stated that laparoscopic repair of perforations is clearly feasible and beneficial in terms of significant lower mean duration of hospital stay. Furthermore randomized controlled trial²² extended advantages of laparoscopic repair in terms of significant benefits to the patient though reduced post-operative pain, lesser chest infections, along with shorter hospital stay and earlier return to normal activities compared to open technique. Though, there is undoubtedly a learning curve for laparoscopy and reduced tactile feedback is present in laparoscopy and great care needs to be taken to avoid damage to adjacent tissues. This study was an attempt to compare results of laparoscopic repair versus open repair so as to determine whether repair of the perforation by laparoscopic approach is better compared to conventional open repair.

OBJECTIVES

The objectives of this study were;

Primary

To Compare result of laparoscopic versus open repair of hollow viscus perforation using time for recovery in days.

Secondary

1. Operative time for the procedure
2. Mannheim peritonitis index

REVIEW OF LITERATURE

The Hippocratic facies seen in terminal stages of peritonitis has been discussed since Hippocrates (460 BC).²³

Aristotle was the first to describe intestinal injury as a consequence of blunt abdominal trauma.²⁴

Anatomy

The stomach is readily recognizable as the asymmetrical, pear-shaped, most proximal abdominal organ of the digestive tract. The part of the stomach attached to the esophagus is called the cardia. Just proximal to the cardia at the gastroesophageal (GE) junction is the anatomically indistinct but physiologically demonstrable lower esophageal sphincter. At the distal end, the pyloric sphincter connects the stomach to the proximal duodenum. The stomach is relatively fixed at these points, but the large midportion is quite mobile.²⁵

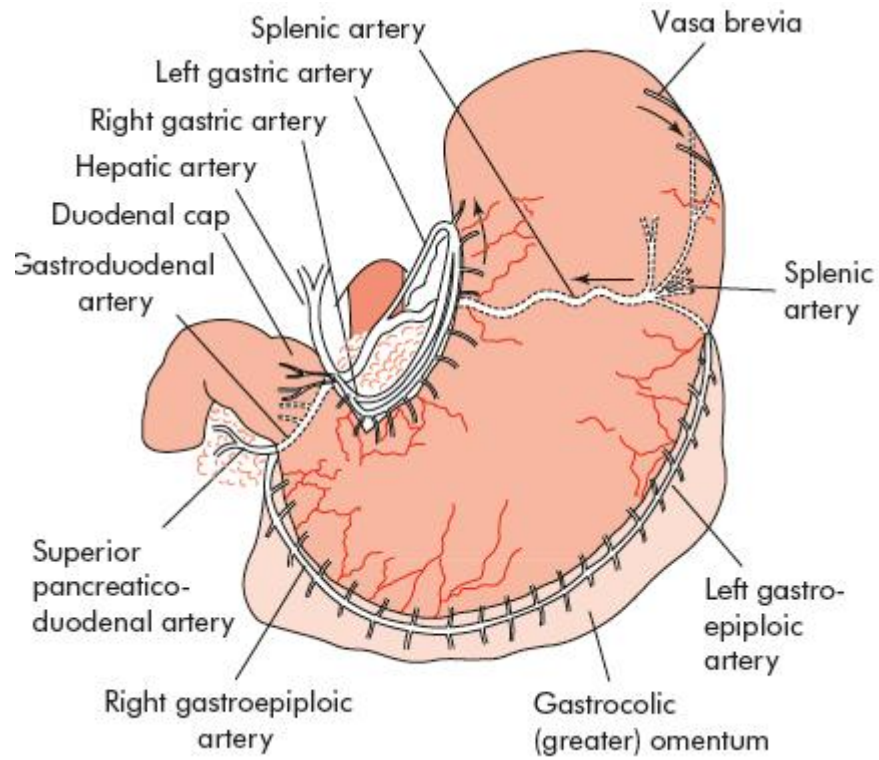


Figure 1. Anatomy of stomach²⁵

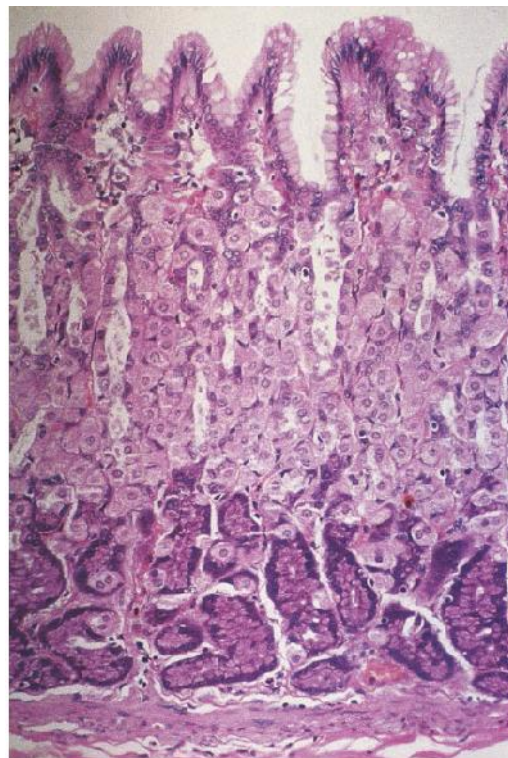


Figure 2. Histology of stomach²⁵

The stomach is the most richly vascularized portion of the alimentary canal. Both the quantity of blood delivered to the stomach and the richness of the intramural gastric vascular anastomotic network are impressive. The large majority of the gastric blood supply is from the celiac axis via four named arteries. The left and right gastric arteries form an anastomotic arcade along the lesser curvature, and the right and left gastroepiploic arteries form an arcade along the greater gastric curvature.²⁵

Both the extrinsic and intrinsic innervation of the stomach play an important role in gastric secretory and motor function. The vagus nerves provide the extrinsic parasympathetic innervation to the stomach, and acetylcholine is the most important neurotransmitter.²⁵

There are four distinct layers of the gastric wall: mucosa, submucosa, muscularis propria, and serosa. The inner layer of the stomach is the mucosa, which is lined with columnar epithelial cells of various types. Beneath the basement membrane of the epithelial cells is the lamina propria, which contains connective tissue, blood vessels, nerve fibers, and inflammatory cells. Beneath the lamina propria is a thin muscle layer called the muscularis mucosa. The epithelium, lamina propria, and muscularis mucosa constitute the mucosa. The epithelium of the gastric mucosa is columnar glandular. The gastric glands are lined with different types of epithelial cells, depending upon their location in the stomach.²⁵

Parietal cells

These are in the body (acid-secreting portion) of the stomach and line the gastric crypts, being more abundant distally. They are responsible for the production

of hydrogen ions to form hydrochloric acid. The hydrogen ions are actively secreted by the proton pump, a hydrogen–potassium-ATPase (Sachs), which exchanges intraluminal potassium for hydrogen ions. The potassium ions enter the lumen of the crypts passively, but the hydrogen ions are pumped against an immense concentration gradient (1000 000:1).²⁵

Chief cells

These lie principally proximally in the gastric crypts and produce pepsinogen. Two forms of pepsinogen are described: pepsinogen I and pepsinogen II. Both are produced by the chief cell, but pepsinogen I is produced only in the stomach. The ratio between pepsinogens I and II in the serum decreases with gastric atrophy. Pepsinogen is activated in the stomach to produce the digestive protease, pepsin.²⁵

Endocrine cells

The stomach has numerous endocrine cells, which are critical to its function. In the gastric antrum, the mucosa contains G cells, which produce gastrin. Throughout the body of the stomach, enterochromaffin-like (ECL) cells are abundant and produce histamine, a key factor in driving gastric acid secretion. In addition, there are large numbers of somatostatin-producing D cells throughout the stomach, and somatostatin has a negative regulatory role.²⁵

The small intestine is a tubular structure within the abdominal cavity that extends from the pylorus of the stomach to the ileocecal valve adjacent to the large intestine.¹³ The length of the small intestine grows with age from about 6.5 feet in a

newborn to almost 20 feet in an adult. The main functions of the small intestine are to aid in digestion, absorption of nutrients, and elimination of waste.²⁶

The small intestine is comprised of 3 portions: the duodenum, the jejunum, and the ileum.^{13,14} The duodenum is distal to the stomach and is the widest portion of the small bowel, has no mesentery and is only partially covered by the peritoneum. It consists of 4 portions: superior, descending, horizontal, and ascending. The jejunum is the center portion of the small intestine with a diameter of approximately 3 cm. The ileum is narrower, thinner, and is less vascular as compared to jejunum, but its aggregated lymph nodules (Peyer's patches) are larger and more numerous.^{26,27}

The blood supply to the proximal duodenum includes the superior pancreatico-duodenal branch of the gastroduodenal artery. The branches of the superior mesenteric artery supply the distal duodenum, jejunum, and ileum with extensive collateralization to prevent ischemic injury. The splenic and superior mesenteric veins drain the duodenum either directly or indirectly, while the superior mesenteric vein drains the jejunum and ileum. The vagus nerves supply parasympathetic nerve activity to the duodenum, and the greater and lesser splanchnic nerves supply sympathetic nerve activity. The superior mesenteric plexus supplies both sympathetic and parasympathetic nerves to the jejunum and ileum.²⁷

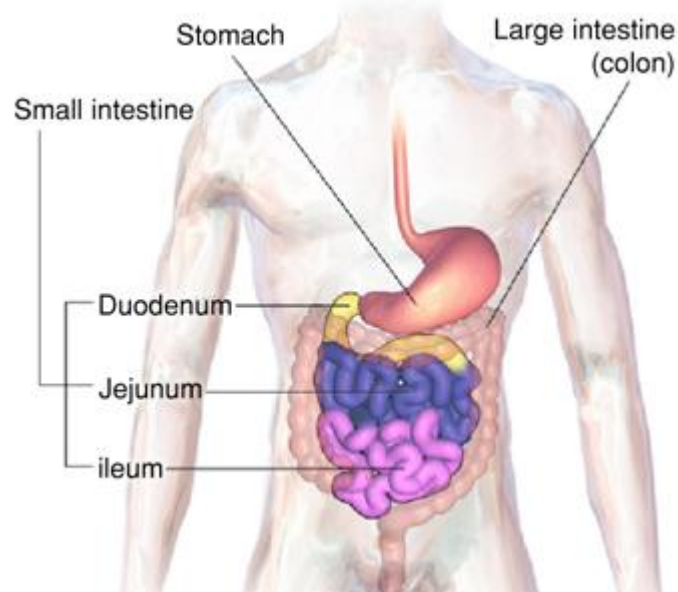


Figure 3. Anatomy of small intestine

The wall of the small intestine is comprised of an inner mucosa layer, submucosa layer, smooth muscular with inner circular and outer longitudinal layers, and the serosa layer (Figure 2).²⁷ The inner walls show mucosal folds called the plicae circulares (also called valves of Kerckring). The plicae are more numerous in the proximal jejunum, reduce in numbers toward the distal jejunum, and are completely absent in the ileum. The circular folds slow the passage of the food along the intestines and increase surface area for absorption. The plicae are covered with small fingerlike projections called villi, which are covered with microvilli. The microvilli absorb fats and nutrients from the partially digested semi-liquid food from the stomach.²⁷

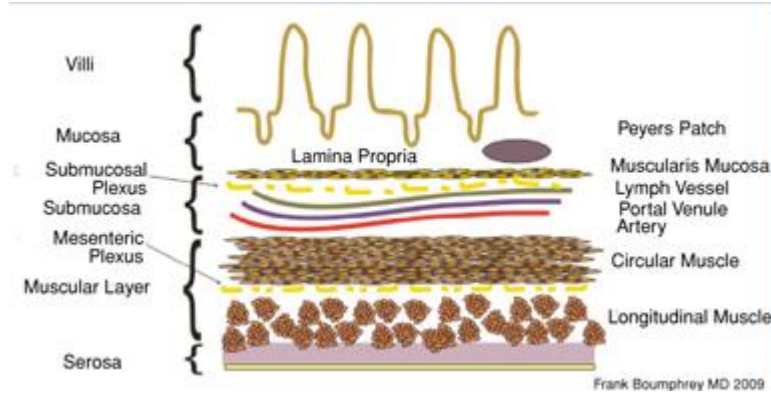


Figure 4. Layers of small intestine

The small intestine is a complex organ with several functions. In fact it is capable of digestion, absorption and secretion, endocrine function and protects the internal environment against noxious ingested substances and against luminal bacteria and their toxins. The potential surface area available for digestion and absorption is amplified 600-times by circular mucosa folds, villus mucosal architecture and the microvillus surface of epithelium. Although specific properties are characteristic of specific segments of small bowel, like bile acid absorption in distal ileum, maximal resections are feasible without a significant morbidity because of the compensatory adaptation of remaining intestine. The small bowel measures about 120 cm in length from pylorus to ileocecal valve. The jejunum begins at ligament of Treitz. Jejunum and ileum are suspended by a mobile mesentery covered by a visceral peritoneal lining that extends onto the external surface of the bowel to form the serosa. Jejunum and ileum receive their blood from the superior mesenteric artery (SMA). Although mesenteric arcades form a rich collateral network, occlusion of a major branch of the SMA may result in segmental intestinal infarction. Venous drain is via the superior mesenteric vein, which then joins the splenic vein behind the neck of the pancreas to form the portal vein. Peyer's patches are

lymphoid aggregates present on the antimesenteric border of distal ileum. Smaller follicles are present through all small bowel. Lymphatic drainage of intestine is abundant. Regional lymph nodes follow the vascular arcades and then drain toward the cisterna chyli. Jejunal and ileal wall consists of serosa, muscularis, submucosa and, innermost, mucosa.²⁸

Hollow viscous perforation

An organ with an abnormal opening often is referred to as a perforated viscus. Viscus technically means a hollow organ found inside the body. Examples of these hollow organs mostly are found in the abdomen such as the stomach, appendix, duodenum, jejunum, ileum and large intestines. The term viscus is a singular form, often used in referring to only one organ involved. Its plural term, viscera, generally is used to denote the involvement of many organs.²⁹

Hollow organs often have several layers of cells in their walls in order to hold materials inside. Food materials consumed during meals and some acids used in digestion typically are found in the stomach. Digested food and waste materials to be excreted out of the body usually are found in the intestines.²⁹

Spilling of these materials inside the abdomen usually happens in the presence of a perforated abdominal viscus. These materials often are toxic inside the body cavity, and can place the life of a patient in danger. Bacteria often reach the blood system in most of these cases, thus immediate medical attention and effective treatment generally are needed in such situations.²⁹

A hollow viscus perforation can occur in many instances. Blunt abdominal trauma such as those that often happen in road accidents can lead to the perforation

of the hollow viscus organ. Penetrating abdominal trauma often can reach important organs like the intestines and stomach as seen in those caused by gunshots and stab wounds. Infections sometimes can lead to viscus perforation such as those that occur in a ruptured appendix, ruptured gallbladder, and as a complication of typhoid fever and peptic ulcer disease.²⁹

The patient with acute abdomen complains of an acute attack of abdominal pain that may occur suddenly or gradually over a period of several hours and presents a symptom complex which suggests a disease that possibly threatens life and demands an immediate or urgent diagnosis for early treatment. Cope in 1921 wrote that ‘the majority of severe abdominal pain which ensue in patients who have been previously fairly well and which lasts as long as 6 hours are caused by conditions of surgical importance.’^{1,2}

Epidemiology

The study of acute abdomen is the most challenging and fascinating subject in surgery. It constitutes about 5 to 10% of all emergency department visits.^{1,3}

The great majority of perforations of the stomach or duodenum are complications of peptic ulcer. Approximately 3% of patients with typhoid experience intestinal perforation.³⁰ The ruptured or perforated viscus challenges the surgeon’s skill as a technician and his knowledge of preoperative, per-operative and postoperative care of the severely ill surgical patient.^{31,32}

The diagnosis and treatment of gastro intestinal perforation remains main problem in our country.^{33,34} Improved medical and surgical care has reduced this problem in North America and the U.K., where vascular lesions and malignancies

are major causes of perforations, while in our country, peptic disease, typhoid, tuberculosis are still preceding malignancies.^{4,11} The first clinical description of perforated peptic ulcer was made by Crisp in 1843. Smoking and use of non-steroidal anti-inflammatory drugs are important risk factors for perforation.³⁶ Especially these days, the inadvertent use of NSAIDs and other over the counter analgesics forms one of the most common risk factors.^{4,13} Perforation of the stomach, duodenum and small bowel form a considerable proportion of emergency work load than colonic perforation.⁴ Perforation of the large intestine represent a major surgical challenge to the clinician, not simply because the technical aspects of the operation may be difficult but more importantly because the situation is rapidly lethal, in the type of compromising patients in whom the condition usually presents. In developed societies most common cause are, the diverticular disease and colonic carcinoma, where as in the developing countries infective conditions such as amoebiasis is important. Perforation of the large intestine is a rapidly fatal condition, death being caused by sepsis from peritoneal contamination with various enteric pathogens both aerobic and anaerobic. Majority of patients present with sudden onset of abdominal pain.⁴

Longo et al.³⁷ reported an incidence of BHVI as 0.6% at a single institution. In a multi-institutional study³⁸ from the Eastern Association for the Surgery of Trauma involving 95 trauma centers, the overall incidence of blunt hollow viscus injury (BHVI), defined as full-thickness perforation, hematoma, or serosal tear, was 1.2% after blunt trauma. Specifically, perforating injury to the small bowel after blunt trauma was even more uncommon, with a reported incidence of, 0.3%.³⁸ Characteristic injury patterns from this series included free perforation, hematomas,

or serosal tears most commonly involving the small bowel, followed by injury to the colon and stomach in decreasing frequency.³⁹

A high index of suspicion is essential to diagnose visceral perforation early as significant morbidity and mortality results from diagnostic delay.^{4,40,41} Thus, an interest is undertaken to find the etiological factors and clinical features, age and sex incidence and also to assess the common type of perforations and their presentations, operative modalities, complications arising postoperatively.⁴

Amongst patients with perforated viscus there are also subsets of variable degrees of risk and therefore a variation in the urgency with which diagnosis and intervention must occur. The outcome in these individuals varies with the site of the perforation, the underlying pathology, the age of the patient, and collateral diseases. The major issue influencing the pace of early management is the degree to which the perforation has been contained or localized by peritoneal defense mechanisms.⁴²

1. Perforation of peptic ulcer; contrast via a nasogastric tube shows evidence of a duodenal ulcer but no continuing leakage.[there may be an option for conservative management]
2. Appendiceal perforation with a localized abscess.
3. A diverticular perforation with a localized pelvic abscess.

Scenarios of greater urgency include:

- a. Perforation of a peptic ulcer with free air and fluid and continuing extravasation demonstrated.

- b. Perforation of the small bowel suspected.
- c. Perforation of the appendix with a spreading peritonitis.
- d. Perforation of a colonic diverticulum with generalized peritonitis.⁴²

Peptic ulcers may perforate through a minute hole or the perforation may be several centimeters in diameter. Gastric ulcers are frequently large.⁴²

Symptoms of hollow viscus perforation start with acute abdominal pain with distention. Vomiting may or may not be present. Guarding and abdominal wall rigidity are frequently present. Liver dullness is obliterated on percussion. The abdominal pain is generalized and mainly continuous in nature.²⁵

Diverticulitis often presents with an acute process in the left lower quadrant. There is localized tenderness and sometimes a tender mass. The condition is manageable by intravenous antibiotics and may resolve completely or at least allow bowel preparation and a single stage resection. Imaging with CT is useful to define the extent of the process. Cases suitable for antibiotic management usually show only thickening of the colon wall, diverticulae, and mesenteric stranding or small pericolic fluid collections.⁴³

Cecal diverticulitis is an uncommon condition, resulting from perforation of a solitary diverticulum of the cecum or ascending colon. These diverticulae do not share the same pathogenesis as the much more common diverticular disease of the colon, usually dominant in the sigmoid. The solitary diverticulum has probably been present from birth, so the complication of inflammation occurs in younger individuals. It is virtually indistinguishable from appendicitis, other than that the

pain begins in the RLQ or flank. Tenderness or mass may be higher than the usual location for the appendix. A CT scan, if done, may demonstrate the normal appendix and the localized inflammation, often lateral to the ascending colon.⁴³

Diagnosis

Because of the potential surgical nature of the hollow visus perforation, an expeditious workup is necessary. The workup proceeds in the usual order—history, physical examination, laboratory tests, and imaging studies.¹ Although imaging studies have increased the accuracy with which the correct diagnosis can be made, the most important part of the evaluation remains a thorough history and careful physical examination. Laboratory and imaging studies are usually needed, but are directed by the findings on history and physical examination.¹⁹

As previously described, the taking of a history, performing a physical examination and initiating monitoring and management may all have to occur simultaneously. Obviously a prolonged interrogation of a hypotensive patient who is vomiting and in severe pain, without attempting any resuscitation or other necessary interventions may not only waste critical time, but also exposes the patient to unnecessary suffering and risk. The duration of the pain and the other associated complaints need to be clearly defined. It is useful to determine the region of the abdomen where the pain was first felt. A description of the rate of onset and how the pain spread throughout the abdomen may also be helpful. If pain is felt in the flanks or in the back, this suggests a retroperitoneal process. Generalized abdominal pain or periumbilical pain suggests that there is an origin in the small bowel, colon, or appendix. Pain which is in the epigastrium and radiates towards the right upper

quadrant or sometimes the scapular area will usually signal biliary origin. Pain arising very abruptly in the upper abdomen, which is severe and spreads rapidly to involve other quadrants may be experienced by patients with a perforated peptic ulcer. Perforation of a sigmoid diverticulum will result in left lower quadrant pain which may spread to a variable extent to involve other quadrants of the abdomen. Appendicitis has a characteristic pattern beginning as periumbilical colic and then shifting to the right lower quadrant where it exhibits features of localized peritonitis.⁴³

Perforated viscus symptoms generally range from moderate to severe manifestations. They mostly include fever, low blood pressure, tachycardia, abdominal pain, nausea, vomiting, and abdominal distention. Patients mostly are in severe pain with their abdomen feeling rigid or board-like when touched. When prompt treatment for a perforated viscus is not given, patients often develop complications that can be life-threatening.²⁹

The diagnosis of BHVI should be made expeditiously to allow appropriate intervention and to provide the best outcome possible for injured patients. A delay in the recognition of BHVI increases morbidity and mortality.^{44,45} Neideree et al.⁴⁵ demonstrated an increase in acute respiratory distress syndrome and sepsis when surgical intervention was delayed for 24 hours. In a more recent study,⁴⁶ an interval of >0.5 hours between presentation and laparotomy was noted to increase the incidence of death 3-fold. A high index of suspicion is essential when evaluating blunt trauma patients to decrease the incidence of missed blunt hollow viscus injuries.

The imaging modalities include X-ray erect abdomen or lateral decubitus, CT scan and Ultrasonography. Free gas under diaphragm is sign of a hollow viscus perforation. A lateral decubitus X-ray abdomen is done if a patient is unable to stand up. However the preferred modality is CT scan of abdomen in which even a very minimal amount of Pneumo-peritoneum is seen

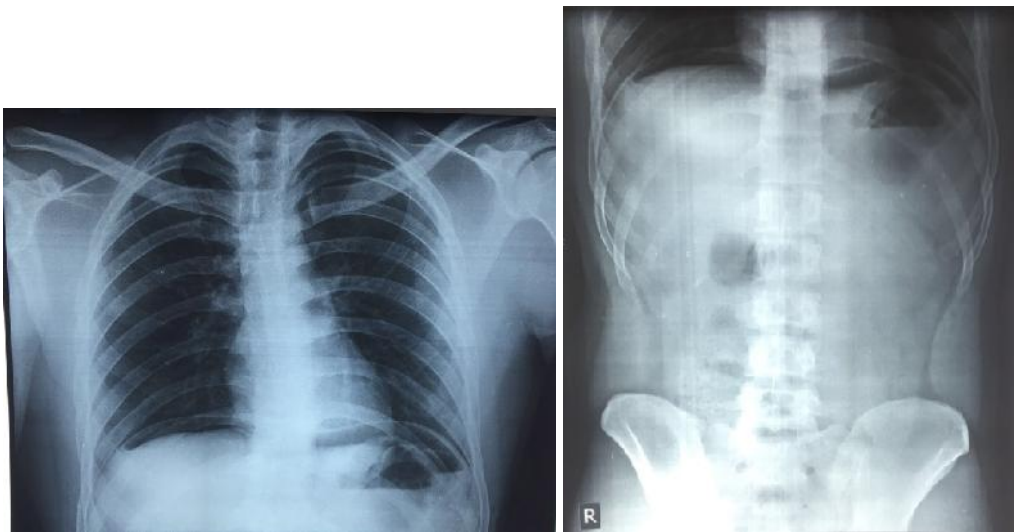


Figure 2. Chest X-ray and erect abdomen X-ray showing free gas under diaphragm



Figure. CT Scan showing pneumoperitoneum

Management

After initial resuscitation and stabilization of a trauma patient, attention is directed toward clinical evaluation. Physical exam findings alone, however, may not accurately establish the diagnosis.³⁹ Abdominal pain or tenderness is an important finding when present, as it may indicate intra-abdominal injury, but its presence does not necessarily indicate the need for surgical intervention. Complicating factors such as the administration of medications for pain or agitation, and intoxication with alcohol or other substances may reduce the reliability of the clinical exam.⁴⁴ This further underscores the need for additional, objective information to identify pathology and to determine if surgical intervention is necessary.³⁹

It is always advantageous to do an early surgery than a late surgery. The investigative procedure involved should be such that, they should give a definite diagnosis in a short-time.⁴⁷ And after a diagnosis is made, the method of management of the case holds prime importance.⁴⁸

Success in treatment depends largely on early diagnosis with early intervention and competent post operative care.¹

Open surgery generally is the standard management for a perforated viscus. The use of laparoscopy, which also is known as minimally invasive surgery, also has been utilized with some patients. The nature of treatment often depends on the cause of the perforated viscus and its location, among numerous other considerations.

The first major trial of conservative treatment was undertaken by Herman Taylor at the King George Hospital, Ilford from 1944 onwards. In 1957 Taylor

reported ten years experience of managing 256 patients with perforated ulcer; 208 of whom had been treated by conservative method.⁴⁹

Nair SK (1981) reported maximum morbidity in the form of wound infections in 52% of patients which was followed by faecal fistula in 16% of patients, septicaemia in 8% of patients and respiratory infections in 4% of patients.⁵⁰

Christiansen J (1987) compared simple closure versus closure and proximal vagotomy in perforated duodenal ulcers. He studied 50 cases and found that no significant difference in morbidity and mortality in early postoperative months. But recurrence was higher of 52% in simple closure against 16% after closure and proximal vagotomy during follow up study.¹⁰

Devi AK, Paul S, Bhattacharjee N (1994) in their study of 171 patients showed that simple closure is safe emergency procedure in all perforated duodenal ulcers. Definite ulcer healing operation may be done in selected cases of perforated chronic duodenal ulcer.⁵¹

Singh BU (2003) concluded that repair of typhoid perforation is a better procedure than temporary ileostomy in enteric perforation due to its cost effectiveness and absence of complications related to ileostomy and ileotransverse bypass should be considered in treatment option in patient with an unhealthy gut.⁵²

Sui WT (2004) concluded that laparoscopic repair of perforated peptic ulcer is a safe emergency procedure in routine clinical practice for patients with perforated pyloroduodenal ulcer.⁵³

Jani K, Saxena AK (2006) showed that omental plugging is a safe and reliable method of treatment for large duodenal ulcer (> 0.5 to 2.5 cm) perforation especially in high risk patients.⁵⁴

The principles of managing blunt and sharp abdominal injuries have been well established. There has been a change towards a more conservative approach in both types of injury. In adult patients, in centres where high volumes of penetrating injuries are seen, initial conservative management with careful observation and the application of set criteria for intervention have been able to reduce the need for operative intervention and negative exploration.⁵⁵

Laparotomy has been the choice of management of gunshot and sharp abdominal injuries. Blunt injuries resulting in hollow viscus perforation can remain undiagnosed up to an average of 18 hours before signs of peritonitis become evident. The incidence of intestinal injury following blunt abdominal trauma has been reported to be 1 - 15%. The most common site is the small intestine, the jejunum being particularly vulnerable due to its anchoring point at ligamentum Treitz. Hollow viscus perforation has a significant mortality of up to 15% in some series.⁵⁵

The place of laparoscopic repair of perforated peptic ulcer followed by peritoneal toilet has been established, although it is not routinely practiced.⁵⁶

Laparoscopic repair of perforated peptic ulcer (PPU) was first reported with the introduction of laparoscopy.⁵⁷ Nevertheless, the use of the procedure in routine practice has been rather limited during the 1990s, largely because of the low incidence of PPU after identification of *Helicobacter pylori* as a prime factor, and because of the effectiveness of pharmacologic treatment in eradication of the

bacterium and prevention of ulcer recurrence.⁵⁸ Nevertheless, several studies have reported the use of the laparoscopic approach in clinical practice. Minimally invasive treatment of PPU involves entering the abdominal cavity after establishing a pneumoperitoneum, closure of the gastric defect, and lavage of the abdomen. Closure of the defect is facilitated either by direct suturing with or without placement of an omental patch or by introduction of biological glue with or without placement of a gelatin sponge.⁵⁷ The concept of sutured closure is the laparoscopic counterpart to the open technique, whereas nonsutured repair does not require laparoscopic suturing skills and has the advantage of shorter operative time.⁵⁹

Despite the use of various anti ulcer agents and eradication therapy hollow viscus perforation are one of the most common complications in surgery. It is one of the most common causes of admission in casualty worldwide and more in developing nations.

Some patients with perforated ulcer can be managed successfully by non-operative means. However the chief hurdles to this treatment are uncertainty or error in diagnosis, the unknown site and pathology of the perforation,⁶⁰ and the unlikely response in elderly patients in whom this treatment is more attractive.' However, routine definitive ulcer surgery in the form of highly selective vagotomy has been suggested in patients with perforated duodenal ulcer because this operation is unlikely to cause long-term side effects and because the prediction of the clinical course after simple repair of the ulcer is unreliable.⁶¹ However, even the strongest advocate for immediate definitive ulcer surgery for perforated peptic ulcer agrees that simple repair is indicated for patients who are poor surgical risks because of major concurrent medical illness or shock, for patients who have heavy bacterial

contamination of the peritoneal cavity because of delay in surgery, and when a surgeon experienced in ulcer surgery is not available.⁶¹

Fewer surgeons currently have acquired enough expertise in performing highly selective vagotomy with advances in medical therapy. Simple closure remains an attractive option for perforation in most centers.^{60,61} Reports of laparoscopic treatment have shown that peritoneal toilet can be performed effectively and perforations can be closed safely.⁶²⁻⁶⁷ Whether repair of the perforation by the laparoscopic approach is better than by conventional open repair is undetermined.⁶⁶

Bhogal RH et al.⁵⁶ evaluated whether the early results from laparoscopic repair resulted in improved patient outcome compared with conventional open repair. In this study All patients who underwent repair of perforated peptic ulcer disease during a 12-month period were included in the study. The primary end points that were evaluated were total operative time, nasogastric tube utilisation, intravenous fluid requirement, total time of urinary catheter and abdominal drainage. Thirty-three patients underwent surgical repair of perforated peptic ulcer disease (19 laparoscopic repairs and 14 open repairs; mean age, 54.2 (range, 32-82) years). There was no increase in total operative time in patients who had undergone laparoscopic repair (mean: 61 minutes laparoscopic versus 57 minutes open). There was significantly less requirement for intravenous/intramuscular opiate analgesia in patients who had undergone laparoscopic repair (mean time to oral analgesia: 1.2 days laparoscopic versus 3.8 days open). In addition there was a significant decrease in the time that the nasogastric tube (mean: 2.1 days laparoscopic versus 3.1 days open), urinary catheter (mean: 2.3 days laparoscopic versus 3.7 days open) and

abdominal drain (mean: 2.2 days laparoscopic versus 3.8 days open) were required during the postoperative period. Patients who had undergone laparoscopic repair required less intravenous fluids (mean: 1.4 days laparoscopic versus 3.1 days open) and returned to normal diet (mean: 2.3 days laparoscopic versus 4.8 days open) and full mobilization significantly earlier than those who had undergone open repair (mean: 2.3 days laparoscopic versus 3.3 days open). In addition, patients who had undergone laparoscopic repair required a shorter in-patient hospital stay (mean: 3.1 days laparoscopic versus 4.3 days open). Study concluded that, Laparoscopic repair is a viable and safe surgical option for patients with perforated peptic ulcer disease and should be considered for all patients, providing that the necessary expertise is available.

A study⁶⁸ on laparoscopic repair of perforations reported that, it is clearly feasible and mean duration of hospital stay is significantly lesser than that of open technique. But there is undoubtedly a learning curve for laparoscopy and reduced tactile feedback is present in laparoscopy and great care needs to be taken to avoid damage to adjacent tissues.⁶⁸

Lau WY et al.⁶⁹ reported that, The place of laparoscopic repair of perforated peptic ulcer followed by peritoneal toilet of the peritoneal cavity has been established. Whether repair of the perforated peptic ulcer by the laparoscopic approach is better than conventional open repair and whether sutured repair is better than sutureless repair are both undetermined. In their study authors compared laparoscopic versus open repair and suture versus sutureless repair of perforated duodenal and juxtapyloric ulcers. One hundred three patients were randomly allocated to laparoscopic suture repair, laparoscopic sutureless repair, open suture

repair, and open sutureless repair. Laparoscopic repair of perforated peptic ulcer (groups 1 and 2) took significantly longer than open repair (groups 3 and 4; 94.3 +/- 40.3 vs. 53.7 +/- 42.6 minutes: Student's test, $p < 0.001$), but the amount of analgesic required after laparoscopic repair was significantly less than in open surgery (median 1 dose vs. 3 doses) (Mann-Whitney U test, $p = 0.03$). There was no significant difference in the four groups of patients in terms of duration of nasogastric aspiration, duration of intravenous drip, total hospital stay, time to resume normal diet, visual analogue scale score for pain in the first 24 hours after surgery, morbidity, reoperation, and mortality rates. Study concluded that, Laparoscopic repair of perforated peptic ulcer is a viable option. Sutureless repair is as safe as suture repair and it takes less time to perform.

Lau H.⁷⁰ in 2004 compared the efficacy and safety of open and laparoscopic repair of perforated peptic ulcers. MEDLINE and EMBASE were searched from January 1990 to December 2002 for studies published in English. The reference lists of identified studies were checked. Thirteen studies (n=658) were included: 2 RCTs (n=161), 4 prospective non-randomised studies (n=228) and 7 retrospective controlled observational studies (n =264). Study concluded that, Laparoscopic repair of perforated peptic ulcer was associated with a significantly lower wound infection, reduced post-operative pain and analgesic consumption, but a longer operating time and higher reoperation rate.

Recently, Antoniou SA et al.⁵⁷ conducted a meta-analysis of randomized trials to test the hypothesis that, minimally invasive approach involves less operative stress and results in decreased morbidity and mortality. Medline, EMBASE, and the Cochrane Central Register of Randomized Trials databases were searched, with no

date or language restrictions. Authors identified 4 randomized trials, with a cumulative number of 289 patients, that compared the laparoscopic approach with open sutured repair of perforated ulcer. Analysis of outcomes did not favor either approach in terms of morbidity, mortality, and reoperation rate, although odds ratios seemed to consistently support the laparoscopic approach. Results did not determine the comparative efficiency and safety of laparoscopic or open approach for PPU. Study concluded that, in view of an increased interest in the laparoscopic approach, further randomized trials are considered essential to determine the relative effectiveness of laparoscopic and open repair of PPU.

The total trauma incurred by the patient undergoing an operation is the sum of access trauma and the surgical procedural trauma. When the access trauma of a midline laparotomy (open surgery) is relatively large compared with the procedural trauma of patch repair for perforation, the benefit of minimal access-laparoscopy will be maximized.

A randomized controlled trial²² concludes that laparoscopic repair confers significant benefits to the patient in the form of reduced post-operative pain, lesser chest infections, a shorter hospital stay and earlier return to normal activities compared to open technique.

A meta-analysis⁵⁷ in which authors identified 4 randomized trials, with a cumulative number of 289 patients that compared the laparoscopic approach with open sutured repair of perforated ulcer. Analysis of outcomes did not favor either approach in terms of morbidity, mortality, and reoperation rate, although odds ratios seemed to consistently support the laparoscopic approach.

A published study⁷¹ concludes that laparoscopic technique for suturing and peritoneal lavage is safe but is technically demanding. Their study showed zero incidence of postoperative leak and intra-abdominal collection.

A study⁷² concluded that postoperative pain was less after laparoscopic repair, wound infection rate was significantly lower but re-operation rate was higher after laparoscopic repair compared to open technique.

Memon and Brow⁷³ reported one of the earliest studies, that laparoscopic closure of perforated duodenal ulcer is technically possible if performed within 6 hours.

Three randomised studies^{64,74,75} shown a decrease in analgesia requirement, but no benefit in terms of length of stay, time to resume normal diet, visual analogue score in first 24 hours or early return to activity.

Overall, laparoscopy provides an accurate diagnosis in patients with perforated peptic ulcer and can be used safely to treat these conditions without resorting to laparotomy. However, large prospective, randomised trials are required to clarify the exact role of this new modality of treatment.

Empirically based risk assessment for important clinical events has been extremely useful in evaluating new therapies, in monitoring resources for effective use and improving quality of care.⁷⁶

Despite advances in diagnosis, management and critical care of patients with peritonitis due to hollow viscous perforation, prognosis remains poor. Early assessment by scoring systems will influence the management and prognosis. The

scoring systems may be helpful in patients with hollow viscous perforation may help in selecting patients for aggressive surgical approach,⁷⁶ grading the severity, decision making and improved therapy in the management.⁷⁷

Many scoring systems have been designed and used successfully to grade the severity of acute peritonitis like, Acute physiology and chronic health evaluation (APACHE) II score, Simplified acute physiology score (SAPS), Sepsis severity score (SSS), Ranson score, Imrite score, Mannheim peritonitis index (MPI).^{78,79}

Mannheim Peritonitis Index (MPI) Score

MPI was developed by Wacha and Linder in 1983.⁸⁰ It was developed based on the retrospective analysis of data from 1253 patients with peritonitis, in which 20 possible risk factors were considered. Of these only 8 proved to be of prognostic relevance and were entered into the Mannheim Peritonitis Index, classified according to their predictive power. Patients with a score exceeding 26 were defined as having a high mortality rate⁸⁰ The Mannheim Peritonitis Index (MPI) is a specific score, which has a good accuracy and provides an easy way to handle with clinical parameters, allowing the prediction of the individual prognosis of patients with peritonitis.⁸¹

Mannheim Peritonitis Index scoring⁸⁰

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
Exudate	
Clear	0
Cloufy purulent	6
Fecal	12

Definitions of organ failure²⁵

- Kidney
 - Creatinin level >177 umol/L
 - Urea level > 167 mmol/L
- Lung PO₂ < 50 mm Hg
- PCO₂ > 50 mm Hg
- Shock – hypodynamic or hyper dynamic
- Interstitial obstruction – Paralysis > 24 hours complete mechanical obstruction.

Recently Muralidhar VA et al.⁷⁶ conducted a prospective study to evaluate Mannheim Peritonitis Index (MPI) score for predicting the outcome in patients with peritonitis. of 50 patients admitted and operated for peritonitis in JSS Medical College Hospital. The structured scoring system i.e. MPI was applied along with

other clinical and biochemical parameters recorded in pre-structured proforma. The overall mortality and morbidity was 14% and 38% respectively. MPI scores of 20, 21-29, and 30 had a mortality of 5%, 14%, and 50% respectively. MPI score of 25 had highest sensitivity of 72.09% and specificity of 71.43% in predicting mortality, 80.65% sensitivity and 57.89% specificity for morbidity. MPI score of > 25 were associated with 6.45 times higher risk of mortality (p=0.03), 5.72 times higher risk of morbidity (p=0.005) compared to patients with MPI score 25. Study concluded that, MPI is disease specific, easy scoring system for predicting the mortality in patients with secondary peritonitis. Increasing scores are associated with poorer prognosis, needs intensive management and hence it should be used routinely in clinical practice.

METHODOLOGY

This one year randomized controlled trial was conducted in the Department of General Surgery, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi from January 2015 to December 2015.

Study design

The study design was hospital based one year randomized controlled trial.

Study period and duration

This study was conducted for the period of one year from January 2015 to December 2015.

Place

The present study was conducted in the Department of General Surgery, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi a tertiary care teaching hospital attached to Jawaharlal Nehru Medical College, Belagavi.

Source of Data

Patients admitted in the Department of General Surgery, KLES Prabhakar Kore hospital and MRC, Belagavi with hollow viscus perforation undergoing either laparoscopic or open repair for hollow viscus perforation were enrolled.

Sample size

A total of 60 patients divided into two groups of 30 each were studied.

Sampling procedure

The sample size was calculated form following formula.

$$\text{Sample size (n)} = 2(Z_{\alpha/2} + Z_{\beta})^2 (S_1^2 + S_2^2) / (\bar{x}_1 - \bar{x}_2)^2$$

assigning ⁽⁵⁾ $\bar{x}_1=26.1, \bar{x}_2=10.4, S_1=6.9, S_2=15.1$

$$n = 2(1.56 + 0.84)^2 (7^2 + 15^2) / 16^2$$

$$n = 20 \text{ per group}$$

However, as the sample size of 20 was small, a sample size of minimum 30 per group was considered arbitrarily.

Randomization

The patients with hollow viscus perforation were randomized sequentially into two groups of 30 each as below;

- Group A: Patients underwent laparoscopic repair for hollow viscus perforation.
- Group B: Patients underwent open repair for hollow viscus perforation.

Selection criteria

Inclusion

- All patients getting admitted to KLES Dr. Prabhakar Kore Hospital, and Medical Research Centre, Belagavi with a diagnosis of hollow viscous perforation, who are willing to participate and giving written informed consent for participation in the study.
- Male/Female patients above 18 years of age.

Exclusion

- Patients who have not given informed and written consent to participate in the trial.
- Patients with associated diseases such as inflammatory bowel syndrome, malignancy, and systemic disorders such as connective tissue disorders, coagulopathies etc.
- Patients with co-morbid conditions like renal failure, liver failure, substance abuse.
- Patients requiring extensive surgical management such as resection anastomosis and perforations like appendicular perforation.

Ethical clearance

Prior to the commencement, the study was approved from the Ethical and Research Committee, Jawaharlal Nehru Medical College, Belagavi

Informed Consent

The patients fulfilling selection criteria were informed about the nature of study and a written informed consent was obtained (Annexure I).

Method of collection of data

Demographic data such as age, sex was obtained through an interview. All the patients interviewed for history. These patients were subjected to physical examination and systemic examination. The findings obtained were noted on a predesigned and pretested proforma (Annexure II).

Investigations

As a standard practice all the selected patients were subjected to the following investigations.

- Complete blood count
- Blood grouping
- Total leukocyte count
- Direct count
- platlet count
- Random Blood Sugar
- Renal function tests
- Serum creatinine
- Urine routine and microscopy
- X-ray - Erect abdomen

Procedure / Intervention

Group A

Patients underwent laparoscopic repair for hollow viscus perforation. Laparoscopic repair was to be performed under general anaesthesia. The patient was placed in Lloyd-davis position and open method was used for insertion of initial 10 mm umbilical port. A 30-degree side-view laparoscope was used in all cases. A second 10mm port is inserted over the left upper quadrant, and a single 5 mm port is placed in the right upper quadrant under laparoscopy. An additional 5 mm port is used for liver and gallbladder retraction as necessary. Perforation was closed primarily with absorbable sutures and omental patch was attached by mobilizing greater omentum. Thorough peritoneal lavage was given and drains kept

Group B

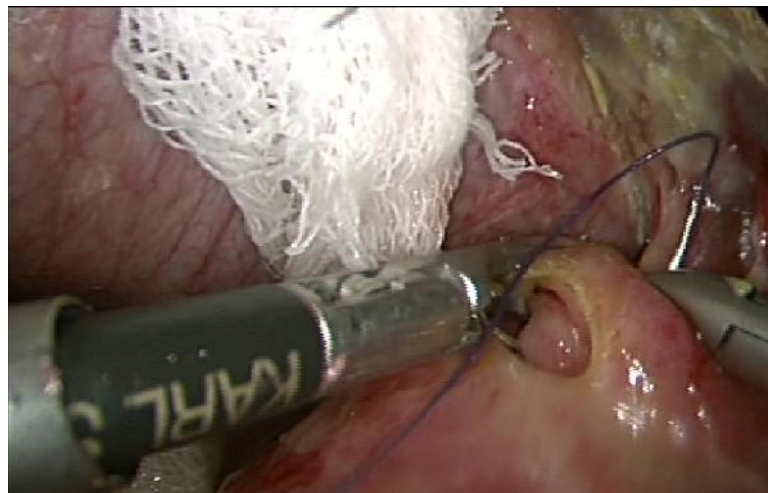
Patients underwent open repair for hollow viscus perforation. All open repairs were performed taking a midline incision and primarily sutured and omental patch was attached. Thorough peritoneal lavage was given and drains kept.



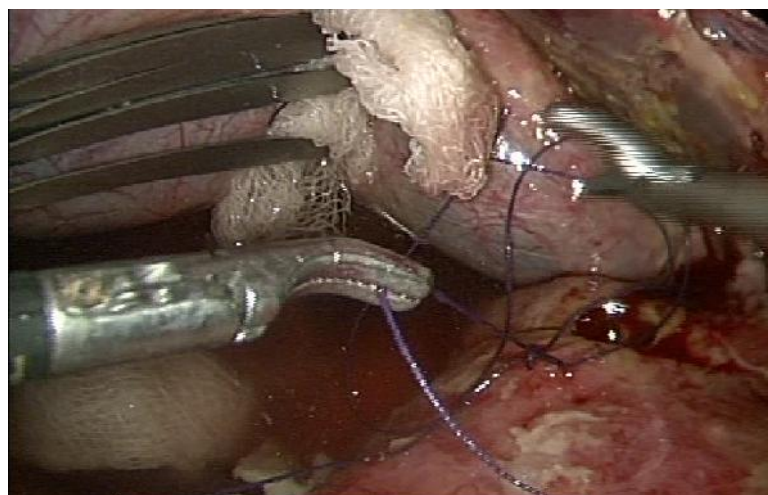
Photograph 1. Patient in Lloyd Davis position with Port placement



Photograph 2. Laparoscopic view of hollow viscous perforation in duodenum



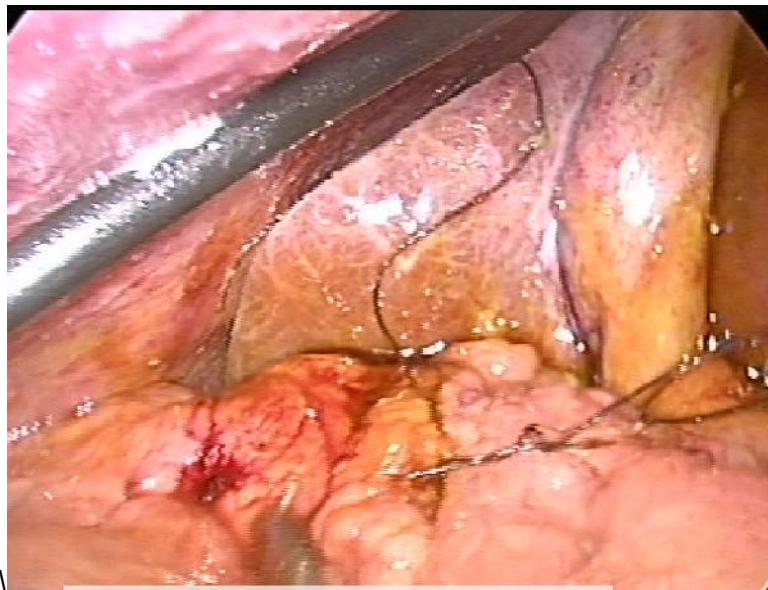
Photograph 3. Laparoscopic repair of hollow viscous perforation – Primary closure



Photograph 4. Laparoscopic knotting



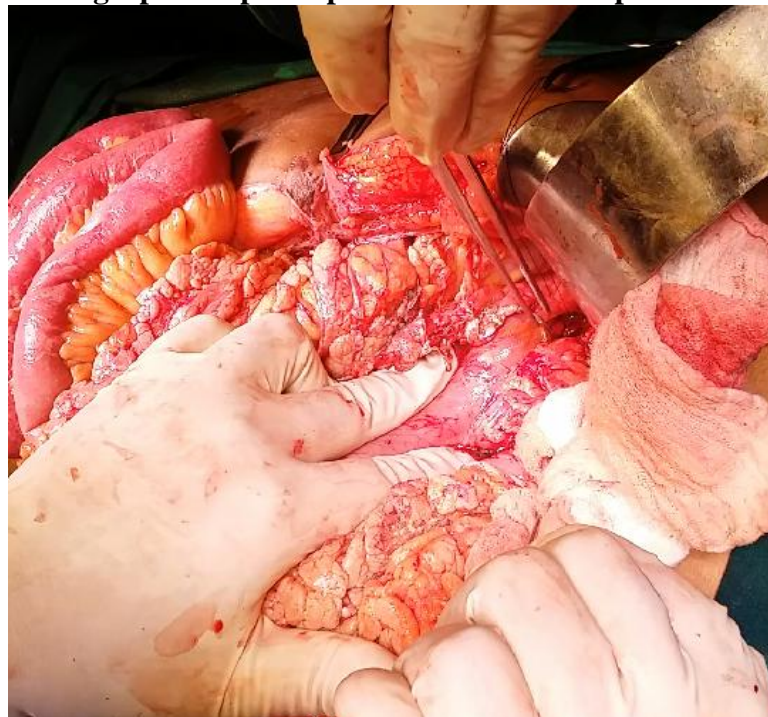
Photograph 5. Laparoscopic repair of hollow viscus perforation after knotting



Photograph 6. Application of Omental patch



Photograph 7. Open repair of hollow viscus perforation



Photograph 8. Open primary closure of hollow viscus perforation

Outcome variables

Patients were evaluate for

- Time taken for resumption of daily activities – Patients were assessed for tolerating normal diet, could fully ambulate and required only oral analgesics.
- Operative time.
- MPI score.

Mannheim Peritonitis Index (MPI)

Mannheim peritonitis index (MPI).^{78,79} MPI was developed by Wacha and Linder⁸⁰ in 1983. It was developed based on the retrospective analysis of data from 1253 patients with peritonitis, in which 20 possible risk factors were considered. Of these only 8 proved to be of prognostic relevance and were entered into the Mannheim Peritonitis Index, classified according to their predictive power. Patients with a score exceeding 26 were defined as having a high mortality rate The Mannheim Peritonitis Index (MPI) is a specific score, which has a good accuracy and provides an easy way to handle with clinical parameters, allowing the prediction of the individual prognosis of patients with peritonitis.⁸¹

Risk factors	Score
Age >50 years	5
Female sex	5
Organ failure	7
Malignancy	4
Preoperative duration of peritonitis >24hrs	4
Origin of sepsis not colonic	4
Diffuse generalised peritonitis	6
Exudate	Clear 0 Cloudy purulent- 6 Fecal-12
Mannheim's peritonitis index	

Statistical analysis

The data obtained was coded and entered in Microsoft Excel Spreadsheet (Annexure III). The data analysed using SPSS software version 20.0. Categorical data was expressed as rates, ratios and percentages and comparison was done using Fishers exact test and/or chi-square test. Continuous data was expressed as mean±standard deviation and comparison was done using independent sample t test. A 'p' value of 0.050 was considered as statistically significant.

RESULTS

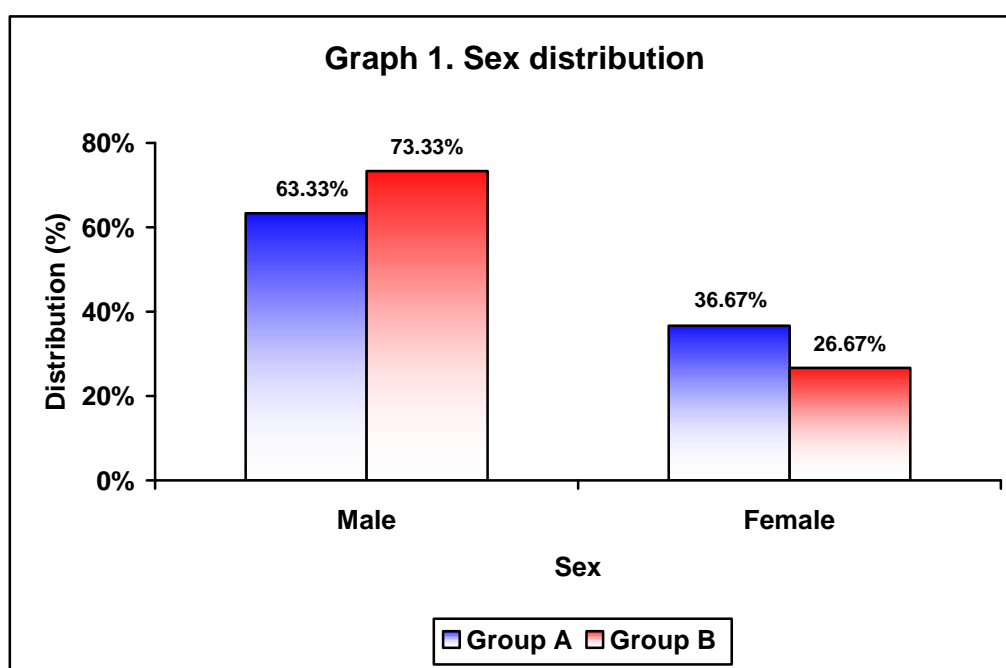
The present hospital based one year randomized controlled trial was done in the Department of General Surgery, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi. A total of 60 Patients admitted with hollow viscus perforation undergoing either laparoscopic or open repair for hollow viscus perforation from January 2015 to December 2015 were studied. These were randomized sequentially into two groups of 30 each as Group A (Patients in this group underwent laparoscopic repair for hollow viscus perforation) and Group B (Patients in this group underwent open repair for hollow viscus perforation).

The data obtained was analysed and the final results and observations were tabulated as below.

Table 1. Sex distribution

Sex	Group A (n=30)		Group B (n=30)	
	Number	Percentage	Number	Percentage
Male	19	63.33	22	73.33
Female	11	36.67	8	26.67
Total	30	100.00	30	100.00

p = 0.405

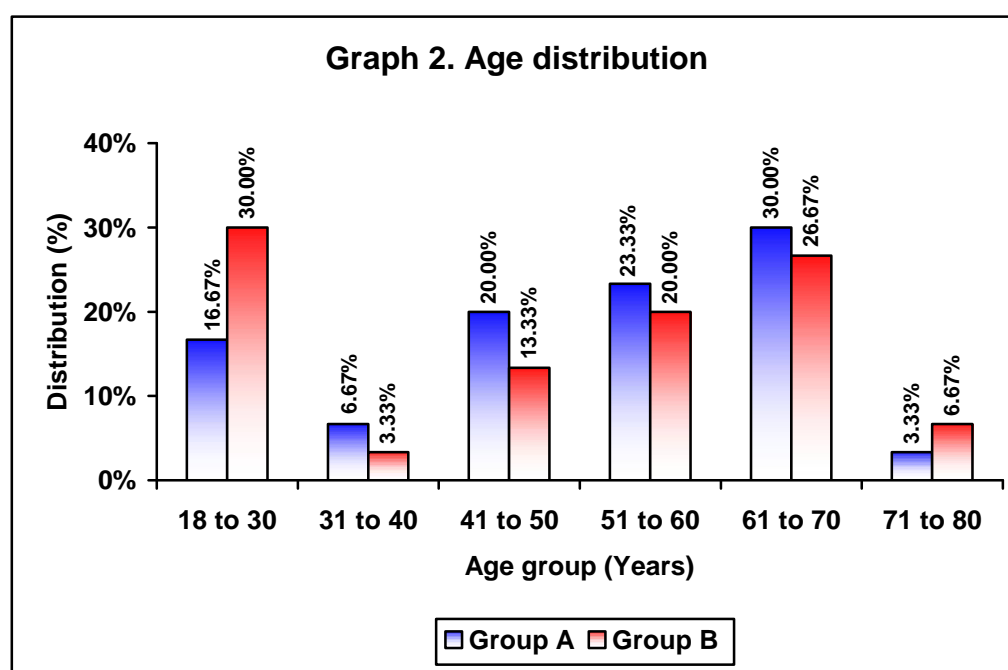


In the present study most of the patients were males in group A (63.33%) as well as in group B (73.33%). The sex distribution in group A and group B was comparable ($p=0.405$).

Table 2. Age distribution

Age group (Years)	Group A (n=30)		Group B (n=30)	
	Number	Percentage	Number	Percentage
18 to 30	5	16.67	9	30.00
31 to 40	2	6.67	1	3.33
41 to 50	6	20.00	4	13.33
51 to 60	7	23.33	6	20.00
61 to 70	9	30.00	8	26.67
71 to 80	1	3.33	2	6.67
Total	30	100.00	30	100.00

$p = 0.800$



In this study most of the patients in group A were aged between 61 to 70 years (30.00%) compared to 18 to 30 years in group B (30.00%). However this difference was statistically not significant ($p=0.800$).

Table 3. Mean age

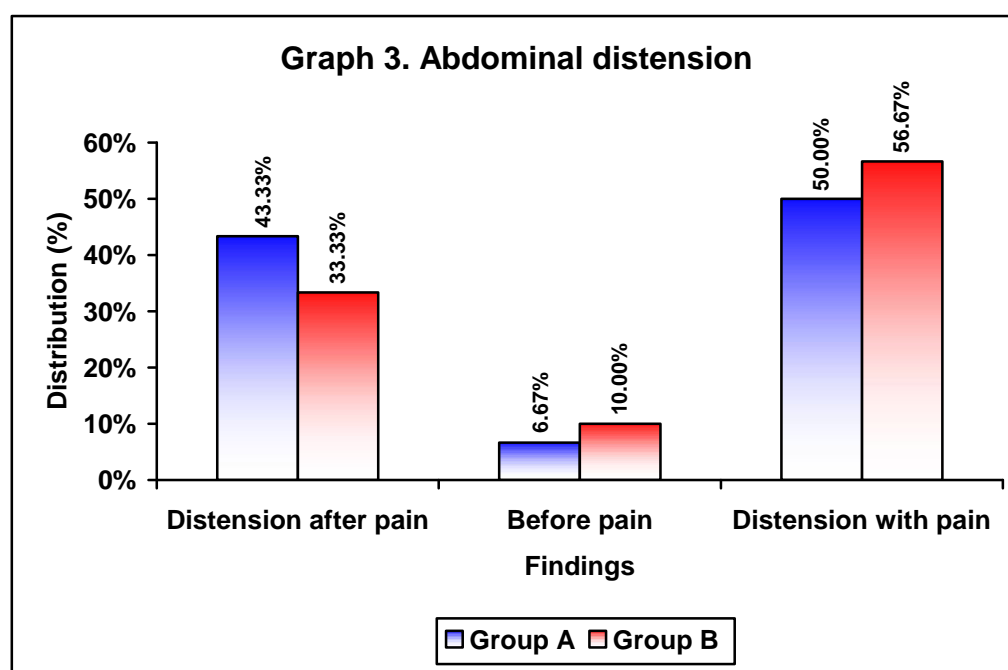
Variables	Group A (n=30)		Group B (n=30)		p value
	Mean	SD	Mean	SD	
Mean age (Years)	48.30	18.23	49.30	15.27	0.819

In the present study the mean age was almost similar in group A (48.30 ± 18.23 years) and group B (49.30 ± 15.27 years) ($p=0.819$).

Table 4. Abdominal distension

Findings	Group A (n=30)		Group B (n=30)	
	Number	Percentage	Number	Percentage
Distension after pain	13	43.33	10	33.33
Before pain	2	6.67	3	10.00
Distension with pain	15	50.00	17	56.67
Total	30	100.00	30	100.00

p = 0.699

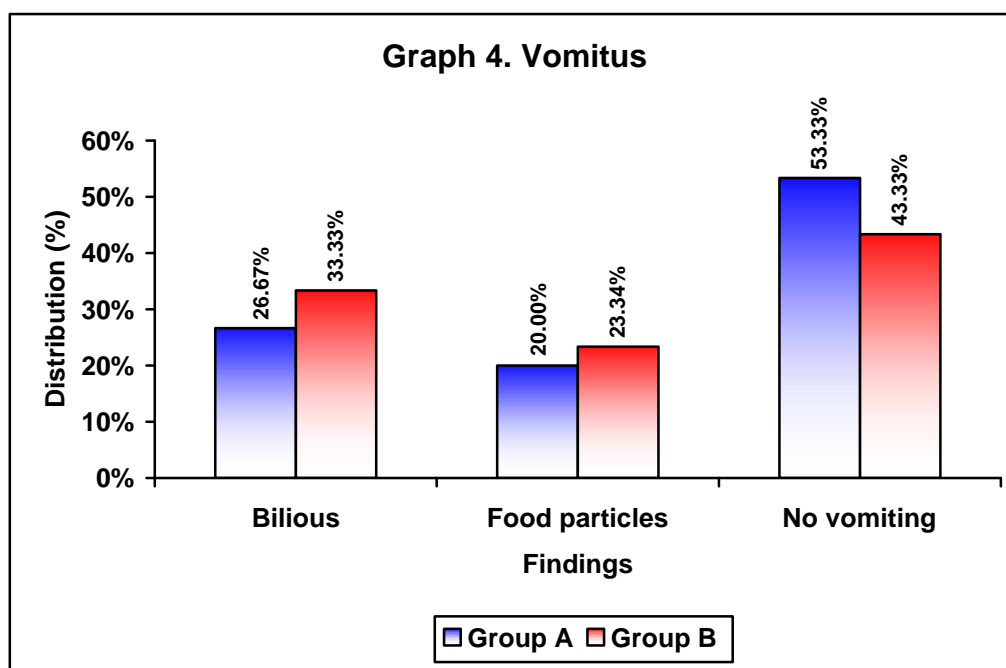


In the present study most of the patients had abdominal distension with pain in group B (56.67%) as well as group A (50.00%) (p=0.699).

Table 5. Vomitus

Findings	Group A (n=30)		Group B (n=30)	
	Number	Percentage	Number	Percentage
Bilious	8	26.67	10	33.33
Food particles	6	20.00	7	23.34
No vomiting	16	53.33	13	43.33
Total	30	100.00	30	100.00

$p = 0.737$

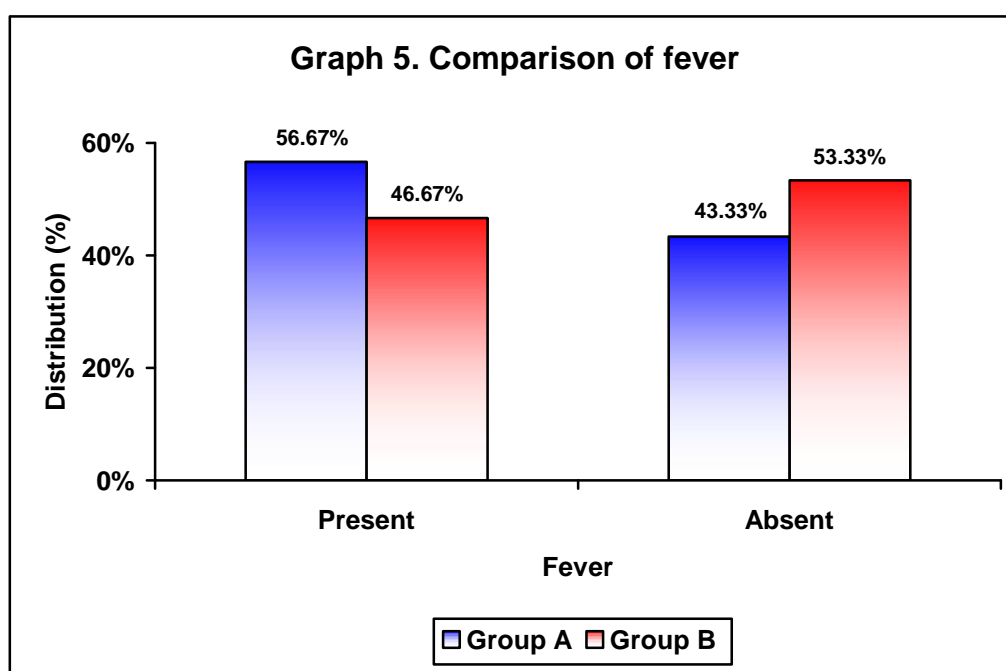


In this study most of the patients in group A (26.67%) and group B (33.33%) had bilious vomitus. However this difference was statistically not significant ($p=0.737$).

Table 6. Comparison of fever

Fever	Group A (n=30)		Group B (n=30)	
	Number	Percentage	Number	Percentage
Present	17	56.67	14	46.67
Absent	13	43.33	16	53.33
Total	30	100.00	30	100.00

p = 0.438

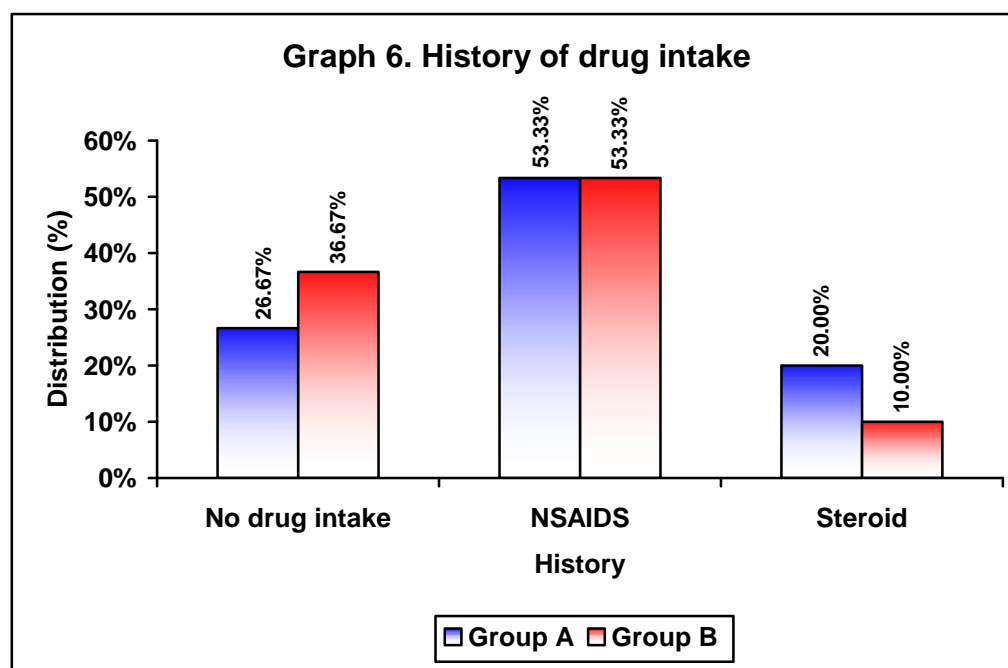


In the present study 56.67% of the patients presented with fever in group A compared to 46.67% of the patients in group B. However, this difference was statistically not significant (p=0.438).

Table 7. History of drug intake

History	Group A (n=30)		Group B (n=30)	
	Number	Percentage	Number	Percentage
No drug intake	8	26.67	11	36.67
NSAIDS	16	53.33	16	53.33
Steroid	6	20.00	3	10.00
Total	30	100.00	30	100.00

p = 0.479

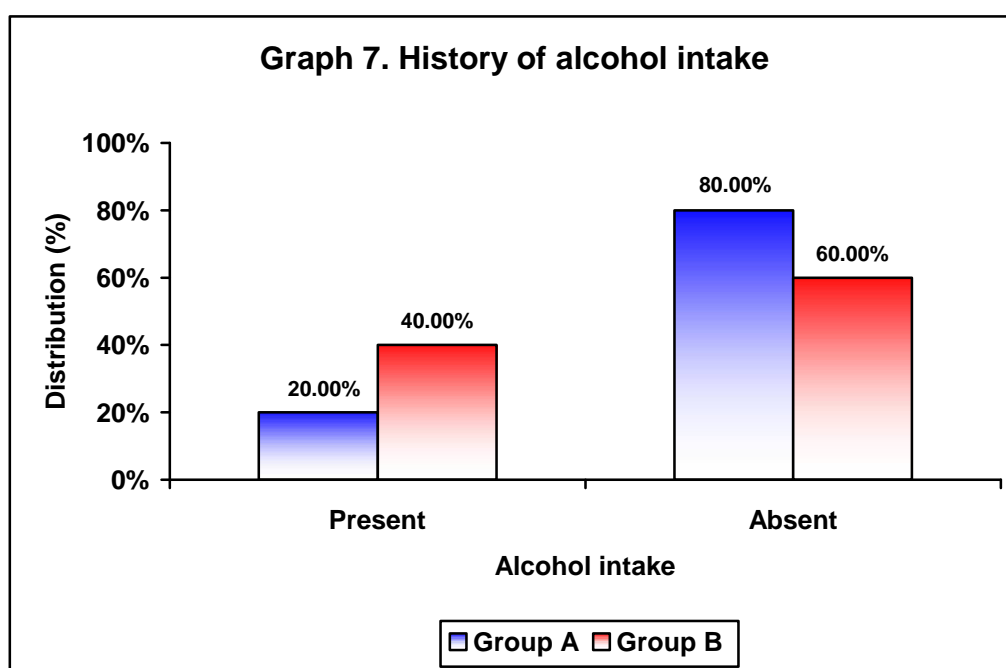


In this study most of the patients had drug history of NSAIDS (53.33% each) in group A as well as group B (p=0.479).

Table 8. History of alcohol intake

Alcohol intake	Group A (n=30)		Group B (n=30)	
	Number	Percentage	Number	Percentage
Present	6	20.00	12	40.00
Absent	24	80.00	18	60.00
Total	30	100.00	30	100.00

p = 0.091

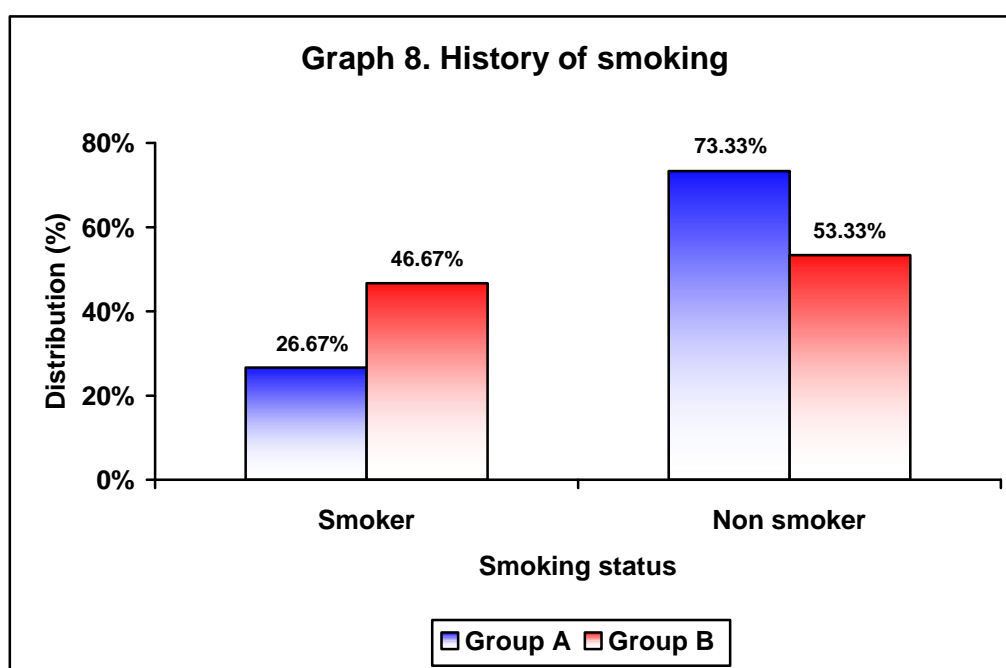


In the present study 40.00% of the patient in group B reported history of alcohol intake compared to 20% in group A. However this difference was statistically not significant (p=0.091).

Table 9. History of smoking

Findings	Group A (n=30)		Group B (n=30)	
	Number	Percentage	Number	Percentage
Smoker	8	26.67	14	46.67
Non smoker	22	73.33	16	53.33
Total	30	100.00	30	100.00

p = 0.108

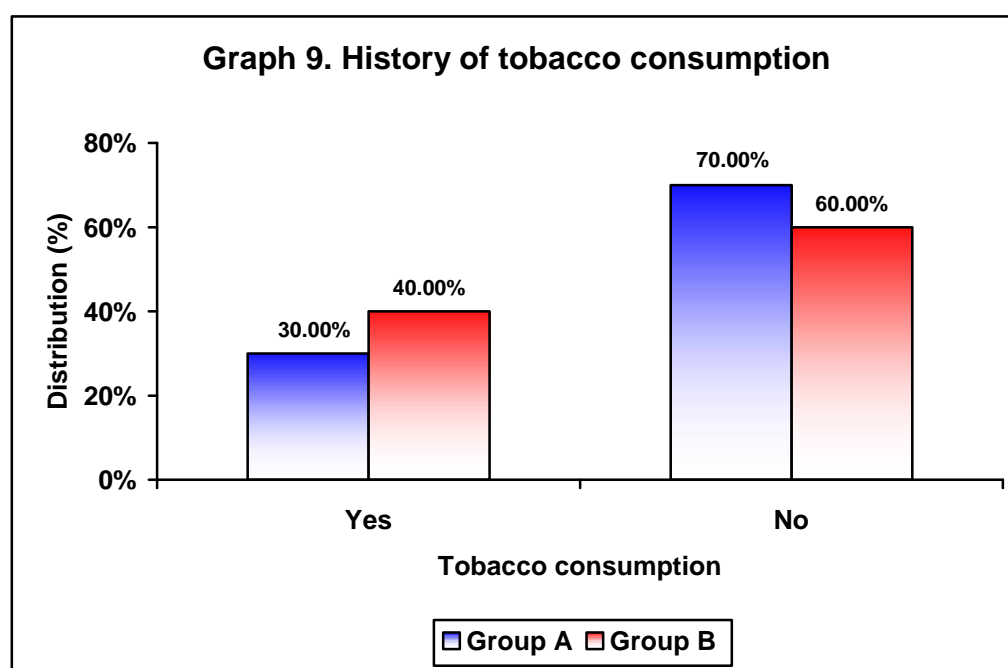


In this study 46.67% of the patients reported history of smoking in group B compared to 26.67% of the patients in group A. However this difference was statistically not significant (p=0.108).

Table 10. History of tobacco consumption

Findings	Group A (n=30)		Group B (n=30)	
	Number	Percentage	Number	Percentage
Yes	9	30.00	12	40.00
No	21	70.00	18	60.00
Total	30	100.00	30	100.00

p = 0.292

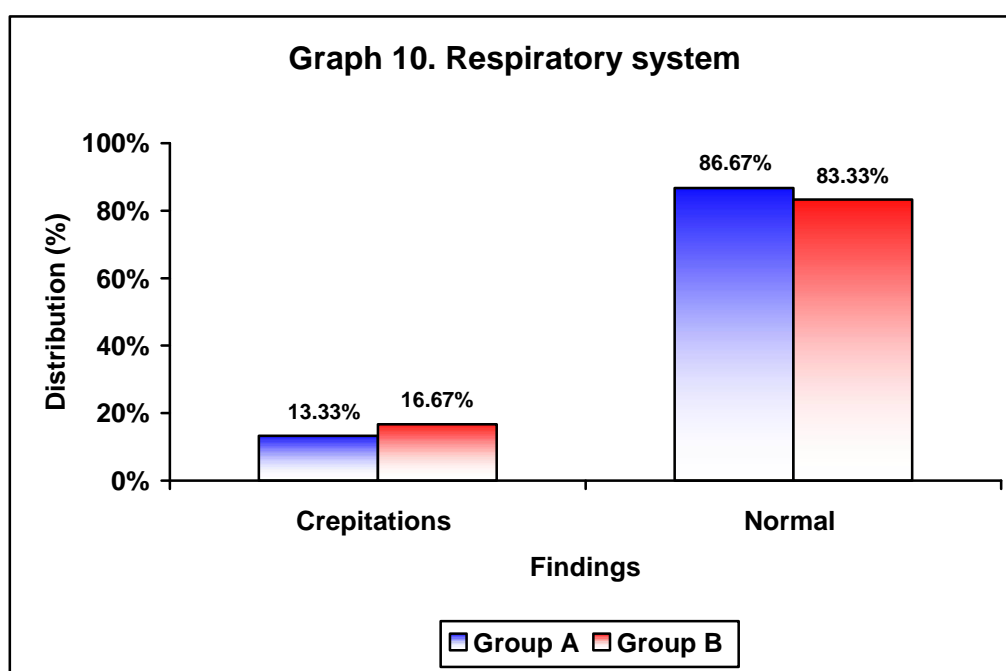


In the present study 40.00% of the patients reported history of tobacco consumption in group B while in group A. it was noted among 30.00% of the patients. However this difference was statistically not significant ($p=0.292$).

Table 11. Respiratory system

Findings	Group A (n=30)		Group B (n=30)	
	Number	Percentage	Number	Percentage
Crepitations	4	13.33	5	16.67
Normal	26	86.67	25	83.33
Total	30	100.00	30	100.00

p = 0.461

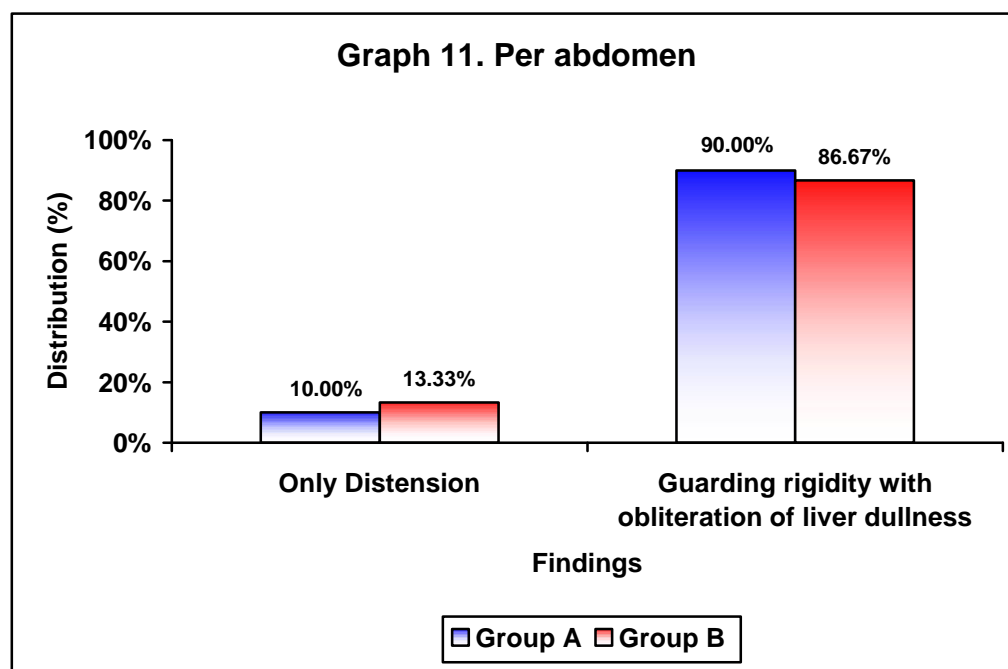


In this study on respiratory system examination, crepitations were noted among 16.67% of the patients in group B compared to 13.33% of the patients in group A (p=0.461).

Table 12. Per abdomen

Findings	Group A (n=30)		Group B (n=30)	
	Number	Percentage	Number	Percentage
Only Distension	3	10.00	4	13.33
Guarding rigidity with obliteration of liver dullness	27	90.00	26	86.67
Total	30	100.00	30	100.00

p = 0.543

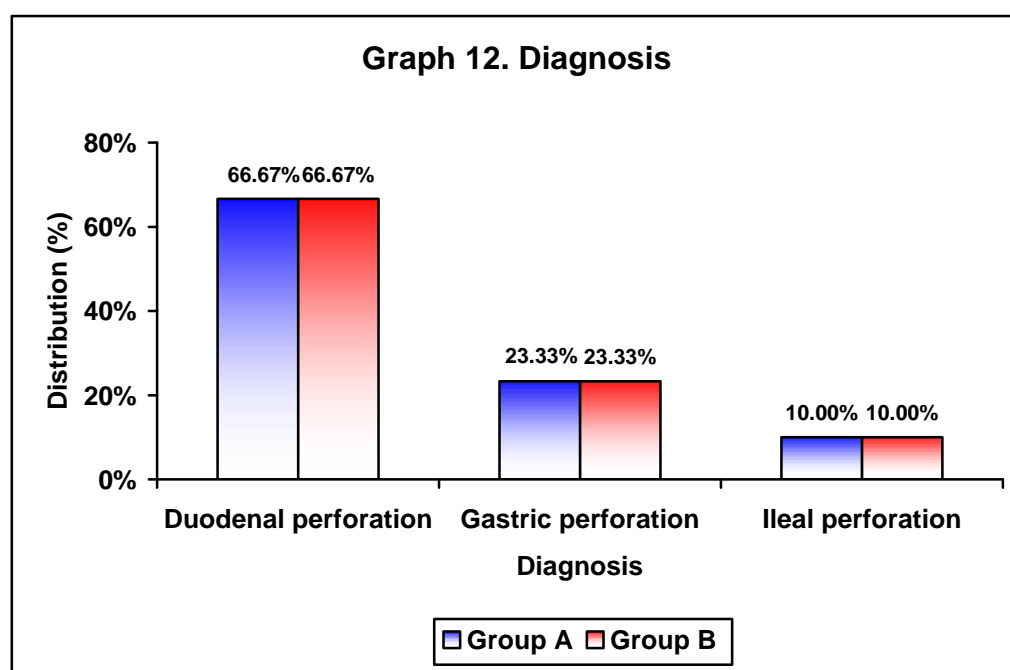


In the present study on abdominal examination guarding rigidity with obliteration of liver dullness related was present in 90.00% of the patients in group A compared to 86.67% of the patients in group B. However this difference was statistically not significant (p=0.543).

Table 13. Diagnosis

Diagnosis	Group A (n=30)		Group B (n=30)	
	Number	Percentage	Number	Percentage
Duodenal perforation	20	66.67	20	66.67
Gastric perforation	7	23.33	7	23.33
Ileal perforation	3	10.00	3	10.00
Total	30	100.00	30	100.00

p = 1.000

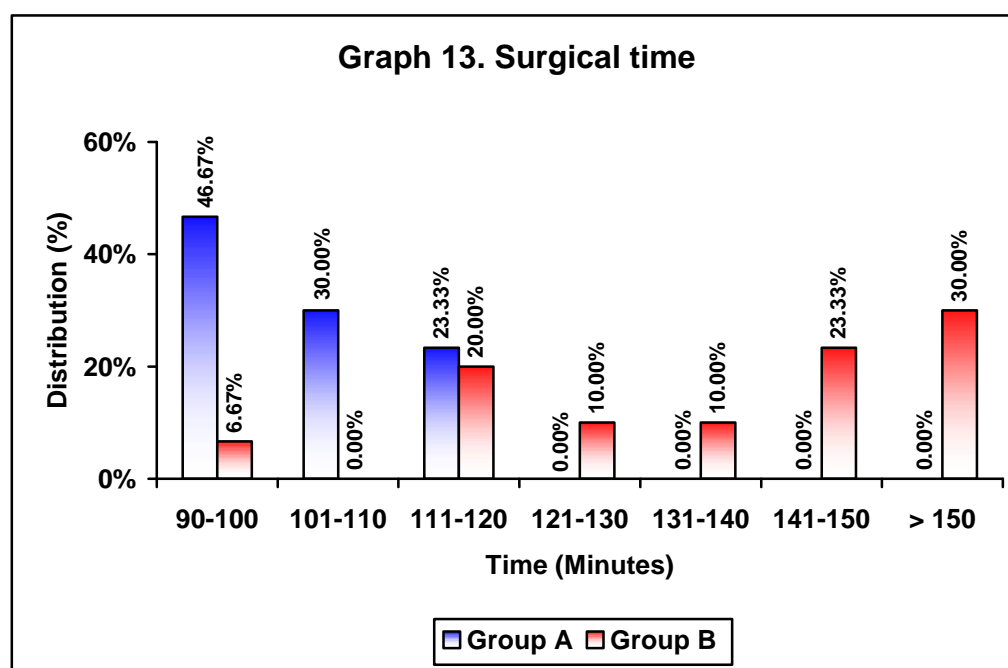


In this study almost equal number of patients had duodenal perforation in group A and group B (66.67% each) while gastric perforation was noted in 23.33% of the patients each in group A and B and ileal perforation was noted in 10.00% of the patients each in both the groups (p=1.000).

Table 14. Surgical time

Time (Minutes)	Group A (n=30)		Group B (n=30)	
	Number	Percentage	Number	Percentage
90-100	14	46.67	2	6.67
101-110	9	30.00	0	0.00
111-120	7	23.33	6	20.00
121-130	0	0.00	3	10.00
131-140	0	0.00	3	10.00
141-150	0	0.00	7	23.33
>150	0	0.00	9	30.00
Total	30	100.00	30	100.00

$p < 0.001$



In the present study among the patients in group A, maximum patients that is, 46.67% required 90 to 100 minutes compared to 6.67% of the patients in group B. This difference was statistically significant ($p < 0.001$).

Table 15. Mean Duration of surgery

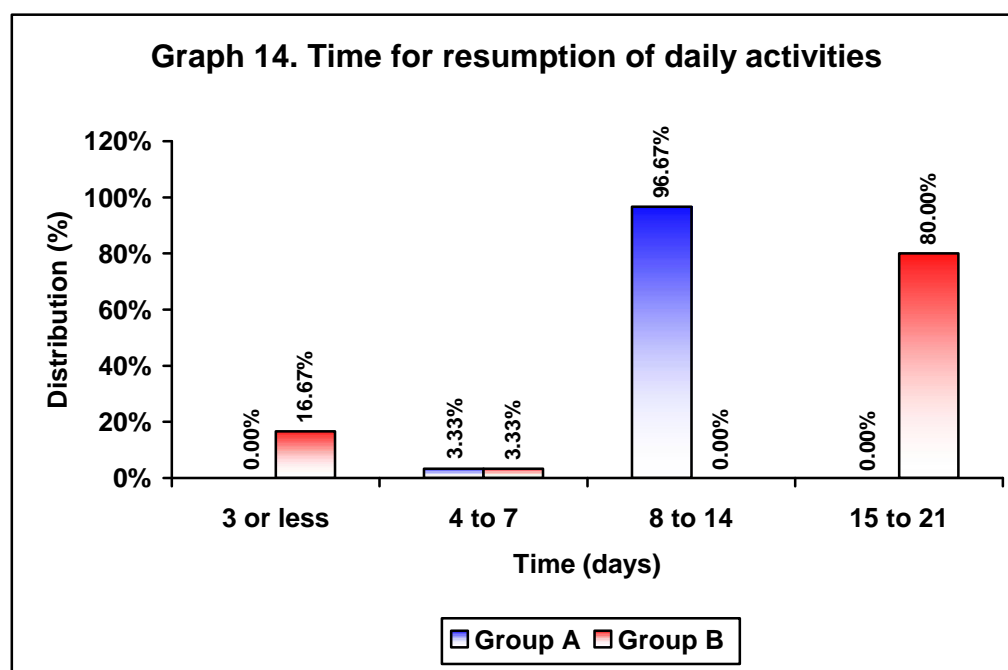
Variables	Group A (n=30)		Group B (n=30)		p
	Mean	SD	Mean	SD	value
Mean duration (Minutes)	105.13	9.57	141.67	20.19	<0.001

In the present study the mean duration of surgery was significantly low in group A (105.13 ± 9.57 minutes) compared to group B (141.67 ± 20.19 minutes) ($p < 0.001$).

Table 16. Time for resumption of daily activities

Time (Days)	Group A (n=30)		Group B (n=30)	
	Number	Percentage	Number	Percentage
3 or less	0	0.00	5	16.67
4 to 7	1	3.33	1	3.33
8 to 14	29	96.67	0	0.00
15 to 21	0	0.00	24	80.00
Total	30	100.00	30	100.00

p<0.001



In the present study time for resumption of daily activities in group A was between 8 to 14 days in 96.67% of the patients while in group B, 80.00% of the patients required 15 to 21 days. This difference was statistically significant ($p<0.001$).

Table 17. Mean Duration for resumption of daily activities

Variables	Group A (n=30)		Group B (n=30)		p value
	Mean	SD	Mean	SD	
Mean duration (Days)	4.53	0.73	11.87	2.93	<0.001

In this study the mean duration of resumption of daily activities was significantly low in group A (4.53 ± 0.73 days) compared to group B (11.87 ± 2.93 days) ($p < 0.001$).

Table 18. Manhiem's peritonitis index

Variables	Group A (n=30)		Group B (n=30)		p value
	Mean	SD	Mean	SD	
Manheim's peritonitis index (Score)	22.07	4.65	21.47	5.39	0.646

In the present study the mean Manheim's peritonitis index score was comparable in group A and group B (22.07 ± 4.65 vs. 21.47 ± 5.39 ; $p = 0.646$).

Table 19. Clinical characteristics

Variables	Group A (n=30)		Group B (n=30)		p value
	Mean	SD	Mean	SD	
Duration of pain abdomen	25.77	11.27	21.73	4.13	0.074
Duration of Vomiting	16.24	10.53	10.86	2.80	0.058
Respiratory rate (minute)	21.33	3.08	22.93	4.48	0.113
Pulse rate (/minute)	107.00	14.34	108.60	9.08	0.608
SBP (mm Hg)	108.20	13.00	111.33	10.42	0.307
DBP (mm Hg)	66.60	9.64	71.00	8.45	0.065
Haemoglobin (gm%)	10.98	2.50	11.35	1.50	0.490
Total count (/Cumm)	1521.04	3673.52	12.11	2.69	0.032
Serum creatinine (mg/dL)	1.35	0.52	1.23	0.44	0.370

The comparison of clinical characteristics is as shown in Table 19.

DISCUSSION

Hollow viscus perforation of abdomen is one of the commonest type of abdominal emergencies which is life threatening and most commonly managed by general surgeons.^{1,4}

Majority of patients presents with sudden onset of abdominal pain. A high index of suspicion is essential to diagnose visceral perforation early, as significant morbidity and mortality results from diagnostic delay.^{1,4}

Evaluation and management of hollow viscus perforations of abdomen provides some of the most challenging experiences for a surgeon with the advent of new technology. Surgeon must continually reassess standard method of treatment and be receptive to new ideas. However, the number of patients who needed surgical intervention for hollow viscus perforation, remained relatively unchanged.^{1,4}

Hollow viscus perforation is a common abdominal disease and laparoscopic surgery has changed the way of management for these emergencies. It is a condition for which the laparoscopic approach has significant attractions. Laparoscopy allows the confirmation of the diagnosis and furthermore allows the identification of the position, site, and size of the perforation.^{72,83,84} The procedure also allows closure of the perforation and adequate peritoneal toilette without the need for a large abdominal incision. Royal College of England stated⁶ that laparoscopic repair of perforations is clearly feasible and beneficial in terms of significant lower mean duration of hospital stay. Furthermore randomized controlled trial⁵ extended advantages of laparoscopic repair in terms of significant benefits to the patient

though reduced post-operative pain, lesser chest infections, along with shorter hospital stay and earlier return to normal activities compared to open technique.⁸² This study was an attempt to compare results of laparoscopic repair versus open repair so as to determine whether repair of the perforation by laparoscopic approach is better compared to conventional open repair in our setting.

This hospital based one year randomized controlled trial was carried out in the Department of General Surgery, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi. A total of 60 Patients admitted with hollow viscus perforation undergoing either laparoscopic or open repair from January 2015 to December 2015 were studied. These were randomized sequentially into two groups of 30 each as Group A (Patients in this group underwent laparoscopic repair) and Group B (Patients in this group underwent open repair).

In the present study males outnumbered females in Group A as well as group B that is, 63.33% of the patients were males in Group A compared to 73.33% of the patients in group B (73.33%) suggesting that, hollow viscous perforation common among males. These observations were similar to the studies by Tim HT et al.¹ to evaluate the etiological factors, clinical parameters, laboratory and Radiological investigations in the diagnosis and to compare and evaluate post operative recovery till time of discharge. Dandapat MC at et al.⁸⁵ stated that out of 340 cases there were 304 male patient (89.41 %) and 36 female patient (10.59 %) with sex ratio of 8.4:1. However, sex distribution pattern in group A and group B was comparable (p=0.405) in the present study.

In this study the most common age group among the patients in group A was 61 to 70 years comprised of 30% of the patients while in group B, 30.00% patients were aged 18 to 30 years ($p=0.800$). The mean age in group A was 48.30 ± 18.23 years compared to 49.30 ± 15.27 years in Group B ($p=0.819$). These findings suggest that, hollow viscous perforation is common in fifth decade of life. These findings were consistent with Dandapat MC et al.⁸⁵ who reported 38.24% of the patients with age 31-40 years and 24.12% with > 40 years while 14.71% were aged < 20 year and 22.94% with age of 21-30 years. However, age distribution pattern ($p=0.800$) and mean age ($p=0.819$) in group A and group B was comparable ($p=0.405$) in the present study.

In the present study clinical presentation in patients with group A and group B were comparable as most of the patients presented with abdominal distension with pain in Group B (56.67%) and group A (50.00%) ($p=0.699$). Most of the patients that is, 26.67% in group A had bilious vomitus (26.67%) compared to 33.33% in group B ($p=0.737$). More than half of the study population in group A (56.67%) presented with fever compared to 46.67% of the patients in group B ($p=0.438$). With regard to treatment history most of the patients had drug history of NSAIDS (53.33%) in Group A as well as Group B ($p=0.479$). personal history of alcohol intake was noted in 40.00% of the patients in group B compared to 20% in group A ($p=0.091$). Furthermore, smoking was reported by 46.67% of the patients in Group B compared to 26.67% of the patients in group A ($p=0.108$). history of tobacco consumption was reported by 40.00% of the patients in Group B while in Group A it was noted among 30.00% of the patients ($p=0.292$).

In this study, on respiratory system examination, crepitations were noted among 16.67% of the patients in Group B compared to 13.33% of the patient in Group A ($p=0.461$). On abdominal examination Guarding rigidity with obliteration of liver dullness related was present in majority of the patients in Group A and Group B (90.00% vs 83.33%; $p=0.543$).

In the present study almost equal number of patients had duodenal perforation in group A and group B (66.67% each) while Gastric perforation was noted in 23.33% of the patients each in group A and B and Ileal perforation was noted in 10.00% of the patients each in both the groups.

Many scoring systems have been designed and used successfully to grade the severity of acute peritonitis like, Acute physiology and chronic health evaluation (APACHE) II score, Simplified acute physiology score (SAPS), Sepsis severity score (SSS), Ranson score, Imrite score, Mannheim peritonitis index (MPI).^{78,79} MPI was developed by Wacha and Linder in 1983.⁸⁰ It was developed based on the retrospective analysis of data from 1253 patients with peritonitis, in which 20 possible risk factors were considered. Of these only 8 proved to be of prognostic relevance and were entered into the Mannheim Peritonitis Index, classified according to their predictive power. Patients with a score exceeding 26 were defined as having a high mortality rate The Mannheim Peritonitis Index (MPI) is a specific score, which has a good accuracy and provides an easy way to handle with clinical parameters, allowing the prediction of the individual prognosis of patients with peritonitis.⁸¹ In the present study the mean Mannheim's peritonitis index Score was comparable in group A and group B (22.07 ± 4.65 vs. 21.47 ± 5.39 ; $p=0.646$) suggesting comparable severity of hollow viscous perforation in both the groups.

The above findings suggest that, the demographic, clinical characteristics and diagnosis of the study population were similar in group A and Group B ($p>0.050$) ruling out the possible bias in study results.

In the present study among the patient in Group A, significantly higher number of patients required lower operative time that is, 46.67% of the patients in Goup A required 90 to 100 minutes while in Group B, most of the patients required > 150 minutes and duration of 90 to 100 minutes was noted among only 6.67% of the patients in group B ($p<0.001$). The mean duration of surgery was significantly low in group A compared to group B (105.13 ± 9.57 minutes vs (141.67 ± 20.19 minutes; $p<0.001$). These findings suggest that, laparoscopic repair of hollow viscus perforation is highly advantages as it consumes lesser time. These findings were consistent with a more recent study by Siu et al,⁷² where significantly reduced mean operating time was demonstrated. In contrast to these findings Antoniou SA et al.⁵⁷ in meta-analysis of randomized trials during 2013, reported that, mean duration of surgery was 62 minutes for the laparoscopic group and 53 minutes for the open group (weighted mean difference 0.38, 95% CI 1.22–1.99, $P=0.639$). In the present study also a difference of 36.5 minutes was observed. Early evidence from prospective studies demonstrated longer operating times for laparoscopic repair of PPU. Longer duration of surgery was reported by Lau et al.⁶⁴ in their trial conducted between 1992 and 1994. It has been suggested that acquaintance with the laparoscopic concept and laparoscopic suturing skills may result in a reduction in the duration of surgery over time.⁸⁶ A prospective analysis of more than 100 cases of laparoscopic treatment of PPU performed by surgical trainees has reported an acceptable mean operating time of 65 minutes, although most the study population

consisted of low-risk patients.⁵³ In contrast, Bertleff MJOE et al.⁸⁹ during last decade in 2009 to evaluate whether laparoscopic closure of a PPU is as safe as conventional open correction reported Operating time was significantly longer in the laparoscopy group (75 min versus 50 min), which was consistent with a study by Lunevicius R, et al.⁸⁷ in the literature. However, it was attributed to the more demanding laparoscopic suturing,⁸⁸ especially if the edges of the perforation are infiltrated and friable. Sutures easily tear out and it is more difficult to take large bites and to tie knots properly. Another reason for the increase in operating time is the irrigation procedure. Irrigating through a 5-mm or even a 10-mm trocar is time consuming, and suction of fluid decreases the volume of gas and reduces the pneumoperitoneum.⁸⁹

In the present study time for resumption of daily activities in group A was quick compared to group B. That is, resumption of daily activities among majority of the patients in Group A (96.67%) was between 8 to 14 days compared to 80% of the patients in Group B with duration of 15 to 21 days. This difference was statically significant ($p < 0.001$). Also, the mean duration of resumption of daily activities was significantly low in group A (4.53 ± 0.73 Days) compared to group B (11.87 ± 2.93 Days) ($p < 0.001$) that is earlier by almost 6 days. These findings show that, laparoscopic repair of hollow viscus perforation is highly beneficial as it helps patient to resume his daily routine activities early. These findings were consistent with a recent study by Zedan AS et al.⁹⁰ in Egypt to compare between laparoscopic and laparotomy repair of perforated duodenal ulcer found that those patients who underwent laparoscopic procedure resumed normal activity earlier than the patients in the laparotomy group, 14 ± 1.9 versus 20.5 ± 3.9 days ($p < 0.001$). Data collected

by Lau⁹⁰ involved six studies that examined that the time taken to resume normal activity by patients underwent laparoscopic surgery was significantly earlier than the patients who underwent open repair, as shown in the studies conducted by Mehendale et al.⁹² and Katkhouda et al.⁸⁴ Siu et al.⁵³ found that patients returned to normal activities after laparoscopic repair within an average of 10 ± 6.9 days, which was significantly earlier than the return of those who underwent open repair 26 ± 15.1 days, and this agreed with the results of our study.

Overall the present study showed that, laparoscopic repair is the ideal procedure for the management of hollow viscous perforation as it results in lower surgical time and early resumption of daily activities. Furthermore, another benefit of the laparoscopic procedure not often mentioned in literature^{89,91} is cosmetic outcome which was not taken into the consideration in this study as the long term follow up was not feasible due to lack of follow up which was limitation of the study. Now a days patients are aware of this benefit, and sometimes this is the reason why they demand laparoscopic surgery.

Further multicentric studies with large sample size considering cosmetic outcome would provide precise information of the laparoscopic repair of hollow viscous perforation.

CONCLUSION

Based on the result of this study it may be concluded that, laparoscopic repair is beneficial procedure for the management of hollow viscous perforation in terms of lower surgical time and early resumption of daily activities

SUMMARY

Perforation of a hollow viscus is an emergency which prompts a rapid and effective treatment. Open surgery is considered as the standard approach, but the use of laparoscopy for diagnostic purposes and treatment in favorable circumstances and locations appears to be a safe alternative with a number of advantages. This study was aimed to compare results of laparoscopic repair versus open repair so as to determine whether repair of the perforation by laparoscopic approach is better compared to conventional open repair.

This one year hospital based randomized controlled trial was conducted in the Department of General Surgery, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi. A total of 60 Patients admitted with hollow viscus perforation undergoing either laparoscopic or open repair from January 2015 to December 2015 were studied. The patients were randomized sequentially into two groups of 30 each as Group A (laparoscopic repair) and Group B (open repair).

Most of the patients were males in Group A (63.33%) as well as group B (73.33%) ($p=0.405$). The most common age group was 61 to 70 years (30.00%) in group A were and 18 to 30 years in Group B (30.00%) ($p=0.800$). The mean age was almost similar in group A (48.30 ± 18.23 years) and Group B (49.30 ± 15.27 years) ($p=0.819$). Most of the patients had abdominal distension with pain in Group B (56.67%) as well as group A (56.67%) ($p=0.515$). Most of the patients in group A had bilious vomitus (26.67%) compared to vomitus with food particles in group B (23.33%) ($p=0.737$). 56.67% of the patients presented with fever in group A compared to 46.67% of the patients in group B ($p=0.438$). Other characteristics

including drug history of NSAIDS ($p=0.479$), alcohol intake ($p=0.091$), smoking ($p=0.108$), tobacco consumption ($p=0.292$), systemic examination findings were comparable between two groups ($p>0.050$). Almost equal number of patients had duodenal perforation in group A and group B (66.67% each) while Gastric perforation was noted in 23.33% of the patients each in group A and B and Ileal perforation was noted in 10.00% of the patients each in both the groups ($p=1.000$). The mean Mannheim's peritonitis index Score was comparable in group A and group B (22.07 ± 4.65 vs. 21.47 ± 5.39 ; $p=0.646$). Maximum patients in Group A that is, 46.67% of the patients required lower surgical time that is, 90 to 100 minutes compared to only 6.67% in group B ($p<0.001$). The mean duration of surgery was significantly low in group A (105.13 ± 9.57 minutes) compared to group B (141.67 ± 20.19 minutes) ($p<0.001$). Time for resumption of daily activities in patients with group A was less that is 8 to 14 days in 96.67% of the patients compared 15 to 21 days in 80% of the patients with Group B ($p<0.001$). The mean duration of resumption of daily activities was significantly low in group A (4.53 ± 0.73 Days) compared to group B (11.87 ± 2.93 Days) ($p<0.001$).

Laparoscopic repair is beneficial procedure for the management of hollow viscous perforation in terms of lower surgical time, and early resumption of daily activities.

BIBLIOGRAPHY

1. Tim HT, Ghata S, Mishra RK, Mohapatra JS, Dugar D. Clinicopathological Study on Hollow Viscus perforation. *J Pharm Biomed Sci* 2015;05(02):100-3.
2. Silen W. *Cope's Early Diagnosis of the Acute Abdomen*, 22nd ed., New York, Oxford University Press, 2010.
3. Kauffman GL. Acute abdomen In: Corson JD Williamson RCN. Editors surgery Mosby, UK 2001.
4. Rao M, AbdulSamee A, Khan SM. Hollow viscus perforation: a retrospectum study. *International J Recent Sci Res* 2015;6(3):3250-4.
5. Achkar E, Richard G. Farmer Bertram Fleshler. *Peptic ulcer disease in clinical gastroenterology* 2nd ed., USA: Lea and Febiger: 1992.
6. Afridi SP, Malik F, Rahman SU, Shameen H, Samo KA. Spectrum of Perforation Peritonitis in Pakistan: 300 cases eastern Experience *World J Emer Surg* 2008;3:31.
7. Arthur DJ, Thomas V, Berne, John DA. Perforated duodenal ulcer - An alternative therapeutic plan. *Arch Surg* 1998;133:1166-70.
8. Beniwal US. Comparative study of operative procedures in typhoid perforations". *Ind J Surg* 2003;65(2):172-6.
9. Ceneviva RC. Simple suture without proximal gastric vagotomy for perforated duodenal ulcer. *Br J Surg* 1986; 73:427-30.

10. Christiansen J. Perforated duodenal ulcer managed by-simple closure versus closure and proximal vagotomy. Prospective study of 50 cases. *Br J Surg* 1987;74(4):286-7.
11. Dorairajan LN, Gupta S, Suryanarayana Deo SV, Chumber S, Sharma LK. Peritonitis in India- A decade's experience. *Trop Gastroenterol* 1995;16:33-8.
12. Inderbir S. Alimentary system Gastrointestinal tract in Human embryology, 5th ed., McMillan India press; 1991.
13. Inderbir S. Oesophagus, stomach and intestine. Textbook of human histology, 2nd edition, published by Jaypee Brothers Medical Publishers. 1994.
14. Kapoor VK. Perforated duodenal ulcer simple is safe. *Ind J Surg* 1988;50: 424-27.
15. Khan S, Khan IU, Aslam S, Haque A. Reterospective analysis of abdominal surgeries at Nepalgunj Medical College, Nepalgunj, Nepal: 2 year's experience. *Kathmandu University Medical Journal* 2004;2:336-43.
16. Mourougayan V. Smile SR, Sibal RN. Morbidity and mortality of definitive surgical procedures in duodenal ulcer perforation. *Ind J Surg* 1994;56(3): 102-8.
17. Nair SK, Singhal VS, Kumar S. Non traumatic intestinal perforation. *Ind J Surg* , 1981;43(5):371-8.
18. Playforth MJ, McMahom MJ. The indication of simple closure of perforated duodenal ulcer. *Br J Surg* 1978;65:699-701.

19. Ronald A. Squires and Russell G. Postier. Acute Abdomen In: Sabiston textbook of surgery: the biological basis of modern surgical practice. 19th ed. Texas: Elsevier; 2016.
20. Shackel Ford's Surgery of the alimentary tract, Charles J Yeo, ed. Philadelphia: Saunders Elsevier; 2007. pp. 1025-35
21. Kaiser AM, Katkhouda N. Laparoscopic management of the perforated viscus. *Semin Laparosc Surg* 2002;9(1):46-53.
22. Robertson GS, Wemyss-Holden SA, Maddern GJ. Laparoscopic repair of perforated duodenal ulcers, The role laparoscopy in generalised peritonitis *Ann R Coll Surg Engl* 2000;82(1):6-10.
23. Jeremy T. The peritoneum, omentum, mesentery and retroperitoneal space. 23rd edition. In: Russel RCG, Williams NS, Bulstrode CJK, Bailey and Love's Short Practice of Surgery. London: Arnold Publishers; 2013.
24. Erwin TR. Abdominal trauma. *The Surgical Clinics of North America*. WB Saunders Co 1990;70(3):517-75.
25. Williams NS, Bulstrode CJK, O'Connell PR (Ed.). *Bailey and Love's - Short practice of surgery*. 26th ed., New York: Taylor and Francis Group, CRC press; 2008. p. 1204-8.
26. Friedman S, Blunberg, RS. Inflammatory bowel disease. In: *Harrison's Principles of Internal Medicine*. 17th ed., New York, NY: McGraw-Hill Companies Inc.; 2008.

27. Gray H. Anatomy of the Human Body. Philadelphia, PA: Lea & Febiger; 2000.
28. Norton JA, Bollinger RR, Chang AE, Surgery. Basic science and clinical evidence. Springer-Verlag New York, Inc.; 2001
29. What is a Perforated Viscus? Available from: URL: <http://www.wisegeek.org/what-is-a-perforated-viscus.htm> Access Date 20.07.2016.
30. Hamer Davidson H, Sherwood L Gorbach. "Infectious diarrhea and bacterial food poisoning" in Sleisenger and Fordtran's Gastrointestinal and Liver Disease pathophysiology/ diagnosis/ management, 7th edition, vol 2, W. B. Saunders Co, 2002: 1882-85, 1889-1901.
31. William Schumer and Sheldon Burman. "The perforated viscous diagnosis and treatment in surgical emergencies". The surgical clinic of North America, edit by Nyhus Lloyd M, 1972; 52 (1):231-8.
32. Vinod Kumar B, Mathew AS. Clinical Study of Abdominal Hollow Visceral Perforation-Non Traumatic. JEMDS 2014; 30 (3):8366-71.
33. Sharma L, Gupta S, Soin AS, Sikora S, Kapoor V. Generalized peritonitis in India-The tropical spectrum. Surg Today 1991;21(3): 272-7.
34. Danvath K, Matta S. Clinical study and management of benign gastrointestinal perforations. J. Evid. Based Med Healthc 2016;3(37):1844-8.

35. Lundy J, Sherlock P, Kurtz R, Fortner JG, Turnbull AD. Spontaneous perforation of the gastrointestinal tract in patients with cancer. *Am J Gastroenterol* 1975;63(6):447-50.
36. Bertleff MJ, Lange JF. Perforated peptic ulcer disease: A review of history and treatment. *Dig Surg* 2010;27:161–9.
37. Longo WE, Degutis LC, Baker CC. Blunt hollow viscus injuries of the digestive tract: a poorly recognized phenomenon. *Conn Med* 1989;53: 451-4.
38. Watts DD, Fakhry SM. Incidence of hollow viscus injury in blunt trauma: an analysis from 275,557 trauma admissions from EAST multi-institutional trial. *J Trauma* 2003;54:289-94.
39. Joseph DK, Kunac A, Kinler RL, Staff I, Butler KL. Diagnosing blunt hollow viscus injury: is computed tomography the answer? *Am J Surg* 2013;205(4):414-8.
40. Sadler TW. "Digestive system" in *Langman's medical embryology*, 7th ed., Williams and Wilkin Company;1994.
41. Sorsi GA, Richard H, Turnage. "Appendicitis in Gastrointestinal and liver disease. Pathophysiology and diagnosis/management 7th ed., edit by Sleisenger and Fordtran's., 2002; 2: 2089-96.
42. Allardice DB, Toews S. Perforated viscus and acute abdomen. Available from: http://gidiseasesandcomplications.com/perforated_viscus/perforated_viscus.pdf Access Date: 22.07.2016.

43. Walker HK, Hall WD, Hurst JW, Clinical Methods: The History, Physical, and Laboratory Examinations. 3rd ed., Boston: Butterworths; 1990.
44. Weinberg JA, Fabian TC. Injuries to the stomach, small bowel, colon, and rectum. Available at: http://www.acssurgery.com/acssurgery/secured/getChapterByIDHTML.action?bookId5ACS&chapId5part07_ch08&type5tab. Accessed January 24, 2013.
45. Neideree MJ, Byrnes MC, Helmer SD. Delay in diagnosis of hollow viscus injuries: effect on outcome. *Amer Surg* 2003;69:293-8.
46. Malinoski DJ, Patel MS, Yakar DO, et al. A diagnostic delay of 5 hours increases the risk of death after blunt hollow viscus injury. *J Trauma* 2010;69:84-7.
47. Harisha NSMS, Vupputuri H, Lakshmi Narayana G. Unravelling Acute Abdomen – A Clinical Study. *International Journal of Science and Research (IJSR)* 2015; 4(6):85-8.
48. Shackel Ford's Surgery of the alimentary tract, Charles J Yeo, ed. Philadelphia: Saunders Elsevier; 2007. p. 1025-33.
49. Munro A. Perforated peptic ulcer. In: Peter JF, Zygmunt KH, George YG, Emergency Abdominal Surgery. 3rd ed., London: Chapman and Hall Medical; 1998. p. 163-76.
50. Nair SK, Singhal VS, Kumar S. Non-traumatic intestinal perforation. *Ind J Surg* 1981;43(5):371-8.

51. Dev AK, Paul S, Bhattacharjee N. Perforated duodenal ulcer, A review of results and efficacy of treatment in 171 cases. *Ind J Surg* 1994;56(5):222-7
52. Singh BU, Jindal D, Sharma J, Jain S, Shyam G. Comparative study of operative procedures in typhoid perforations. *Ind J Surg* 2003;65(2):172-6.
53. Sui WT, Chau CH, Law BKB, Tang CN, Ha PY, Li MKW. Routine use of laparoscopic repair of perforated peptic ulcer. *Br J Surg* 2004;91:481-4.
54. Janikalpesh, Saxena AK. Omental plugging for large sized duodenal peptic perforation: A prospective randomized study of 100 patients. *Southern Medical Journal* 2006;99(5):467-71.
55. Numanoglu ALP. The use of laparoscopy is well established in paediatric abdominal trauma. *Laparoscopy in abdominal trauma*. 2010; 28(3):119-21.
56. Bhogal RH, Athwal R, Durkin D, Deakin M, Cheruvu CN. Comparison between open and laparoscopic repair of perforated peptic ulcer disease. *World J Surg*. 2008;32(11):2371-4.
57. Antoniou SA, Antoniou GA, Koch OO, Pointner R, Granderath FA. Meta-analysis of laparoscopic versus open repair of perforated peptic ulcer. *JSLs* 2013;17(1):15-22.
58. Coghlan JG, Gilligan D, Humphries H, et al. *Campylobacter pylori* and recurrence of duodenal ulcers—a 12-month follow-up study. *Lancet* 1987;2:1109-11.

59. Lau WY, Leung KL, Zhu XL, Lam YH, Chung SC, Li AK. Laparoscopic repair of perforated peptic ulcer. *Br J Surg* 1995;82:814-6.
60. Sawyers JL. Acute perforations of peptic ulcer. In: Scott HW, Sawyers JL, eds. *Surgery of the Stomach, Duodenum and Small Intestine*. 2nd ed., Boston, MA: Blackwell Scientific; 1992. p. 566-72.
61. Hugh TB. Perforated peptic ulcers. In: Schwarz S, Ellis HA, eds. *Maingot's Abdominal Operations*. 9th ed. Norwalk, CT: Appleton and Lange; 1990. p., 627-645.
62. Nathanson LK, Easter DW, Cuschieri A. Laparoscopic repair/peritoneal toilet of perforated duodenal ulcer. *Surg Endosc* 1990;4(4):232-233.
63. Périssat J, Collet D, Edey M. Therapeutic laparoscopy. *Endoscopy* 1992;24(1-2):138-43.
64. Mouret P, François Y, Vignal J, Barth X, Lombard-Platet R. Laparoscopic treatment of perforated peptic ulcer. *Br J Surg*. 1990;77(9):1006.
65. Benoit J, Champault GG, Labhar E, Sezeur A. Sutureless laparoscopic treatment of perforated duodenal ulcer. *Br J Surg* 1993;80:1212.
66. Sunderland GT, Chisholm EM, Lau WY, Chung SC, Li AK. Laparoscopic repair of perforated peptic ulcer. *Br J Surg* 1992;79:785.
67. Tate JJT, Dawson JW, Lau WY, Li AKC. Sutureless laparoscopic treatment of perforated duodenal ulcer. *Br J Surg* 1993;80:235.

68. Critchley AC, Phillips AW, Bawa SM, Gallagher PV. Management of perforated peptic ulcer in a district general hospital. *Ann R Coll Surg Engl* 2011;93(8):615-9.
69. Lau WY, Leung KL, Kwong KH, Davey IC, Robertson C, Dawson JJ, et al. A randomized study comparing laparoscopic versus open repair of perforated peptic ulcer using suture or sutureless technique. *Ann Surg* 1996;224(2):131-8.
70. Lau H. Laparoscopic repair of perforated peptic ulcer: a meta-analysis. Database of Abstracts of Reviews of Effects (DARE): Quality-assessed Reviews [Internet]. Available from: URL: <https://www.ncbi.nlm.nih.gov/pubmedhealth/PMH0061788/> Access Date 18.08.2016
71. Sze Li Siow, Mahendran HA. Laproscopic repair of perforated peptic ulcers : the sutured omental patch and focused sequential lavage technique (*Surg Laparosc Endosc Percutan Tech* 2014;24;134-13
72. Siu WT, Leong HT, Law BKB, Chau CH, Anthony CN, Fung LKH et al. laparoscopic repair for perforated peptic ulcer. *Ann Surg.* Mar 2002;235(3):313-9.
73. Memon MA, Brow G. Laparoscopic closure of acutely perforated duodenal ulcer- an early experience. *Ir Med J* 1993;86(3):106-7.
74. Miserez M, Eypasch E, Spangenberger W, Lefering R, Troidl H. Laparoscopic and conventional closure of perforated peptic ulcer. A comparison. *Surg Endosc* 1996;10:831-6.

75. So JB, Kum CK, Fernandes ML, Goh P. Comparison between laparoscopic and conventional omental patch repair for perforated duodenal ulcer. *Surg Endosc* 1996;10(11):1060-3.
76. Muralidhar VA, Madhu CP, Sudhir S, and Srinivasarangan M. Efficacy of Mannheim Peritonitis Index (MPI) Score in Patients with Secondary Peritonitis. *J Clin Diagn Res* 2014; 8(12): NC01–3.
77. Ponting GA, Sim AJW, Dudley HAF. Comparison of local and systemic Sepsis in predicting survival. *Br J Surg* 1987;74:750-2.
78. Kologlu M, Elker D, Altun H, Sayek I. Validation of MPI and PIA II in two different groups of patients with secondary peritonitis. *Hepatogastroenterology*. 2001;48:147-51.
79. Bosscha K, Reijnders K, Hulstaert PF, Algra A, van der Werken C. Prognostic scoring systems to predict outcome in peritonitis and intra-abdominal sepsis. *Br J Surg* 1997;84(11):1532-4.
80. Wacha H, Linder MM, Feldman U, Wesch G, Gundlach E, Steifensand RA. Mannheim peritonitis index – prediction of risk of death from peritonitis: construction of a statistical and validation of an empirically based index. *Theoretical Surg*. 1987;1:169-77.
81. Correia MM, Thuler LCS, Velasco E, Vidal EM, Schanaider A. Peritonitis Index in oncologic patients. *Revista Brasileira de Cancerologia*. 2001;47(1):63-8.

82. Di Saverio S, Bassi M, Smerieri N, Masetti M, Ferrara F, Fabbri C. Diagnosis and treatment of perforated or bleeding peptic ulcers: 2013 WSES position paper. *World J Emerg Surg* 2014;9:45.
83. Naegaard JM, Edwin B, Reiertsen O. Laparoscopic and open operations in patients with perforated peptic ulcer. *Eur J Surg* 1999;165:209-14.
84. Katkhouda N, Maver E, Mason R. Laparoscopic repair of perforated duodenal ulcers. Outcome and efficacy in 30 consecutive patients. *Arch Surg* 1999;134:845-50.
85. Dandapat MC. Gastrointestinal perforation review of 340 cases. *Indian J Surg* 1991;53(5):189-93.
86. Lunevicius R, Morkevicius M. Systematic review comparing laparoscopic and open repair for perforated peptic ulcer. *Br J Surg*. 2005;92:1195-207.
87. Lunevicius R, Morkevicius M. Comparison of laparoscopic vs open repair for perforated duodenal ulcers. *Surg Endosc* 2005;19:1565-71
88. Kirshtein B, Bayme M, Mayer T, Lantsberg L, Avinoach E, Mizrahi S. et al Laparoscopic treatment of gastroduodenal perforations. *Surg Endosc* 2005; 19:1487-90.
89. Bertleff MJ, Halm JA, Bemelman WA, van der Ham AC, van der Harst E, et al. Randomized clinical trial of laparoscopic versus open repair of the perforated peptic ulcer: the LAMA Trial. *World J Surg*. 2009;33(7):1368-73.

90. Zedan AS, Lolah MA, Badr ML, Ammar MS. Laparoscopic versus open repair of perforated duodenal peptic ulcer: a randomized controlled trial. *Menoufia Med J* 2015;28:62-8
91. Lau H. Laparoscopic repair of perforated peptic ulcer: a meta-analysis. *Surg Endosc* 2004; 18:1013-21.
92. Mehendale VG, Shenoy SN, Joshi AM, Chaudhari NC. Laparoscopic versus open surgical closure of perforated duodenal ulcers: a comparative study. *Indian J Gastroenterol* 2002;21:222-4.

ANNEXURE I

CONSENT FOR PARTICIPATION IN RESEARCH STUDY

Mr. /Mrs. /Miss. _____ we are requesting you to enroll yourself in study titled “A ONE YEAR RANDOMISED CONTROLLED TRIAL TO COMPARE LAPAROSCOPIC REPAIR VS OPEN REPAIR FOR THE TREATMENT OF HOLLOW VISCUS PERFORATION AT KLES PRABHAKAR KORE HOSPITAL BELAGAVI” conducted by Dr. ***** *****, Post Graduate in M.S. General Surgery under the guidance of Dr. ***** ***, Associate Professor, Department of General Surgery, Jawaharlal Nehru Medical College, Belagavi under KLE University, Belagavi.

Respected Sir/Madam,

We request you to enroll yourself to participate in our study as you are eligible for participating in the study. During the study your operative outcome will be accessed by some questions which will be answered by your operating surgeon.

Purpose of the study

- 1) Time taken to return to routine activities (in days)
- 2) Operating time

Procedure Involved

If you agree to enroll yourself in my study, I will ask your present& past history. You will undergo step wise physical examination and your routine blood and urine investigation will be carried out. You will be divided in either of the groups for respective treatment.

Risks and Benefits

There is no risk involved.

Benefit-It will help in decreasing post op complications.

Alternatives

Even if you decline the participation in the study, your operative outcomes will not be documented. Your participation in this research is voluntary. You may choose not to enroll yourself in this study. Your decision will not change present or future health care services offered to you at K.L.E.S. Dr. Prabhakar Kore Hospital. If you decide to participate you are free to withdraw at any time.

Privacy and Confidentiality

The only people to know that you are a research subject are members of the research team. No information about you or information provided by you during the research will be disclosed to other without your written permission except:

In emergency to protect your rights and welfare.

If required by law.

Institutional/sponsors policy

There is no possibility of any harm or injury during your participation in this study.

Authorization to Publish Results

When the results of the research are published or discussed, in a conference, no information will be displayed that would disclose your identity. Any information that is obtained in connection with this study and that can be identified with your identity remaining confidential.

Questions

In case you have any questions related to the study, in future or in case of study related injury or illness, you can contact Dr. ***** *****, Department of General Surgery, KLES Hospital and MRC, Belagavi, phone number: *****. Or Dr. *** *****, MS, *****, Associate Professor, Department of General Surgery, KLES Hospital and MRC, Belagavi.

If you have any queries about your rights as a study subject, you may call Dr. *****, Professor, Department of Pathology and Chairman, Jawaharlal Nehru Medical College Institutional Ethical Committee for Human Subjects Research, Phone number- *****, or extension ***** at J.N. Medical College, Belagavi.

“A ONE YEAR RANDOMISED CONTROLLED TRIAL TO COMPARE LAPAROSCOPIC REPAIR VS OPEN REPAIR FOR THE TREATMENT OF HOLLOW VISCUS PERFORATION AT KLES PRABHAKAR KORE HOSPITAL BELAGAVI.”

Consent for participation in prospective study

I, Mr. /Ms / Mrs. _____ voluntarily agree for the participation as a subject of study. By signing this consent form I am not giving up any of my legal rights, I may withdraw from the study anytime. I am signing the consent form after having read or been read for me in vernacular language, including the risks and the benefits and having all my questions answered.

Subject Name : _____

Signature or the Left Thumb Print of Subject : _____

Date:

Witness Name: _____

Signature: _____

Date:

Investigators Name: _____

Signature: _____

Date:

Place: _____

ANNEXURE II – PROFORMA

**"A ONE YEAR RANDOMISED CONTROLLED TRIAL TO
COMPARE LAPAROSCOPIC REPAIR VS OPEN REPAIR FOR
THE TREATMENT OF HOLLOW VISCUS PERFORATION AT
KLES PRABHAKAR KORE HOSPITAL BELAGAVI."**

Name & Address of the patient:

In Patient Number : _____

Sex : _____

Age of the Patient : _____

Weight of Patient : _____

Operating Surgeon : _____

Date of Admission : _____

Date of Surgery : _____

Date of Discharge : _____

Presenting history and symptoms

Complaints

a) Pain abdomen

Duration :

Time of onset :

Mode of onset :

Site :

Nature : intermittent/ colicky/ dull aching/

Continuous /vague/radiating

Intensity of pain: mild/moderate/ severe

Radiation :

Reliving/ Aggravating factors:

b) Distension of abdomen

Mode of onset : with/ after/ before the pain onset

c) Vomiting: Duration

Frequency :

Amount :

Vomitus : blood/ food particles/ bile/

Faecal matter :

d) Diarrhea

Duration

e) Malena

f) Constipation

g) Fever

Intermittent / continuous / undulant / duration

h) Drugs taken

Cortisone's / antihistaminics /aspirin /analgesics

i) Other complaints

Past history

Pain abdomen

Site / nature / associated symptoms & duration

H/O NSAID's or steroid intake

Personal History

Food habits : vegetarian/ non vegetarian/ mixed
Alcohol intake : chronic / occasional / non alcoholic
Smoking : No. of beedies / cigarettes per day & duration
Socio-economic status: low / middle / high
Bowel / Bladder :

Family History

General Physical Examination

Vitals

Pulse rate: SBP:

Respiratory rate: DBP:

Temperature:

Respiration:

a) Rate &Regularity: b) Type: Abdominal / Thoracic

Systemic examination

Respiratory System :

Cardiovascular System:

Skeletal system :

Per abdomen :

A) Inspection

Shape / movements of quadrants with respiration

Peristaltic movements:

Skin :

Umbilicus :

Engorged veins :

B) Palpation

Tenderness and site of tenderness :
Rebound tenderness :
Liver/ spleen / other masses :
Muscular rigidity/guarding :
Distension :

C) Percussion

Obliteration of liver dullness/shifting dullness

D) Auscultatio

Peristaltic sounds: Heard / not heard / Borborygmi

E) Measurements

Central nervous system: Appearance:
Built : Nourishment:
Facies : Hydration: good / dehydrated:
Anemia : Jaundice:
Cyanosis : Pedal edema:
Lymphadenopathy

8) Investigations

Routine :
Blood :
Hb% :
B.T Blood grouping :
RBS :
TLC :
DLC :
Platlet count :

Widal test :

Blood urea :

S. Creatinine :

Urine :

Sugar Albumin Microscopy:

Erect X- ray abdomen :

Air under diaphragm : present / absent

Bilateral / Unilateral

Diagnosis

Proposed Surgery

Laparoscopic or open repair for hollow viscus perforation

Variables

- Time to return to daily activities (In days)
- Operating time
- Mannheim's peritonitis index

Risk factors	Score
Age >50 years Female sex	
Organ failure	
Malignancy	
Preoperative duration of peritonitis Origin of sepsis not colonic Diffuse generalised peritonitis	
Exudate Mannheim's peritonitis index	

Signature of operating surgeon

ANNEXURE III – KEY TO MASTER CHART

/Cumm	-	Per cubic millimeter
/minute	-	Per minute
a	-	Abdominal distension after pain
b	-	Abdominal distension before pain
bl	-	Bilious vomiting
c	-	Continuous pain
CNS	-	Central nervous system
crp	-	Crepitations
d	-	Dull aching pain
D	-	Distension
DBP	-	Diastolic Blood Pressure
DuP	-	Duodenal perforation
F	-	Female
f	-	Food particles in vomitus
g	-	Generalized pain
gm%	-	Gram in percentage
gp	-	Gastric perforation
grp	-	Guarding and rigidity present with obliteration of liver Dullness
GUD	-	Gas under diaphragm
Ilp	-	Ileal perforation
Lap	-	Laparoscopic
M	-	Male

m	-	Moderate
mg/dL	-	Milligram per deciliter
mmHg	-	Millimeter of mercury
n	-	No
nad	-	No abnormality detected
nsd	-	Non steroidal anti-inflammatory drugs
Rx	-	Treatment
s	-	Severe
SBP	-	Systolic Blood Pressure
strd	-	Steroid
w	-	Abdominal distension with pain
y	-	Yes