

EVALUATION OF CASES OF HOLLOW VISCUS PERFORATION

Dissertation submitted in partial fulfilment of

M.D. DEGREE EXAMINATION

M.S. GENERAL SURGERY - BRANCH I

CHENGALPATTU MEDICAL COLLEGE, CHENGALPATTU



THE TAMILNADU DR .M.G.R. MEDICAL UNIVERSITY

CHENNAI, TAMILNADU

APRIL 2013

CERTIFICATE

This is to certify that this dissertation titled

“ EVALUATION OF CASES OF HOLLOW VISCUS PERFORATION ”

has been prepared by **DR.K. ANAND** under my supervision in the department of general surgery, Chengalpattu Medical College , Chengalpattu, during the academic period 2010 – 2013, and is being submitted to The Tamilnadu Dr.M.G.R. Medical University, Chennai, in partial fulfillment of the University regulation for the award of the Degree “Master Of Surgery” (M.S.,General Surgery) and his dissertation is a bonafide work.

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ACKNOWLEDGEMENT

I wish to express my sincere thanks to Dr.P.R.Thenmozhi Valli M.D , Dean, Chengalpattu Medical College & Hospital, Chengalpattu, for having kindly permitted me to utilize the hospital facilities.

I wish to express my grateful thanks to Prof. Dr. G.Raja Billy graham M.S, Professor & Head of the Department, Department of General Surgery , Chengalpattu Medical College ,Chengalpattu for his immense help, encouragement and constant supervision .

And I wish to thank my unit chief Dr. V.Mohanraj M.S ,and Dr. M.Abdul kader M.S, for their immense help and guidance and great care and attention to prepare this dissertation.

I wish to thank my unit assistant professors Dr.V.T.Arasu, M.S, Dr.P.Sangameshwaran M.S, for their valuable suggestions and utmost care to prepare this dissertation.

I owe great debt of gratitude to all the Assistant Professors and Tutors for their able help and support. They have been a source of great encouragement throughout my Post graduate course.

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S.NO	CONTENTS	PAGE.NO
1	INTRODUCTION	1
2	AIM OF THE STUDY	2
3	MATERIALS AND METHODOLOGY	3
4	REVIEW OF LITERATURE	7
5	OBSERVATION AND RESULTS	49
6	DISCUSSION	78
7	CONCLUSION	88
8	BIBLIOGRAPHY	90
9	ANNEXURES	
	Appendix	100
	Proforma	101
	Clinical photos	102
	Master charts	107

INTRODUCTION

Hollow viscus perforation is defined as the perforation of any hollow viscus in a patient who presents with acute abdomen with the presence of extra luminal air radiologically. The Causes of hollow viscus perforation includes peptic ulcer disease, perforation of a gastrointestinal neoplasm [benign or malignant], acute appendicitis with perforation, and acute colonic or small bowel diverticulitis, including Meckel's diverticulitis. Some rare causes may include iatrogenic perforations caused by endoscopes or catheters, or spontaneous rupture of the distal esophagus (Boerhaave's syndrome), and foreign body ingestion as well as ischemia leading on to loss of bowel wall integrity.

In my study,I have statistically analysed the incidence of hollow viscus perforation at different sites in GIT (excluding appendicular perforation) and to know about the various etiological factors,size of perforation and the other associated pathologies of various causes in Chengalpattu government hospital in the period of OCT 2010-OCT 12 .

AIMS OF THE STUDY :

To statistically analyse the incidence of the perforation at different sites of Gastrointestinal Tract and their Etiologies.

To know the site, size of the perforation and the surrounding induration and the associated pathology intraoperatively .

Objectives of the Study

1. To study etiological factors of perforation like
 - a. Traumatic (blunt)
 - b. Non-traumatic that is due to
 - Peptic ulcer disease,
 - Infections (typhoid fever ,tuberculosis,) ,
 - Neoplasms and other
 - Rare causes.
2. The relationship of smoking and alcohol with acid peptic disease.
3. To study about the site, size and presentation(acute or chronic) of various hollow viscus perforation.
4. To find out the commonest type of perforation in our set up and etiological factors for the same.
5. To study the various mode of complications.

MATERIALS AND METHODS :

The patients who are taken up for study are analysed from their case details from the medical registry and the details of the patient and their clinical details are analysed. Among the 166 patients taken up for study, they are prospectively and retrospectively analysed and follow up were done and they are filed up to give the study regarding hollow viscus perforation. I have attached the proforma of the patients.

METHODOLOGY :

Patients satisfying the inclusion criteria and who gave consent are taken up for the study .A clinical history and appropriate investigations are done as mentioned in the proforma enclosed.Based on the risk factors and clinical manifestations, definite surgery or plan for conservative management will be decided.

First all the patients and the details of their age and occupation and the place from they hailed are documented. The patients are asked for the details of the previous admission to the hospital for peptic ulcer or intake of antiulcerogenic drugs from the counter directly. If so details of the ulcer, duration of pain , aggravating and relieving factors are enquired and taken up for study.

The history regarding the loss of weight and loss of appetite are asked to work up for tumour cases.

Smoking history regarding number of cigarettes or beedis are obtained both from medical registry and eliciting the history from the patient.

The history of alcoholism and the details of chronic alcoholism were enquired and studied for the peptic ulcer and the different sites of perforation.

If the patients suffers from blunt abdominal trauma either from trauma or fall of heavy object over the abdomen , after asking the details of trauma from the patients or from the attenders, patient is taken up for laparotomy after the clinical and radiological pictures were suggestive of hollow viscus perforation.the findings in laparotomy were noted and the degree of injury and various factors regarding size of perforation, surrounding induration, sites in various parts of gastrointestinal tract and associated pathology and the associated morbidities regarding mesenteric tear, hemoperitoneum were taken up for study.

Intraoperatively the site and the size of the perforated ulcers and the various associated pathologies regarding the intraoperative findings were elicited and worked up for the extent of disease and the assessment of morbidity and mortality.

The blood parameters were analysed and the hemodynamic condition of the patients were improved before taken up for surgery.

All the patients were taken up for laparotomy and the above mentioned intraoperative findings were taken up for study and documented and analysed for the study.

The details of the patient with their age and sex and the etiological factors and various other modes of etiologies and the adverse social habits are also considered and the laparotomy findings regarding the site, size and the associated pathologies and complications of the patients and morbidity and mortality were analysed and enlisted in master chart (1-6).

Then all the details are centralised and processed through Microsoft excel.

INCLUSION CRITERIA :

Patients aged between 20 and 70 years admitted with

A) Obliteration of liver dullness

B) Radiologically by free air under the right dome of diaphragm in x-ray abdomen erect view.

EXCLUSION CRITERIA :

- 1) Patients aged below 20 years and above 70 years.
- 2) Patients with stab injury abdomen.
- 3) Patients with appendicular perforation(diagnosed USG or intraoperatively)
- 4) Patients who did not give consent for the study.
- 5) Patients who on chronic treatment with NSAIDS, antiplatelet drugs and the steroids.

REVIEW OF LITERATURE

HISTORY

Hippocrates describes the faces of terminal stages of peritonitis as Hippocratic facies since 460 BC.¹

Aristotle first describes blunt trauma to the abdomen may cause intestinal injury²

Conservative management was taken as a treatment modality by Herman Taylor at the King George Hospital, Ilford as a treatment of perforation, since 1944

In 1957 Taylor published his ten years experience of managing 256 patients with hollow viscus perforation, of which he treated 208 patients by conservative management.³

In 1981 SK Nair 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.

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.⁴

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.

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.

Sui WT (2004) explained that perforated duodenal ulcer can be managed by laparoscopic approach even in emergency setting.⁵

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.⁶

HOLLOW VISCUS PERFORATION :

I. PREVALENCE AND EPIDEMIOLOGY :

The epidemiology of hollow viscus perforation depends on the underlying cause. Although the incidence and prevalence of the various causes varies greatly, the morbidity and mortality of hollow organ perforation are significant in all cases, given the possibility of progression to peritonitis and the resultant complications.

The most common cause of hollow viscus perforation is gastroduodenal peptic ulcer disease. Peptic ulcer disease is more common disease, with a lifetime incidence of ten percent⁷ and with variable prevalence internationally, depending mainly on its association with the use of NSAIDs or H. pylori infection.

The incidence of perforation has been reported to be 2% to 5% in patients with peptic ulcer disease. Perforated peptic ulcer disease carries significant morbidity and mortality in old aged patients, because many patients are elderly and have associated comorbidities.⁸

The overall reported mortality rate varies between 1.3 to 20 %, and Factors such as old age, associated co morbid disease, preoperative shock, size of the perforation, delay in presentation and surgical procedure⁹⁻¹⁰, are the various risk factors for mortality in such a situation. Although the size of a perforation is an

important measure in determining the outcome, any definition of small perforation and giant perforations of duodenal ulcers is not exactly elicited in any of the world journals or literature.

The duodenum is the commonest site of perforation, followed by the ileum.

The differential diagnosis of a perforation in the small gut includes

S1	<u>SOLITARY PERFORATION</u>	<u>MULTIPLEPERFORATIONS</u>
1	Typhoid,trauma, duodenal perforation	Trauma, typhoid
2	Ascariasis, ambiasis, actinomycosis, tuberculosis	Tuberculosis, amebiasis (also in large bowel)
3	Tumours (primary and secondary)	Leukemia (CML)

Approximately 90 % of the patients with peptic ulcer disease give the history of previous ulcer or dyspepsia or intake of proton pump inhibitors or antacids..

II . PEPTIC ULCER DISEASE :

Focal defect in gastric or duodenal mucosa extending on to submucosa or deeper plane¹¹ or a break in epithelium of esophagus, stomach, duodenum, or Meckel's diverticulum due to the acid peptic disease or infections like H.pylori.

It may be acute or chronic. PPD peaks around people of old ages.¹¹

Prevalence is around two to ten percent.¹¹

Though the treatment of peptic ulcer disease has improved drastically. Emergency surgery and death rate has not decreased dramatically.

Sl.no	BENEFICIAL FACTORS	DETRIMENTAL FACTORS
1	 HELICOBACTER  INFECTIONS	NSAIDS AND ASPIRIN CONSUMPTION
2	MEDICAL MANAGEMENT	NO ULCER PROPHYLAXIS
3	 OPERATIVE MANAGEMENT	AGING POPULATION

The ultimate pathway in peptic ulcer disease is the acid peptic injury to duodenal mucosa

There is a remarkable difference in more recent studies from those of the 1990s, in which gender as a simple factor, now demonstrates a very slight preponderance in men. The previous studies suggested that men constituted 80% of patients with perforated duodenal ulcer. Equally remarkable is that more recent studies demonstrate a significant increase in mean age in these patients, being reported as high as 67 years in men and 77 years in women, a statistically significant difference.

There are three conditions which fall in the category of peptic ulcer disease:

Chronic ulcers in the duodenum occurs in duodenal bulb.

1. Chronic gastric ulcer
2. Chronic duodenal ulcer
3. Erosive gastritis

There are two types of chronic gastric ulcer;

- ❖ Type 1 → body of stomach ulcers
- ❖ Type 2 → ulcers develop in the antrum, or pyloric canal

Perforation is a life threatening complication of peptic ulcer disease and occurs in approximately 2 to 10 percent of peptic ulcers.

The first clinical description of perforated peptic ulcer was made by Crisp in 1843.

The change in the incidence_:

During the 19th century , ulcer perforation was a rare disease that occurred mainly in young women, with perforation mainly near the cardia region .

.During the early 20th century, the incidence of ulcer perforations, increased and ulcers were situated in the duodenum of males of middle age.

Non operative management of perforated peptic ulcer disease was first described in 1935 by Wangensteen and can be applied even now.

Mikulicz¹² introduced closure of perforation by suture in space 1885¹² when he closes a perforated gastric ulcer.

Cellan Jones first explained the use of pedicled Omental patch as a rapid method of treatment in duodenal perforation in 1929.¹³

Graham also described the use of free graft of omental patch to repair the perforation in 1937. Sharma have described free omental plug in form of

mushroom(serosal patch technique), for the closure of perforation greater than 2.5cm (giant peptic perforation).

III . PERFORATED GASTRIC ULCER :

The great majority of perforated gastric ulcers are located in the immediate prepyloric area.

They have the property as perforated duodenal ulcers, and same etiological and risk factors are applicable. However, perforation of ulcers elsewhere in the stomach introduces the possibility of malignancy, and immediate definitive resections of the stomach are recommended. If the patient's condition is poor, and only a simple closure is advised and the biopsy specimens should be taken from the margins of the ulcer, even in the suspected gastric ulcer and send for biopsy. If the biopsy resulted it to be malignant, the definite surgery has to be done .

If the patient is inoperable and there is increased risk of surgery related complications, and the patient is inoperable for surgery, palliative surgery such as anterior gastrojejunostomy can be done. Divine exclusion gastrojejunostomy can be done but is an obsolete procedure.

IV. NSAIDS AND THE PERFORATION :

The Non steroidal anti inflammatory drugs has been implicated as a treatment modality for patients of rheumatoid arthritis and osteoarthritis, which is considered as one of the important etiology for peptic ulcer and subsequently lead on to perforation.

The incidence of NSAID induced perforation is more in gastric region than duodenum and the prevalence is around ten to 15 %

The cause of APD is increased thrice in patients who on NSAIDS than control. whereas risk increases 5 fold in old aged patients of 60 years and above as the intake of drugs is more for pain and osteoarthritis.

Consumption of steroidal anti inflammatory drugs have increased the incidence of perforation 6- 8 times and contribute towards a quarter of perforation patients..

Recent research has confirmed the association of NSAIDs as a cause of peptic ulcer disease, the reduction in the gastrointestinal side effect of NSAIDS can be controlled by limiting the intake of ulcerogenic drugs, counselling and prescription of anti ulcer medications (proton pump inhibitors and the use of H₂ blockers) , prostaglandins, and antisecretory medicines), and prescription of NSAIDs with minimal gastrointestinal side effects to patients at risk of developing gastrointestinal complications.¹⁴

A recent study of lumiracoxib¹⁵ showed a three to four fold(79 %) reduction in ulcer complications compared with other NSAIDs in the treatment of patients with osteoarthritis.

But selective NSAIDs cost significantly more than nonselective agents. In the long term, refinement of NSAIDs and improved treatment protocols should further reduce the incidence of peptic ulcer disease and its complications.

There is now more uniform agreement in recent reports concerning the incidence of nonsteroidal anti-inflammatory drugs (NSAIDs) used by patients presenting with perforated ulcers;

These vary from of 32% to 60% in those patients with perforated ulcer in whom NSAID usage was implicated as a major factor.

So NSAIDS are accepted as iatrogenic cause of the peptic ulcer disease and for future perforation.

V. CIGARETTE SMOKING :

Cigarette smoking has been mainly implicated and a strong independent risk factor in the pathogenesis of peptic ulcer disease and its complications.¹⁶

The complications implicated in cigarette smoking are due to

- a) Decreases healing¹⁷

- b) Impairs response to healing¹⁷
- c) Increases complications as perforation.

But the exact mechanism is not known

Proposed mechanisms:

- Altered gastric emptying.
- Decreased bicarbonate production
- Increased H.pylori infection
- Noxious free radical production

Smokers have a three fold higher mortality from peptic ulcer than nonsmokers.

The proposed mechanism in smokers is that smoking causes reduction in the blood supply to gastric mucosa due to vasoconstriction, leading on to ischemia and that ischaemia reduces mucosal resistance against, for instance, the action of acid and ulcerogenic contribute to ulcer perforation.. Tobacco smoking is a well known risk factor for uncomplicated peptic ulcer.. the risk of peptic ulcer progressively increased with increasing pack years cigarettes.¹⁸

VI. MECKEL'S DIVERTICULITIS WITH PERFORATION :

Meckel's diverticulum is the most frequent ¹⁹ congenital anomaly of the GI tract, affecting approximately 2% of the total population. There is a male predominance in ratio of 3 : 2. Meckel's diverticula are designated true diverticula because their walls contain all the layers found in normal small bowel. They are usually found in the ileum within 100 cm of the ileocecal valve. Nearly 60% of Meckel's diverticula contain heterotopic mucosa, of which over 60% consist of gastric mucosa.

Pancreatic acini are the next most common; others include Brunner's glands, pancreatic islets, colonic mucosa, endometriosis, and hepatobiliary tissues. Hildanus in 1598 described Meckel's as "DISEASE OF TWOS":²⁰

- A. 2% prevalence,
- B. 2 feet proximal to the ileocecal valve in adults, and are
- C. 2 years of age. (symptomatic).
- D. Two mucosa (gastric and pancreatic)

The complications arising from Meckel's diverticulum is found to be four to six %.

Meckel's diverticula are asymptomatic unless associated complications arise¹⁹. The most common presentations associated with symptomatic Meckel's diverticula are

bleeding, intestinal obstruction, and diverticulitis ; Diverticulitis is present only in 2 % of the symptomatic patients²¹.They rarely presents with perforation.

INVESTIGATIONS :

The sensitivity of

[A] CT scanning for the detection of Meckel's diverticula is too low to be clinically useful.

[B] Enteroclysis is associated with an accuracy of 75%, but usually is not applicable during acute presentations of complications related to Meckel's diverticula.

[C] Radionuclide scans (99mTc-pertechnetate) can be helpful in the diagnosis of Meckel's diverticulum

The accuracy of radionuclide scanning is reported to be 90% in pediatric patients but less than 50% in adults

Treatment of symptomatic meckels: wedge resection or limited resection and anastomosis of the involved bowel

SMALL BOWEL PERFORATION :

1. Typhoid enteritis caused by *Salmonella typhi* can lead to overt intestinal bleeding and perforation, most often affecting the terminal ileum.
2. The distal ileum and caecum are the most common sites of intestinal involvement by infection due to *Mycobacterium tuberculosis* . This condition can result in intestinal inflammation, strictures, and fistula formation, similar to those seen in Crohn's disease.
3. CMV can cause intestinal ulcers, bleeding, and perforation.²²

VII. JEJUNAL PERFORATION :

Jejunal diverticula which may present with diverticulitis has been explained as the cause of jejunal perforation in literature. why the blunt injury to the abdomen causes perforation to jejunum is explained later.

Jejunal diverticula are rare with an incidence of less than 0.5% .

Pathologically, they are pseudo diverticula of the pulsion type, due to increased intraluminal pressure and weakening of the bowel wall. These outpouchings only contain only mucosa and submucosa (in contrast to meckels diverticulum of the terminal ileum, which is a true diverticulum as it contains all four layers of the small bowel)

Presentation :

Most cases of jejunal diverticulosis remaining completely asymptomatic²³,. These include chronic abdominal pain, malabsorption, hemorrhage, diverticulitis, obstruction, abscess formation and rarely diverticular perforation²⁴.

Complications in jejunal diverticulum : 10- 30 %

Etiology :

The exact etiology of jejunal diverticulosis is unknown, but thought to develop from factors such as

1. Intestinal Dyskinesia,
2. Abnormal Peristalsis
3. Increased intraluminal pressures.

These diverticula arise on the mesenteric border where there is weakening of the bowel wall where the mesenteric vessels penetrate the jejunum.

Jejunal diverticulosis can cause chronic nonspecific abdominal symptoms or, can present as an acute presentation such as perforation. Jejunal diverticulosis in the elderly can lead to significant morbidity and mortality in patients with abdominal pain and diarrhoea., initial management should be conservative line of

management mainly to reduce pain and reduce the symptoms and to reduce the risk of complications associated with diverticular disease.

Surgical management is the treatment of choice if jejunal diverticulum present as diverticulitis or intestinal perforation.

Solitary jejunal diverticulum²⁵ on mesenteric side is a rare clinical finding detected incidentally on thorough laparotomy. It should be considered in the differential diagnosis of acute abdomen, especially in the elderly patients.

Diverticulectomy with or without segmental bowel resection is the surgical management of choice.

VIII. ILEAL PERFORATION :

Enteric or typhoid fever is a systemic febrile condition which is caused by *Salmonella typhi*. Infection is by either direct contact with an infected individual or indirect contact via contaminated water or food. Ileal perforation is a late complication which occurs in the third week of typhoid fever. The main pathology is due to ileocecal lymphatic hyperplasia of the Peyer's patches, which may occur with secondary bacteremia and peritonitis. Peyer's patches undergo swelling and ulceration and can progress to capillary thrombosis and subsequent necrosis. These ulcerations are always located in anti mesenteric

border of the intestine and may perforate or bleed, third to fourth week of the disease though they usually heal without scar formation.

Majority of the patients are in the lower socioeconomic status and it mainly affects the younger age group and in third week of typhoid fever.

Most case of typhoid fever have seasonal variation and occurs especially in the period of summer or autumn.

The mortality rate in the literature was 9.9% (10-80²⁶ %).The mortality and the complications in ileal perforation due to typhoid fever are directly proportional to duration of infection, the onset of peritonitis and time of hospitalisation, and the time of surgery after hospitalisation.²⁷

Although conservative medical line of management reduces the mortality in typhoid perforation, early limited surgery is warranted to give good results to the patient²⁸

IX. TUBERCULAR PERFORATION :

Abdominal tuberculosis is the sixth²⁹ most common of extrapulmonary tuberculosis.

The presentation of abdominal tuberculosis is non specific.. Patients may present with abdominal pain, distention, nausea and vomiting and altered bowel

habit. Perforation is rare but serious complication of intestinal tuberculosis. The incidence of perforation due to tuberculosis is 1-15 % .The low incidence of tubercular perforation of the bowel is due to reactive fibrosis of the peritoneum, the organisms are trapped in lymphoid aggregation of the bowel wall, which undergoes inflammatory enlargement and ulceration of the overlying mucosa.

Tuberculosis of the small intestine may cause multiple perforations. the main pathology in tuberculosis of the bowel is due to the vasculitis which causes the ischemia of the involved bowel, leading on to the perforation. The perforation of small bowel due to tubercular etiology is rare ³⁰

X. APPENDICULAR PERFORATION :

Surgical therapy for appendicitis was first initiated secondary to the treatment of appendicular perforation. Hancock describes the treatment of appendicitis in 1848. He did it for acute appendicitis where there is no complications like appendicular abscess.

McBurney³¹ published a paper in 1894 describing the McBurney point as follows: "maximum tenderness, when one examines with the fingertips is, in adults, one half to two inches inside the right anterior spinous process of the ilium on a line drawn to the umbilicus."

The main etiology is due to fecoliths.

1. Acute appendicitis – 40 %
2. Gangrenous appendicitis without rupture - 65 %
3. Gangrenous appendicitis with rupture – 90 %

The strong association between delay in presentation and appendiceal perforation supported the proposition that appendiceal perforation is the reason for advanced stage of acute appendicitis. Recent studies describes that non perforated appendicitis and perforated appendicitis are two different modes of disease and should be treated in different manner.

Immediate appendectomy has long been the recommended treatment for acute appendicitis because of the presumed risk of progression to rupture. Children and old age people are more susceptible for appendicular perforation. The overall rate of perforated appendicitis is 25.8%. Children <5 years of age and patients >65 years of age have the highest rates of perforation (45 and 51%, respectively).the literature explains delay in presentation of appendicitis is a major cause for appendicular perforation.

Appendiceal rupture occurs most commonly in antimesenteric border of appendix distal to obstruction, the obstruction is mostly a fecolith in 90 % of the patients.

Appendicular rupture should be diagnosed if a patient with

- a temperature of >39°C (102°F)³³
- a white blood cell count of >18,000 cells/cu.mm³³

Children <5 years of age have 25% negative appendectomy rate and 45 % appendicular perforation rate.

The wound infection rate in children is 11% for the treatment of perforated appendicitis. Perforated appendicitis patients are more prone for intra abdominal abcesses.

The treatment regimen for perforated appendicitis generally includes immediate appendectomy and irrigation of the peritoneal cavity..IV antibiotics are preferred treatment in postoperative till the white blood count becomes normal or patient is afebrile for 24 hours period.

The dreaded complication of appendicular perforation is septic portal vein thrombosis which carries high mortality.³²

XI. GIST TUMOUR WITH PERFORATION :

Gastrointestinal stromal tumors are rare malignancies.

Although they are the most common sarcoma of the gastrointestinal (GI) tract, they represent only 0.2% of all GI tumors.³⁴

The term GIST was first employed in 1983 by Mazur and Clark to describe nonepithelial tumors of the GI tract that lacked the ultrastructural features of smooth muscle cells as well as the immunohistochemical characteristics of Schwann cells.³⁴

A defining feature of GIST is their gain of function mutation of oncogene KIT, a receptor tyrosine kinase. pathologic kit signal transduction is believed to be a main event in GIST pathogenesis. KIT expression is assessed by staining the tissues for CD117 Antigen and present in 95% of GISTS.³⁵

GISTs are usually asymptomatic, detected incidentally by laparotomy for some other reasons³⁴. Small bowel obstruction, nausea vomiting . abdominal distension and crampy pain is the most common mode of presentation. Hemorrhage is the second most common mode of presentation.³⁵

Obstruction of the GI tract is occasionally a presenting condition, sometimes may lead to perforation.³⁴

Surgery remains the standard therapy for all resectable non metastatic tumors.

Small intestinal GISTs should be treated with segmental resection. If the diagnosis is known before resection, wide lymphadenectomy can be avoided as GISTs are rarely associated with lymphnodal metastasis [4]

Imatinib mesylate, known commonly as Gleevec, usually used to treat chronic myelogenous leukemia (CML). This orally administered drug also inhibits KIT and PDGFRA protein tyrosine kinases. Imatinib inhibits KIT activity by lodging in an ATP-binding pocket that forms upon receptor dimerization³⁶

XII.COLONIC PERFORATION:

Infective colitis:

Infective organisms like cytomegalovirus and many organisms can cause colitis leading on to severe bloody colitis, toxic megacolon, sometimes may lead on to colonic perforation.³⁷

Caecal perforation :

Caecal perforation is most commonly due to a complication or consequence of colonic carcinoma presenting with stenotic growth or obstruction in any of the large bowel .There is conspicuous diffusion in segments proximal to the colonic obstruction (it usually determines an insidious onset of the benign or malignant neoplastic disease.it was previously thought as the chronic constipation which is leading on to caecal perforation. The high rate of mortality in caecal perforation is most commonly due to the leakage of faecal material in the peritoneal cavity that develops into septic and toxic peritonitis ³⁸

Obstruction at the ileocecal valve by growth or obstruction produces the symptoms suggestive of small bowel perforation. The obstruction of the distal colon is depending mainly on the competence of ileocaecal valve. The ileocecal valve is incompetent in 10 – 30% of individuals, and colonic pressure is relieved by reflux into the ileum. If colon is not decompressed through the ileocaecal

valve, a closed loop is formed between the obstruction of the bowel and ileocaecal valve. The colon distends progressively because the ileum continues to empty gas and fluid into the obstructed segment, circulation will be impaired and the gangrene and perforation of the caecal gangrene occurs. The wall of the right side of colon is thinner than left side of colon and have a large luminal diameter and caliber, according to the law of Laplace, the risk of caecal perforation is greater, there is a high risk of caecal perforation if the caecal diameter exceeds 10-12 cm.

Subtotal colectomy is indicated as a treatment for caecal perforation³⁹

Colonic diverticulitis :

A colonic diverticulum is a pulsion or false diverticulum, it does not contain all four layers of the bowel wall. Mucosa herniates through the muscle, covered by serosa. Diverticula occur where the arterial supply (vasa recta) penetrate the circular muscle layer

.(1) obstruction of the ostia of the diverticula lead to increased intradiverticular pressure and perforation develop in four "rows" at the points of the colonic circumference

.(2) Increase in intraluminal pressure erode the wall of the diverticulum.

Inflammation and necrosis result in colonic perforation.

The diverticulitis may be simple or complicated .

Investigation of choice is CT Abdomen.

Surgical treatment holds good but primary resection anastomosis or staged procedures can be done depending on faecal contamination and the stability of the patient.

Segmental Enteritis:

- Non-occlusive infarction of small or large bowel without any established etiology.
- Suspected etiology is either due to endotoxins or exotoxins or functional cause.
- Mainly affects the small bowel especially jejunum
- Presentation:
 - ❖ Abdominal pain with distension
 - ❖ Persistent tachycardia
 - ❖ Prolonged hypotension
 - ❖ Reduced bowel sounds

XIII. STERCORAL PERFORATION :

Stercoral perforation is the perforation of the large bowel with resultant leaking of fecal matter into the peritoneal cavity. The spillage of fecal material has got the highest mortality rate, the most common causes explained are diverticular disease and colorectal tumour. The main treatment is based on the surgery and intensive care management.

Colonic carcinoma is one of common causes of stercoral peritonitis. There are two main sites of perforation: growth in the proximal parts of the bowel, usually ascending colon, mainly due to diastatic perforation from long-lasting distant complete obstruction.

The second common site of perforation is a distant part of the colon, mostly a sigmoid colon. Sigmoid colon is the most common site for diverticular disease and malignancy, stercoral perforation can also occur in other parts of the bowel, either in appendix. The right colon is usually involved by diastatic perforation by colonic wall necrosis due to impaired blood perfusion through narrowed and elastic vessels, the risk of the perforation is higher if the caecum distends above 10-12 cm.

Diverticular perforation can occur also on the right or transverse colon despite that fact, that diverticulas localised there are only few or even

single. Foreign bodies or impacted hard stools may also cause stercoral perforation in the distal bowel.

Colonic diverticular perforation can be treated even by laparoscopic⁴² measures unless there is gross fecal contamination and iv fluids and higher antibiotics.

Cecostomy as a treatment for colonic perforation decreases the mortality of the second operation.

J.C.Goligher wrote more than thirty years ago that "treatment of the carcinoma of the colon complicated by perforation and peritonitis make very melancholic reading", documenting this opinion by 90% mortality by patients with stercoral peritonitis from perforation of stercoral ulcer and 70 % mortality after perforation of growth"

Chemical peritonitis/contamination :

The perforation of the bowel initially leads to chemical peritonitis. There may be contamination of the micro organisms. The presence of acid from the stomach or duodenum sterilizes gastroduodenal contents; the contamination is there if there is reduction of acid contents either due to antacids or antiulcer medications or due to gastric tumour. Spillage of gastroduodenal contents is usually diffuse but may be

localized in the upper abdomen by adhesions or the omentum. Spillage along the right paracolic gutter into the right lower quadrant

Intermediate stage:

After 6–12 hours, patient feels relief of pain due to the dilution of the irritating duodenal and gastric contents by the peritoneal exudate.

Intra-abdominal infection.

After 12–24 hours intra-abdominal infection supervene.. Any perforation should be operated on with a delay of more than 12 hours as infection. So therefore these patients are subjected to prophylactics some patients may present a few days after the perforation in the stage of septicemia and profound shock... Untreated perforations eventually succumbed to an early “septic” death from diffuse peritonitis or from intra-abdominal abscess.

XIV. INVESTIGATIONS :

BLOOD INVESTIGATIONS :

Complete Blood Count : HB, TC , DC, PCV, ESR (look for signs of anemia, dehydration and hemoconcentration.)

Renal Function Tests : urea, creatinine with full electrolytes ,dehydration and

Hypokalemia

Random Blood Sugar : diabetic keto acidosis may mimick severe abdominal pain.

Serum Amylase : may be elevated.{ also in pancreatitis }

Arterial blood gases (ABGs) and serum lactate : Suspect mesenteric ischemia

Blood grouping and cross matching :

Electrocardiogram (ECG) : To rule out chest pain and for anaesthetic purposes.

Imaging studies : An erect chest x-ray (CXR) will show free air under the diaphragm in 70-80% of perforations A lateral decubitus film may show free air if the erect CXR is normal. It should be differentiated from chilatadis sign .

Computed tomography (CT) with water soluble thin contrast is indicated if there is no pneumoperitoneum on plain radiology. CT may also help to localise the site of the perforation if not confirmed clinically.

CT is also a valuable investigation in blunt trauma to detect the bowel perforation and mesenteric tears, it is more accurate than diagnostic peritoneal lavage in diagnosing abdominal injuries due to blunt trauma.

This helps the surgeon to decide for the upper abdominal or lower abdominal incision. With the advent of CT, contrast studies are rarely required.

With ultrasound, free intraperitoneal fluid may be the most important indication of the perforation the free air in the chest x ray could not be detected in pyloric or duodenal perforation in 8 % perforations . There is also evidence that CT examination is of little or no diagnostic value until at least 6 hours from the onset of symptomatology in the absence of pneumoperitoneum on plain abdominal film or ultrasound study

XV. BLUNT TRAUMA :

Bowel and mesentery injuries occur in 5% of blunt trauma cases As these injuries are most commonly seen in motor vehicle accident victims, the early diagnosis is important. In patients with duodenal perforation,surgery performed within 24 hr of injury has a 5% mortality rate, whereas delayed diagnosis and treatment leads to a mortality rate of 65% clinical signs may be asymptomatic or subtle.

Traumatic duodenal perforation has an incidence of 1%-17% ⁴⁴ of the patients with blunt injury abdomen. Its prognosis correlates to the nature of injury, associated injuries, size of perforation and delayed diagnosis

CT findings of bowel and mesenteric injury include

1. Free air under the diaphragm
2. extravasation of oral contrast material,

3. peritoneal fluid or
4. retroperitoneal fluid,
5. thickened bowel wall, ⁴⁵
6. high-density clot⁴⁵

(sentinel clot) adjacent to the involved bowel, and focal mesentenc infiltration

Free air in either the peritoneal cavity or the retroperitoneum from injury to the retroperitoneal portion of the duodenum,

. The most common location to detect free intraperitoneal air is in the subdiaphragmatic area .. The common sites of blunt injury abdomen are jejunum (proximal portion), near the ligament of trietz, and in the ileum near ileocaecal junction

The main reasons contributed are

- as the fixed and mobile parts of the bowel are in continuity.
- Susceptible to mechanical shearing force.

Delayed diagnosis of bowel and mesenteric Injuries results in increased morbidity and mortality, usually because of hemorrhage and peritonitis that leads to sepsis and mortality.

Sudden deceleration against a relatively fixed restraining seat belt can result in mesenteric tears, avulsions and perforations occur most commonly in the small bowel. The other structures injured in this manner include

- a) The Retroperitoneal Duodenum,
- b) Pancreas,
- c) Kidney,
- d) Hepatic And
- e) Renal Veins; And Intrapertoneal Viscera Such As
- f) Liver,
- g) Spleen,
- h) Gravid Uterus And
- i) Greater Omentum..

Avulsions and tears occur at points of fixation of otherwise mobile viscera and mesenteries;.

The redundant portion of sigmoid colon, subject to injury by avulsion and explosive mechanism due to proximity to lumbar vertebral column

XVI. RETROPERITONEAL PERFORATION :

The majority (90-95 per cent.) of duodenal ulcers are situated in the duodenum first part, pars horizontalis superior, and most near the pylorus. If such an ulcer

perforates will result is peritonitis. A small number of duodenal ulcers are situated further down in the duodenum, in its pars verticalis or (even more rarely) in its pars horizontalis inferior. If an ulcer in these parts occurs on the back wall, where there is no peritoneal coverage., and perforates, the perforation leads to inflammation in the retroperitoneal tissue. The complication of retroperitoneal perforation is abscess or phlegmon formation, which is sometimes confirmed by biopsy.

The duodenal ulcers on perforation, give rise to retroperitoneal suppurations are most frequently on posterior wall of the pars verticalis duodeni Acute retroperitoneal perforation leads to an inflammatory process in the retroperitoneal tissue which in different cases may behave differently. '

XVII. SIZE OF THE PERFORATION :

A giant duodenal ulcer is defined as an ulcer more than 2 cm in diameter, usually found in the posterior aspect of the duodenal bulb, penetrating into the Pancreas, where it is associated with a significant risk of bleeding from the underlying gastroduodenal artery. Morbidity and mortality rates are higher with giant duodenal ulcers than with smaller ulcers. Such patients are treated by definitive procedures like Vagotomy with distal gastrectomy⁴⁷ and

Gastrojejunostomy with or without tube Duodenostomy provided the patient is fit and the available of experienced surgeons or gastroenterologists.

. If the General condition of the patient is not fit and an experienced Surgeon is not available, Triple ostomy comprising of

- 1) Feeding Jejunostomy,
- 2) Controlled Tube Dudenostomy ⁴⁸ And
- 3) Gastrostomy can be done

Omental plugging was a safe method of treatment for large sized any peptic perforation. ⁴⁹

XVIII. Complication Of The Perforation :

Peritonitis remains a potentially fatal threatening condition. Peritonitis refers to an inflammatory response of the peritoneum in the abdominal cavity in terms of activation of local mediator cascades by different stimuli. Bacterial, viral and chemical agents may cause inflammation of the peritoneal layer, leading on to peritonitis

Peritonitis can be classified in to types based on the cause of the inflammatory process:

- Primary,

- Secondary And
- Tertiary Peritonitis.

Primary peritonitis is defined as a diffuse bacterial infection of the peritoneal cavity occurring without any loss of integrity of the alimentary tract. It usually responds to medical treatment and does not require surgical intervention.

The most common form, **Secondary peritonitis** is usually due to spillage of gastrointestinal or genitourinary microorganisms into the peritoneal cavity as a result of loss of integrity of the mucosal barrier., and is the consequence of a local infectious process within the abdominal cavity, patient with hollow viscus perforation can lead to diffuse peritonitis. It requires timely surgical treatment with appropriate antimicrobial therapy⁵⁰

Tertiary peritonitis is defined as persistent or recurrent peritonitis after initial adequate treatment for secondary peritonitis and treated well.

XIX. CONSERVATIVE MANAGEMENT OF PERFORATION :

Not all patients with perforated PUD require intervention ,some patients will seal off the perforation with omentum almost immediately and so be suitable for a conservative approach .But the standard of care remains emergency laparotomy after adequate resuscitation and improvement of

hemodynamic status with appropriate repair using omentum as the primary closure of the perforation.

Laparoscopy and washout alone may be sufficient for sealed cases of PUD. laparoscopy and simple wash is also sufficient if there is no gross contamination and there is no much peritoneal exudate. When the surgeon is not satisfied about the lavage, convert the laparoscopy in to laparotomy for copious wash with normal saline

In cases of colonic perforation, resection of the affected bowel is the appropriate management. As primary anastomosis is not advised and too contraindicated in the presence of gross contamination; there needs to be a diversion procedure as stoma, the major example is Hartmann's procedure

When there is no gross contamination or iatrogenic injury during colonoscopy or when there is adequate preparation of the bowel. Primary closure of the colonic perforation can be done..

Appropriate attention to the haemodynamic state of the patient is required.

Antibiotics should be continued for a therapeutic course. Eradication of H Pylori is recommended in those with duodenal ulcers.

Laparoscopic repair of the gastric outlet obstruction with perforation is difficult and so the management of giant ulcer is difficult to manage by laparoscopic measures.

Post operative period is very crucial. Early mobilization of the patient and adequate antibiotics is necessary for the survival of the patient and to avoid major post operative complications,.

A high level of surveillance for infectious complications (e.g. abscess) is also necessary.

The nonoperative treatment of perforated peptic ulcer was used sporadically for half an-century,. This was due to progress in the development of proton pump inhibitors and recognition of H. pylori as a causative factor for duodenal ulcer. donovan also explains that half the perforations are sealed at opening the abdomen and these are to be gently released to remove the adhesions and for firm closure of the perforation.

Patients who are stable and there is no clinical evidence of contamination, patient has to be advised to do gastroduodenogram for further management.

Impression :

1. If there is no leak or limited to a small area adjacent to the duodenum as confirmed by the contrast duodenogram with water soluble material, the

patient can be treated with nasogastric suction, intravenous fluids, antibiotics, and bed rest.

2. If the perforation allows contrast to disseminate in the subhepatic or paraduodenal space, operation to close the defect with or without a definitive ulcer operation is immediately undertaken. The patient should be evaluated for *H. pylori*, and, if positive, triple or quadruple antibiotic and drug therapy is the treatment of choice.
3. In patients not operated on for the perforation, elective definitive surgical management should be considered.

SURGICAL MANAGEMENT :

1. Definite procedure : Large perforations (>2.0 cm): vagotomy, antrectomy, Billroth II reconstruction
2. Synchronous bleeding and perforation: generally require vagotomy, pyloroplasty, U-stitch control of posterior bleeding
3. Chronic ulcer symptoms, *H. pylori* negative: patch closure, parietal cell vagotomy, *or* vagotomy, pyloroplasty with ulcer excision
4. NSAID dependence: patch closure, parietal cell vagotomy, *or* vagotomy, pyloroplasty, ulcer excision
5. Previous *H. pylori* treatment failure or known *H. pylori* negative patients:

patch closure, parietal cell vagotomy, *or* vagotomy, pyloroplasty, ulcer excision

6. Previous ulcer complications: patch closure, parietal cell vagotomy, *or* vagotomy, pyloroplasty, ulcer excision
7. Perforated gastric ulcer (more than 1-2 cm proximal to pyloric vein): antrectomy, with or without vagotomy, Billroth I reconstruction
8. Previous operation for duodenal ulcer: if previous vagotomy, requires 60% “70% gastric resection, Billroth II anastomosis; if previous adequate gastrectomy, requires truncal vagotomy, possible resection (P.O., investigate whether Z-E tumor)
9. Young patients (under 40 years): patch closure, parietal cell vagotomy, *or* vagotomy, excision of ulcer, pyloroplasty

Contraindications for definite procedure :

There are contraindications to definitive ulcer surgery at the time of closure of perforation Serious concurrent medical illness myocardial infarction, history of congestive heart failure, uncontrolled diabetes,, pulmonary disease with abnormal blood gas analysis, and marginal or patients in chronic or acute renal failure,the simple procedure of direct closure with omental patch is sufficient.. it is also the

procedure if the patient is hemodynamically unstable or poor cardio pulmonary status.

XX. EMERGENCY SETUP :

Perforated duodenal ulcer patch closure has been called the Graham closure by Roscoe Graham in 1938. The open approach of Graham's closure has been proven to be successful in majority of patients. The perforation or ulcer is identified either during diagnostic laparoscopy or laparotomy. In the open approach, omental patch is brought in to the perforated edges and three or four sutures are taken and the suture material is preferably either absorbable or non absorbable suture material.

A small, half-circle needle with the swaged-on suture is placed through the edge of the defect, approximately 0.5 to 1.0 cm from the edge of the perforation.

One wall is sewn first, the tip of the needle being brought out through the edges of perforation, and, and the needle holder is applied for passage of the needle through the opposite edge of the perforation. The utmost care should be taken to prevent the complication of passing the needle through the posterior mucosa the needle should be passed parallel to the anterior wall of the duodenum, and it is very unlikely to involve the posterior duodenal mucosa in the sewn edges.

XXI. ELECTIVE / DEFINITE PROCEDURE :

The size of the perforation may be from 2-3 mm to 2-3cm . When the abdomen is opened, the perforation may have sealed spontaneously, covered sometimes by adjacent omentum or with the leakage of food particle or bile leak from the perforation.

Definitive operation can be carried out successfully at the time of the great majority of operations for giant perforation. Appropriate procedures include

- gastric resection (with and without truncal vagotomy),
- gastrojejunostomy or pyloroplasty
- bilateral truncal vagotomy, and

if there is no ideal condition for major procedure, or the patient general condition is poor with alteration in the hemodynamic status, the operating surgeon must choose a simple closure of the perforation as a life saving method(damage control surgery)

if necessary a definitive procedure can be performed thereafter improving the condition. Follow up of the patients after simple plication show that nearly one third of all patients remain free of symptoms, and about half of those with recurrent symptoms require a definitive operation for ulcer disease⁵¹

Laparoscopic closure of perforated ulcer is practiced reasonably common all over the world.

Advantages of laparoscopic repair ⁵²:

- ✓ Alleviation of pain
- ✓ Decreased wound morbidity.

. This approach is as safe and effective as open repair. Laparoscopic Graham Steele patch repair of perforated duodenal or justapyloric ulcer is beneficial for patients if there is no associated risk factors. But the operative time is prolonged and there is higher incidence of conversion to open surgery.

XXII. PREDICTORS OF MORTALITY :⁵³

- ✓ Major Medical Illness,
- ✓ Preoperative Shock, And
- ✓ Longstanding Perforation (More Than 24 Hours)
- ✓ Old Age
- ✓ Coexisting Cardiac And Pulmonary Diseases
- ✓ Time lapse between the entry and time taken for surgery.

OBSERVATION AND RESULTS :

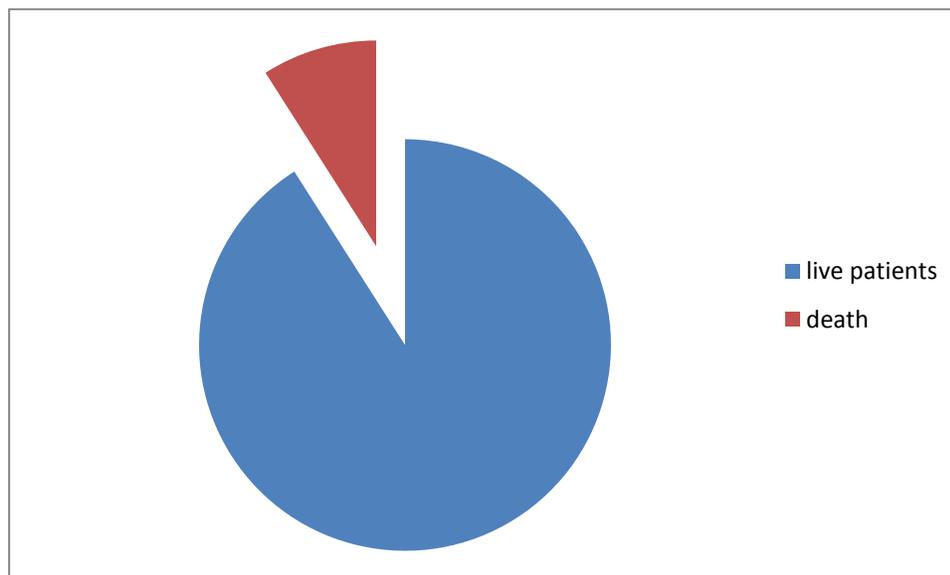
Among the hollow viscus perforation of all the 166 patients admitted in the period of OCT 2010-12 are statically analysed. All the risk factors, etiological factors and their adverse social habits are taken from the patients history and from the medical registry , and the operative findings regarding the site, size , induration and the associated morbidity and post operative complications and the mortality of the patients are charted out in proforma and all the results are summed up to give the statistical analysis of perforation of various sites of gastrointestinal tract.

TABLES AND ANALYSIS :

TABLE 1 : MORTALITY RATE :

❖ Total number of patients	160	100 %
❖ No of Deaths in study	15	9.4%

MORTALITY RATE : 9.4 %

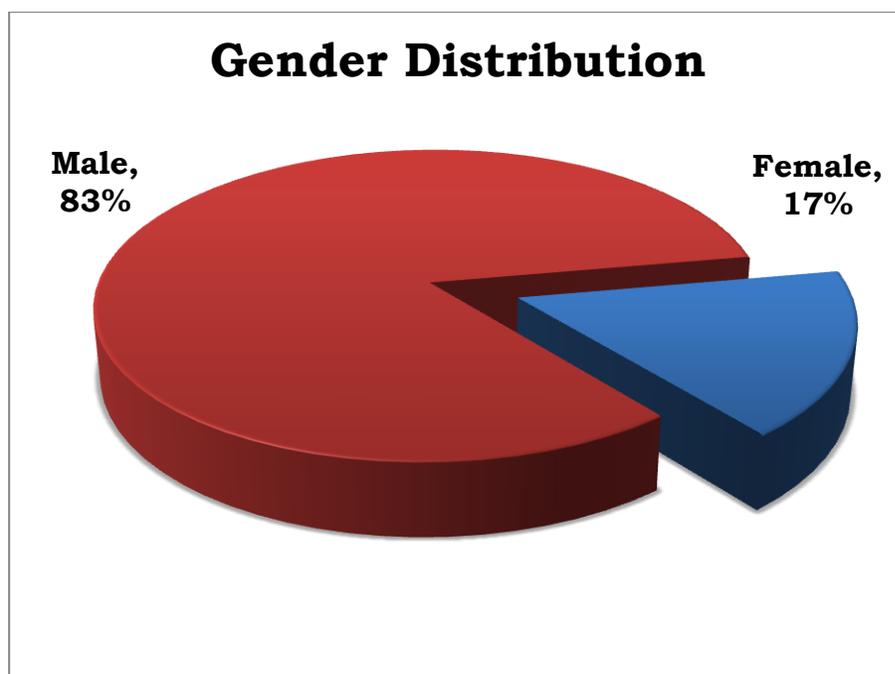


AMONG the 166 patients taken up for study with the evidence of perforation suspected clinically and confirmed intraoperatively, 15 patients expired due to complications of perforation and delayed admission in hospital.the mortality rate in the entire study is 9.4 % (10 % approx.)

TABLE 2 : SEX INCIDENCE :

Sex	No. of Patients	Percentage
Male	133	83.12 %
Female	27	16.88%
Total	160	100%

MALE : FEMALE :: 4.9 :1



Of the total number of perforations taken up for study,, males outnumber women in the ratio of 4.9 : 1..only 16% of the total number of patients with perforation were females.In my study, comparing male with female sex, there is an significant increase in the incidence of perforation among males compared to females.

TABLE 3 : AGE INCIDENCE :

SL NO	AGE OF THE PATIENTS	NUMBER OF PATIENTS	PERCENT
1	20-30	35	21.8%
2	31-40	31	19.3%
3	41-50	30	18.7%
4	51-60	30	18.7%
5	61-69	34	21.2%

THE MEAN AGE = 43.5 YEARS

Among the patients taken up for study in the age of 20- 70 years { exclusion criteria : < less than 20 years and the patients above 70years of age },

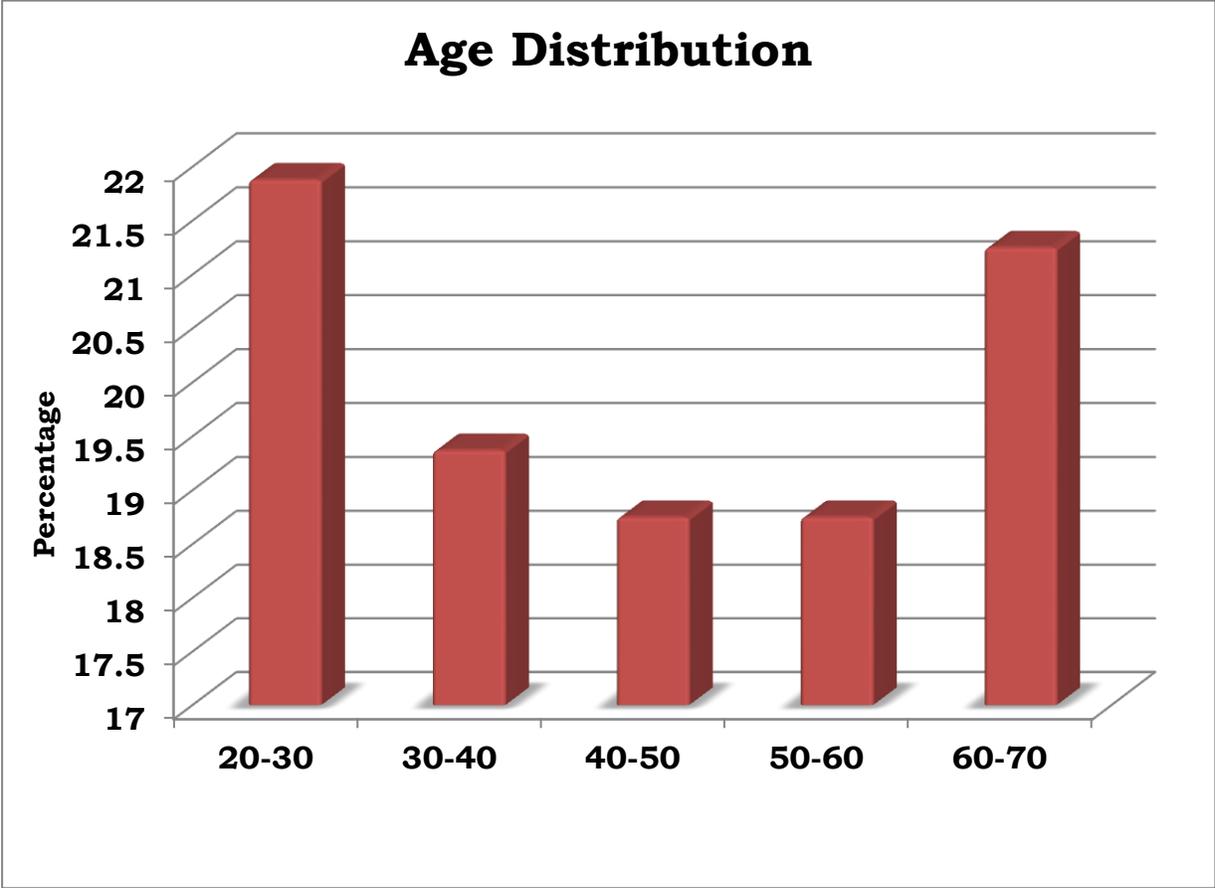
perforations are relatively more common in the age group of 20-30 years , as more number of younger generations suffer from peptic ulcer either due to H.pylori or adverse social habits and accidental traumas. 22 % of the total number of perforation patients are in younger age group comparing to middle and old age people .

The second most common age group of perforations in my study are in the age group of 60- 70 years , due to relative increase in the tumour related causes and

peptic ulcer, these patients are to be followed up for further management , after correction of primary pathology.

.The least common group in my study attributes to middle age and above 50 years population . here the incidence and perforations are mainly due to peptic ulcer and infections.

The mean age of perforation in my study population of 160 patients is **43.5 years**.



Etiological factors among the perforation :

The study of Etiological factors in my study, as expected goes in favour of acid peptic disease, which contributes about 60 % of total number of study people with perforations.

The current literature also suggest as peptic duodenal ulcer disease as the most common cause of perforation world wide due to strong evidence of ulcerogenic factors and contributory factors like smoking , alcohol and intake of ulcerogenic drugs.

The blunt (exclusion: penetrating or stab) trauma to the abdomen either in form of road traffic accidents, fall injury and fall of heavy object over the abdomen contribute to third most common cause of perforation.

The tuberculosis and typhoid fever as the cause of intestinal perforation contributes very little (< 10 %) to the study population.

TABLE 4 : ETIOLOGICAL FACTORS AMONG THE PERFORATION

PATIENTS :

ETIOLOGICAL FACTORS	NO. OF PATIENTS	PERCENTAGE
APD	99	61.88 %
TUMOUR	15	9.38 %
INFECTIONS	9	5.62 %
BLUNT	16	10.00 %
RARE CASES	21	13.13%
	160	100 %

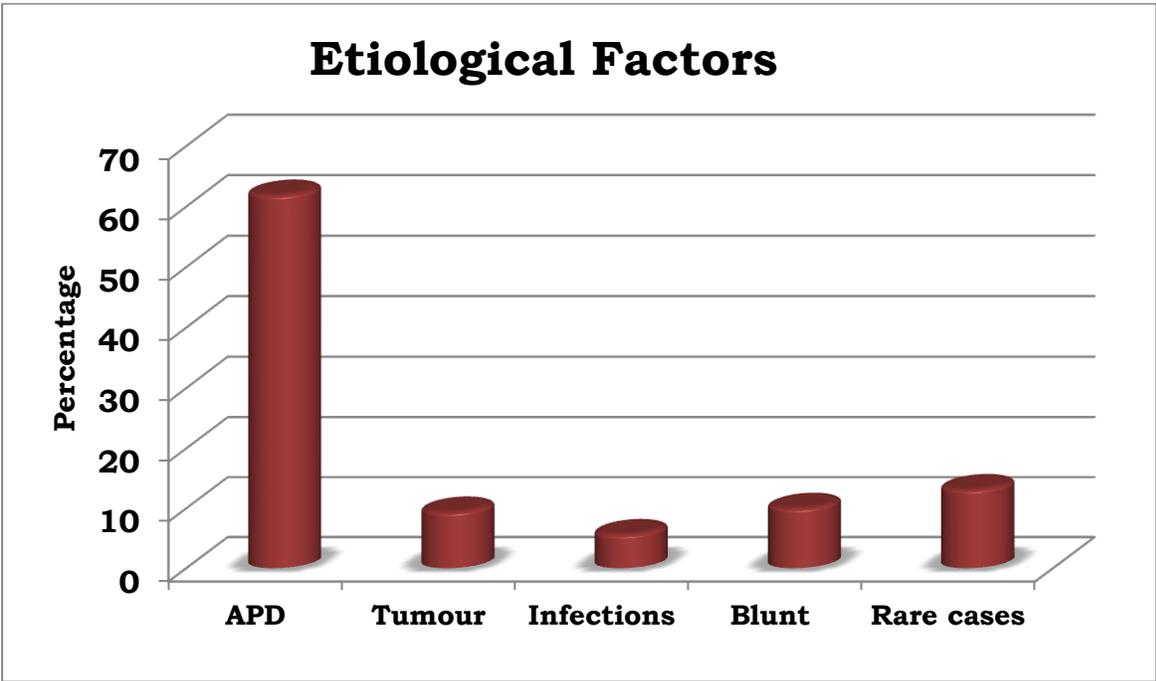
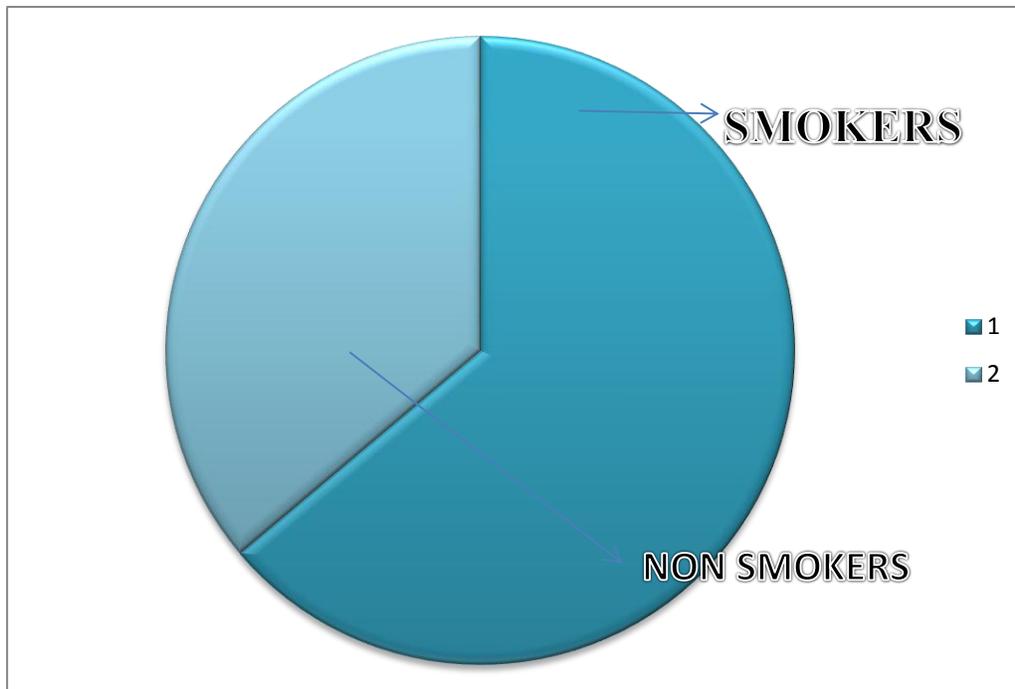


TABLE : 5 : ACID PEPTIC DISEASE IN SMOKERS VS NON SMOKERS

AMONG MALE PATIENTS

Risk factors	No. of male patients	Percent
Smokers	53	63.85 %
Non Smokers	30	36.14%
Total	83	100

P VALUE : 0.163

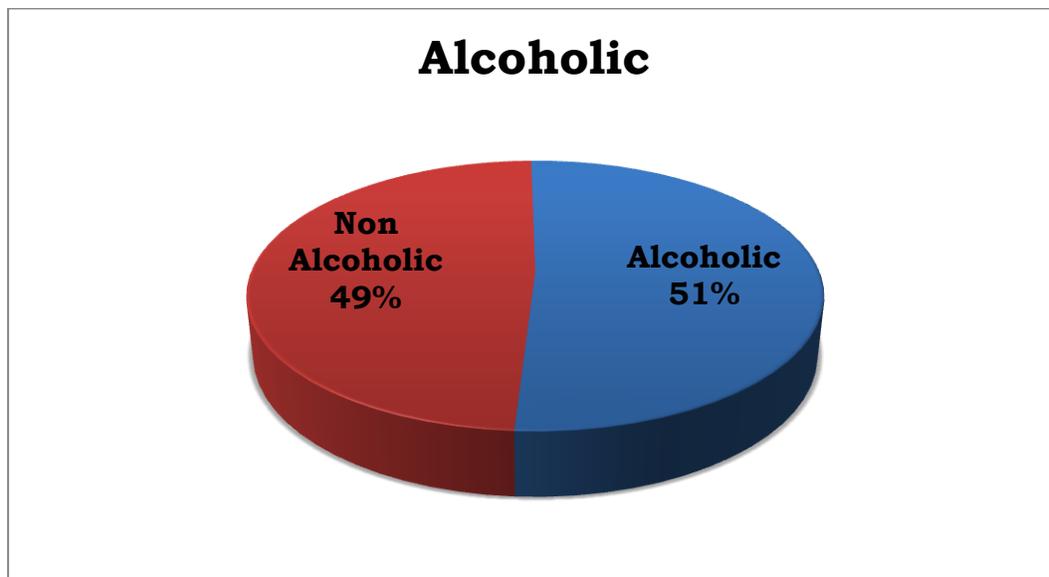


Among male patients in the study, smokers have an increased risk of acid peptic disease and subsequent duodenal ulcer and perforation. In this study, all 27

females were excluded and 133 male patients are studied to derive a causal relationship of smokers against acid peptic disease.

TABLE 6 : ALCOHOL – AN ADVERSE FACTOR :

Adverse social habits	No. Of patients	Percent
Alcoholic	82	51.25
Non Alcoholic	78	48.75
Total	160	100

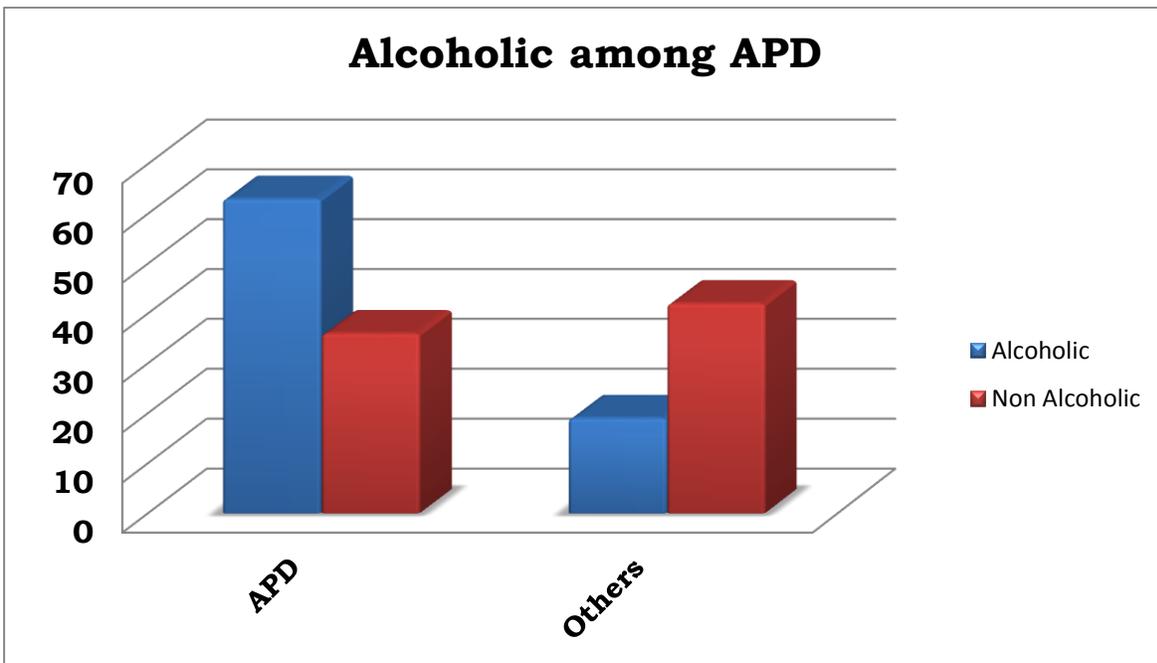


It is evident from the table that majority of patients with perforations in the study (51 %) were alcoholics as compared to 49 % of non alcoholics. But there is no significant difference between two group of populations.

TABLE 7 : ALCOHOL - AN ETIOLOGY FOR ACID PEPTIC DISEASE

	Alcoholic	Non Alcoholic	Total	P value
APD	64	35	99	<0.05
Others	19	42	61	
Total	82	78	160	

There is a significant association between alcohol consumption and APD which is statistical Significant with p value <0.05.



In this table, there seems to be an increased risk of acid peptic disease among alcoholics than non alcoholics. There is a definite increase in risk of acid peptic

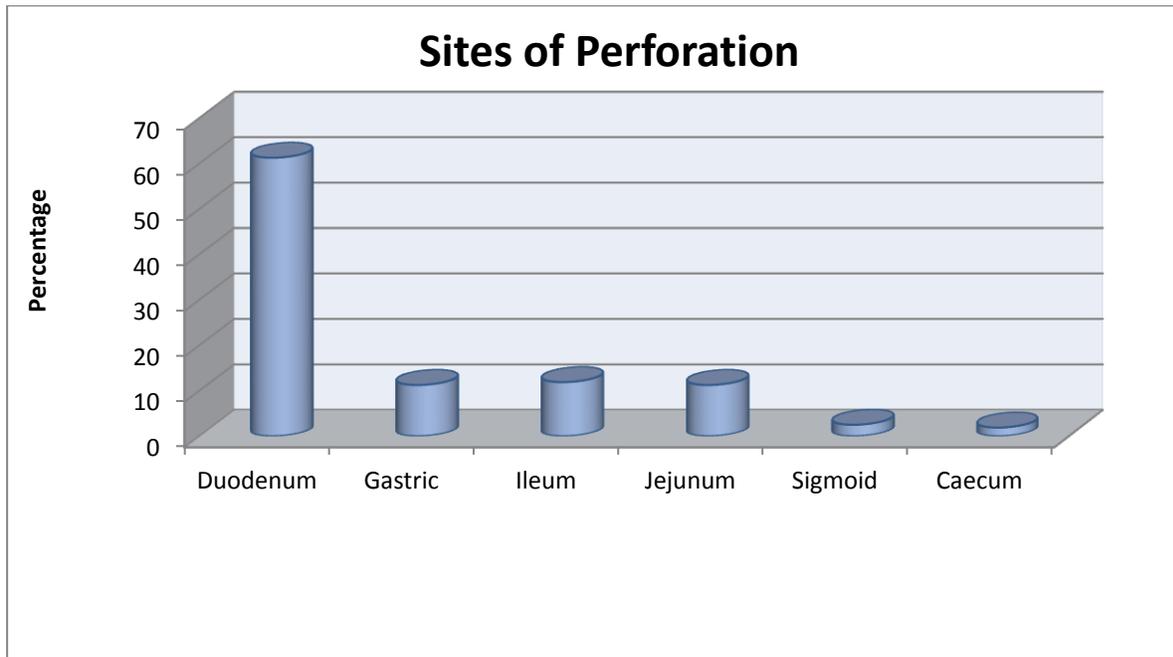
disease due to alcohol, which is also statistically significant, evident from the P value.

TABLE 8 : THE INCIDENCE IN VARIOUS SITES:

SITE OF PERFORATION	NO. OF PATIENTS	PERCENT
DUODENUM	98	61.25
GASTRIC	18	11.25
ILEUM	19	11.88
JEJUNUM	18	11.25
SIGMOID	4	2.5
CAECUM AND TRANSVERSE	3	1.88
TOTAL	160	100

Among the study population of 160 among the cases with perforations, duodenal ulcer contributes to majority of the perforations and so the second part of duodenum is the most common site of perforation contributing about 62 % of total perforations.

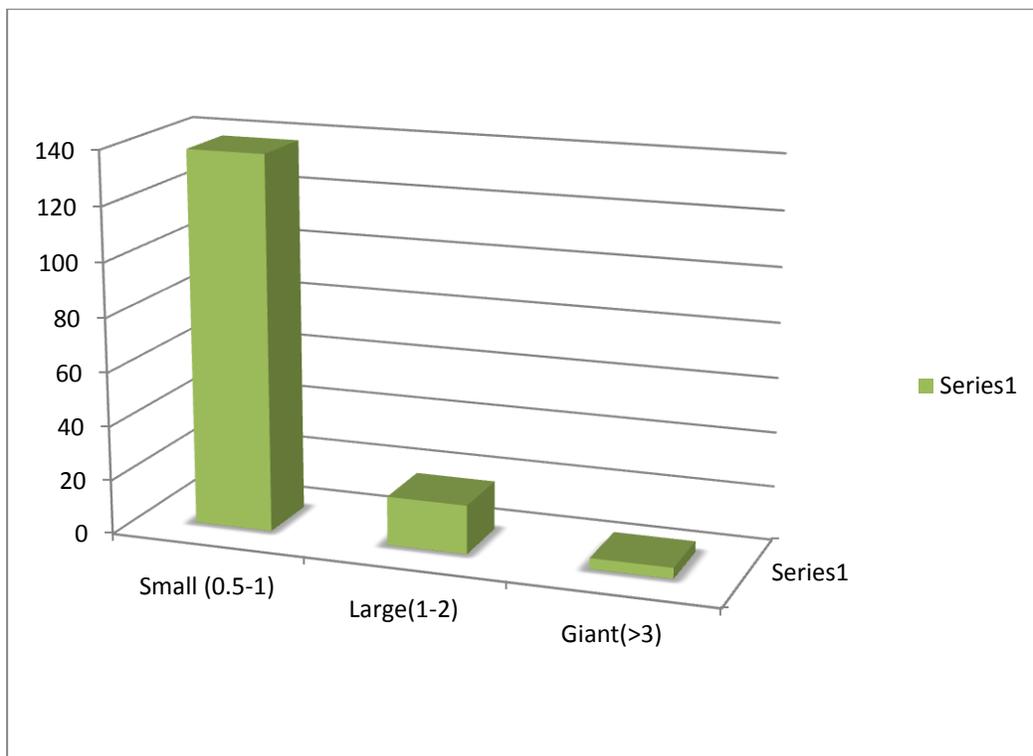
Ileum which holds the second most common site to duodenum contributes around 12 % of the total perforations, the common causes attributed to be are the infections (typhoid and tuberculosis) and tumour causes(benign or malignant)



The jejunum attributed to be the third most common cause due to blunt trauma (exclusion : penetrating and stab) as it is freely mobile and an intraperitoneal organ.

TABLE : 9 : VARIOUS SIZE INCIDENCE :

SIZE OF PERFORATION	NO. OF PATIENTS	PERCENTAGE
SMALL (0.5-1)	138	86.25%
LARGE(1-2)	18	11.25%
GIANT(>3)	4	2.5%
TOTAL	160	100%



Among the various sites of the perforation enlisted, the size of the perforation is also taken as the variable of prognostic factor and treatment modality.

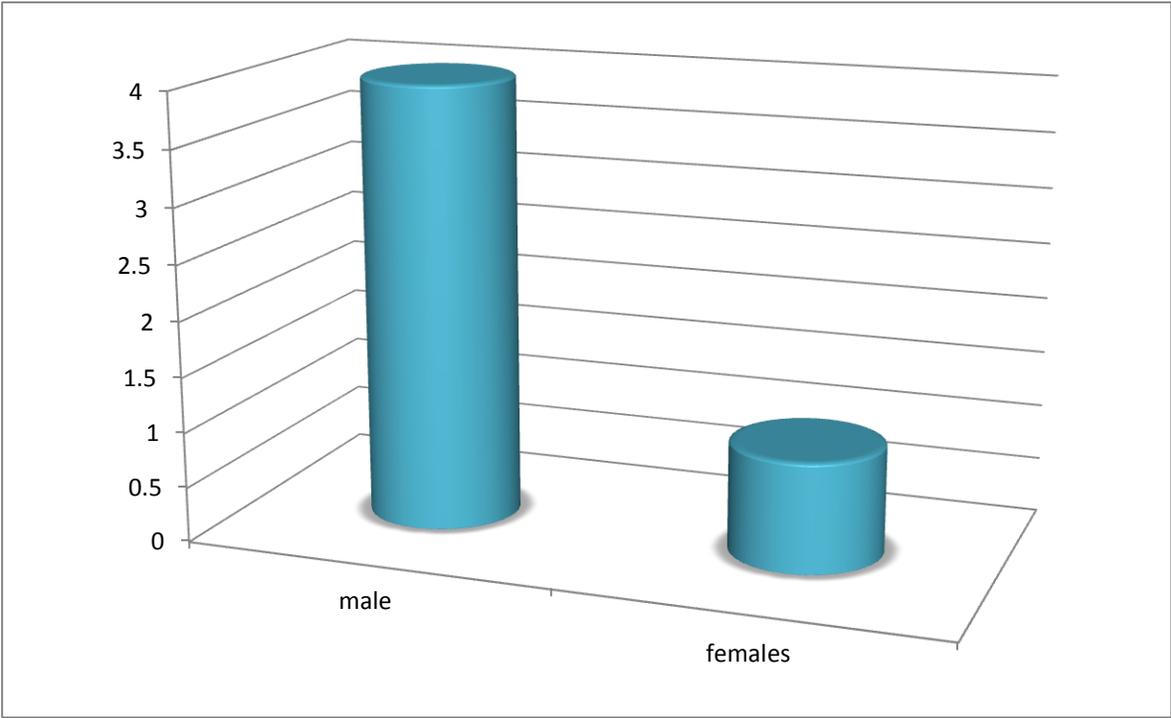
Approximately 86 % of the patients have small sized and 11 % of large size perforations were treated by primary closure. In patients of giant perforation of above 3 cms, either due to trauma or multiple perforations or stercoral perforations, the procedure resection anastomosis or primary closure of the bowel can be done depending on the nature of abdominal exudates and condition of the patient.

TABLE : 10 : TYPHOID PERFORATION AMONG BOTH SEXES :

Typhoid perforations, in my study are more common in males and contribute to about 80 % of the perforation, compared to only about 20 % incidence in females

SL NO	SEX	TYPHOID PERFORATION	PERCENTAGE
1	MALES	4	80%
2	FEMALES	1	20%
	TOTAL	5	100%

TYPHOID PERFORATION AMONG BOTH SEXES

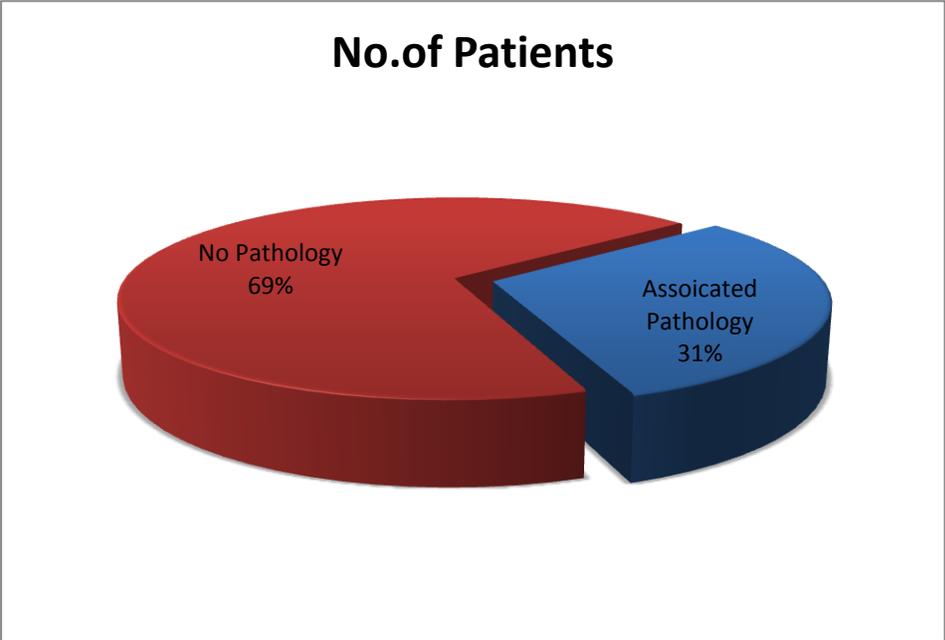


ASSOCIATED PATHOLOGY:

Table : 11 : Various Associated Pathology In Perforation Patients

SL NO	NO. OF PATIENTS	PERCENT
ASSOCIATED PATHOLOGY	49	30.62%
NO PATHOLOGY	111	69.38%
TOTAL	160	100%

In my study , nearly about 30 % of the perforation patients are associated with various pathologies. Which is also considered for the further management.



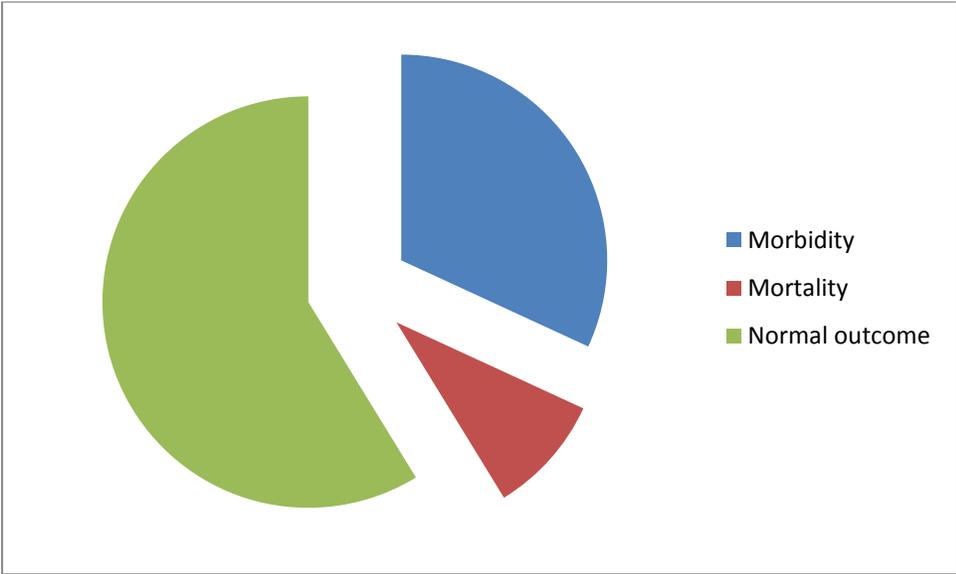
Regarding the perforation and its etiologies, the associated pathology are also considered for the management of the cause. In blunt trauma patients, where there is the associated mesenteric tear, hemoperitoneum , the associated pathologies are treated depending upon the underlying condition..

In Patients with chronic gastric ulcer with induration, the associated pathology may be a metastatic nodule in the liver or peritoneal mets, then the patient may be subjected to palliative procedure.

If the patient is with gastrointestinal tumour with perforation, the search is for the various other sites of GIST and ascites.

TABLE : 12 :THE CLINICAL OUTCOME :

Outcome	No. of Patients	Percent
Morbidity	51	31.88%
Mortality	15	9.38%
Normal outcome	94	58.75%
Total	160	100

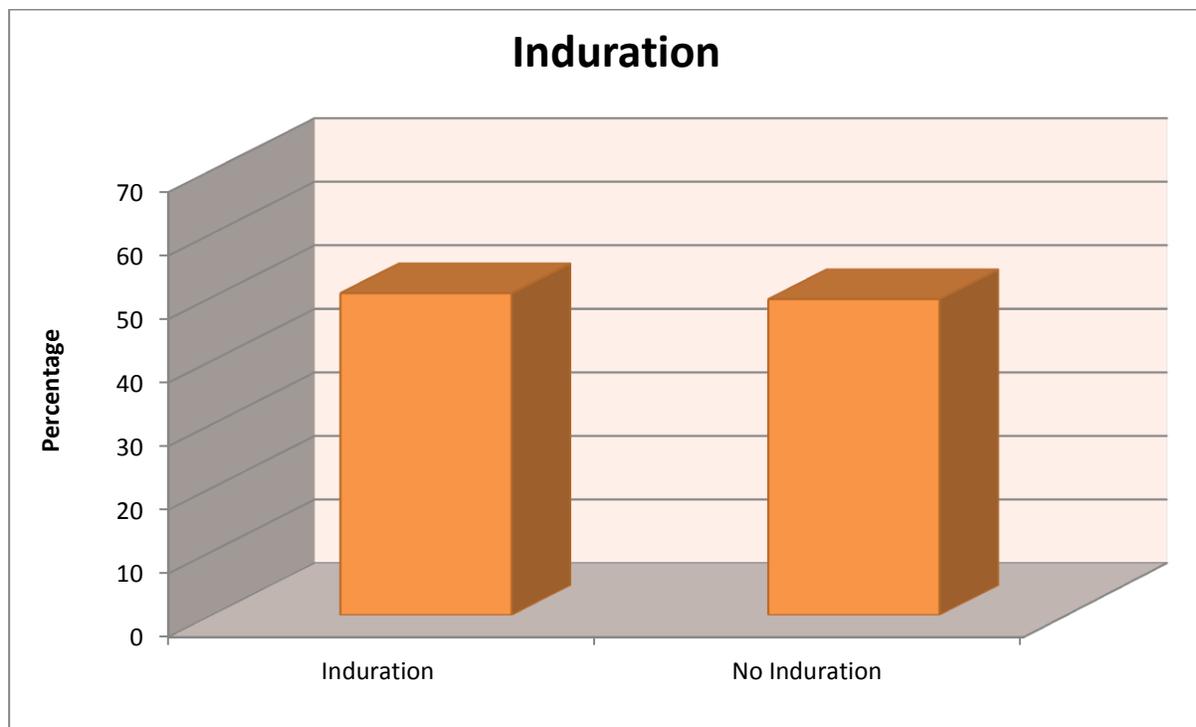


Among the study population, there is 10% mortality due to perforation and its consequences. 32 % of the population suffer from morbidity . 59 % of the study people recovered normally without any morbidity or post operative complications.

INDURATION OF THE SURROUNDING AREA :

TABLE 13 : INDURATION OF THE PERFORATED EDGES AMONG PERFORATION PATIENTS

SL NO	NO. OF PATIENTS	PERCENT
INDURATION	81	50.62%
NO INDURATION	79	49.68%



Among the total number of perforations studied , about 81 cases (51 %) of the patients presents with induration due to chronicity of duodenal ulcer and the degree of dyspepsia. Induration is considered as the pre morbid risk for perforation of a peptic ulcer. And for tumour induced cases, the rest 49 % of the people , there is no induration, these are mainly due to the acute nature of the ulcer or with the effects of blunt trauma. Induration also explains the chronicity of the disease.but there is no significant difference between the two groups of population.

TABLE 14 : GENDER DISTRIBUTION AMONG VARIOUS SITES OF PERFORATION :

Gender	Male	Female	Total
Duodenum	78	20	98
Gastric	16	2	18
Ileum	16	3	19
Jejunum	18	0	18
Sigmoid	2	2	4
Caecum	3	0	3
Total	133	27	160

P value =0.121

In comparing gender distributions among various sites of perforations, males outnumber females in all regions of perforation. The blunt trauma is more common in males as they are more exposed to the motor vehicle accidents. The infections typhoid and tuberculosis disease and the perforations are more in the male population due to consumption of unhygienic foods and migrant population.

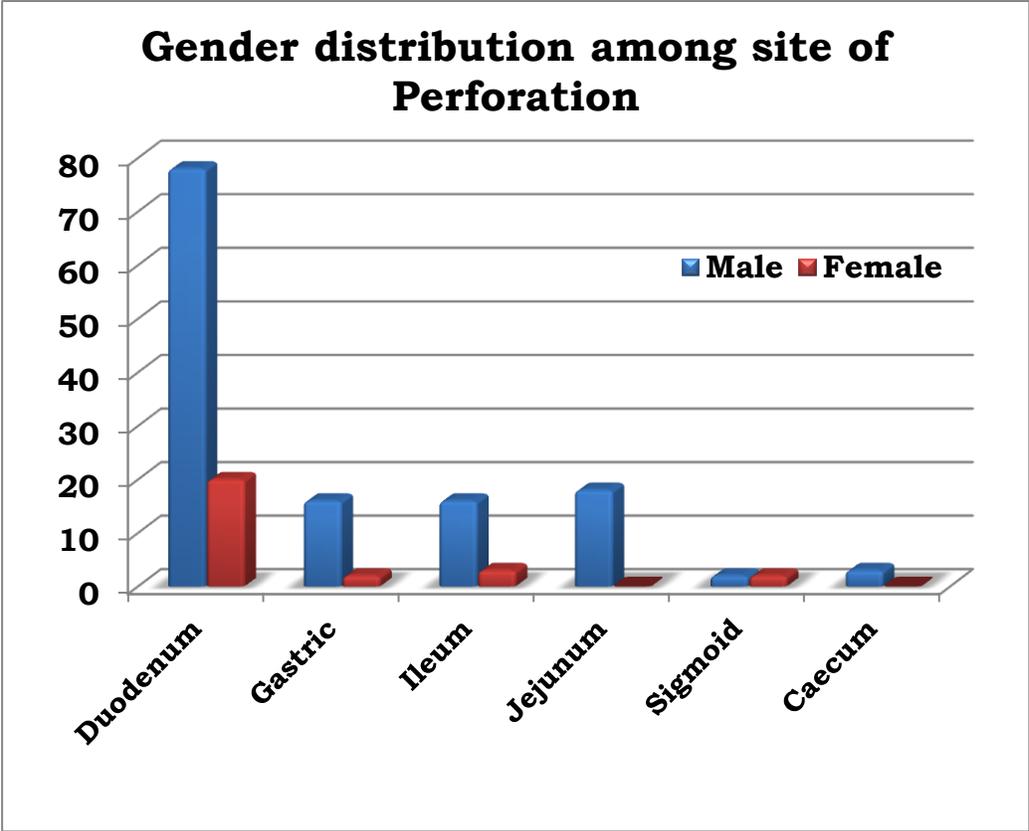
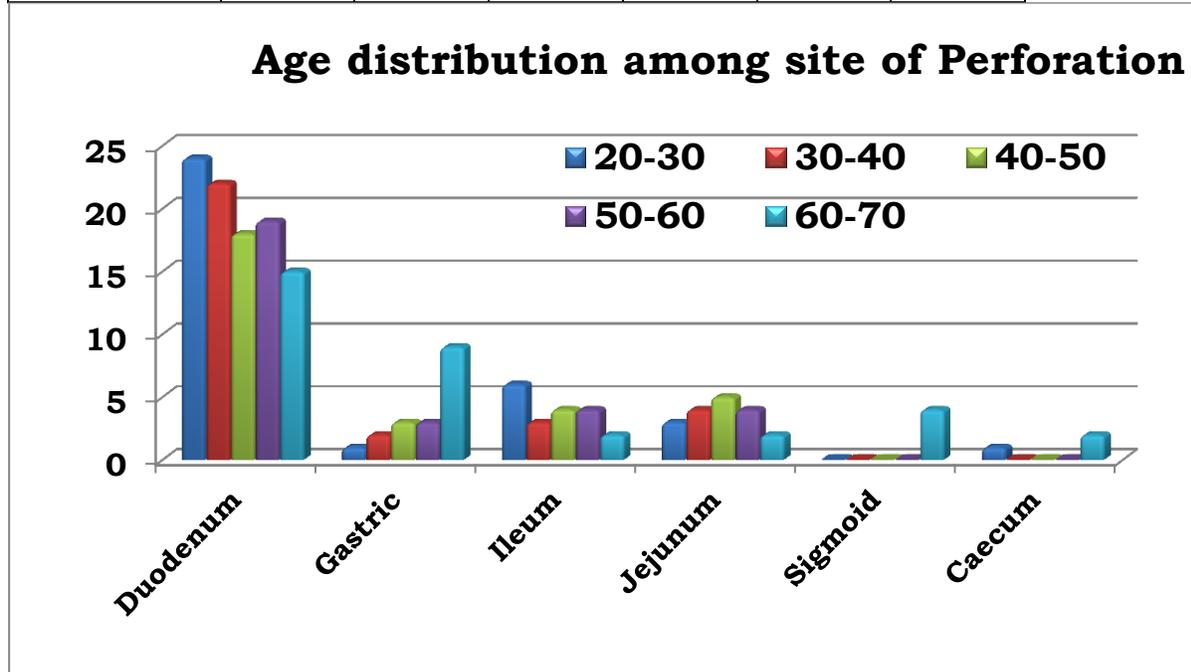


TABLE 15 : SITES IN DIFFERENT AGE GROUPS :

	20-30	30-40	40-50	50-60	60-70	Total
Duodenum	24	22	18	19	15	98
Gastric	1	2	3	3	9	18
Ileum	6	3	4	4	2	19
Jejunum	3	4	5	4	2	18
Sigmoid	0	0	0	0	4	4
Caecum	1	0	0	0	2	3
Total	35	31	30	30	34	160

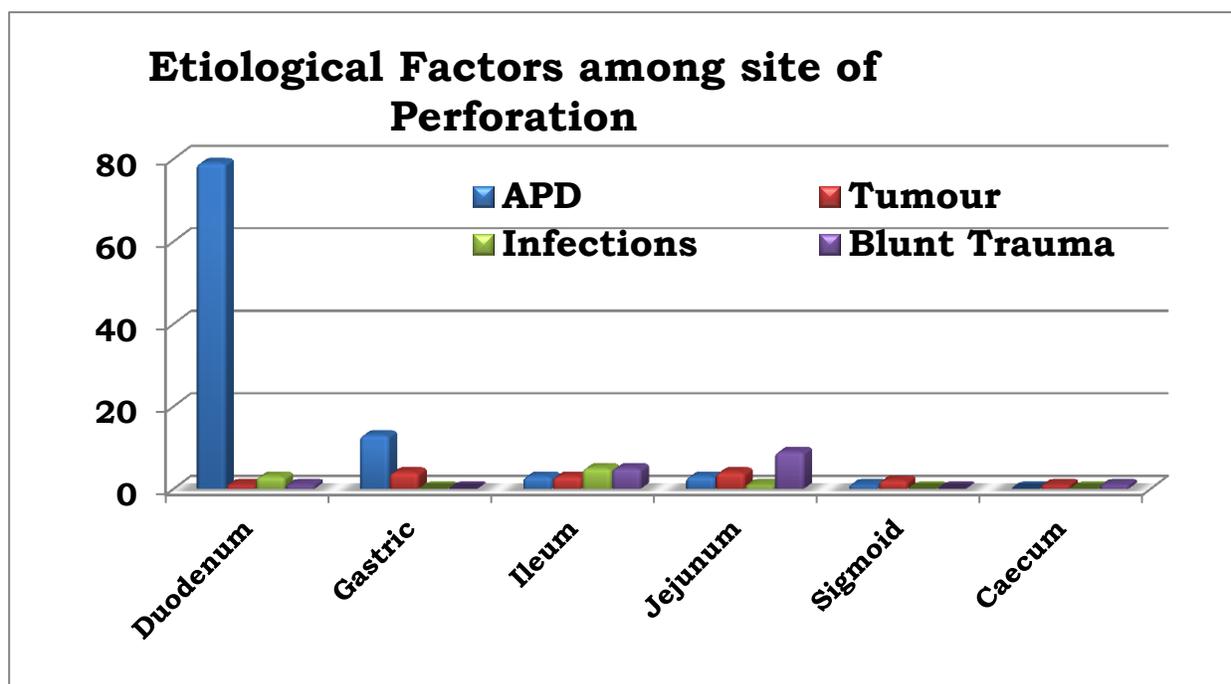


It is clearly evident from the table and chart that duodenal perforation occurs more in young males and sigmoid and caecal perforations are more in old ages.

TABLE 16 : ETIOLOGICAL FACTORS IN COMPARISON TO VARIOUS

SITES OF PERFORATION :

	Duodenu	Gastric	Ileum	Jejunum	Sigmoi	Caecu	Total
APD	79	13	3	3	0	0	99
Tumour	1	4	3	4	2	1	15
Infections	0	0	6	3(M)	0	0	9
Blunt Trauma	1	0	5	9	0	1	16

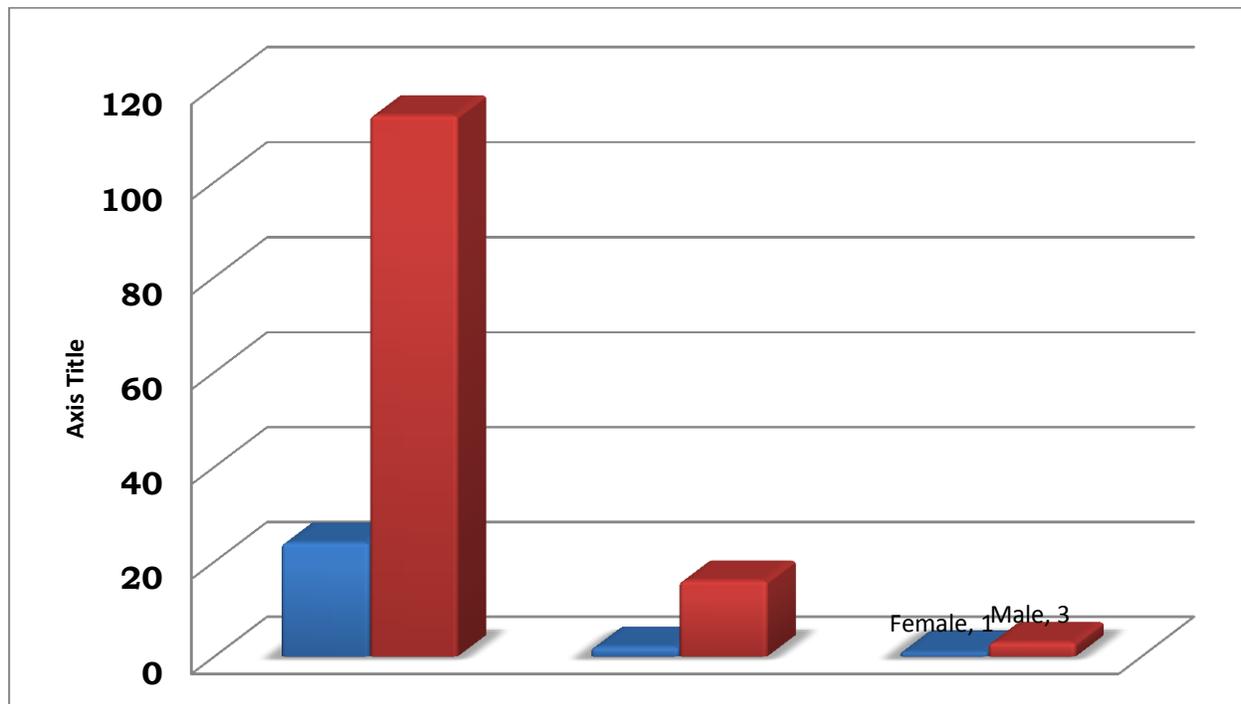


It is evident from the chart that blunt traumas are more common in small bowel especially the jejunum and infections are more confined to jejunum and ileum.

TABLE 17 : PERFORATION SIZES IN COMPARISON TO BOTH SEXES

	Small (0.5 -1)	Large (1-2)	Giant (>3)	Total
Male	114	16	3	133
Female	24	2	1	27
Total	138	18	4	160

P=0.726

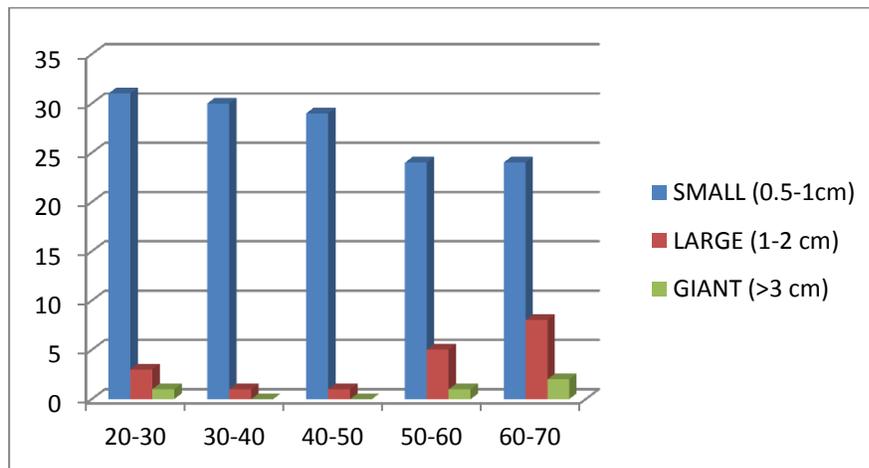


As the number of perforations are more common in male sex, the size of perforation is also proportional to the age group. But there is no statistically significant relationship between size of the perforation and the sex status.

Table 18 : Perforation Sizes Among Different Age Groups

SL NO	SMALL (0.5-1cm)	LARGE (1-2 cm)	GIANT (>3 cm)	TOTAL
20-30	31	3	1	35
30-40	30	1	0	31
40-50	29	1	0	30
50-60	24	5	1	30
60-70	24	8	2	34
Total	138	18	4	160

P=0.079

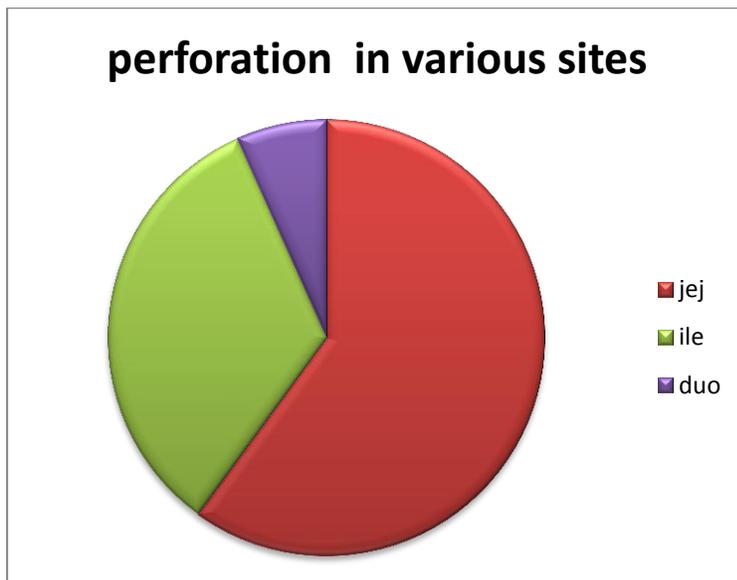


The giant perforations are limited more in old age group, as the constipation and malignancy of the bowel are common cause at old age group. The people of old

ages are more succumbed to the perforations of larger and giant size as the tumour related causes and stercoral perforation are more common in their age groups

TABLE 19 : BLUNT TRAUMA AND VARIOUS SITES OF PERFORATION

SL NO	SITE OF INJURY	PATIENTS	PERCENT
1	JEJUNUM	9	60.0%
2	ILEUM	5	33.33%
3	DUODENUM	1	7.14%



Jejunum is the most common site of perforation in bowel trauma followed by ileum and duodenum.

TABLE 20 : POSTOPERATIVE COMPLICATIONS :

SL NO	COMPLICATIONS	NUMBER OF PATIENTS	Percent
1	WOUND INFECTION	15	37.5%
2	RESPIRA COMPROM	4	10 %
3	DYSELECTROLYTEMIA	3	7.5%
4	SEPSIS	6	15%
5	BED SORES	3	7.5%
6	ABDOMINALABCESS	3	7.5%
7	FECAL FISTULA	2	5%
8	INCISIONAL HERNIA	2	5%
9	BURST ABDOMEN	2	5%

Among the patients taken up for study, postoperative complications are there in 32 % of the patients. The most common post operative complication is wound

infection which is there in 15 perforation patients , approximating to around 37 %

.The most dreaded complications are fecal fistula and abdominal abscesses

which was there in around 10 % of the patients.

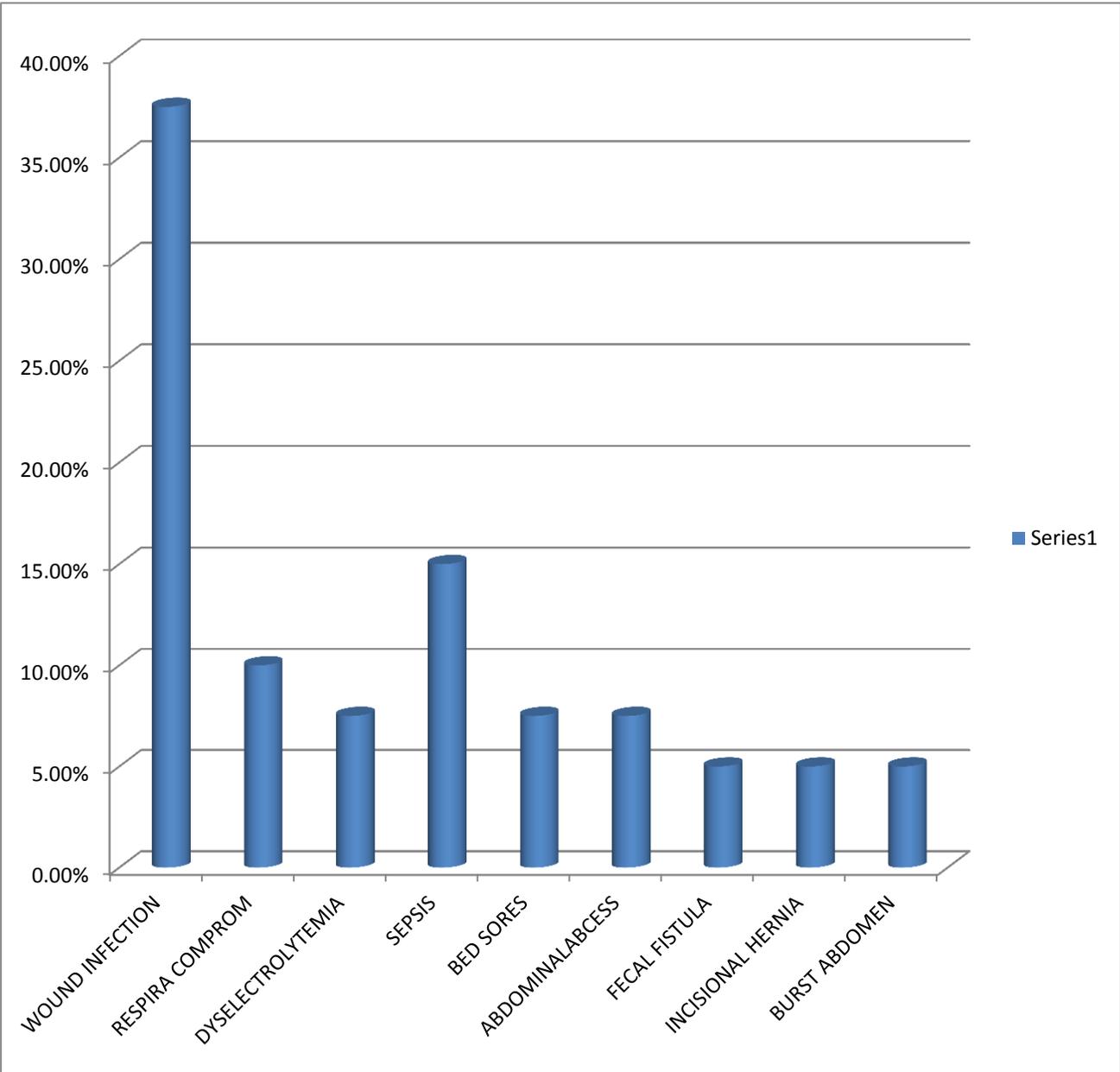


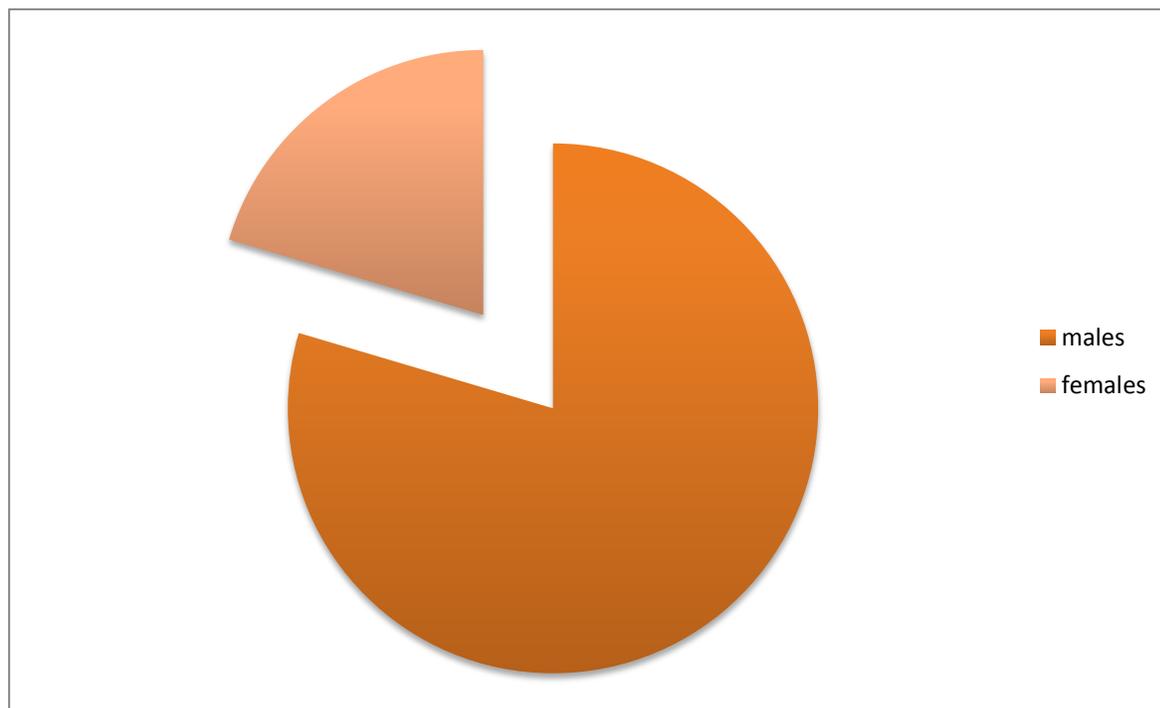
TABLE 21 : GENDER DISTRIBUTION AMONG DUODENAL

PERFORATIONS :

Gender	Male	Female	Total
Duodenal perforation	78	20	98
percentage	79.5%	20.5%	100%

P value : 0.134

DUODENAL PERFORATION AMONG BOTH SEXES



It was revealed that male contributes about 80 % of duodenal perforation than females who contribute only 20 % of the perforation.

DISCUSSION :

In my study of hollow viscus perforation in Chengalpet government hospital in the period of OCT 2010-2012, the various etiological factors, adverse habits are taken into account and the various intra operative findings and complications of the patients are analysed in the post operative period. These are summed up in the charts and are compared with literature studies.

In the patients with bowel perforations regarding the survival rate of the patients, the mortality of the perforations in my study is about 9.4 %. The literature and various studies also coincides with the survival rate in our locality population, the literature reviled it to be 10 % . nearly 15 patients succumbed to death

The overall mortality in a similar study at Ghana⁵⁴ explains about there is 11 % of mortality studied among 326 patients ,which is comparable to my study.

i. SEX INCIDENCE :

In my study there is an increased ratio of perforation in male patients compared to females. Males contribute about 80 % of the duodenal perforation than females of only 20 %.

Studies too reveal there is increase in perforation among male patients .

There is a remarkable difference in more recent studies from those of the 1990s, in which as simple a factor as male gender now demonstrates a very slight preponderance, previous studies suggested that males constituted 80% of patients with perforated duodenal ulcer..⁵⁵

ii. ETIOLOGICAL FACTORS IN PERFORATION :

My study in perforation patients clearly delineates that there is increased incidence of perforation in duodenal ulcer patients which contributes around 62 % of the patients with perforation.

Other studies also explains that there is perforation incidence more among the patients of peptic ulcer especially duodenal ulcer⁵⁶.

A study in JIPMER in INDIA regarding hollow viscus perforation explains there is increase rate of perforation in duodenal followed by enteral perforations⁵⁷

iii. AGE INCIDENCE :

In my study, the lower age group and young patients were more affected by perforated gastroduodenal disease than in the quoted studies. The main reasons that affects the younger generation is due to lack of parental guidance and premature self dependence , thus predisposing the patients to adverse social factors and the risk of gastroduodenal perforation.

The perforations of about 35 patients (approx. 22.5 %) of the perforations are in the younger age group compared to middle age and old aged people.

This is comparable to a study in UNIVERSITY OF ADEN where young generations (29-40) are more frequently affected⁵⁸ than other age groups.

Smoking vs perforation :

iv. CIGARETTE SMOKING

In this study among 166 patients, the majority of cases (52 %) were smokers , but this is not significant comparable to 48% of non smokers. but when the comparison was made between acid peptic disease among male smokers and non smokers, it was found that 64 % of male smokers were associated with acid peptic disease, which is valuable and clinically significant. current smoking was strongly associated with gastroduodenal perforation. It was found that current smoking was a significant contributor and an independent risk factor for cigarette smoking.

SVANES in his study explains current smoking increased the risk for ulcer perforation 10-fold and there is a significant dose-response relationship⁵⁹.

ANDERSEN⁶⁰ in his study explains the association between smoking and the risk of peptic ulcer perforation and found that smoking more than 15 cigarette per day increased the risk of perforation more than 3-fold.⁶⁰

SILVERSTEIN in his study explains effects of the toxic constituents of cigarette smoke in relation to peptic ulcer and impairment of wound healing⁶¹

Smokers must be advised to stop smoking prior to elective surgery or when treating for wounds resulting from trauma, disease, or emergent surgery.

A study in Denmark studied smoking in relation to the perforated ulcer explains that smokers who smoke 15 cigarettes per day are more prone for getting perforation.

The risk of both gastric and duodenal ulcers progressively increased with increasing pack-years of cigarette smoking⁶²

v. ALCOHOLICS AND PERFORATION :

The studies showed that majority of cases (51 %) were alcohol drinkers as compared to 49 % of non alcoholics, but this is not significant , but when the comparison was made between alcoholics and non- alcoholics in relation to acid peptic disease,alcoholics(64) were high risk of developing acid peptic disease than non alcoholics(35)

Alcohol contributes an important risk factor and independent risk factor for duodenal perforation.The current alcohol drinkers were at least three times increased risk of perforation as compared to nonalcoholics.

Similar findings were reported by Andersen⁶⁰ who explains the association between intake of alcohol and the risk of peptic ulcer perforation,

Alcohol is known to impair wound healing through a variety of mechanisms: nutritional deficiencies leading to impaired wound healing and alcoholic disinhibition leads to increased risk behavior and more prone for duodenal perforation than non drinkers

It is evident from anderson study and british study of relationship of alcohol vs peptic ulcer⁶³

Chronic alcoholism is also associated with the presence of gastric metaplasia. both clinically and experimentally, alcohol had been shown to affect the mucosal barrier and histology and altering gastric mucosal defense Mechanisms⁶⁴ These Ulcerogenic Effects Play A Crucial Role in the study of perforations done in other parts of the world

vi. ACID PEPTIC DISEASE VS PERFORATION :

The study of etiological factors in my study, as expected goes in favour of acid peptic disease, which contributes about 63 % of total number of study people with perforations.

The current literature also suggest as peptic duodenal ulcer disease as the most common cause of perforation world wide due to strong evidence of ulcerogenic factors and contributory factors like smoking , alcohol and intake of ulcerogenic drugs.⁶⁵ Although the majority of peptic ulcer disease are controlled by proton pump inhibitors and anti ulcer medications, the emergency in still debate continues to be the perforated peptic ulcer disease

A study in arab emirates states that patients with history of dyspepsia and previous peptic ulcer disease should take prophylactic anti ulcer medications to avoid ulcer related complications

vii. GATRIC ULCER VS DUODENAL ULCER :

Gastric ulcer location, hemodynamic instability and larger ulcer size were factors associated with increased rates of mortality.

In my study, among 18 gatric ulcer with perforations, there is two mortalities comparing with 6 perforations of duodenal perforation with complications.the percentage varies between 11 % of gastric ulcer with 6% of duodenal ulcer perforations.so there is definite increase in morbidity and mortality in gastric perforations compared to duodenal perforations.

It is evident from the maingots abdominal operations, there is higher mortality rate for gastric ulcer than duodenal perforation in the range of 15- 20 %⁶⁵.

Among the gastric ulcer perforation edges sent for histopathological examination, two biopsies were positive for malignancy .(adenocarcinoma) so biopsy is must in all cases of gastric perforations.

viii. SIZE OF THE PERFORATION :

Larger ulcer size were factors associated with increased rates of mortality.the giant ulcers of more than 3 cms are more prone for obtaining post operative complications, and some succumbed to death.If the perforation size is large, and it is not amenable for primary omental patch closure, patient can be subjected for controlled tube duodenostomy as an emergency management.⁴⁸

ix. BLUNT TRAUMA VS PERFORATION :

Jejunal perforation is relatively common following focal blunt abdominal trauma. The mortality rate remains in the region of 30%. The main factors affecting mortality and morbidity are delay of more than 24 hours and multiple perforations with associated injuries.

There is an delay in presentation and diagnosis of traumatic bowel perforation following blunt trauma to abdomen. Signs of peritoneal sepsis remain the most common findings in our environment. The mortality and morbidity following

blunt trauma and bowel perforation are high because of established peritonitis. Delayed presentation or large leak of bowel content into the abdominal cavity determines the prognosis and the associated complications⁶⁶CT has an important role in identifying the hemoperitoneum, air fluid levels., mesenteric injury and bowel perforations.CT is better than diagnostic peritoneal lavage in better assessment of cases before posted for surgery⁶⁷

x. TYPHOID PERFORATION IN MALE SEX :

Typhoid infection and perforation are more common in male sex^{68a}. The exact etiological reason remains unclear why the typhoid perforations occur more common in males. My study clearly explains that there is increased incidence in male to female ratio of 4 :1 ,. Surgery is better treatment for modality than medical management. The factors which are going to alter the morbidity or mortality are multiple perforation and fecal contamination.^{68b}

xi. TUBERCULAR PERFORATION :

The incidence of the perforation due to tuberculosis is about 2.5 % which is comparable to other studies which elicit about 2 % of the total perforations. The main pathology attributed toward the gut tuberculosis is vasculitis⁶⁹ and that occurs most commonly in ileum.Among the 4 patients with tubercular

perforation, two patients died and two patients are with multiple perforations. the mortality rate among tubercular perforation in my study was around 50 %.

As the mortality is very high, patient should be subjected for resection of the involved segment and anastomosis. Endoscopic biopsy of the suspected lesions should be done and sent for TB-PCR and histopathological study to distinguish it from other differential diagnosis⁷⁰

xii. Induration in perforation study :

Among the study population. Induration is there in 52 % of the patients which is less significant compared to 48 % of the patients with no induration .Induration mainly explains the chronic nature of the disease pathology

xiii. Morbidity and complications :

In this study, nearly 32 % of the perforation people are associated with post operative complications, these complications arise due to the contamination of the peritoneal cavity due to the contents of the perforated bowel, late admission to the hospital and delay in surgical intervention. The most common post operative complication is wound infection, followed by sepsis and bed sores.

MISCELLANEOUS CAUSES :

A) we reported a rare cause of lightning induced ileal perforation⁷¹, a lightning strike over the abdominal wall and burning of abdominal wall hairs with a contact burn over the antimesenteric border of distal ileum leading on to the perforation, the presentation is very rare and primary closure of the perforation done.

B) A case of sigmoid volvulus with perforation at its apex: sigmoid volvulus is defined as the twisting of sigmoid colon⁷² along its mesenteric axis leading on to ischemia, perforation and death. We reported a cause of sigmoid volvulus with gangrenous bowel and the patient died due to septicemia.

C)segmental enteritis :we also reported a case of segmental enteritis with gangrenous segment of the small bowel and the presented with perforation. Resection of the gangrenous segment was done. But the exact etiology is not known

D) A case of meckels diverticulitis with perforation of the ileum.we also reported a case of meckels diverticulitis in 20 year old female about 70 cms proximal to ileocaecal junction, segmental resection and anastomosis was done

CONCLUSION :

There are various indices mentioned in literature to predict morbidity and mortality due to sepsis. We in our study conclude that the

- ❖ **AGE OF THE PATIENT,**
- ❖ **GENDER STATUS ,**
- ❖ **SMOKING ,**
- ❖ **ALCOHOL CONSUMPTION and**
- ❖ **PREVIOUS HISTORY OF ACID PEPTIC DISEASE.**

are independent predictors of morbidity and mortality in patients with hollow viscus perforation. The mortality and morbidity can be best avoided by monitoring the patients perioperatively and to give high quality of care by anaesthesiologists for risk assessment of the cases and to give goal directed therapy.

- The most frequent cause of Hollow viscus perforation encountered in my study was peptic ulcer perforations, which was observed in 64% of cases.
- The highest incidence of bowel perforation (22 %) was observed in the age group 21 to 30 years, followed by 60-69 years (21 %).
- Males were predominantly affected, with a male to female ratio of 5 :1.
- The mortality rate in my study was around 10 %.

- The most important factor clearly deciding the fate of the patient is eliminating the source of infection.
- The omental patch procedure was a simple and very effective method for closure of any size of perforations. Perforation of peptic ulcer was the most commonly encountered perforation (62 %), followed by small bowel perforations.
- Gastric ulcer perforations carry higher mortality risk than duodenal ulcer patients. Irrespective of the etiologies, all gastric ulcer perforations should be sent for biopsy from the perforation edges.
- Typhoid fever should be treated with appropriate antibiotics to prevent enteric perforation and if perforation occurs, there should not be any delay in surgical intervention
- Perforations due to tuberculosis are solitary or multiple and carry worst prognosis , so these patients should be subjected to resectional procedure of the involved segment.
- Colonic perforations should be treated surgically by primary closure or two staged procedures depending up on the condition of the patient and fecal contamination of the peritoneal cavity.

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ABBREVIATIONS:

APD	--	Acid Peptic Disease
PUD	--	Peptic Ulcer Disease
NSAIDS	--	Non Steroidal Anti Inflammatory Drugs
CT	--	Computerised Tomogram
MRI	--	Magnetic Resonance Imaging
USG	--	Ultrasonogram
CML	--	Chronic Myeloid Leukemia
H.Pylori	--	Helicobacter Pylori
PPI	--	Proton Pump Inhibitors
GIST	--	Gastrointestinal Stromal Tumour.
DUO	--	Duodenum
ILE	--	Ileum
JEJ	--	Jejunum
GAST	--	Gastric Region / Stomach

A STUDY ON HOLLOW VISCUS PERFORATION DURING THE PERIOD OF

OCT 2010- OCT 2012

NAME :

OCCUPATION :

IP NO :

AGE :

SEX :

SL	ETIOLOGICAL FACTORS	YES	NO
1	Acid Peptic Disease		
2	Tumour (Benign / Malignant)		
3	Infections		
4	Trauma (Blunt)		
5	Miscellaneous Causes		

ADVERSE SOCIAL HABITS :

SL NO	CONSUMPTION	YES	NO
1	SMOKING		
2	ALCOHOL		

TREATMENT HISTORY : NSAIDS / ANTIPLATELET DRUGS / STEROIDS / NIL

INVESTIGATIONS :

A] Hemogram : Hb:

Tc:

Dc:

B] Renal (RFT) : Sugar :

Urea:

Creatinine :

electr:

C] Chest X –Ray : Pneumoperitoneum { Yes / No }

D] USG ABDOMEN AND CT ABD :

OPERATIVE FINDINGS :

Site Of The Perforation : Stomach / Duodenum/ JEJ/ ILE/COLON

Size Of The Perforation : Small/ Large / Giant

Surrounding Induration : Yes / No

Associated Pathology :

MORBIDITY / MORTALITY

:

HPE REPORT (IF AVAILABLE)

:

Fig 1 : Pneumoperitoneum (Air Under Both Domes Of Diaphragm)

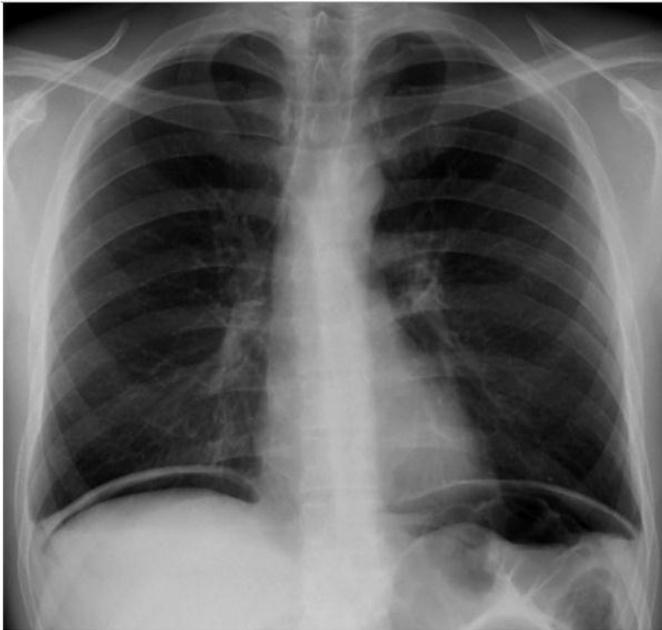


FIG 2 : MALIGNANT GASTRIC PERFORATION



Fig 3 : Jejunal Diverticulitis With Perforation



Fig 4 : TYPHOID PERFORATION OF THE ILEUM

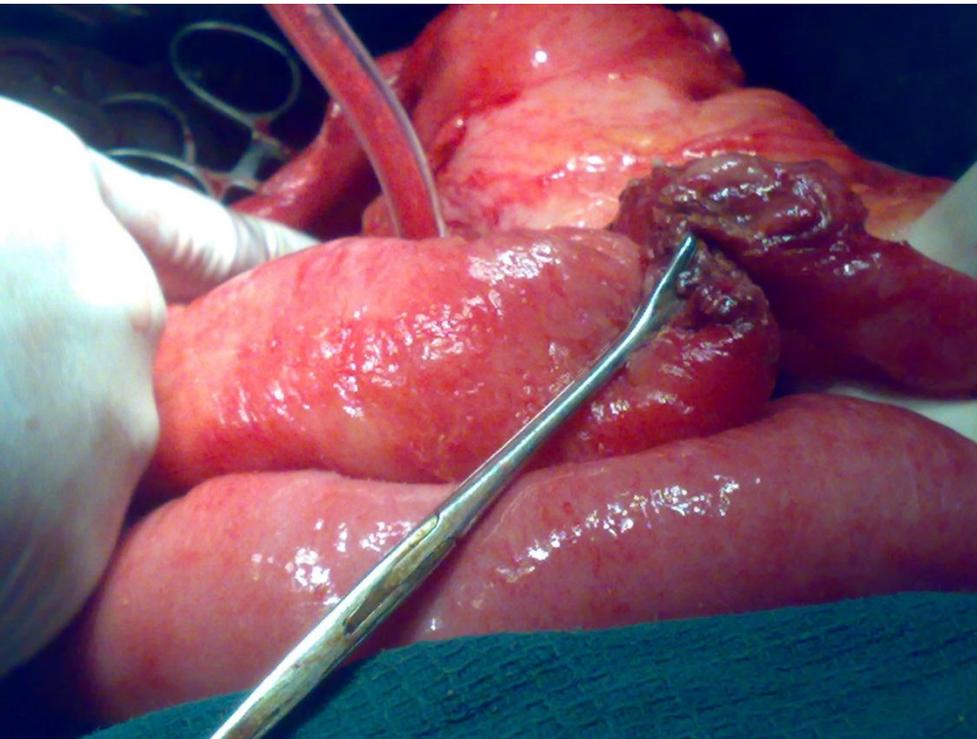


FIG 5 : JEJUNAL GIST WITH PERFORATION



Fig 6 : Stercoral Perforation Of The Rectosigmoid Region

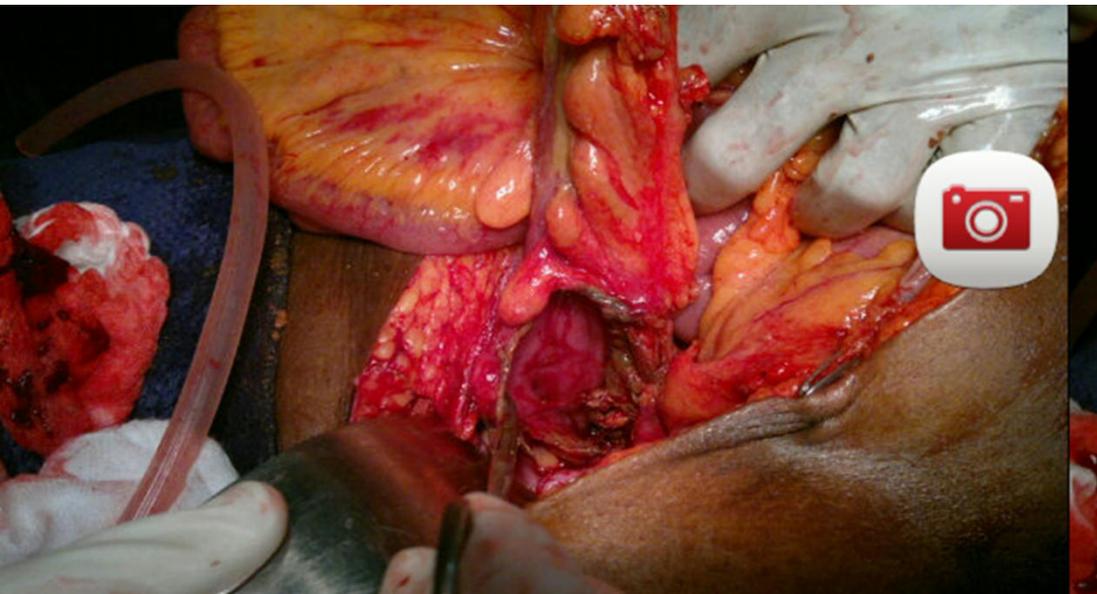


FIG 7: LIGHTNING STRIKE – ILEAL PERFORATION



FIG 8 : BLUNT TRAUMA-MULTIPLE JEJUNAL PERFORATIONS





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Submission time	18-Dec-2012 11:15PM
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EVALUATION OF CASES OF HOLLOW VISCUS PERFORATION Dissertation submitted in partial fulfilment of M.D. DEGREE EXAMINATION M.S. GENERAL SURGERY - BRANCH I CHENGALPATTU MEDICAL COLLEGE, CHENGALPATTU THE TAMILNADU DR .M.G.R. MEDICAL UNIVERSITY CHENNAI, TAMILNADU APRIL 2013 1 INTRODUCTION Hollow viscus perforation is defined as the perforation of any hollow viscus in a patient who presents with acute abdomen with the presence of extra luminal air radiologically. The Causes of hollow viscus perforation includes peptic ulcer disease, perforation of a gastrointestinal neoplasm [benign or malignant], acute appendicitis with perforation, and acute colonic or small bowel diverticulitis, including...



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EVALUATION OF CASES OF HOLLOW VISCUS PERFORATION

BY ANAND 22101271 M.S. GENERAL SURGERY



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Dissertation submitted in partial fulfilment of
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