THE CLINICAL STUDY ON FACTORS INFLUENCING OUTCOME OF DISEASE IN GASTRO- DUODENAL PERFORATIONS

Dissertation submitted in partial fulfillment of the regulation for the award of **M.S. DEGREE IN GENERAL SURGERY**

(BRANCH I)

THE TAMILNADU

DR. M.G.R MEDICAL UNIVERSITY

CHENNAI-600 032

APRIL 2012

CERTIFICATE

This is to certify that the dissertation titled **"THE CLINICAL STUDY ON FACTORS INFLUENCING OUTCOME OF DISEASE IN GASTRO DUODENAL PERFORATIONS"** is a bonafide research work done by **DR.RAJESWARAN A** and submitted in partial fulfillment of the requirements for the Degree of **M.S, GENERAL SURGERY, BRANCH I** of the **TAMILNADU DR. M.G.R MEDICAL UNIVERSITY, CHENNAI.**

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DECLARATION

I solemnly declare that the dissertation titled **"THE CLINICAL STUDY ON FACTORS INFLUENCING OUTCOME OF DISEASE IN GASTRO DUODENAL PERFORATIONS"** was done by me from 2009 – 2012 under the guidance and supervision of **PROF. Dr. S. NATARAJAN M.S.** This dissertation is submitted to the **TAMILNADU DR. M.G.R MEDICAL UNIVERSITY** towards the partial fulfillment of the requirement of award of **M.S DEGREE IN GENERAL SURGERY (BRANCH I).**

Place:

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First I thank The God for the forgiveness and blessings.

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CONTENTS

Introduction

INTRODUCTION

Gastro duodenal perforations following peptic ulcer disease(PUD) is one of the most common surgical emergency encountered in surgical practice. After the introduction of proton pump inhibitors(PPI) need for elective surgery is virtually eliminated. But the emergency perforation surgery is not much in the downfall.

Perforation occurs in 2-10% of patients with PUD and accounts for more than 70% of deaths associated with PUD[1,2]. Often perforation is the first clinical presentation of PUD. The incidence of duodenal perforation is 7-10 cases/ 100.000 adults per year.[1.2,15,25,27,28]. The perforation site usually involves the anterior wall of the duodenum (60%), although it might occur antral (20%) and lesser-curvature gastric ulcers (20%)[27]. Duodenal ulcer is the predominant lesion of the western population, whereas gastric ulcers are more frequent in oriental countries, particularly in Japan. Gastric ulcers have a higher associated mortality and a greater morbidity resulting from hemorrhage, perforation and obstruction[26]. PPU used to be a disorder mainly of younger male patients

By ordered the frequency duodenal perforation being the commonest and accounts for 60-70%.[1] In Duodenal perforations 80-90% due to the H.pylori[1,2,10].

Second being the gastric perforations 10-20%[1.2]among which 70-80 due to H.pylori. Gastro-duodenal perforations covers most of the perforations. Other factors contributing are NSAID Ingestion, ,smoking,, alcoholism and other causes includes malignancy, Zollinger Ellison syndrome, stab injury abdomen, tabesdorsalis,, porphyria, familial mediterian fever, sickle cell disease and rarely cocaine abuse [1.2]causes juxta pyloric perforations.

Eventhough peptic ulcer perforation are common surgical emergency ,the outcome is painstaking for the patients and also for surgeons. Previously peptic ulcer is so common and it was complicated by bleeding, perforation and duodenal obstruction. Nowadays it was almost nil and but perforation incidence was rising due to NSAID like aspirin and other COX-2 inhibitors[15`].

Prescription of non-steroidal anti-inflammatory drug (NSAID)-[1.2]induced ulcers is increasing with the result that perforated peptic ulcers will continue to present despite modern medical management of PUD[2].smoking is believed to be one of the most important etiological factors in the development of peptic ulcers especially in the young and increases the risk tenfold in both men and women. It is estimated that smoking may account for 77% of all ulcer perforations in those younger than75 years, whereas in the older population, smoking is of much less importance. The use of NSAIDs is another well-documented and important risk factor for ulcer perforations. It has been estimated to increase the risk by 5–8 times. However, the use of NSAID is less common in the population than smoking and therefore accounts for a smaller number of perforations. The role of *H. pylori* infection in ulcer perforation cannot be confirmed but this continues to be a well-debated subject. Current evidence shows that treatment for eradication of *H. pylori* significantly reduces the peptic ulcer recurrence rate. Recurrent ulcer rates were 6 and 4% for duodenal and gastric ulcers when *H. pylori* was eradicated compared with 67 and 59%, respectively, when the organism was not eradicated. Other risk factors include alcohol, stress with burns leading to Curling's ulcer, neurological insult (Cushing's ulcer) and major surgery. Familial association[1,.2] with a threefold increase in incidence of duodenal ulcers in relatives and duodenal ulcers are most common in HLA-[B1.2] and people with blood[2]group[O1.2].

Aims and Objectives

AIMS AND OBJECTIVES

Inspite of improvement in modern era of surgical therapeutics, with higher spectrum of antibiotics-still perforative peritonitis seems to be a grave disease with high mortality and morbidity.

- ✓ To study the factors which influence the outcome of disease.
- ✓ To study the prognosis of the disease.
- ✓ To study the prognosis of the surgery.
- ✓ To study the complications of the surgery

Able to improve the outcome of disease by reducing the mortality and morbidity

Review of Literature

REVIEW OF LITERRATURE

HISTORICAL DATA

In the last hundred years much has been written on peptic ulcer disease and the treatment options for one of its most common complications: perforation. The reason for reviewing literature was evaluating most common ideas on how to treat perforated peptic ulcers in general, opinions on conservative treatment and surgical treatment and summarizing ideas about necessary pre- per and postoperative proceeding

Fig.1Henrietta-anne

King Charles I's daughter, Henrietta-Anne (fig.1), died suddenly in 1670 (at age 26) after a day of abdominal pain and tenderness[3]. Since poisoning was suspected autopsy was performed and revealing peritonitis and a small hole in the anterior wall of the stomach. However, the doctors had never heard of a perforated peptic ulcer (PPU) and attributed the hole in the stomach to the knife of the dissector.[3, 4]

In 1729 **Christopher Rawlinson** reported one case and one by **Jacob Peneda** in 1795.

In 1835 **Cruveilheir** described clinical features of peritonitis.

Brinton[7]made a collection of 234 cases of peptic ulcer perforations with his Atlas in 1857.**Hensner-kriedge**[7] -1st surgeon to perform simple closure of perforated peptic ulcer in 1892**. Heberer**[7] 1919 reported a successful Gastric resection**.**

Birches (1925)-1s[7] performed selective vagotomy.

Zollinger-Ellison (1955)-described islet cells tumors and peptic ulcer.

PUD-A rare disease some 100 years ago**.**

Rodney maingot's reported the mortality of 30% -30 years back. In modern surgical Era reduced drastically

Cellan-Jones published an article in 1929entitled "a rapid method of treatment in perforated duodenal ulcers". Treatment of choice at that time was, after excision of friable edges if indicated, the application of purse string sutures and on top and omental graft[8]. An encountered problem was narrowing of the duodenum. To avoid this, he suggested omentoplasty without primary closing of the defect. His technique consisted of placing 4-6 sutures, selecting a long omental strand passing a fine suture through it, the tip of the strand is then anchored in the region of the perforation and finally the sutures are tied of.

It was not until 1937 that **Graham**[9] published his results with a free omental graft. He placed three sutures with a piece of free omentum laid over these sutures, which are then tied. No attempt is made to actually close the perforation. The omental graft provides the stimulus for fibrin formation. His approach has been the golden standard since. Very often surgeons mention they used a Graham patch, but they actually mean they used the pedicled omental patch described by Cellan-Jones[8,9]

Anatomy & physiology

Wallace P. Ritchie, Jr. called ***"the stomach an elegant organ, once thought to be the seat of the soul, always handy to bring to the dinner table, and a recognized source of ecstasy and grief"***

Stomach is an asymmetric dilatation of proximal gastro intestinal tract, responsible for the initial digestion and storage of food. Stomach's capacity in adults is 1.5-2.5 litres. Stomach is divided into cardia, body, fundus, antrum and pylorus[1]

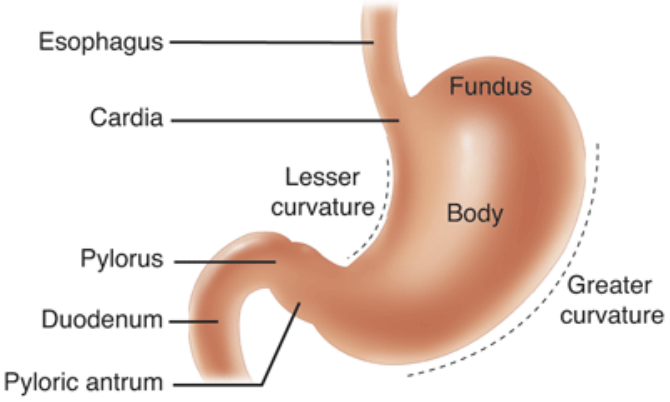

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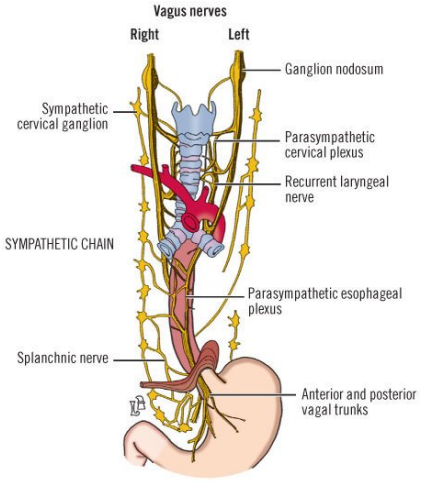
Fig.2 Stomach

Vagus nerve[1] are motor and sensory supply of the stomach (fig3a, b.)



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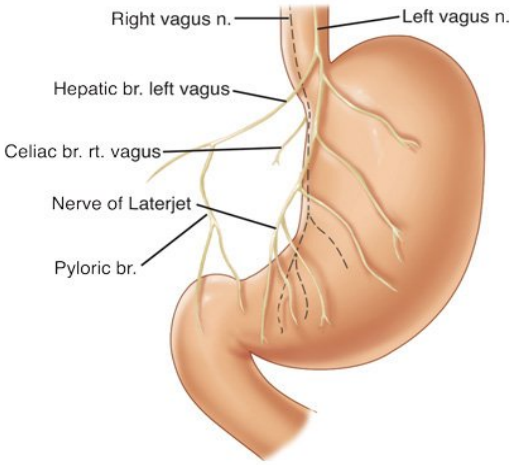

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Fig3a.Vagus anatomy

Fig.3b Vagal Innervation of stomach

Histology[1]

Gastric epithelial cells-columnar type and filled with mucinous granules-responsible for lubrications of contents.

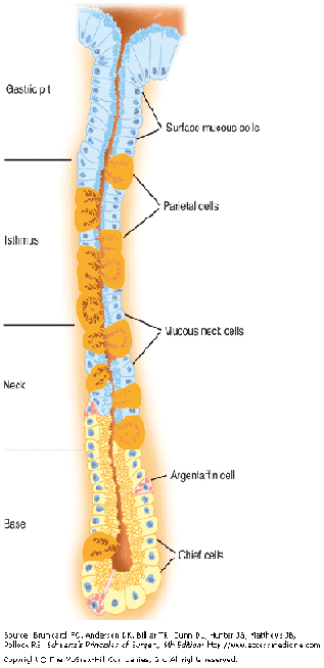

Fig4 Gastric gland from the body of the stomach

Parietal cells found in the body of the stomach lie in gastric crypts. Responsible for H^+ ion secretion.

Chief cells found mainly in the fundus and responsible for secretion of pepsinogen.

Endocrine cells G and D cells mainly. G cells secrets gastrin mainly found in antrum. D cells secrets somatostatin involved in negative feed back of gastric acid secretion.

Physiology stomach has reservoir, secretory and motor function. In the stomach ingested food mixed with the acid and other digestive enzymes and through pyloric antrum‘ the chime’ slowly released into the duodenum. In the duodenum the pancreatic and duodenal secretion brought the content into neutral PH. The rate at which the chime delivered from the stomach is dependent upon osmolality and fat and caloric contents of the chyme through receptors in the proximal duodenum.

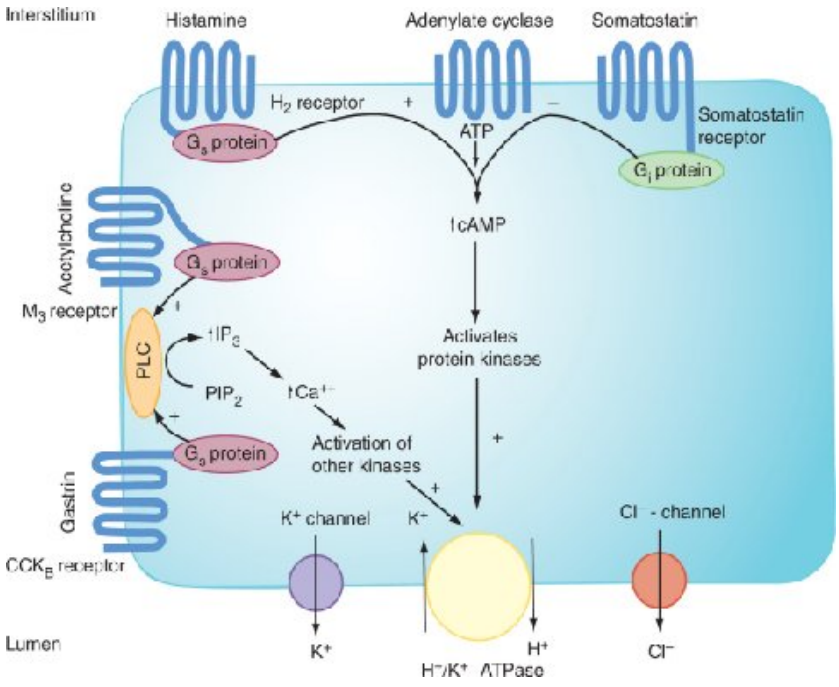

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Fig5 Control of acid secretion in the parietal cell.

Secretion of gastric juice[1]consists of pepsin, intrinsic factors and other organic solutes in the dilute HCl. Mainly secretion in 3 phases **cephalic** ,**gastric and intestinal.**

Mucosal barrier[1]*gastric mucous* forms the protective barrier which mucosa from the damage from the acid. The protective function is mainly mucous secreted by surface mucous cells in the antral area. mucous is unstirred layer of viscio-glycoproteins in the luminal area. *Bicarbonate* secretion causes the cellular PH of 5 in against to the luminal PH of 2. Damage occurs when luminal PH is <1.4 and mucosal barrier is damaged due to NSAIDs.

Prostaglandins

PGE have been shown to have potent antiulcer activity, acting through two major mechanisms—**1.Inhibition of acid secretion 2.Enhancement of duodenal mucosal cytoprotection**. Cytoprotective features of prostaglandins involve multiple PGE-mediated actions, including (1) increased mucus production, (2) increased duodenal alkaline secretion, (3) increased duodenal mucosal blood flow, (4) increased gastric mucosal sulfhydryl compounds, (5) increased lysosomal instability, (6) increased surface phospholipids, and (7) stabilization of mast cell membranes.

Basal Acid Secretion[1] basal level of acid secretion that is roughly 10% of maximal acid output. Basal acid secretion also exhibits a circadian variation, with night-time acid secretion greater than daytime. Under basal conditions, 1 to 5 mmol/hour of hydrochloric acid is secreted, and this is reduced by 75% to 90% after vagotomy or administration of atropine. These findings suggest that acetylcholine plays a significant role in basal gastric acid secretion. However, H_2-receptor blockade diminishes the magnitude of acid secretion by 90%, suggesting that histamine also

plays an important intermediary role in this process. Thus, it appears likely that basal acid secretion is due to a combination of cholinergic and histaminergic input.

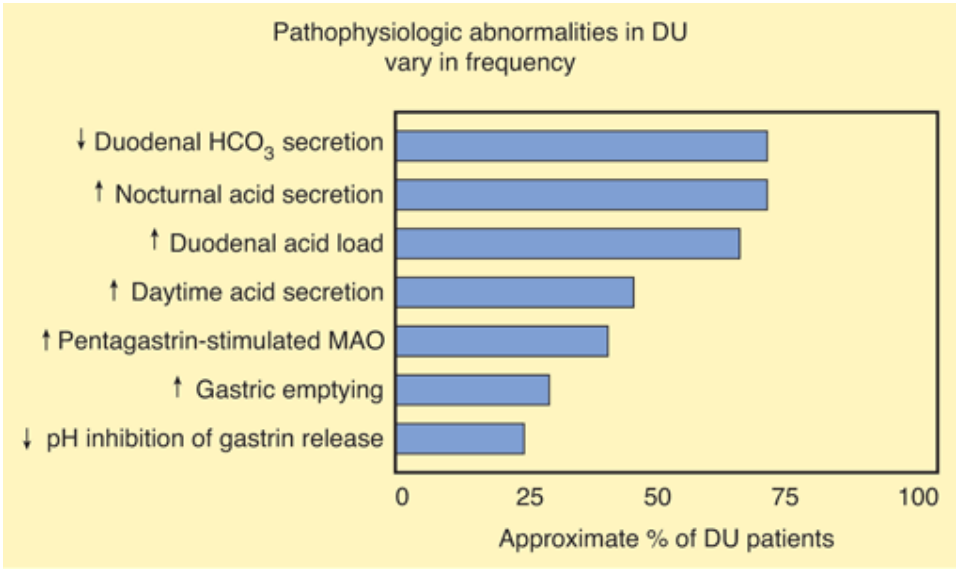

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Fig.6.Frequency of physiologic abnormalities in patients with duodenal ulcer

Types of gastro-duodenal perforations[1]

Currently**, five types** of gastric ulcer are described, although the original **Johnson classification** contained three types- The most common, Johnson **type I gastric ulcer**, is typically located near the incisura angularis on the lesser curvature, close to the border between the antrum and the body of the stomach with normal or decreased acid secretion. **Type II gastric ulcer** is associated with active or quiescent duodenal ulcer disease, and **type IIIgastric ulcer** is prepyloric ulcer disease. Type II and type III gastric ulcers are associated with normal or increased gastric acid secretion. **Type IVgastric ulcers** occur near the GE junction, and acid secretion is normal or below normal. **Type V gastric ulcers** are medication induced and may occur anywhere in the stomach. NSAIDs and aspirin have similar effects

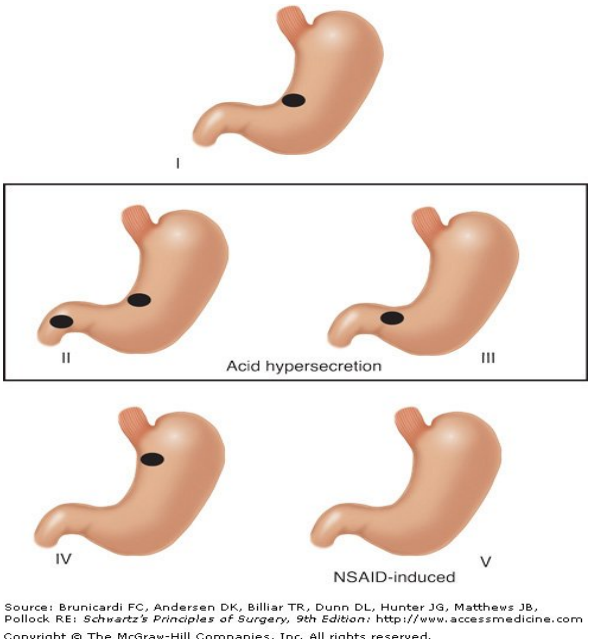

Fig.7. Types of Peptic Ulcer

Patho-physiology of ulcer formation[1]

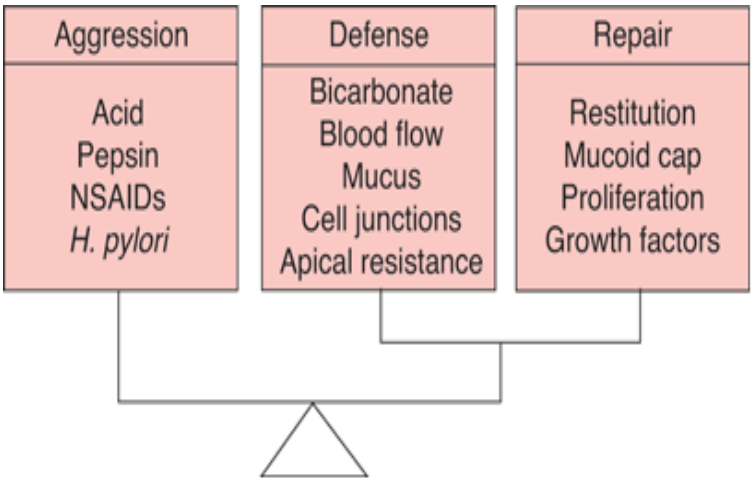

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Fig.8 Peptic ulcer pathophysiology[50].

Imbalance between defense and acid secretion leads to duodenal ulceration[1]

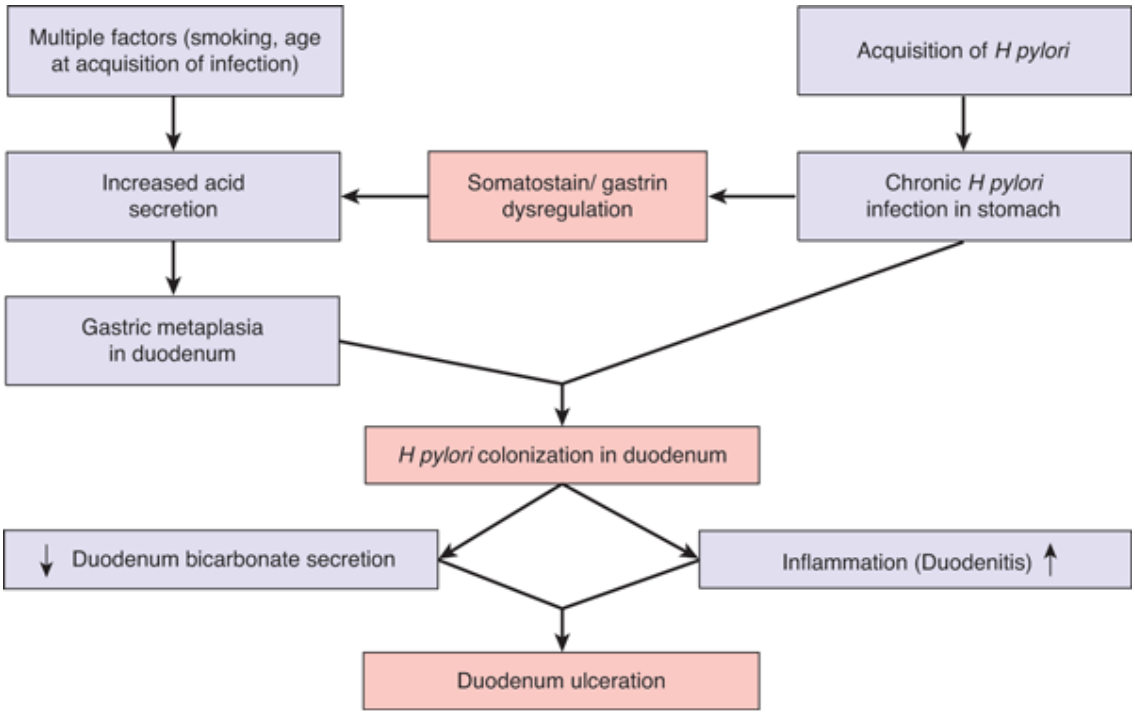

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Fig 9.Model of *H. pylori*–induced effects on duodenal ulcer pathogenesis[49]

ETIOPATHOGENESIS OF GASTRO-DUODENAL PERFORATIONS

90% Duodenal perforations[11] and 75-85% Gastric perforations[11] are due to H. Pylori[11,12].Other causes are malignancy, Zollinger-ellison syndrome, trauma especially stab injury, caustic ingestion and rarely tabesdorsalis, porphyria, familial mediterrian fever, sickle cell disease. Cocaine abuse may cause juxta pyloric perforation. Male is most common victim of the life threatening disease.

The pathogenesis of PUD may best be considered as representing a complex scenario involving an imbalance between defensive (mucus-bicarbonate layer, prostaglandins, cellular renovation, and blood flow) and aggressive factors (hydrochloric acid, pepsin,

ethanol, bile salts, some medications, etc.) [12] In recent years Helicobacter pylori is the prime culprit

(H.pylori)[12] infection and NSAIDs[12] have been identifed as the two main causes of peptic ulcer. The use of crack cocaine[15] has also led to an increase in PPU, but with a different underlying mechanism since PPU secondary to the use of crack cocaine is caused by ischemia of the gastric mucosa and treatment of these perforations do not require acid reducing defnitive surgery.

Three clinical phases in the process of PPU can be distinguished[16] .

Phase 1: **Chemical peritonitis/ contamination**:[16] The perforation causes a chemical peritonitis. Acid sterilizes gastroduodenal contents; it is only when gastric acid is reduced by treatment or disease (gastric cancer) that bacteria and fungi are present in the stomach and duodenum.

Phase 2: Intermediate stage:[16]after 6-12 hrs many patients obtain some spontaneous relief of the pain. This is probably due to the dilution of the irritating gastroduodenal contents by ensuing peritoneal exudates(fig-10).

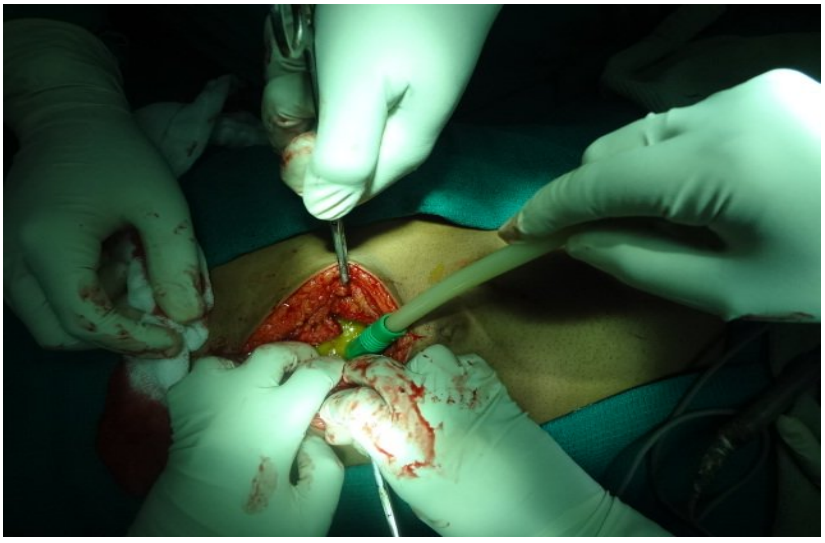

Fig.10 Sero-Purulent Fluid

Phase 3: Intra-abdominal infection[16]: After 12-24 hrs intra abdominal infection supervenes.

Helicobactor pylori [12,18,21]

Micro--aerophilic, Spiral/ Helical, Gram negative with 4-6 flagella. Resides gastric epithelium beneath mucous layer(fig..2).H.pylori—produces urease which split urea to ammonia&HCo3-creatingalkaline micro environment –Breaks MUCOSAL BARRIER

Mechanism of injury.

1.Toxin-causes local injury.

2.Induce local immune response.

3.Raises Gastrin level-increses acid hyper secretion due to reduced antral D cells.

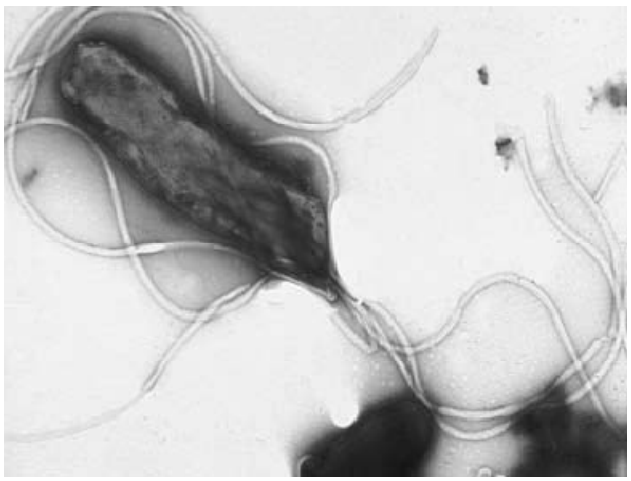

Fig.11-Helicobactor pylori

Higher incidence found in low socio-economic status. Transmitted by person to person. Serology-(IgG.)90% speficity& sensitivity

Patho-physiology of ulcer formation-due to H. pylori infection[1]

Helicobacter strains that lack flagella are unable to navigate through the unstirred mucus layer to get to the apical membrane of the SEC for attachment, and are

nonpathogenic. One of the mechanisms by which *Helicobacter* causes gastric injury may be through a disturbance in gastric acid secretion. This is due, in part, to the inhibitory effect that *H. pylori* exerts on antral D cells that secrete somatostatin, a potent inhibitor of antral G-cell gastrin production. *H. pylori* infection is associated with decreased levels of somatostatin, decreased somatostatin messenger RNA production, and fewer somatostatin-producing D cells. These effects are probably mediated by *H. pylori*–induced local alkalinization of the antrum (antral acidification is the most potent antagonist to antral gastrin secretion), and *H. pylori*–mediated increases in other local mediators and cytokines. The end result is hypergastrinemia and acid hypersecretion (Fig. 26-26).[17]Thishypergastrinemia presumably leads to the parietal cell hyperplasia seen in many patients with duodenal ulcer. The acid hypersecretion and the antral gastritis are thought to lead to antral epithelial metaplasia in the postpyloric duodenum. This duodenal metaplasia allows *H. pylori* to colonize the duodenal mucosa and, in these patients, the risk of developing a duodenal ulcer increases 50-fold. [1,17]

When *H. pylori* infection is successfully treated, acid secretary physiology tends to normalize. Other mechanisms whereby *H. pylori* can induce gastroduodenal mucosal injury include the production of toxins (vacA and cagA), local elaboration of cytokines (particularly interleukin-8) by infected mucosa, recruitment of inflammatory cells and release of inflammatory mediators, recruitment and activation of local immune factors, and increased apoptosis

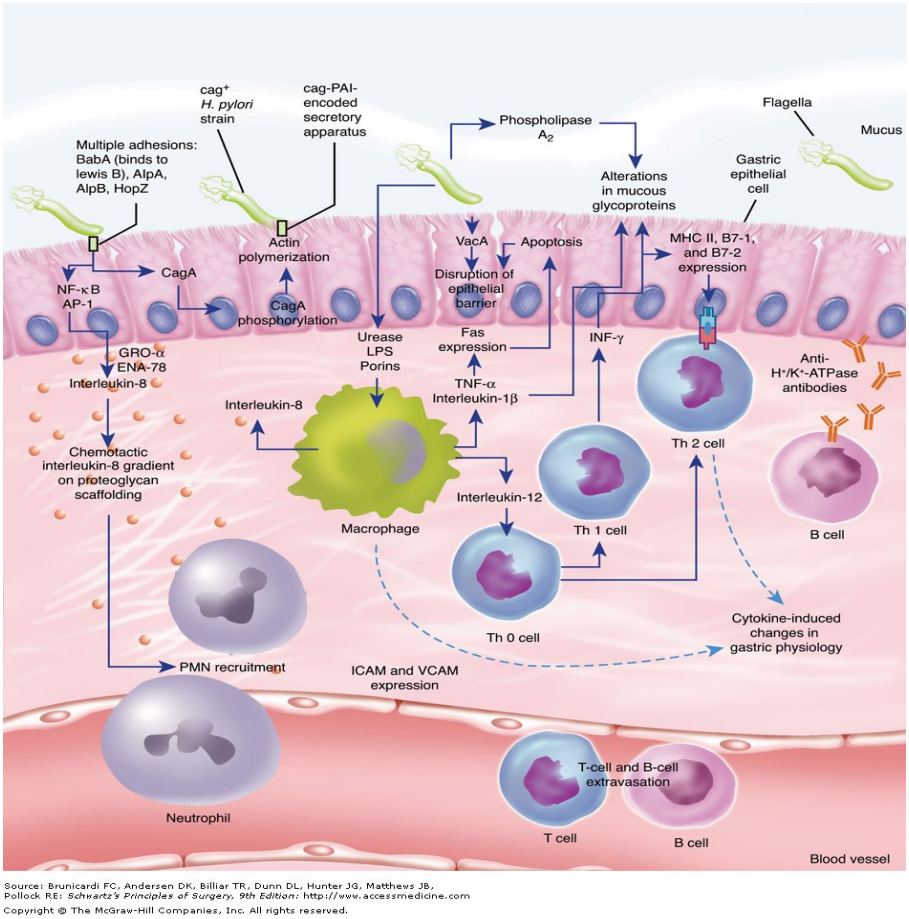

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Fig12.Pathogen-host interactions in the pathogenesis of *Helicobacter pylori* infection.[52]

ICAM = intercellular adhesion molecule-1; INF = interferon-; LPS = lipopolysaccharide; NF B = nuclear factor B; PAI = pathogenicity island; PMN = polymorphonuclear neutrophil; TNF- = tumor necrosis factor alpha; VCAM = vascular cell adhesion molecule.

NSAID INGESTION(5-10%perforations)[1,2,3,19,21]

NSAIDs (including aspirin) are inextricably linked to PUD. Patients with rheumatoid arthritis and osteoarthritis who take NSAIDs have a 15 to 20% annual incidence of peptic ulcer, and the prevalence of peptic ulcer in chronic NSAID users is about 25% (15% gastric and 10% duodenal). Complications of PUD (specifically

haemorrhage and perforation) are much more common in patients taking NSAIDs. More than half of patients who present with peptic ulcer haemorrhage or perforation report the recent use of NSAIDs, including aspirin. Many of these patients remain asymptomatic until they develop these life-threatening complications.

The overall risk of significant serious adverse GI events in patients taking NSAIDs is more than three times that of controls. This risk increases to five times in patients more than age 60 years old. In elderly patients taking NSAIDs, the likelihood that they will require an operation related to a GI complication is 10 times that of the control group, and the risk that they will die from a GI cause is about four and one-half times higher. This problem is put into perspective when one realizes that approximately 20 million patients in the United States take NSAIDs on a regular basis; perhaps as many regularly take aspirin. Persons who take NSAIDs also have a higher hospitalization rate for serious GI events than those who do not.

SMOKING & ALCOHOL[2.1]

Smoking has associate with type-I &II gastro-duodenal ulcer. Epidemiologic studies suggest that smokers are about twice as likely to develop PUD as nonsmokers. Smoking increases gastric acid secretion and duodenogastric reflux. Smoking decreases both gastroduodenal prostaglandin production and pancreaticoduodenal bicarbonate production the observed association between smoking and PUD. Although difficult to measure, both physiologic and psychologic stress undoubtedly play a role in the development of peptic ulcer in some patients. In 1842, Curling described duodenal ulcer and/or duodenitis in burn patients. Decades later, Cushing described the appearance of acute peptic ulceration in patients with head trauma (Cushing's ulcer). Even the ancients recognized the undeniable links between PUD and stress. Patients still present with ulcer complications (bleeding,

perforation, and obstruction) that are seemingly exacerbated by stressful life events. The use of crack cocaine[1,2] has been linked to juxta pyloric peptic ulcers with a propensity to perforate. Alcohol is commonly mentioned as a risk factor for PUD, but confirmatory data are lacking.

ZOLLINGER-ELLISON SYNDROME[1,3,4]

1.Acid hyper secretion

2.Severe peptic ulcer disease

3. .Non-β Islet cell tumors

ZES is caused by the uncontrolled secretion of abnormal amounts of gastrin by a duodenal or pancreatic neuro endocrine tumor (i.e., gastrinoma). Most cases (80%) are sporadic, but 20% are inherited. The inherited or familial form of gastrinoma is associated with multiple endocrine neoplasia type 1 (MEN1), which consists of parathyroid, pituitary, and pancreatic (or duodenal) tumors. Gastrinoma is the most common pancreatic tumor in patients with MEN I. Patients with MEN I usually have multiple gastrinoma tumors, and surgical cure is unusual. Sporadic gastrinomas are more often solitary and amenable to surgical cure. Currently, about 50 to 60% of gastrinomas are malignant, with lymph node, liver, or other distant metastases at operation. Five-year survival in patients presenting with metastatic disease is approximately 40%. The larger the primary gastrinoma, the higher the likelihood of metastatic disease. More than 90% of patients with sporadically, completely resected gastrinoma will be cured.

The most common symptoms of ZES are epigastric pain, GERD, and diarrhea. More than 90% of patients with gastrinoma have peptic ulcer. Most ulcers are in the typical

location (proximal duodenum), but atypical ulcer location (distal duodenum, jejunum, or multiple ulcers) should prompt an evaluation for gastrinoma

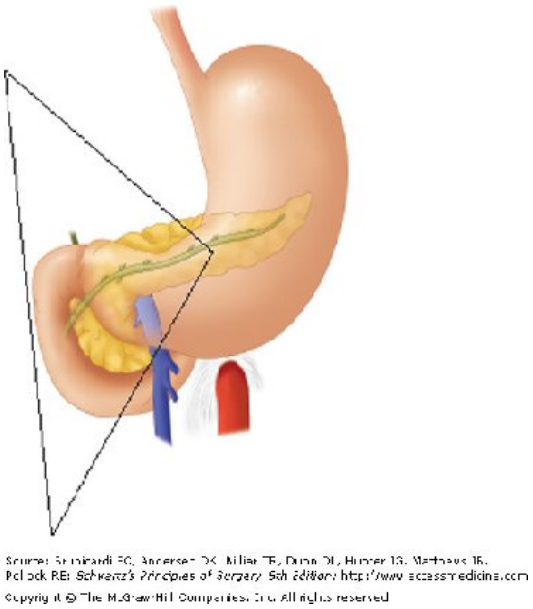


Fig13. Gastrinoma Triangle

Gastrinoma also should be considered in the differential diagnosis of recurrent or refractory peptic ulcer, secretory diarrhea, gastric rugal hypertrophy, esophagitis with stricture, bleeding or perforated ulcer, familial ulcer, peptic ulcer with hypercalcemia, and gastric carcinoid. The majority of patients with ZES have been symptomatic for several years before definitive diagnosis and, in general, patients with ZES and MEN1 are diagnosed in their 20s and 30s, while those with sporadic ZES more typically are diagnosed in their 40s and 50s.

ZES is an important part of the differential diagnosis of hypergastrinemia. All patients with gastrinoma have an elevated gastrin level, and hypergastrinemia in the presence of elevated BAO strongly suggests gastrinoma. Patients with gastrinoma usually have a BAO >15 mEq/h or >5 mEq/h if they have had a previous procedure

for peptic ulcer. Acid secretory medications should be held for several days before gastrin measurement, because acid suppression may falsely elevate gastrin levels. Causes of hypergastrinemia can be divided into those associated with hyperacidity and those associated with hypoacidity Fasting Gastrin level->1000 pg/dl is diagnostic. On stimulation with secretin ->100 pg/dl is diagnostic.Values<100pg/dl excludes the diagnosis of ZES.

90% of gastrinoma's present in this GASTRINOMA TRIANGLE.(fig.13)

Malignancy causing-perforation

Malignant disease causing perforation.Either by directly or distal obstruction which leads to proximal perforation indirectly. Disease causing perforation occurs anywhere in the GIT directly .Indirectly usually in small bowel or the caecum.

Trauma 1.Blunt injury

2.Penetrating injury

Blunt injury usually will not cause gastro duodenal perforations

Penetrating injury-abdomen

a)Stab injury

b)Gunshot

c)Missile

Any suspicion of intra abdominal injury-Laparotomy.

Indication for surgery

*Peritonitis *Hemodynamic instability *Hemoperitoneum

*Bowel perforation *splenic/liver lacerations

Caustic Ingestion[1]Because gastric injuries are more common and severe after the ingestion of liquid alkali and strong acid solutions compared with crystalline lye and other caustic substances, knowledge of the ingested agent is critical. Burns of the

upper mouth and pharynx, drooling, and upper airway compromise often indicate a significant caustic ingestion. There is no absolute correlation between oropharyngeal and esophagogastric injury, and it has been reported that up to 20% of patients with injuries to the esophagus and stomach have no evidence of oropharyngeal involvement.

The most useful modality for the diagnosis of the presence or extent of caustic injuries to the esophagus and stomach is endoscopic examination of the aerodigestive tract. In cases of alkali ingestion, endoscopy is terminated at the point at which deep, circumferential burns are noted, because gastric injury is common in the presence of second- or third-degree circumferential injury, and further passage of the scope may produce additional injury.The passage of a nasogastric tube and measurement of intragastric pH may be prognostic, because it has been reported if the gastric pH is more than 7, gastric injury is likely. Some have advocated laparotomy with direct inspection of the stomach in all cases of lye ingestion producing deep, circumferential esophageal burns, but this policy is not universally accepted.[94] In acid ingestion, the endoscope can usually be passed directly into the stomach to assess the presence or degree of gastric injury, because esophagus injury is usually absent.

Careful examination of the abdomen is paramount in patients with caustic ingestion. Because abdominal signs and symptoms may develop in a delayed fashion, due to progressive necrosis, serial examinations are indicated. Marked abdominal tenderness or peritonitis should trigger laparotomy. If any doubt exists as to the potential for severe gastric injury, laparotomy or laparoscopy should be performed

Iatrogenic

1.Accidental injury

2.Cautry injury to bowel

Treatment- Immediate Laparotomy

Other causes**stress**[3,2,1] with burns leading to Curling's ulcer, neurological insult (Cushing's ulcer) and major surgery. There are also some associated diseases that include alcoholic cirrhosis, chronic renal failure (CRF), chronic obstructive pulmonary disease (COPD) and hyperparathyroidism which increase serum calcium and subsequent gastrin production. There is a familial association with a threefold increase in incidence of duodenal ulcers in relatives and duodenal ulcers are most common in HLA-B5[2] and people with blood group O.[2]

Clinical presentation and investigation

King Charles I's daughter, Henrietta Anne(fig.1),died suddenly in 1670 (at age 26) after a day of abdominal pain and tenderness. Since poisoning was suspected autopsy was performed and revealing peritonitis and a small hole in the anterior wall of the stomach. However, the doctors had never heard of a perforated peptic ulcer (PPU) and attributed the hole in the stomach to the knife of the dissector.

Johan Mikulicz-Radecki (1850-1905), often referred to as the first surgeon who closed a perforated peptic ulcer (PPU) by simple closure said[22]:

" Every doctor, faced with a perforated duodenal ulcer of the stomach or intestine, must consider opening the abdomen, sewing up the hole, and averting a possible inflammation by careful cleansing of the abdominal cavity"[22]

Epidemiology

Perforation occurs in 2-10% of patients with PUD and accounts for more than 70% of deaths associated with PUD[1,2]. Often perforation is the first clinical presentation of PUD. The incidence of duodenal perforation is 7-10 cases/ 100.000 adults per year[15,25,27,28]. The perforation site usually involves the anterior wall of the duodenum (60%), although it might occur antral (20%) and lesser-curvature gastric ulcers (20%)[27]. Duodenal ulcer is the predominant lesion of the western population, whereas gastric ulcers are more frequent in oriental countries, particularly in Japan. Gastric ulcers have a higher associated mortality and a greater morbidity resulting from hemorrhage, perforation and obstruction[26] PPU used to be a disorder mainly of younger patients (predominantly males)-peak age is 40-60[25].

peptic ulcers are still responsible for about 20.000-30.000 deaths per year in Europe[27,29] . This may be due to an increase in use of aspirin and/ or NSAIDs.[15]

In 1843 Edward Crisp was the first to report 50 cases of PPU and accurately summarized the clinical aspects of perforation; concluding:

"The symptoms are so typical, I hardly believe it possible that anyone can fail to make the correct diagnosis.[23]"

Patients with PPU have a typical history of sudden onset of acute, sharp pain usually located in the epigastric area and sometimes with referred shoulder pain, indicating free air under the diaphragm[24]. . The typical patient with PPU is male with an average age of 48 years. He may have a history of peptic ulcer disease (29%), non steroidal anti-inflammatory drugs (NSAIDs) usage (20%). Vomiting and nausea are present in 50% of cases. At physical examination pulse might be quickened. About 5-10% of patients experience shock with a mean arterial pressure of less than 80 mm of hg. Hypotension is a late finding. Obliteration or complete absence of liver dullness

was only noted in 37%, so as a diagnostic tool, this has its limitations[26] . In blood analysis a moderate leucocytosis will be found. Main reason for taking a blood sample is excluding other diagnosis like for instance pancreatitis. An X-ray of the abdomen/thorax in standing position will reveal free air under diaphragm in about 80-85 %[30,32]

Some centres perform abdominal Ultrasonography, or computerized tomography (CT) scans with oral contrast[31]. With current radiological techniques 80-90% of cases are correctly diagnosed[13,30].

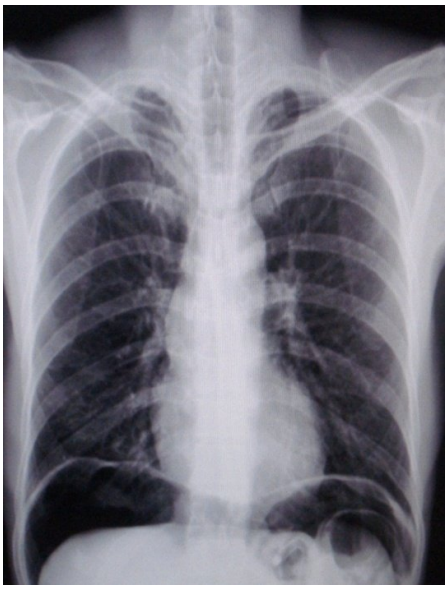


Fig.14. Air under diaphragm-x-ray chest[2]

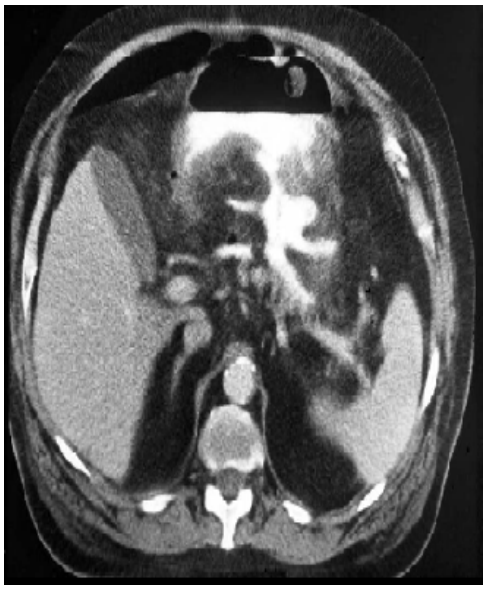


Fig.15.Extraluminal air in CT-scan abdomen

Plain abdominal X-ray may reveal free gas by the presence of gas on both sides of the bowel wall **(Rigler's sign)[2].**

Some centers perform abdominal Ultrasonography, or computerized tomography (CT) scans with oral contrast.. With current radiological techniques 80-90% of cases are correctly diagnosed.

Boey risk factors[2]

The Boey score can be used for risk stratification in patients undergoing open repair for perforated duodenal ulcer as well as being valid for laparoscopic repair. Boey score is defined as the sum of the Boey risk factors scoring one point for the presence of each of the following:

- Shock on admission (systolic blood pressure <90 mm Hg)
- Severe medical illness (ASA III-IV)
- Delayed presentation (duration of symptoms over 24 hours)

Postoperative mortality rates of patients with various

Boey scores have been documented as follows:

0–1.5%

1–14.4%

2–32.1%

3–100%

As soon as diagnosis is made resuscitation is started with large volume crystalloids, nasogastric suction to empty the stomach; and administration of broadspectrum antibiotics. Surgery is indicated and simple omental patch closure is sufficient

Current management of PPU

Non operative management

Conservative treatment **Taylor method** and consists of nasogastric aspiration, antibiotics, intravenous fluids and nowadays H.pylori triple therapy[29,33]. In 1946 Taylor presented the 1ST series of successfully outcome of conservatively treated

patients with PPU, based on the theory that effective gastric decompression and continuous drainage will enhance selfhealing[34] .

Crisp who in 1843 noted that perforations of the stomach were filled up by adhesions to the surrounding viscera which prevented leakage from the stomach into the peritoneum[33,35].It has been estimated that about 40-80% of the perforations will seal spontaneously and overall morbidity and mortality are comparable[27,29,33].

Nonoperative therapy[55] of perforated duodenal ulcers, incorporating nasogastric suction, antibiotics, and fluid resuscitation, had in the past been reserved for poor-risk patients in whom operative treatment would carry undue risks. Nonoperative therapy has also been proposed for lower-risk patients when (1) an upper GI series with water-soluble contrast shows no leak, (2) the patient has never been evaluated or treated for *H. pylori*, and (3) there is no clinical deterioration.[57] Patients managed non operatively require continuous physical examination, meticulous attention to nasogastric tube function, and early mandatory documentation by water-soluble contrast study that there is no communication with the free peritoneal cavity.[58] In patients treated non operatively, constant reassessment is required, and immediate reconsideration of the decision to use nonsurgical therapy is needed in the face of continuing major third-space fluid losses, progressive signs of peritonitis, or increasing pneumoperitoneum. In cases of perforated duodenal ulcer treated surgically or non operatively, expeditious determination of *H. pylori* status must be made. Infected patients should start eradication therapy as soon as is practical. Delaying the time point of operation beyond 12h after the onset of clinical symptoms will worsen the outcome in PPU[27,34]. Also in patients > 70 years Conservative treatment has high mortality[34,36].

When the patients is in shock or is the time point between perforation and "start treatment" > 12 hours simple closure should be first choice oftreatment.

b. Simple suture Open repair technique upper midline incision is performed. Identifcation of the site of perforation is not always easy: sometimes a perforation has occurred at the dorsal site of the stomach, only to be detected afer opening of the lesser sac through the gastrocolic ligament .Double perforations can occur. In case of a gastric ulcer a biopsy is taken to exclude gastric cancer. Simple closure of the perforation can be done in different ways simple closure of the perforation by interrupted sutures without omentoplasty or (free) omental patch, simple closure of the perforation with a pedicled omentum sutured on top of

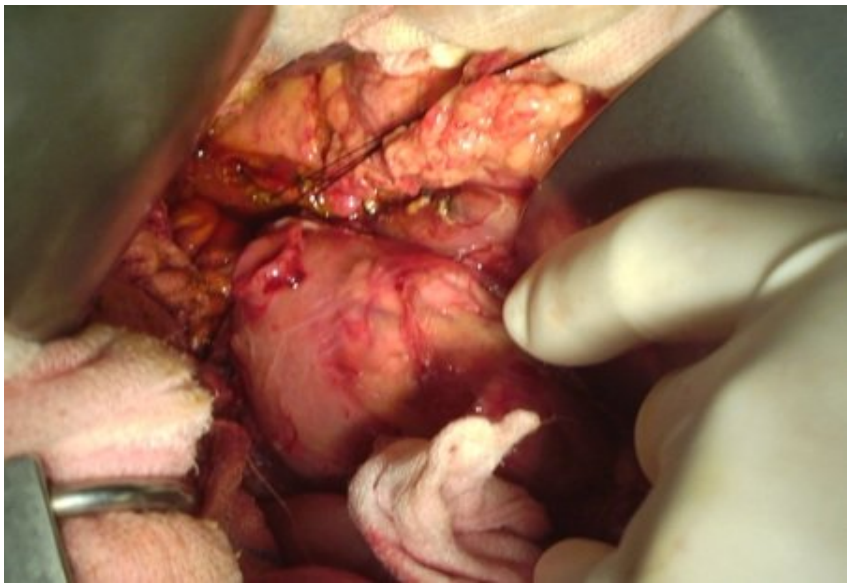


Fig 16.simple omental patch closure

the repair, respresenting omentoplasty, a pedicled omental plug drawn into the perforation after which the sutures are tied over it and finally the **free omental patch after Graham**[38]. . Thorough peritoneal toilet followed is then performed. A drain is not routinely left**.**

Cellan-Jones[37]published an article in 1929 entitled "a rapid method of treatment in perforated duodenal ulcers''

Different suture techniques for closing perforation

Primary closure by interrupted sutures Primary closure by interrupted sutured covered with pedicled omentoplasty

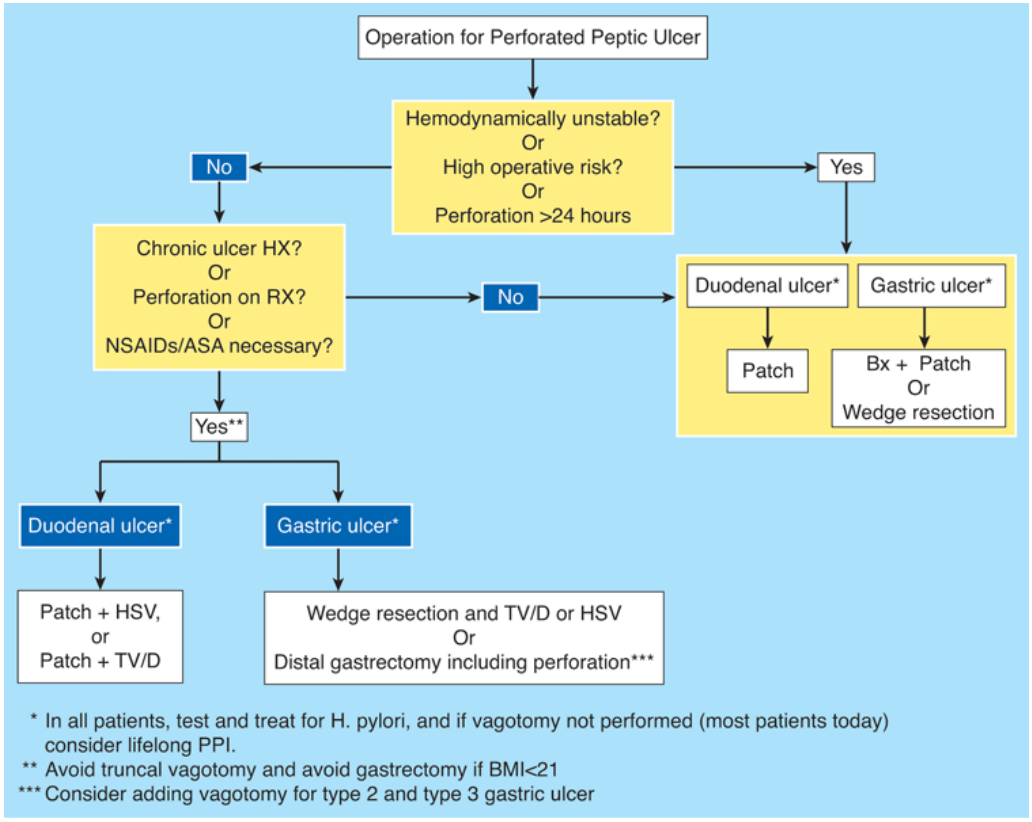
Cellan-Jones repair: plugging the perforation with pedicled omentoplasty

Graham patch: plugging the perforation with free omental plug[38]

d. Laparoscopy Laparoscopic surgery offers several advantages.Laparoscopic repair are postoperative pain reduction and less consumption of analgesics and a reduction in hospital stay and reduction in wound infections, burst abdomen and incisional hernia due to shorter scars has been noted

Suture less techniques have been tried, in which fibrin glue alone or a gelatine sponge has been glued into the ulcer. The downside of this technique is that is only can be used to close small perforations. To overcome this problem a biodegradable patch, that can be cut into any desirable size, has been tested in rats, with good results and still in the experimental level.

Combined laparoscopic-endoscopic repair attempted with good results and only disadvantage is distension of abdomen due to air.



Source: Brunicardi FC, Andersen DK, Billiar TR, Dunn DL, Hunter JG, Matthews JB, Pollock RE: *Schwartz's Principles of Surgery, 9th Edition:* http://www.accessmedicine.com
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Fig 17.Algorithm for operation for perforated peptic ulcer.

ASA = acetylsalicylic acid; BMI = body mass index; Bx = biopsy; HSV = highly selective vagotomy; Hx = history; PPI = proton pump inhibitor; Rx = treatment; TV/D = truncalvagotomy and drainage.

Complication

Postoperative complications The postoperative complication most common observed was pneumonia, followed by wound infection. An overview of all complications and their incidences, based on reviewing literature are[25,32,27,39,40,41,42,43].

Pneumonia	3.6-30%
Wound infection	10-17%
Urinary tract infection	1.4-15%
Suture leak	2-16%

Abscess formation	0-9%
Heart problems (myocardial infarction, heart failure)	5%
Ileus	2-4%
Fistula	0.5-4%
Wound dehiscence	2.5-6%
Biliary leak	4.9
Bleeding	0.6%
Re-operation	2-9%
Sepsis	2.5%
Stroke	4%
Death	5-11%

Respiratory complication is the most common post-operatively- this is due to restriction of movements in basal area due to basal atelectasis and orthostic pneumonia

Materials and Methods

MATERIALS AND METHODS

Patients admitted and operated for gastro duodenal perforations **in Coimbatore Medical College Hospital, Coimbatore** are analysed, and followed-up for the period of 6 months

Total number of cases -50

Gastric perforations -14

Duodenal perforations -36

Study period From November 2009 to April 2011

Inclusion criteria

Patient should be inpatient and operated in the Coimbatore Medical College Hospital

Age >20 yrs.

Patient should be operated for gastro- duodenal perforation

Surgery for gastro-duodenal perforation alone included in this study

Simple omental patch closure done for gastro duodenal perforations

Patients followed-up for the period of 6 months

Exclusion criteria

Patients who were not operated in the Coimbatore Medical College Hospital

Age <19 yrs.

Patients who were treated conservatively

Patients who were treated with laparoscopic closure

During surgery unable to find the perforation, or perforation other than gastro duodenal perforation.

The cases are analysed with following factors.

Factors

Age

Sex

Age of perforations(<24/>24 hrs.)

Site of perforations(gastric/Duodenal)

Size of perforations(<1/1-2/>2 cms)

Presence or absence of shock(systolic BP<100 mm hg)

Medical co morbidities- Respiratory infection, DM, SHT and IHD

Patient taking NSAID

Smoker and Consumer of alcohol.

Patients are followed up for the period of 6 months.

Studied the complication, incidence and also the mortality and morbidity associated with these factors and analyzed statistically.

Complication includes

Respiratory complications

Wound infections

Wound dehiscence

Pelvic collection

Enterocutaneous fistula

Deep vein thrombosis

Recurrence

Relaparotomy

Death

During post op period all perforations are given H.pylori eradication for 6 weeks and advicedlife long PPI. After 6 weeks all perforations closure patients will be

followed up with upper GI scopy to look for PUD, decide about definitive procedure as well as the need for life long PPI. All the patients were upper GI scopy found to be normal.

Statistical analysis was made with **Fisher Exact test** to find the significance of proportion of incidence of complication in association with risk factors.

TABLE NO -1
Fisher Exact test

	Class-I	**Class-II**	**Total**
Sample 1	a	b	a+b
Sample2	c	d	c+d
Total	**a+c**	**b+d**	**n**

If there were no systematic association between the variables A and B within the population from which the cell frequencies are randomly drawn, the probability of any particular possible array of cell frequencies, a, b, c, d, given fixed values for the marginal totals a+b, c+d, etc., would be given by the hypergeometric rule

$$\frac{\frac{(a+c)!}{a!c!} \times \frac{(b+d)!}{b!d!}}{\frac{n!}{(a+b)!(c+d)!}}$$

which for computational purposes reduces to

$$\sum p \qquad \frac{(a+b)!(c+d)!(a+c)!(b+d)!}{n!a!b!c!d!} \quad =$$

Also, the degree of disproportion within any array of cell frequencies—in effect, the degree of ostensible association between variables A and B within the sample—can be measured by the absolute difference

Two-tailed probability would be that sum plus the sum of the separate probabilities for the arrays of equal or greater disproportion at the other extreme.

Results and Discussion

RESULTS OF THE STUDY AND DISCUSSION

The patients who were operated for PPU by simple omental patch closure along with peritoneal toileting were taken as a sample for the study and analysed.

Patients with PPU have a typical history of sudden onset of sharp pain usually located in the epigastric area and sometimes referred to shoulder, indicating free air under the diaphragm[24]. Patients may give h/o smoking, alcohol and NSAID ingestion (20%)[1,2,10]. 29% may have h/o of PUD. [1,2,10]

Obliteration or complete absence of liver dullness may be only noted in 37%, so as a diagnostic tool, this has its limitations[30].We found the typical obliteration of liver dullness clinically in 60%of patients

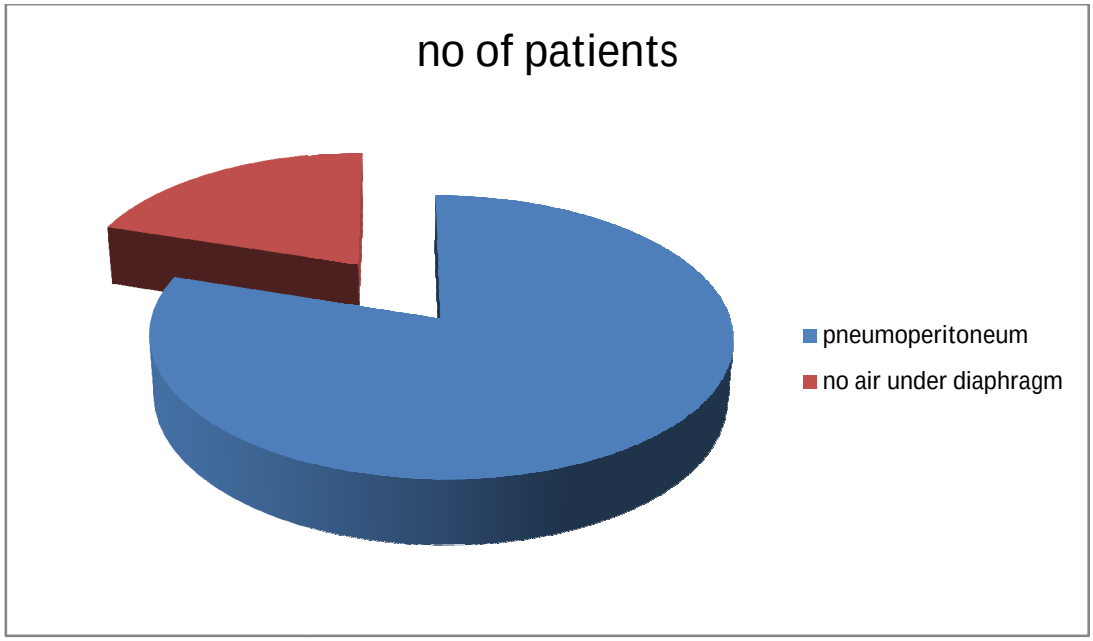

Fig.18 Distribution of air under diaphragm in radiography

X-ray of the abdomen/thorax in standing position will reveal free air under diaphragm in about 80-85 % [30,32.] In our study Chest X ray showed air under diaphragm in 80 % of patients.

We routinely don't go for USG abdomen, and if Xray does not reveals the air under diaphragm and if in strong suspicion of perforative peritonitis, abdominal sonography is done to demonstrate free fluid as well as pneumoperitoneum.

AGE DISTRIBUTION

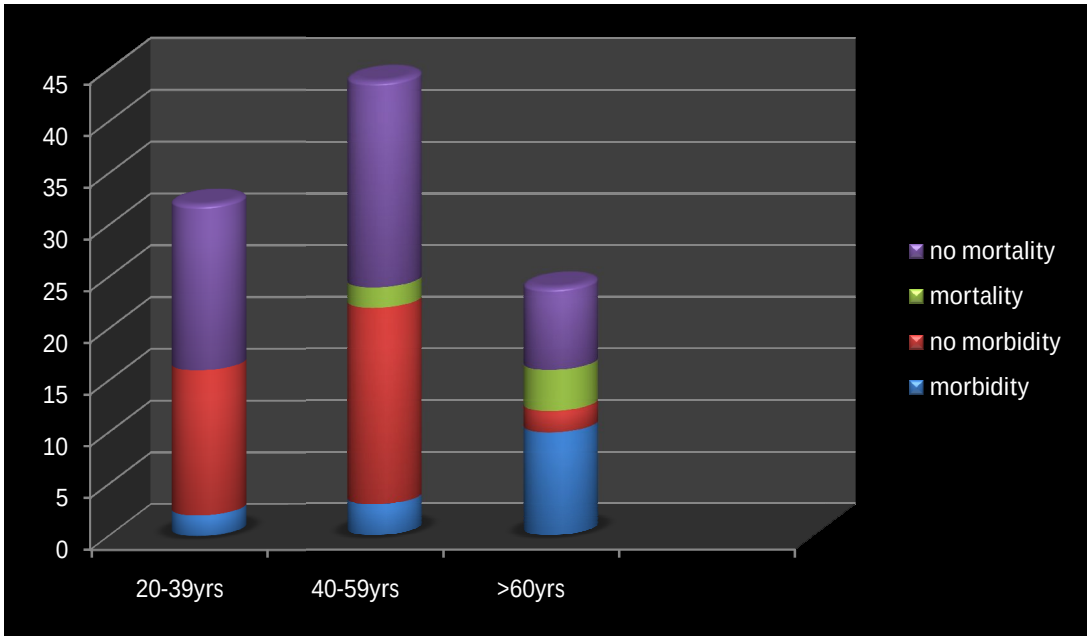

Fig-19. Age Distribution

Though PPU is predominantly a disease of younger age group, its incidence has been increasing with increase in age group. Incidence of PPU commoner between age group of 40-60yrs [2,25].

Mean age of PPU is 48 yrs[2,1,25]. In the study showed 45.34 as mean age.

Moreover it is found in the study, that the age of patient has a significant(P=0.239 &0.5168) effect on the outcome of disease in terms of morbidity and mortality. Age of patient beyond 60 years increases the mortality markedly[60,10]. Mortality rate

following surgery for PPU is 3-5 times higher in the elderly (41%) when compared to the general population (19%).[44,60]

TABLE NO -2

Age group with the outcome

	Morbidity	**No morbidity**	**Mortality**	**No mortality**
<60yrs	12	30	2	36
>60yrs	3	5	4	8

This can be explained by the occurrence of concomitant medical diseases but also by difficulties in making the right diagnosis with a delay of 24 hrs[44].

SEX DISTRIBUTION

PPU is a disorder of male preponderance(80-90%)[1,2].In our study the incidence of PPU, was found to be more commoner in males(92%).

No increase in morbidity and mortality(P=1.0000 &0.6488)with respect to sex of the patients was found in our study (Table-3).

TABLE NO -3

Sex distribution with outcome

	Morbidity	No morbidity	Mortality	No mortality
Male	13	33	6	40
Female	2	2	0	4

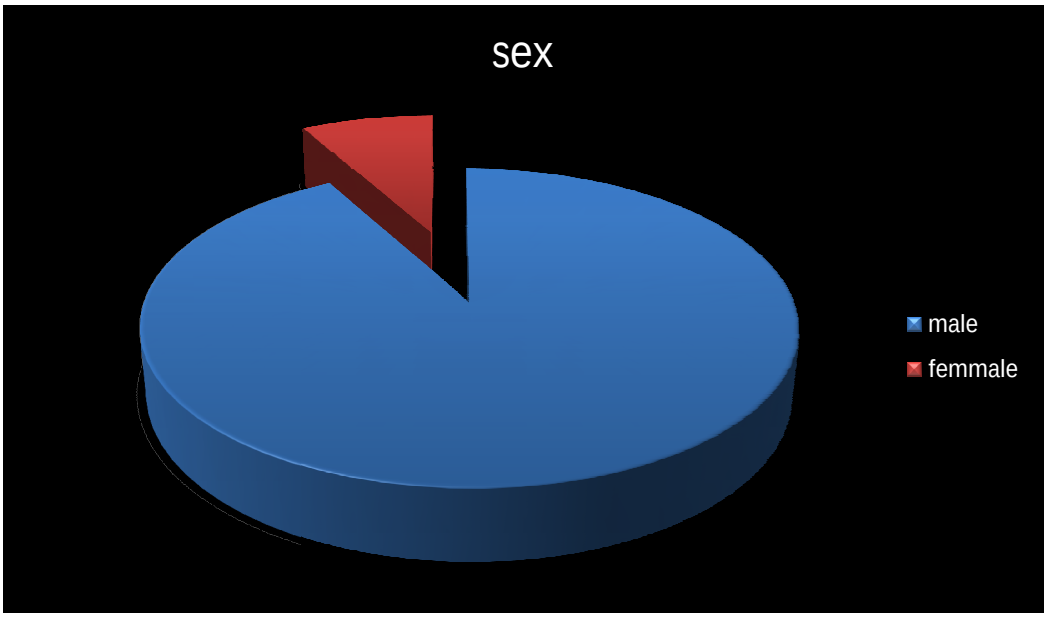

Fig 20.Sex Distribution .

SITE OF PERFORATION

Perforation occurs in 2-10 % of population with PUD of which 60-70% of perforation occurs in duodenum and 10-20% occurs in stomach[2].

TABLE NO -4

Site of perforation with outcome

	Morbidity	No morbidity	Mortality	No mortality
GP	4	10	3	11
DP	11	25	3	33

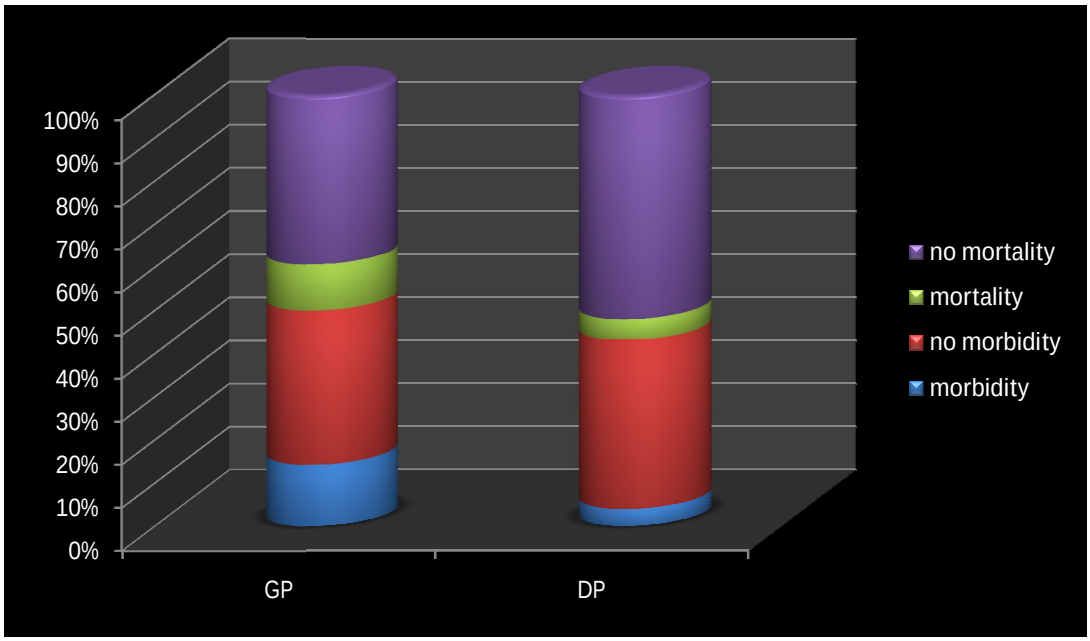

Fig-21 Site of perforation with outcome GP-gastric perforation, DP-duodenal perforation.

The perforation site usually involves the anterior wall of the duodenum (60%), although it may occur in antral (20%) and lesser-curvature gastric ulcers (20%)[27]. Duodenal ulcer is the predominant lesion of the western population, whereas gastric ulcers are more frequent in oriental countries, particularly in Japan. Gastric ulcers are associated with higher mortality and a greater morbidity resulting from hemorrhage, perforation and obstruction[26]. But in this study there is no statistically significant differences noted in the morbidity and mortality among the two groups.

The study showed there is no increase in morbidity and mortality(P=1.000 &0.4491) between the two groups (Table-4).

SIZE OF THE PERFORATIONS

The size of perforations has significant impact in the outcome in gastrodudenal perforations. Greater the size of perforation (giant ulcer >2cms), more is the morbidity and mortality of the disease.

A giant duodenal ulcer is defined as an ulcer more than 2 cm in diameter.[53,54] This ulcer is 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 rates are higher with giant duodenal ulcers than with smaller ulcers, and mortality rates of 8 to 40% have been reported. Fortunately, giant duodenal ulcers are uncommon.

In the past, operative intervention had been used liberally in the management of giant duodenal ulcer[55]. Two studies suggest that the newer medical therapies are more effective in healing duodenal ulcers**.** Simeone et al[**56**] reviewed 75 patients with giant duodenal ulcers during a 5-year period. Eighty-four percent of the patients were successfully managed non operatively with standard modern antiulcer therapies.

TABLE NO - 5

Size of perforation with outcome

	Morbidity	**No morbidity**	**Mortality**	**No mortality**
>2 cms	10	3	4	9
<2 cms	5	32	2	35

In the study it becomes evident that perforation size of >2 cm is associated with increase in morbidity & mortality which is confirmed by test of significance (P=0.0001 &0.0362).

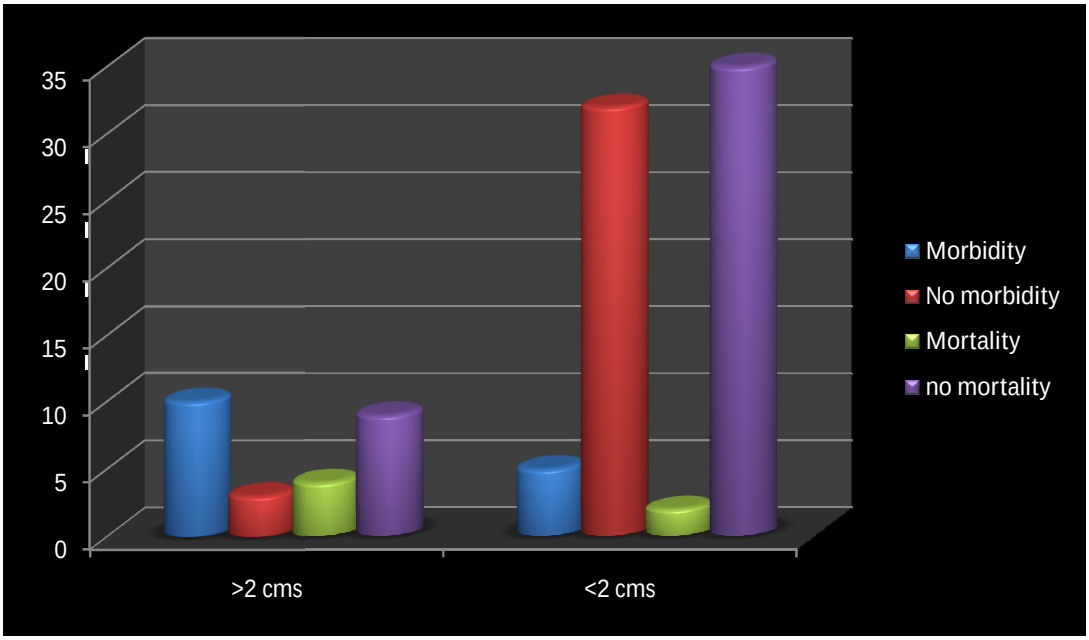

Fig.22 Size of perforation with outcome

Fischer and colleagues [57] reported on 28 patients with giant duodenal ulcers who were treated with omeprazole at a dosage of 40 mg/day. Seventy-one percent of these patients were successfully managed non operatively, with the finding most predictive of failure being an adherent clot or visible vessel at the initial endoscopy. Of the 15 patients with these findings, 47% required an operation, whereas in the remaining 13 patients (without adherent clot or a visible vessel), operation was only necessary in 1 patient (8%).

When ulcer healing fails or an urgent complication occurs, operative intervention is indicated. When possible, truncalvagotomy, antrectomy, and Billroth II reanastomosis should be performed.

Because of the large size of the ulcer and the involvement of the pancreas, resection may not alwaysbe possible. In these circumstances, truncal vagotomy combined with gastrojejunostomy provides a safe, effective alternative.

Based on our study it is evident that the size of perforation>2 cm will adversely affect the outcome(Table-4). There are3 patients with perforation of size>2 cm developed enterocutaneousfistula following surgery for PPU.

AGE OF PERFORATION

Mortality after surgery for perforated peptic ulcer is between 6-10%.[10,27,35,45] There are four main factors which can increase this mortality rate even up to 100%[35].

These are

- age > 60 years,
- delayed treatment (>24hrs)
- shock at admission (systolic BP < 100 mmHg)
- Concomitant disease[27,45].

TABLE NO -6

Age of the perforations with outcome.

	Morbidity	**No morbidity**	**Mortality**	**No mortality**
>24 hrs	10	10	5	15
<24 hrs	5	25	1	29

If patient presented with >24 hrs, the mortality and morbidity is increasing. This may be due to advancement of the disease and this is the adverse prognostic indicators[27,35,45].

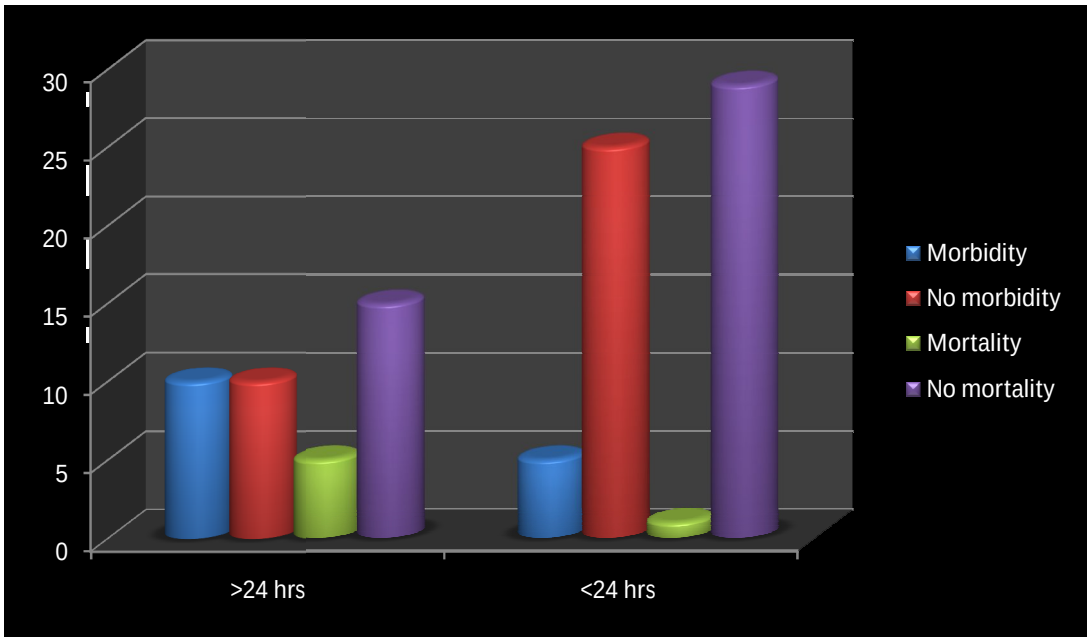

Fig23.Age of the perforations with outcome

In our study also showed the delayed presentation >24hrs affect the outcome of the disease adversely(P=0.0277 & 0.0341)(Table-6).

Influence of concomitant medical disease

The co-existent medical illness also plays a vital role in the prognosis of PPU , as in any other disease. As already mentioned, even one of the four risk factors is associated with high mortality[27,35,45].

TABLE NO -7

Concomitant medical illness with outcome

	Morbidity	**No morbidity**	**Mortality**	**No mortality**
Co morbidity	7	4	5	6
No co morbidity	8	31	1	38

The medical co morbid condition which adversely affects the outcome of disease are- SHT, DM, IHD, Respiratory infection and Anesthetic risk grade(ASA >II).

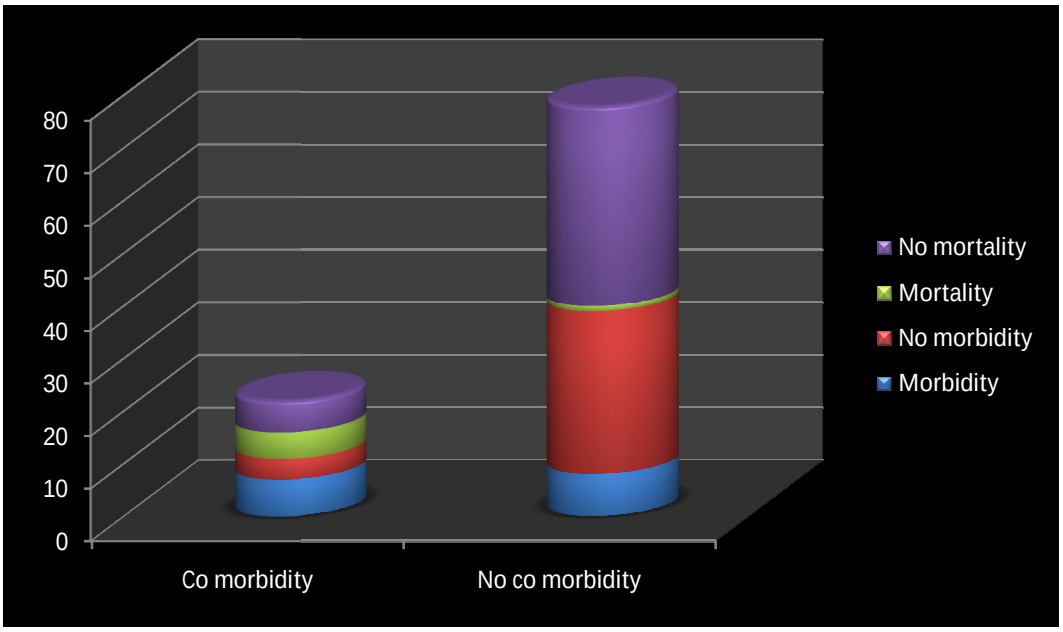

Fig.-24. Concomitant medical illness with outcome

In our study of 50 patients, the following conditions are taken into considerations and were analysed with the outcome of disease[1,2,10].

- Systemic HT
- Uncontrolled DM
- Respiratory tract infections
- Ischemic heart disease
- American society of Anaesthesiologist risk grade –III & IV

With above said co morbidities taken into consideration, it is found to have significant(P=0.0114&0.0012) effect in the outcome of the diseases mentioned below (table-6)

- Delay in recovery from anaesthesia
- Wound dehiscence & SSI
- Requiring supportive measures to maintain stable hemodynamic status
- Respiratory complication-pneumonia, ARDS.
- Increased days of hospital stay.

SMOKING AND OUTCOME OF DISEASE

Smoking has been associated with type-I &II gastro-duodenal ulcer[10]. Epidemiologic studies suggest that smokers are about twice as likely to develop PUD as nonsmokers[1].

Smoking increases gastric acid secretion and duodenogastric reflux. Smoking decreases both gastroduodenal prostaglandin production and pancreaticoduodenal bicarbonate production -the observed association between smoking and PUD[2].

Although difficult to measure, both physiologic and psychologic stress undoubtedly play a role in the development of peptic ulcer in some patients. [2]

TABLE NO - 8

Smoking with its outcome.

	No Morbidity	**Morbidity**	**No mortality**	**Mortality**
Smoker	8	12	15	5
Nonsmoker	27	3	29	1

Smoking is believed to be one of the most important etiological factor in the development of peptic ulcers especially in the young and increases the risk tenfold in both men and women[2]. It is estimated that smoking may account for 77% of all ulcer perforations in those younger than 75 years, whereas in the older population, smoking is of much less importance[2].

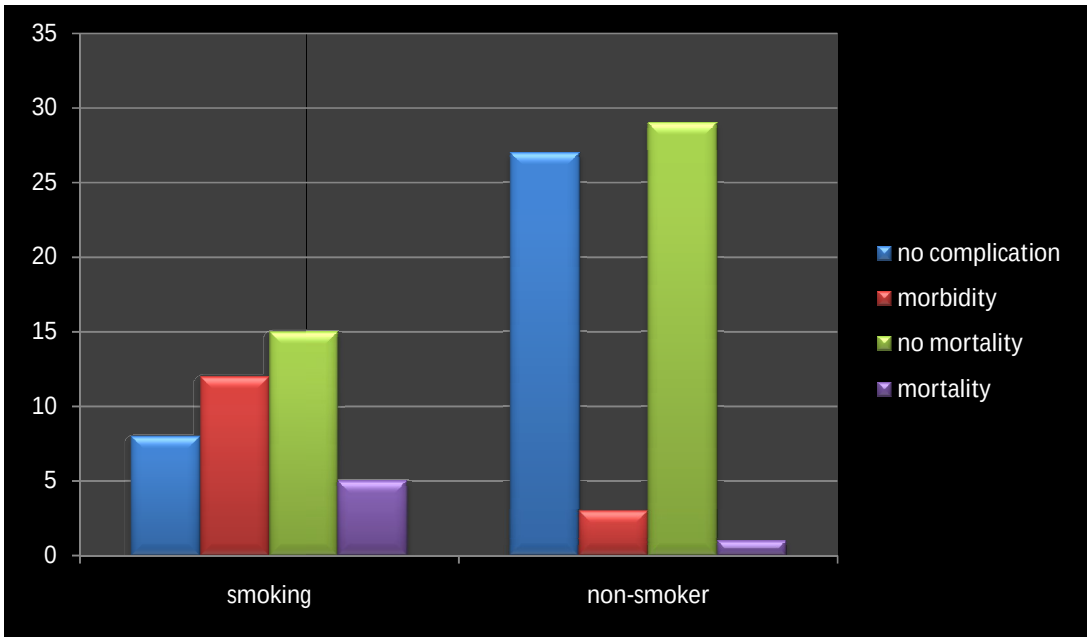

Fig25.smoking with outcome

In our study smoking significantly (P=0.0003& 0.0317) increases morbidity and mortality. It may has a synergistic effect with NSAID and increases the chance of perforation .

ALCOHOL CONSUMPTION

In our study we found that there is no significantly(P=1.000& 0.5142) increased morbidity &mortality found in relation with alcohol consumption. Even though alcohol is considered as a constant risk factor for Perforation, statistically proven data is still lacking.

TABLE NO -9

Alcohol and outcome

	No morbidity	**Morbidity**	**No mortality**	**Mortality**
Alcoholic h/o(+)	6	10	2	14
Alcoholic h/o(-)	9	25	4	30

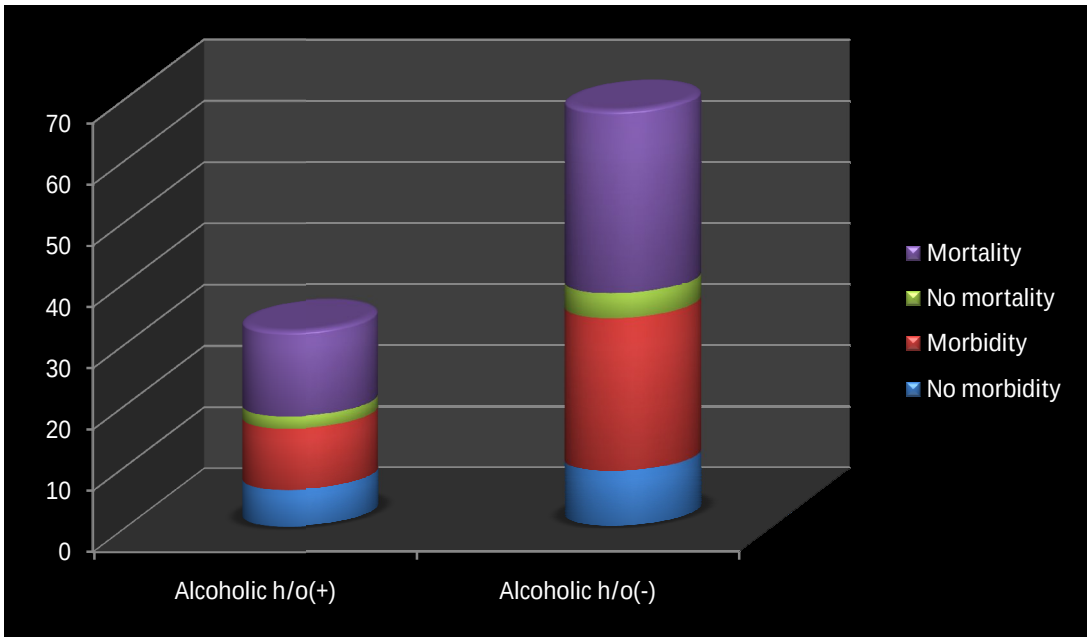

Fig.26. Alcohol and outcome

NSAID INGESTION

NSAIDs represent a group of the most commonly used medications in the United States. More than 30 billion over-the-counter tablets and over 100 million prescriptions are sold yearly in the United States alone. In fact, after the introduction of COX-2 inhibitors in the year 2000, the number of prescriptions written for NSAIDs was >111 million at a cost of $4.8 billion[1,2].

Side effects and complications due to NSAIDs are considered the most common drug-related toxicities in the United States[2.1]. The spectrum of NSAID-induced morbidity ranges from nausea and dyspepsia (prevalence reported as high as 50–60%) to a serious gastrointestinal complication such as endoscopy-documented peptic ulceration (15–30% of individuals taking NSAIDs regularly)[2] complicated by bleeding or perforation in as many as 1.5% of users per year[2,1,10] . About 20,000 patients die each year from serious gastrointestinal complications fromNSAID[1,2]. Over 80% of patients with serious NSAID-related complications did not have preceding dyspepsia.

In our study, 12% of patients gave the h/o NSAID ingestion.

TABLE NO -10

NSAID intake &outcome

	Morbidity	No morbidity	Mortality	No mortality
H/o NSAID intake(+)	2	4	1	5
H/o NSAID intake(-)	13	31	5	41

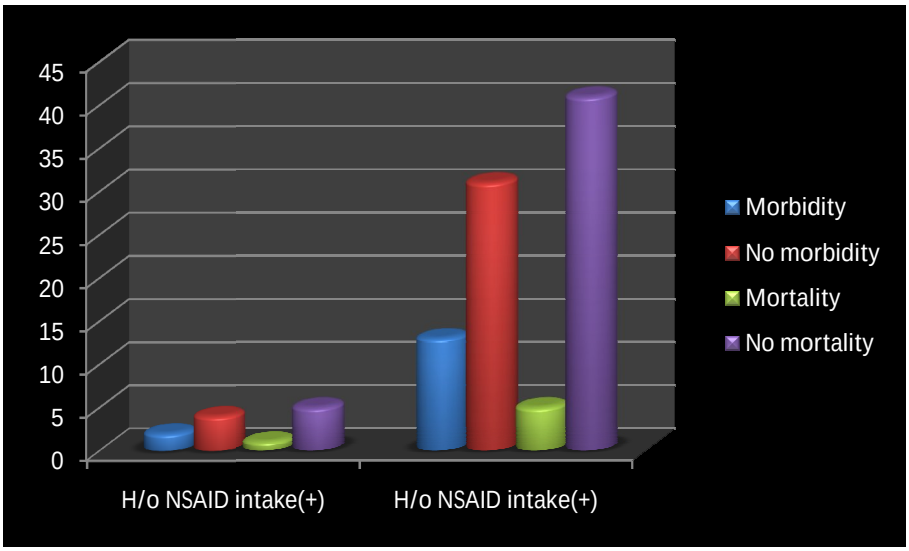

Fig.27. NSAID intake & outcome

In view of the lack of warning signs, it is important to identify patients who are at increased risk for morbidity and mortality related to NSAID usage. Even 75 mg/d of aspirin may lead to serious gastrointestinal ulceration; thus, no dose of NSAID is completely safe[1].

Established risk factors include advanced age, history of ulcer, concomitant use of glucocorticoids, high-dose NSAIDs, multiple NSAIDs, concomitant use of anticoagulants, and serious or multisystem disease[1].

In our study the there is no statistical difference (P=1.000 & P=0.5399) noted in the outcome of disease between the two groups-who had the NSAID and who had not

PPU PATIENT PRESENTED WITH SHOCK

A PPU patient presenting with shock indicates the patient is progressed to advanced stage of peritonitis. As soon as the patient got admitted, we resuscitate with large volume of crystalloids and vigorously correct the renal parameter if the patient is in shock

TABLE NO -11

Initial shock and out come

Initial shock	Morbidity	No morbidity	Mortality	No mortality
Present	8	22	5	21
Absent	7	13	1	29

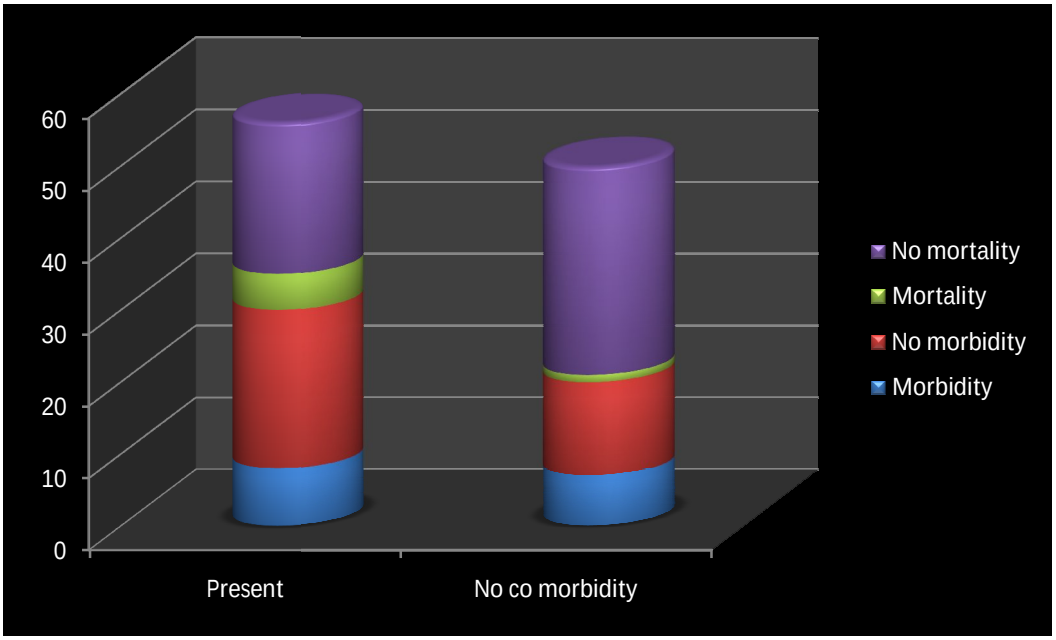

Fig28. Initial shock with outcome

Presentation in shock is one of the prime risk factor predicting the outcome of disease and even it may increase the mortality to 100%[35,10,45].

In our study a patient admitted with shock had adverse prognosis. There is statistically significance(P=0.0704&P=0.5467) noted that the patient who presented with shock at the time of admission had higher morbidity and mortality.

Postoperative complications The postoperative complication which most commonly observed was pneumonia, followed by wound infection. An overview of all complications and their incidences listed below[25,27,32,35,40,41,42,43,46,47,48]

Pneumonia	3.6-30%
Wound infection	10-17%
Urinary tract infection	1.4-15%
Suture leak	2-16%
Abscess formation	0-9%
Heart problems (myocardial infarction, heart failure)	5%
Ileus	2-4%
Fistula	0.5-4%
Wound dehiscence	2.5-6%
Biliary leak	4.9%
Bleeding	0.6%
Re-operation	2-9%
Sepsis	2.5%
Stroke	4%
Death	5-11%

TABLE NO -12

Incidence of complications

Complications	Percentage in the study(%)
Respiratory complication	30
Wound infection	20
Wound dehiscence	2
Pelvic collection	2
Enterocutaneous fistula	6
Relaparotomy	4
Death	12

Respiratory complication is the most common complication noted post operatively[25,27,32,35,40,41-48], this may be due to restriction of movements in basal area,synpneumonic effusion and due to bedridden post-op period causing orthostatic pneumonia.

Wound infection is a next factor causing morbidity to the patient. In our study the complication listed were -wound dehiscence , pelvic abscess, bilious leak and death.

Incidenceof complications in our study were listed below in the table(table-12)

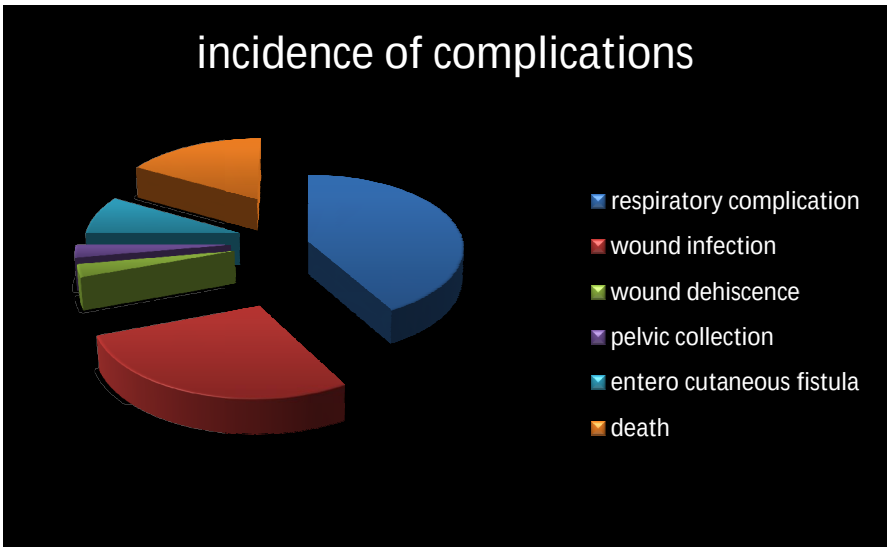

Fig 29 Incidence of complications

In our study burst abdomen (wound dehiscence-2%) patients were treated surgically with tension suturing and the patient improved.(Fig.30.)

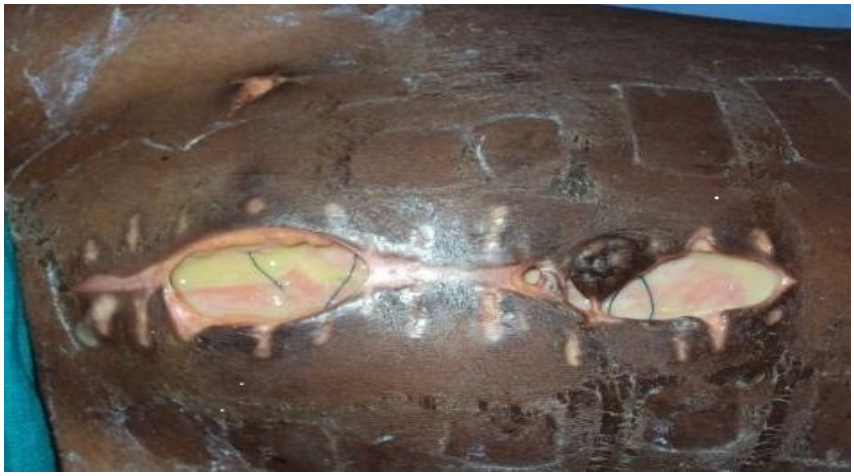

Fig.30.Wound dehiscence

In our study we came across **Entero cutaneous fistula** in 3 patients and 2 patients we done relaparotomy and had 100% mortality. Another one patient we managed conservatively was improved.

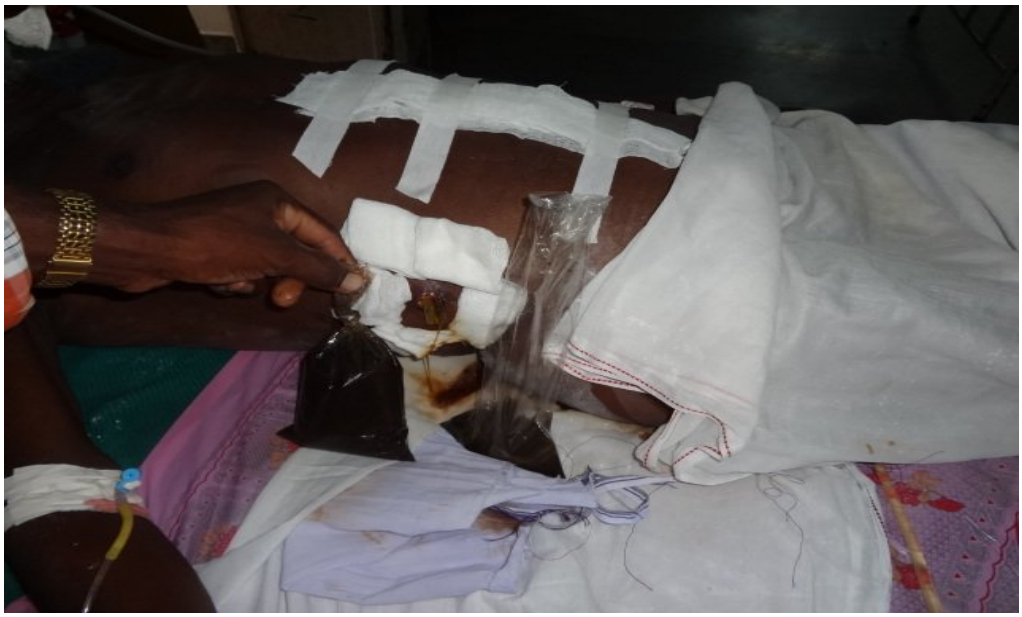

Fig.31 bilious leak following surgery

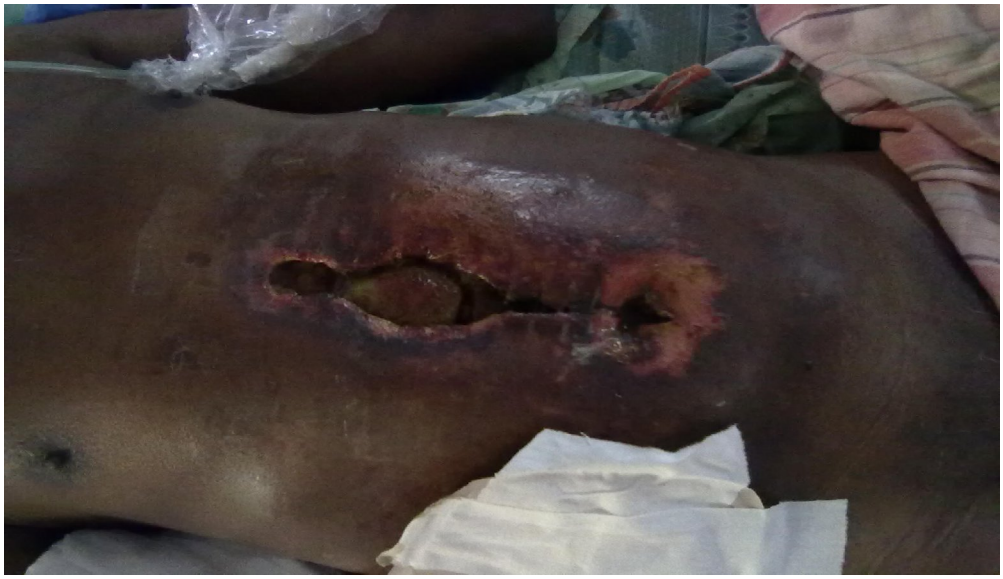

Fig.32.Giant gastric ulcer perforation-primary closure done with wound dehiscence and Entero Cutaneous Fistula

(Fig31)Picture shows that following laparotomy and simple omental patch closure for duodenal ulcer perforation, and post-operatively patient developed bilious leak and with conservative treatment patient improved well.

(Fig32)Another case of giant duodenal ulcer perforation was operated simple omental patch closure, and patient developed entero cutaneous fistula and wound dehiscence. Patient was taken up for relaparotomy and post operatively died of respiratory complication.

We found **pelvic collection** in only 2% of patients. We were routinely using either tube drain or corrugated drain post operatively and removed usually on 4^{th} -6^{th} post-op day.

Hence we ambulate the patients on the day of surgery or the next day, We never came across **deep vein thrombosis (**DVT**)** in the post op period. As well as routinely we were not advocating the practice of giving DVT prophylaxis.

ASSCIATION OF RISK FACTORS TO POST OPERATIVE COMPLICATION

In our study the following statistical significant risk factors were found :

- Age >60
- Presented late (>24 hrs) ,
- Size of perforation >2 cms
- Concomitant medical disease
- Presented with shock

Among these- age, medical co morbidity, delayed presentation , presented with shock is the important risk factor which can individually rise the mortality upto 100%.[25,27,32,40-48]

In our study also found that the following factors statistically increases the morbidity& mortality.(table-11)

TABLE NO -11

Demographic values of association between factors and outcome

Factors	Value		P value
Age	<60yrs	Morbidity	P=0.5168
	>60yrs	Mortality	P=0.0239
Sex	M 92%	Morbidity	P=0.6488
	F8%	Mortality	P=1.0000
Site of perforations	Gp-4	Morbidity	P=1.0000
	Dp-11	Mortality	P=0.4494
Age of perforations	<24hrs	Morbidity	P=0.0277
	>24hrs	Mortality	P=0.0341
Size of perforations	>2cms	Morbidity	P=<0.0001
	<2 cms	Mortality	P=0.0362
Smoker	Yes	Morbidity	P=<0.0003
	No	Mortality	P=0.0317
Comorbidity	Present)	Morbidity	P=0.0114
	Absent	Mortality	P=0.0012
Alcohol	H/o present	Morbidity	P=0.5142
	H/o absent	Mortality	P=1.0000
NSAID	H/o (+)	Morbidity	P=1.0000
	H/o (-)	Mortality	P=0.5399
Initial shock	Present	Morbidity	P=0.5467
	absent	Mortality	P=0.0704

Mortality in our study was 12%.

Table-19 comparison with other series

Risk factors influencing outcome mortality after surgery for perforated peptic ulcer is between 6-10%[35,27,4,25,25,29,42]. There are four main factors which can increase this mortality rate even up to 100%. These are age > 60 years, delayed treatment (>24hrs), shock at admission (systolic BP < 100 mmHg) and concomitant medical diseases . Also gastric ulcers are associated with a two- to threefold increased mortality risk[26] .

TABLE NO -12

Comparisons of mortality with other series

Author	Year	Mortality rate(%)
Swayer	1977	6.7
M.C.Dandapat	1991	10.5%
Schwartz's	9th edition	10-40%
Maingot's	11th edition	19%(40% in elderly)[60]
Present series	2011	12%

In our study also showed the delayed presentation >24hrs affect the outcome of the disease adversely (Table-6).

Conclusion

CONCLUSION

In our study we studied the 50 patients who operated for gastroduodenal perforations are included and analysed the risk factors which adversely affect the outcome of disease

1. In patients with gastro-duodenal perforations 20% not showed the pneumoperitoneum in X ray, and CT scan may helpful in those situations which is more sensitive in finding the pneumoperitoneum
2. Patients age is the important determinant of the outcome.
3. Mean age of PPU is 45.34
4. The gastro-duodenal perforations is the disease of the male preponderance, but the incidence in female is increasing.
5. Gastric perforations has the poor outcome ,but in our study not showed significance.

In our study we found the following factors were significantly increases the morbidity and mortality.

- Age >60 yrs
- Size of perforations >2 cms
- Delayed presentation >24 hrs
- Patient presented with shock (BP<100 mm of Hg)
- Concominant medical illness
- Smoking

The following factors were not significantly affect the outcome of disease in view of mortality and morbidity, in our study.

- Sex of the patient.
- Site of perforation
- H/o alcohol consumption
- H/o NSAID ingesion

Hence there is most of the gastro-duodenal ulcer s are due to H.pylori, H.pylori eradication in PUD has important role. We are treating every gastroduodenal perforations post-operatively with H.pylori eradication and life long PPI, we never found ulcer recurrence, or other complications of PUD.

Annexures

PROFORMA

Name :

Age /sex :

Occupation :

Address :

Chief complaints :

Abdominal pain –duration-

H/o presenting illness:

Past h/o

H/o SHT, DM, Respiratory tract infection, Ischemic heart disease

H/o PUD

Personal h/o

Smoker

Alcoholic

H/o NSAID ingestion

Family h/o

Drug h/o

General Examination

Consciousness

Oriented or not

Hydration- dehydration (moderate, severe dehydration)

Pallor

Cyanosis

Clubbing

Icterus

Lymphadenopathy

Edema

Vital signs

Pulse :

BP (<100mm of hg />100 mmof hg)

Peripheries cold and clammy- yes /no

Abdominal examination

Soft/tenderness

Guarding +/-

Rigidity +/-

Free fluid +/-

Liver dullness obliterated or not

Cardio vascular systems

Respiratory systems.

Central nervous system.

Chest X ray : air under diaphragm +/-

USG abdomen :

CT abdomen :

ECG :

Provisional diagnosis:

Treatment:

Surgery:

- Simple omental patch closure.
- Omental patch closure with gastro duodenostomy
- Omental patch closure with feeding jejunostomy

Complications	Respiratory complications

- Wound infection
- Wound dehiscence
- Pelvic abscess
- Entero cutaneous fistula
- DVT, Death

CONSENT FORM

TITLE OF PROJECT: A CLINICAL STUDY ON FACTORS INFLUENCIING OUTCOME OF DISEASE IN GASTRO- DUODENAL PERFORATIONS

GUIDE: Prof. Dr.Natarajan.MS

P. G. STUDENT: Dr. Rajeswaran. A

PURPOSE OF RESEARCH:

I have been informed that this study will analyse the risk factors which may affect the outcome adversely following gastro duodenal perforations. This study will thus help the investigator better understand for the management of above condition.

PROCEDURE:

I am aware that in addition to the ordinary post operative care received, I will be examined and asked a series of questions by the investigator. I have been asked to undergo the necessary investigations, which would help the investigator as a part of routine management.

RISKS AND DISCOMFORTS:

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

BENEFITS:

I understand that my participation in the study will have no direct benefit to me other than potential benefit of the treatment.

CONFIDENTIALITY:

I understand the medical information produced by this study will become part of my hospital record and will be subject to the confidentiality. Information of sensitive personal nature will not be part of the medical record, but will be stored in the investigator's research file. If the data are used for publication in the medical literature or for teaching purpose, no names will be used and other identifiers, such as photographs will be used only with my special written permission. I understand that I may see the photographs before giving the permission.

REQUEST FOR MORE INFORMATION:

I understand that I may ask more questions about the study at any time and Dr. Rajeswaran at the department of Surgery is available to answer my questions or

concerns. I understand that I will be informed of any significant new findings discovered during the course of the study, which might influence my continued participation. A copy of this consent form will be given to me to keep for careful reading.

REFUSAL OR WITHDRAWAL OF PARTICIPATION:

I understand that my participation is voluntary and that I may refuse to participate or may withdraw consent and discontinue participation in the study as any time without prejudice. I also understand that Dr. Rajeswaran may terminate my participation in this study at any time after he has explained the reasons for doing so.

INJURY STATEMENT:

I understand that in the unlikely event of injury to me resulting directly from my participation in this study, if such injury were reported promptly then appropriate treatment would be available to me. But no further compensation would be provided by the hospital. I understand that by my agreement to participate in this study and not waiving any of my legal rights. I confirm that Dr.Rajeswaran has explained to me the purpose of the research, the study procedure that I will undergo and the possible risks and discomforts as well as benefits that I may experience in my own language. I have been explained all the above in detail in my own language and I understand the same. Therefore I agree to give my consent to participate as a subject in this research project.

_____ _____

(Witness to signature) (Date) (Participant) (Date)

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

_____ _____

(Investigator) (Date)

MASTER CHART

Name	Age	Sex	Ipno.	Diagnosis GP/DP	Procedure-omental patch-OP	1NSAID 2Smoking 3Alcohol	<24/> 24 Hrs-	Shock Present Y/N	comorbidity	Size of Perforat-cms	Complication
Subban	65	M	35882	Malignant GP	GJ+OP	-	>24	Y	SHT	>2	Rc, death
vijayakumar	48	M	37498	DuP	OP	1,3	<24	N	-	<1	nil
Rayappan	64	M	42955	Gp	Op	2,3	>24	Y	LRTI	>2	Ecf,Wi,rc death
Raja	23	M	42989	DP	Op	2	<24	N	-	1-2	nil
Kumar	27	M	42987	Gp	Op	-	<24	N	-	<1	NIL
Backyam	60	F		Dp	Op	-	>24	N	-a	<1	RC
	60	M	52480	Dp	Op	2,3	<24	N	-	<1	Nil
Kanagaraj	5o	M	52579	Dp	Op	2,3	>24	Y	SHT ,Dm	1-2	WI,RC,PA
Sekar	58	M	61393	DP	Op	2	>24	N		1-2	RC,WI
Kittusamy	56	M		Dp	op	2,3	>24	Y	DM, IHD	>2	ECF,WI, death
Gandhi	45	M	51608	Ca-stomach On ct-Gp	Op+feedingjejunostomy	-	>24	y	SHT ,IHD	>2	ECF,WI ,death

Murali	22	M	50147	Dp	Op	2,3	<24	N		<1	Nil
Muthu	60	M	52480	Gp	Op	3	<24	N		<1	nil
Sengotayyan	40	M	44477	Gp	AGJ+op	2,3	<24	Y	LRTI	>2	nil
Velusamy	37	M	43887	Dp	Op	1	>24	Y	BA	>2	Wd
Mohammad kani	62	M	43203	Dp	Op	2,3	<24	N	-	1-2	RC,WD
Rajeev	55	M		Gp	Op	1,2	>24	Y	-	>2	Rc,Wi
Govindhan	55	M	60823	Stab-gp	Prim.clo	2	<24	N	-	>2	Rc, Wi
Nachi	60	M	67687	Dp	Op	2,3	<24	N	-	1-2	Nil
Ramachandr an	484	M	70644	Dp	Op	3	<24	N	-	1-2	nil
Marappan	58	M	3872	Gp	Op	2	<24	n	-	<1	nil
Veerasamy	25	M	3872	Dp	Op	1	<24	y	-	1-2	death
Murugesan	45	M	3972	Dp	Op	2	<24	N	-	<1	Nil
Ganesan	32	M	8342	Dp	Op	2	<24	N	-	<1	Nil
Selvam	60	M	8422	Dp	Op	2	<24	Y	-	>2	Wi,Rc
Nagendren	49	M	13321	Dp	Op	1	<24	N	-	<1	Nil
Muruganand ham	42	M	13341	Dp	Op	2,3	<24	N	LRTI	<1	Rc
Manikandan	42	M	14421	Dp	Op	-2	<24	Y	LRTI	>2	nil
Aanandhan	40	M	14431	Gp	Op	-	<24	Y	-	<1	Nil
Chinnamal	56	F	14444	Dp	Op	2	>24	Y	-	1-2	Rc,WI,Pa
Pappathy	34	F	14456	Dp	Op	-	<24	N	-	1-2	nil
Vnodkumar	26	M	8383	Dp	Op	1	>24	Y	-	1-2	Nil
Thangamani	42	F	8447	Gp	Op	-	>24	Y	-	>2	Nil
Dhandapani	49	M	71320	Dp	Op	2	<24	N	-	1-2	Nil
Nachiappan	45	M	1281	Dp	Op	2	<24	Y		1-2	Nil

Palaniappan	75	M	1310	Dp	Op	2	<24	N	SHT	1-2	Rc,Death
Nagaraj	50	M	31743	dp	op	3	<24	Y	DM	1-2	Nil
Selvaraj	30	M	6660	Gp	Op	3	<24	Y	-	1-2	Nil
Loganathan		M	27134	Dp	Op	3	<24	N	-	<1	Nil
Krishnamoorthi	27	M	64418	Dp	Op	-	<24	N	-	1-2	Nil
Saravanan	40	M	10242	Dp	Op	-	>24	Y	-	1-2	Nil
Sandeep	35	M	15629	Dp	Op		>24	N	-	<1	Nil
Kamaraj	45	M	32500	Gp	Op		>24	Y	-	1-2	Nil
Perumal	43	M	33709	Dp	Op		<24	Y	-	1-2	Nil
Vijay	50	M	53271	Dp	Op	2,3	<24	N	-	>2	Rc,wi
Rathinasamy	51	M	63864	Gp	Op	-	>24	N	-	1-2	Nil
Mariappan	34	M	14889	Dp	Op	-	>24	N	-	<1	Nil
Karthik	24	M	60348	Dp	Op	-	>24	N	-	1-2	Nil
Manoharan	51	M	33127	Dp	Op	-	>24	Y	-	1-2	Nil
Bagavathirao	42	M	46217	Dp	Op	2	>24	Y	-	<1	Rc

Table-I master chart.
Key words: Dp-duodenal perforation;Gp-Gastric perforation;
Op-omental patch closure;
1-H/o NSAID ingestion; 2-Smoker; 3-Consumer of alcohol;
Y-shock present; N-shock absent;
Wi-Wound infection;
Rc- Respiratory complication;
Wd-Wounddehiscence;
Ecf-Entero cutaneous fistula;
PA-pelvic abscess

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