STUDY TO COMPARE THE EFFICACY OF FREE OMENTAL PATCH CLOSURE VERSUS GRAHAMS LIVE OMENTAL PATCH CLOSURE IN PATIENTS WITH DUODENAL PERFORATION

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DECLARATION

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This dissertation is submitted to the Tamilnadu DR MGR Medical University,Chennai in partial fulfillment of the university regulations for the award of M.S degree(General Surgery),branch-1 examination to be held in April 2015.

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CERTIFICATE

*This is to certify that the dissertation entitled "***STUDY TO COMPARE THE EFFICACY OF FREE OMENTAL PATCH CLOSURE VERSUS GRAHAMS LIVE OMENTAL PATCH CLOSURE IN PATIENTS WITH DUODENAL PERFORATION***" is a bona fide work done by DR.Renganathan.M post graduate(2012-2015) in the department of general surgery Govt.Stanley Medical College and hospital,Chennai under my direct guidance and supervision, in partial fulfillment of the regulations of the Tamilnadu Dr.MGR Medical University Chennai for the award of M.S degree(General surgery) Branch-1 examination to be held in April 2015*

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ABSTRACT

Duodenal ulcer perforations are most common cause of peritonitis. For almost a century, duodenal perforations have been closed by Omentopexy. In this, pedicled omentum is mobilised over the perforation and secured with full thickness sutures placed on either side of the perforation. this is the "gold standard" for the treatment of duodenal perforations. However, occasionally we have to come across large perforations of the duodenum, in such cases there is possibility of post-operative leakage following closure by this method. Usually, duodenal ulcer perforations are less than 1 cm in greatest diameter, and as such, are amenable to closure by omentopexy.Most of the surgeons feel that mobilization of the pedicled omentum from the colon, and placement of full thickness sutures into the normal duodenum around the perforation makes the efficiency of omental patch safe even in the presence of large sized perforations. However there is controversial evidence from some of the studies conducted which proved free omental graft superior to pedicled omental graft. Moreover there is evidence from studies which concluded that a Free Omental Plug can be used safely and reliably to treat large duodenal perforations that are more than 25 mm in size.

Objective:

To compare the efficacy of Free Omental Graft with GRAHAMS live Omental Graft in patients with duodenal ulcer perforations of size up to 20 mm.

Methodology:

A series of 60 cases of duodenal perforations were studied and analyzed. Among them 30 patients underwent closure of duodenal perforation by Grahams Pedicled Omental Patching and 30 patients underwent Free Omental Patching. The cases were followed up for 1 month. The results were analysed and the two groups were compared with post-operative leak rates, post-operative hospital stay, complications & mortality.

Results:

In this study we found 26.66% of post-operative leak (8 patients), 60% of wound infection (18 patients) in patients treated with Free Omental Patch and 6.66% of post-operative leak (2 patients), 33.33% wound infection (10 patients) in patients treated with Grahams live Omental Patch. we found 13.33% mortality(4 patients) in patients treated with Free Omental Patch and no mortality in patients treated with Pedicled Omental Patch. However the mortality rate was statistically insignificant. The average hospital stay in our series was 11.93 days for Grahams live Omental Patch and 17.03 days for Free Omental patch.

Conclusion:

Grahams live omental Patching was found to be a superior surgical technique over Free Omental Patching for the closure of duodenal perforations measuring upto 20 mm.

Key words: Duodenal perforation, Grahams live omental graft, Free Omental graft.

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INTRODUCTION

Despite the widespread use of gastric antisecretory agents and eradication therapy, incidence of perforated peptic ulcer has changed little.It is one of the most easily diagnosed acute abdominal conditions, provided that the symptoms are known and appreciated. Many surgeries have been proposed to compensate for duodenal perforation.But none of the surgeries is accepted as the best to solve the problem.The reasons cited for disruption of duodenal closure are high intra luminal pressure,tendency of duodenal mucosa to protrude through closures, break down from autodigestive enzymes of pancreas and bile.

A variety of surgical techniques have been advocated for the management of peptic perforation. However these are not without drawbacks, particularly in managing large perforations,late presentation,advanced age,etc..mortality rates of upto 18% have been reported while managing patients with risk factors by standard techniques.Thus there is a need to find,evaluate and apply methods of managing the catastrophes.

AIM OF THE STUDY

To compare the efficacy of Free Omental Graft against Pedicled Omental Graft in patients with duodenal ulcer perforations of size up to 20 mm.

REVIEW OF LITERATURE

HISTORY:

Galen (AD 131-201) and Aegineta (AD 625-690) were aware of peptic ulcer disease and described its complications[1].

Perforated peptic ulcers as a disease entity has been known since 1670[2].

1726 – George Hamberger, Germany described a duodenal ulcer[2].

1793 – Jacopo Penada, Italy, first recorded a duodenal perforation[2].

1880 – Miculicz reported the first operative attempt to close the perforation[1].

1886 – Heineke, did the first pyloroplasty[3].

1888 – Mikulicz redefined the pyloroplasty by Heineke[3].

1891 – Heusen achieved the first successful operative closure of perforated ulcers[2].

1893 – Codivilla reportedly did the first gastrojejunostomy for a duodenal ulcer[3].

1896 – Bennet suggested sealing a large perforation with omentum[2].

1902 – Finney reported the second type of pyloroplasty[3].

1929 – Cellan-Jones and Graham in 1937 emphasized the utility of an omental patch for the treatment of perforated peptic ulcer[4, 5].

1997 – Raj BR et al found that reliability of the omental plug closure of large duodenal perforation[6].

2000 – Sharma D et al found that, the omental plug is a simpler procedure in an extremely large defect of duodenal perforation which cannot be closed by simple technique[7].

2005 – Lam PWF et al concluded that “three stitch” laparoscopic Graham’s patch repair for duodenal perforation was safe and efficient, and might be the choice for laparoscopic repair relatively large perforations[8].

2006 – KalpeshJani, A.K. Saxena suggested that omental plugging can be safely performed in managing duodenal peptic perforation[9].

ANATOMY

The duodenum is about 20-25cm long, is the shortest, widest and most fixed part of the small intestine. The proximal 2.5cm of the duodenum is intraperitoneal and remainder is retroperitoneal. It extends from the pylorus to the duodeno-jejunal flexure, making a 'C' shaped curve, which embraces the head of the pancreas. It lies between L_1 and L_3 entirely above the level of the umbilicus. It has four parts[10]

1. First (superior) part.
2. Second (descending) part.
3. Third (horizontal) part.
4. Fourth (ascending) part.

First (superior) part:

The first part of the duodenum is one of the commonest sites for peptic ulcer, possibly because of direct exposure of this part to the acidic contents reaching it from the stomach.

The patient is usually an overbusy young person with a tense temperament. The ulcer pain located at the right half of epigastrium is relieved by meals and reappears on an empty stomach.

The first part of duodenum is overlapped by the liver and gall bladder, either of which may become adherent to, or even ulcerated by a duodenal ulcer.

The first part begins at the pylorus, and passes backwards, upwards and to the right to meet the second part at the superior duodenal flexure. Its relations are as follows.

Peritoneal Relations

1. The proximal 2.5 cm is movable. It is attached to the lesser omentum above, and to the greater omentum below.
2. The distal 2.5 cm is fixed. It is retroperitoneal. It is covered with peritoneum only on its anterior aspect.

Visceral Relations

Anteriorly:

Quadrate lobe of liver, and gall bladder .

Posteriorly.

Gastroduodenal artery, bile duct and portal vein.

***Superiorly* :**

Epiploic foramen.

***Interiorly* :**

Head and neck of the pancreas

Second Part

Course

This part is about 7.5 cm long. It begins at the superior duodenal flexure, passes downwards to reach the lower border of the third lumbar vertebra, where it curves towards the left at the inferior duodenal flexure, to become continuous with the third part. Its relations are as follows.

Peritoneal Relations

It is retroperitoneal and fixed. Its anterior surface is covered with peritoneum, except near the middle, where it is directly related to the colon.

Visceral Relations

Anteriorly :

(a) Right lobe of the liver;

(b) transverse colon,

(c) root of the transverse mesocolon,

(d) small intestine

***Posteriorly*:**

(a) Anterior surface of the right kidney near the medial border,

(b) right renal vessels,

(c) right edge of the inferior vena cava,

(d) right psoas major.

***Medially* :**

(a) Head of the pancreas and

(b) the bile duct .

***Laterally* :**

Right colic flexure.

The interior of the second part of the duodenum shows the following special features.

1. The *major duodenal papilla* is an elevation present posteromedially, 8 to 10 cm distal to the pylorus. The hepatopancreatic ampulla opens at the summit of the papilla.

2. The *minor duodenal papilla* is present 6 to 8 cm distal to the pylorus, and presents the opening of the accessory pancreatic duct .

Third *Part*

Course

This part is about 10 cm long. It begins at the inferior duodenal flexure, on the right side of the lower border of the third lumbar vertebra. It passes almost horizontally and slightly upwards in front of the inferior vena cava, and ends by joining the fourth part in front of the abdominal aorta. Its relations are as follows .

Peritoneal Relations

It is retroperitoneal and fixed. Its anterior surface is covered with peritoneum, except in the median plane, where it is crossed by the superior mesenteric vessels and by the root of the mesentery.

Visceral Relations

Anteriorly :

(a) Superior mesenteric vessels and

(b) root of mesentery.

Posteriorly:

(a) Right ureter,

(b) right psoas major,

(c) right testicular or ovarian vessels,

(d) inferior vena cava, and

(e) abdominal aorta with origin of inferior msenteric artery.

Superiorly :

Head of the pancreas with uncinate process

Inferiorly :

Coils of jejunum

Fourth Part

Course

This part is 2.5 cm long. It runs upwards on or immediately to the left of the aorta, up to the upper border of the second lumbar vertebra, where it turns forwards to become continuous with the jejunum at the duodenojejunal flexure. Its relations are as follows.

Peritoneal Relations

It is mostly retroperitoneal, and covered with peritoneum only anteriorly. The terminal part is suspended by the uppermost part of the mesentery, and is mobile.

Visceral Relations

***Anteriorly* :**

(a) Transverse colon,

(b) transverse mesocolon,

(c) lesser sac, and

(d) stomach.

***Posteriorly* :**

(a) Left sympathetic chain,

(b) left psoas major,

(c) left renal vessels,

(d) left testicular vessels, and

(e) inferior mesenteric vein.

***To the right*:**

Attachment of the upper part of the root of the mesentery.

***To the left*:**

(a) Left kidney and

(b) left ureter.

***Superiorly* :**

Body of pancreas.

ARTERIAL SUPPLY:

The duodenum develops partly from the foregut and partly from the midgut. The opening of the bile duct into the second part of the duodenum represents the junction of the foregut and the midgut. Upto the level of the opening, the duodenum is supplied by the *superior pancreaticoduodenal artery,* and below it by the *inferior pancreaticoduodenal artery* .

The first part of the duodenum receives additional supply from:

(a) The right gastric artery;

(b) the supraduodenal artery of Wilkie, which is usually a I branch of the hepatic artery;

(c) the retroduodenal branches of the gastroduodenal artery; and

(d) some I branches from the right gastroepiploic artery.

Venous Drainage

The veins of the duodenum drain into the splenic, superior mesenteric and portal veins.

Lymphatic Drainage

Most of the lymph vessels from the duodenum end in the pancreaticoduodenal nodes present along the inside of the curve of the duodenum, i.e. at the junction of the pancreas and the duodenum. From here the lymph passes partly to the hepatic nodes, and through them to the coeliac nodes; and partly to the superior mesenteric nodes and ultimately via intestinal lymph trunk into the cisterna chyli. Some vessels from the first part of the duodenum drain into the pyloric nodes, and through them to the hepatic nodes. Some vessels drain into the hepatic nodes directly. All the lymph reaching the hepatic nodes drains into the coeliac nodes.

Nerve Supply

Sympathetic nerves from thoracic ninth and tenth spinal segments and parasympathetic nerves from the vagus, pass through the coeliac plexus and reach the duodenum along its arteries.

Histology:

The wall of the duodenum consists of four parts[11]:

1. Mucous membrane
2. Sub mucous layer
3. Muscular layer
4. Serous layer

Mucous membrane:

The mucosa is thrown up into large crescentic folds that project into the intestinal lumen transverse to its long axis. These folds are absent in the proximal 2.5 to 5 cm of the duodenum. The epithelial surface of the villi contains columnar absorptive intestinal cells, goblet cells, Paneth cells, argentaffin cells, and a variety of endocrine polypeptide-secreting cells, not all of which are yet understood. Between the villi projecting from the surface into the lumen are openings of simple tubular glands (crypts of Lieberkühn) extending into the lamina propria[11].

Sub mucous layer

The submucosa is filled with the coiled tubular glands of Brunner that pierce the muscularis mucosa and open into the bottoms of the crypts. These glands, which are characteristic of the duodenal portion of the small intestine, become less frequent, and finally disappear, in its distal segment. Their secretion is alkaline, probably to neutralize the acid gastric secretion of the stomach. The submucosa is bounded by the muscularisexterna. Meissner's plexus is found in the submucosa along with a network of loose connective tissue rich in lymphatics and small blood vessels[11].

Muscular layer

This layer is having a deep layer of circular smooth muscle and a superficial layer of longitudinal smooth muscle. These two layers form the contractile basis of peristalsis. The myenteric plexus of Auerbach lies between these two layers[11].

Serous layer

This is the peritoneal covering. This is absent over the posterior surface except first 2.5cms and over anterior surface where it is crossed by superior mesenteric artery[11].

The Omentum and its Special Properties:

Greater Omentum

This is a large fold of peritoneum which hangs down from the greater curvature of the stomach like an apron and covers the loops of intestines to a varying extent. It is made up of four layers of peritoneum all of which are fused together to form a thin fenestrated rhembrane containing variable quantities of fat.

Attachments

The anterior two layers descend from the greater curvature of the stomach to a variable extent, and fold upon themselves to form the posterior two layers which ascend to the anterior surface of the head, and the anterior border of the body the pancreas. The folding of the omentum is such that the first layer becomes the fourth layer and the second layer becomes the third layer. In its upper part, the fourth layer is partially fused to the anterior surface of the transverse colon and of the transverse mesocolon. The part of the peritoneal cavity called the lesser sac between the second and third layers gets obliterated, except for about 2.5 cm below the greater curvature of the stomach.

Contents

(1) The right and left gastroepiploic vessels anastomose with each other in the interval between the first two layers of the greater omentum a little below the greater curvature of the stomach. (2) It is often laden with fat.

Functions

(1) It is a storehouse of fat.

(2) It protects the peritoneal cavity against infection because of the presence of macrophages in it. Collections of macrophages form small, dense patches, known as *milky spots,* which are visible to the naked eye.

(3) It also limits the spread of infection by moving to the site of infection and sealing it off from the surrounding areas. On this account, the greater omentum is also known as the *policeman of the abdomen.* The greater omentum forms a partition between the supracolic and infracolic compartments of the greater sac.

Lesser Omentum

Definition

This is a fold of peritoneum which extends from the lesser curvature of the stomach and the first 2 cm of the duodenum to the liver. The portion of the lesser omentum between the stomach and the liver is called the *hepatogastric ligament,* and the portion between the duodenum and the liver is called the *hepato-duodenal ligament.* Behind the lesser omentum there lies a part of the lesser sac. The lesser omentum has a free right margin behind which there is the epiploic foramen. The greater and lesser sacs communicate through this foramen.

Attachments

Interiorly, the lesser omentum is attached to the lesser curvature of the stomach and to the upper border of the first 2 cm of the duodenum. Superiorly, it is attached to the liver, the line of attachment being in the form of an inverted 'L'. The vertical limb of the 'L' is attached to the bottom of the fissure for the ligamentum venosum, and the horizontal limb to the margins of the porta hepatis.

Contents:

(A) The right free margin of the lesser omentum contains :

(a) The hepatic artery proper;

(b) the portal vein;

(c) the bile duct;

(d) lymph nodes and lymphatics; and

(e) the hepatic plexus of nerves, all enclosed in a perivascular fibrous sheath.

(B) Along the lesser curvature of the stomach and along the upper border of the adjoining part of the duodenum it contains:

(a) The right gastric vessels;

(b) the left gastric vessels

(c) the gastric group of lymph nodes and lymphatics' and

(d) branches from the gastric nerves

PHYSIOLOGY:

Duodenal motility:

Intrinsic Control

The intrinsic rhythm of small intestinal contractions probably originates within intestinal smooth muscle itself. This intrinsic activity is modified by neural input and by hormones working in an endocrine, paracrine, or neurocrine fashion. Baseline duodenal peristalsis generally occurs at higher frequency (10 to 12/min) than in jejunum or ileum. Thus, in a sense, the duodenum is the "pacemaker" for the distal segments of small intestine[11].

Extrinsic Control

The extrinsic control of intestinal motility is largely under the control of the autonomic nervous system. The sympathetic innervation consists mainly of preganglionic axons originating from spinal roots T9 and T10 run in the splanchnic nerves and synapse with the coeliac ganglia. The sympathetic innervation consists mainly of preganglionic axons originating from spinal roots T9 and T10. These run in the splanchnic nerves and synapse with the celiac ganglia.

The duodenum is innervated by sympathetic fibers from both the celiac ganglia (proximal duodenum) and the superior mesenteric ganglia (distal duodenum). Sympathetic fibers to the small intestine are both cholinergic and noradrenergic[11].

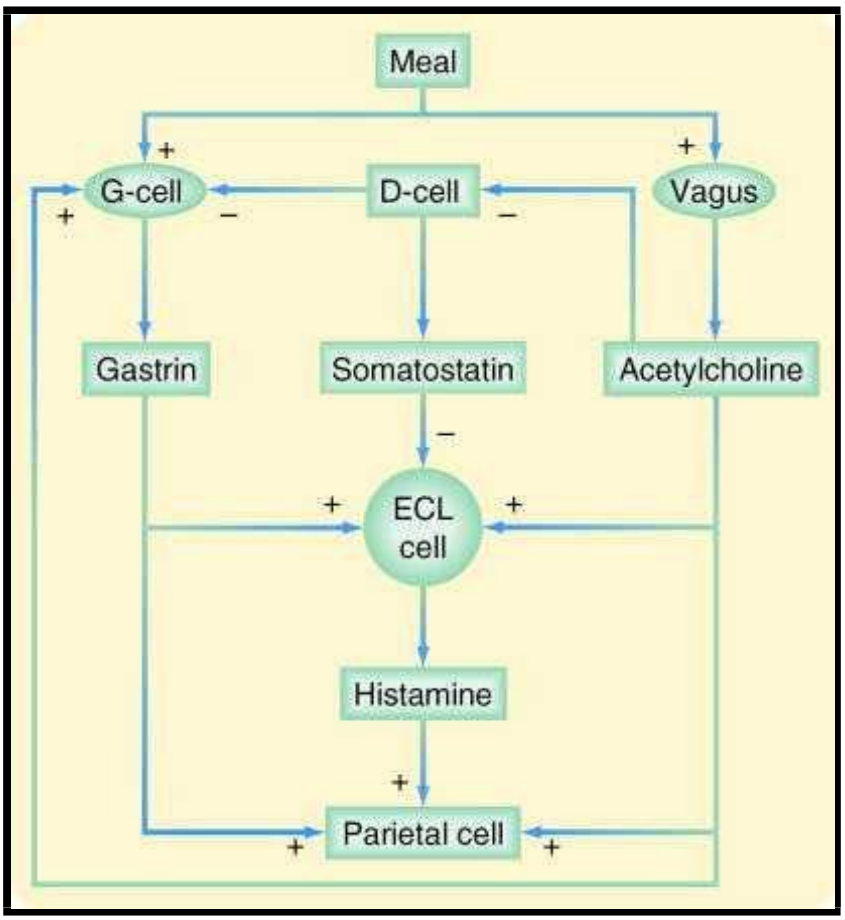


Figure 1 Influence of ECL cells on parietal cells in acid secretion**[12]**

Table 1:Major Actions of Duodenal Peptides[11].

Cholecystokinin	Gallbladder contraction
	Stimulation of pancreatic exocrine and endocrine secretion
	Stimulation of bicarbonate secretion from stomach and duodenum
	Inhibition of gastric emptying
	Growth of pancreas
	Satiety effect
Secretion	Stimulation of pancreatic water and bicarbonate secretion
	Stimulation of biliary water and bicarbonate secretion
	Stimulation of serum parathormone
	Stimulation of pancreatic growth
	Stimulation of gastric pepsin secretion
	Stimulation of colonic mucin
	Inhibition of gastric acid secretion
	Inhibition of gastric emptying and gastrointestinal motility
	Inhibition of lower esophageal sphincter tone
Somatostatin	Inhibition of gastric acid and biliary secretions
	Inhibition of pancreatic exocrine, and enteric secretions
	Inhibition of secretion and action of gastrointestinal endocrine secretion/actions
	Inhibition of gastrointestinal motility and gallbladder contraction

	Inhibition of cell growth
	Small bowel increased reabsorption of water and electrolytes
Neurotensin	Stimulation of pancreatic secretion
	Mesenteric vasodilation
	Decreased lower esophageal sphincter pressure
	Inhibition of gastric acid secretion
Gastric inhibitory polypeptide	Glucose-dependent release of insulin
	Inhibition of gastric acid secretion
Motilin	Initiation of migrating motor complex ("housekeeper") of small intestine
	Increased gastric emptying
	Increased pepsin secretion

Basal Acid Secretion

In humans, The *control of gastric secretions* chiefly occurs in one of the following 3 ways:

1. **Cephalic phase**— is stimulated by the sight, smell, taste or even thought of food. A neural reflex is initiated via branches of the vagus nerve that promotes the release of hydrochloric acid, pepsinogen and mucus.
2. **Gastric phase**— is triggered by the mechanical and chemical stimuli

i) *Mechanical stimulation* comes from stretching of the wall of the stomach and conveying neural messages to the medulla for gastric secretion.

ii) *Chemical stimulation* is by digested proteins, amino acids, bile salts and alcohol which act on gastrin-producing G cells. Gastrin then passes into the blood stream and on return to the stomach promotes the release of gastric juice.

3. Intestinal phase—is triggered by the entry of proteinrich food in the small intestine. An intestinal hormone capable of stimulating gastric secretion is probably released into the blood stream.

1. Tests for Gastric Acid Secretions

The conventional fractional test meal (FTM) has been totally superseded by newer tests. These tests are based on the principle of measuring basal acid output (BAO) and maximal acid output (MAO) produced by the stomach under the influence of a variety of stimulants, and then comparing the readings of BAO and MAO with the normal values. Quantitative analysis is performed after an overnight fast.

The stomach is intubated and gastric secretion collected in 4 consecutive 15-minute intervals. This unstimulated, one-hour collection after titration for the acid concentration in it, is called BAO, expressed in mEq 1-hour. Subsequently, the stomach is stimulated to secrete maximal acid which is similarly collected for one hour and the acid content called as MAO, expressed in mEq-1-hour.

Two highest 15-minute acid outputs are added and then multiplied by 2; this givesthe peak acid output (PAO).

The tests for gastric acid secretion are named after the stimulants used for MAO. Some of the commonly used substances are as under:

i) HISTAMINE. Histamine was the first standard stimulant used for gastric acid secretion test. Subcutaneous injection of histamine phosphate (0.04 mg/kg body weight) is given with simultaneous administration of antihistaminic agent to prevent the untoward side-effects of histamine.

ii) HISTALOG (BETAZOLE). Subcutaneous injection of histalog (1-15 mg/kg body weight) is preferable over histamine due to fewer undesired side-effects and no need for administration of antihistaminic agent.

iii) PENTAGASTRIN (PEPTAVLON). Pentagastrin is currently the most preferred agent administered in the dose of 6 μg/kg body weight. Its activity is similar to gastrin.

iv) INSULIN MEAL (HOLLANDER TEST). This test is based on the fact that in a state of hypoglycaemia, direct vagal action on the parietal cell mass is responsible for acid secretion. Hypoglycaemia induced by intravenous insulin (15 IU soluble insulin) can be used as a test for evaluating the completeness of vagotomy. No increase in acid production should occur if the vagal resection is complete.

v) TUBELESS ANALYSIS. A resin-bound dye, diagnex blue, is given orally. The release of dye by the action of gastric acid and its appearance in the urine indicates the presence of gastric acid. The test can be repeated after giving stimulant of gastric secretion.

SIGNIFICANCE

Normal value for BAO is 1.5-2.0 mEq 1-hour and for MAO is 12-40 mEq 1-hour. In gastric ulcer, the values of BAO and MAO are usually normal or slightly below normal. *Higher values* are found in:

duodenal ulcer; Zollinger-Ellison syndrome (gastrinoma); anastomotic ulcer.

Low value or achlorhydria are observed in: pernicious anaemia (atrophic gastritis); and achlorhydria in the presence of gastric ulcer is highly suggestive of gastric malignancy.

PEPTIC ULCERS

Peptic ulcers are the areas of degeneration and necrosis of gastrointestinal mucosa exposed to acid-peptic secretions. Though they can occur at any level of the alimentary tract that is exposed to hydrochloric acid and pepsin, they occur most commonly (98-99%) in either the duodenum or the stomach in the ratio of 4:1. Each of the two main types may be acute or chronic.

Acute Peptic (Stress) Ulcers

Acute peptic ulcers or stress ulcers are multiple, small mucosal erosions, seen most commonly in the stomach but occasionally involving the duodenum.

ETIOLOGY. These ulcers occur following severe stress. The causes are as follows:

i) *Psychological stress*

ii) *Physiological stress* as in the following:

- Shock
- Severe trauma
- Septicaemia

Extensive burns (Curling's ulcers in the posterior aspect of the first part of the duodenum).

Intracranial lesions (Cushing's ulcers developing from hyperacidity following excessive vagal stimulation).

Drug intake (e.g. aspirin, steroids, butazolidine, indomethacin). Local irritants (e.g. alcohol, smoking, coffee etc).

PATHOGENESIS. It is not clear how the mucosal erosions occur in stress ulcers because actual hypersecretion of gastric acid is demonstrable in only Cushing's ulcers occurring from intracranial conditions such as due to brain trauma, intracranial surgery and brain tumours. In all other etiologic factors, gastric acid secretion is normal or below normal.

In these conditions, the possible hypotheses for genesis of stress ulcers are as under:

1. Ischaemic hypoxic injury to the mucosal cells.
2. Depletion of the gastric mucus 'barrier' rendering the mucosa susceptible to attack by acid-peptic secretions.

Chronic Peptic Ulcers (Gastric and Duodenal Ulcers)

If not specified, chronic peptic ulcers would mean gastric and duodenal ulcers, the two major forms of 'peptic ulcer disease' of the upper GI tract in which the acid-pepsin secretions are implicated in their pathogenesis. Peptic ulcers are common in the present-day life of the industrialised and civilised world.Gastric and duodenal ulcers represent two distinct diseases as far as their etiology, pathogenesis

and clinical features are concerned. However, morphological findings in both are similar and quite diagnostic.

INCIDENCE. Peptic ulcers are more frequent in middle-aged adults. The peak incidence for duodenal ulcer is 5th decade, while for gastric ulcer it is a decade later (6th decade). Duodenal as well as gastric ulcers are more common in males than in females.

Duodenal ulcer is almost four times more common than gastric ulcer; the overall incidence of gastroduodenal ulcers being approximately 10% of the male population.

ETIOLOGY. The immediate cause of peptic ulcer disease is disturbance in normal protective mucosal 'barrier' by acidpepsin, resulting in digestion of the mucosa. However, in contrast to duodenal ulcers, the patients of gastric ulcer have low-to-normal gastric acid secretions, though true achlorhydria in response to stimulants never occurs in benign gastric ulcer.

Besides, 10-20% patients of gastric ulcer may have coexistent duodenal ulcer as well. Thus, the etiology of peptic ulcers possibly may not be explained on the basis of a single factor but is

multifactorial. These factors are discussed below but the first two—*H. pylori* gastritis and NSAIDs-induced injury are considered most important.

1. *Helicobacter pylori* gastritis. About 15-20% cases infected with *H. pylori* in the antrum develop duodenal ulcer in their life time while gastric colonisation by *H. pylori* never develops ulceration and remain asymptomatic.

2. NSAIDs-induced mucosal injury. Non-steroidal anti-inflammatory drugs are most commonly used medications in the developed countries and are responsible for direct toxicity, endothelial damage and epithelial injury to both gastric as well as duodenal mucosa.

3. Acid-pepsin secretions. There is conclusive evidence thatsome level of acid-pepsin secretion is essential for the development of duodenal as well as gastric ulcer. Peptic ulcers never occur in association with pernicious anaemia in which there are no acid and pepsin-secreting parietal and chief cells respectively.

4. Gastritis. Some degree of gastritis is always present in the region of gastric ulcer, though it is not clear whether it is the cause or the effect of ulcer. Besides, the population distribution pattern of gastric ulcer is similar to that of chronic gastritis.

5. Other local irritants. Pyloric antrum and lesser curvature of the stomach are the sites most exposed for longer periods to local irritants and thus are the common sites for occurrence of gastric ulcers.

Some of the local irritating substances implicated in the etiology of peptic ulcers are heavily spiced foods, alcohol, cigarette smoking, unbuffered aspirin.

6. Dietary factors. Nutritional deficiencies have been regarded as etiologic factors in peptic ulcers e.g. occurrence of gastric ulcer in poor socioeconomic strata, higher incidence of duodenal ulcer in parts of South India. However, malnutrition does not appear to have any causative role in peptic ulceration in European countries and the U.S.

1. There is generally *hypersecretion of gastric acid* into the fasting stomach at night which takes place under the influence of vagal stimulation. There is high basal as well as maximal acid output (BAO and MAO) in response to various stimuli.

2. Patients of duodenal ulcer have *rapid emptying* of the stomach so that the food which normally buffers and neutralises the gastric acid, passes down into the small intestine, leaving the duodenal mucosa exposed to the aggressive action of gastric acid.

3. *Helicobacter* gastritis caused by *H. pylori* is seen in 95-100% cases of duodenal ulcers. The underlying mechanisms are as under:

i) Gastric *mucosal defense is broken* by bacterial elaboration of urease, protease, catalase and phospholipase.

ii) *Host factors: H. pylori*-infected mucosal epithelium releases *proinflammatory cytokines* such as IL-1, IL-6, IL-8 and tumour necrosis factor-α, all of which incite intense inflammatory reaction.

iii) *Bacterial factors:* Epithelial injury is also induced by cytotoxin-associated gene protein *(CagA),* while vacuolating cytotoxin *(VacA)* induces elaboration of cytokines

COMPLICATIONS.

Acute and subacute peptic ulcers usually heal without leaving any visible scar. However, healing of chronic, larger and deeper ulcers may result in complications. These are as follows:

1. Obstruction.

Development of fibrous scar at or near the pylorus results in pyloric stenosis. In the case of healed duodenal ulcer, it causes duodenal stenosis.

Healed ulcers along the lesser curvatures may produce ‘hourglass’ deformity due to fibrosis and contraction.

2. Haemorrhage.

Minor bleeding by erosion of small blood vessels in the base of an ulcer occurs in all the ulcers and can be detected by testing the stool for occult blood. Chronic blood loss may result in iron deficiency anaemia. Severe bleeding may cause ‘coffee ground’ vomitus or melaena. A penetrating chronic ulcer may erode a major artery (e.g. left gastric, gastroduodenal or splenic artery) and cause a massive and severe hematemesis and sometimes death.

3. Perforation.

A perforated peptic ulcer is an acute abdominal emergency. Perforation occurs more commonly in chronic duodenal ulcers than chronic gastric ulcers.

Following sequelae may result:

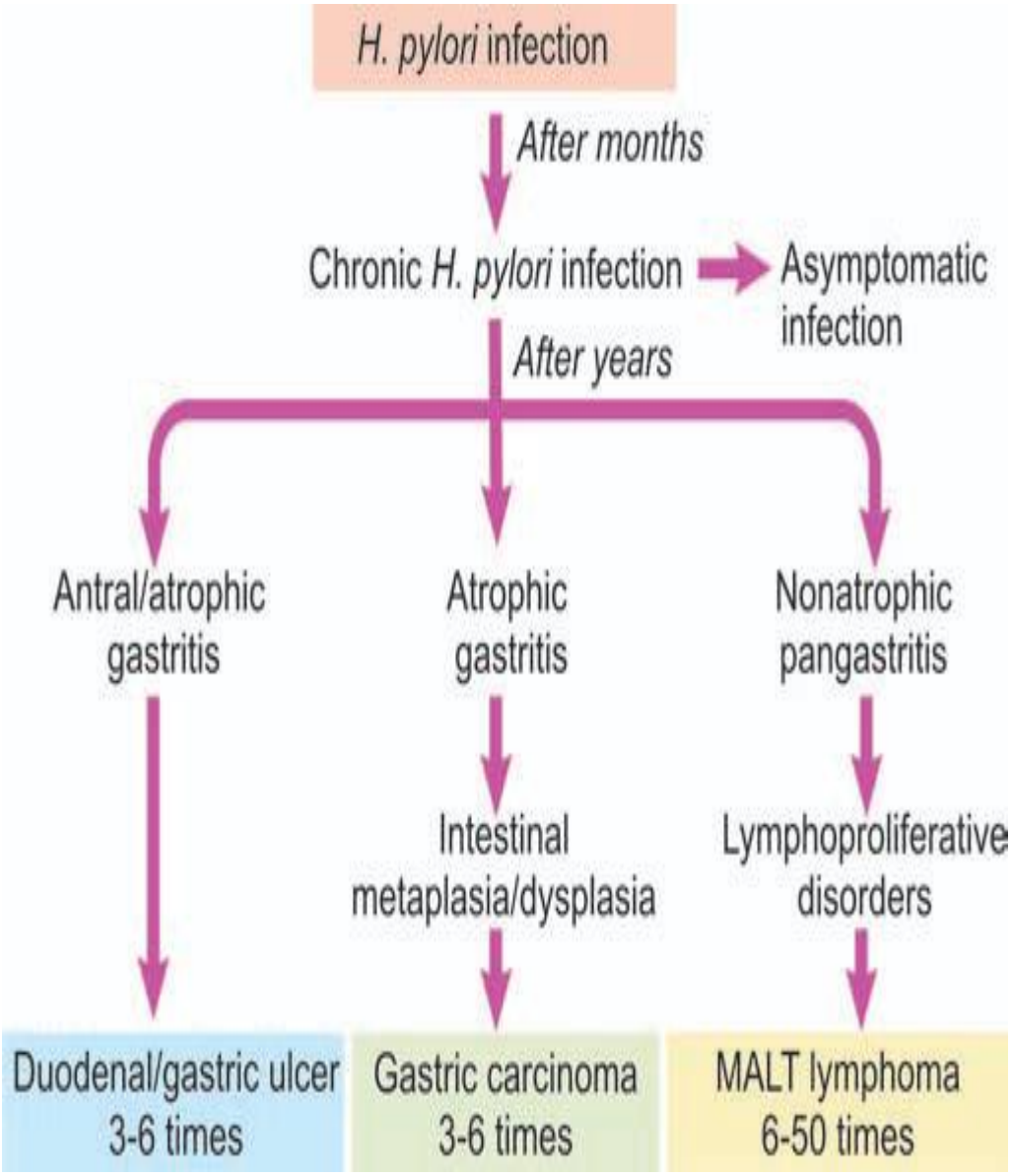
i) On perforation the contents escape into the lesser sac or into the peritoneal cavity, causing *acute peritonitis.*

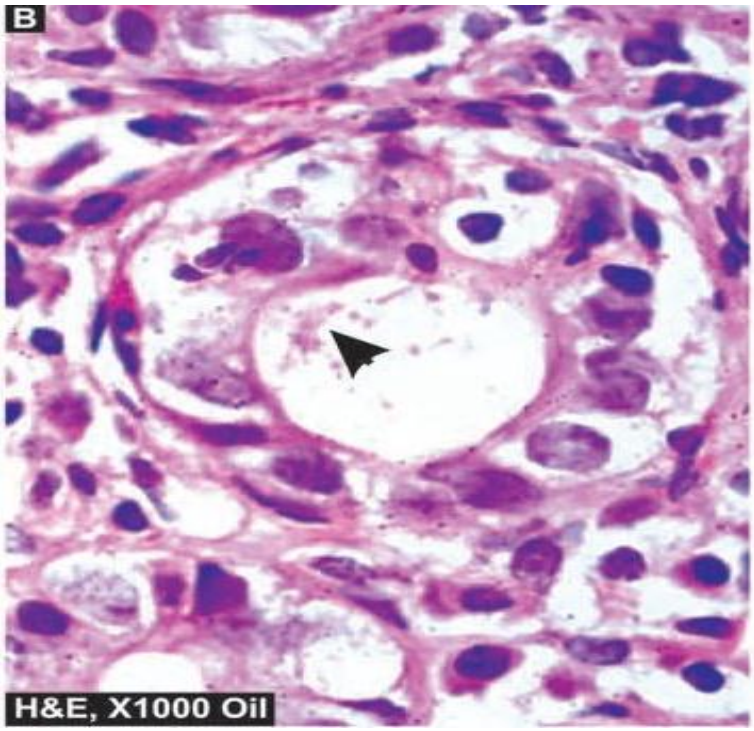
ii) Air escapes from the stomach and lies between the liver and the diaphragm giving the characteristic radiological appearance of *air under the diaphragm.*

iii) *Subphrenic abscess* between the liver and the diaphragm may develop due to infection.

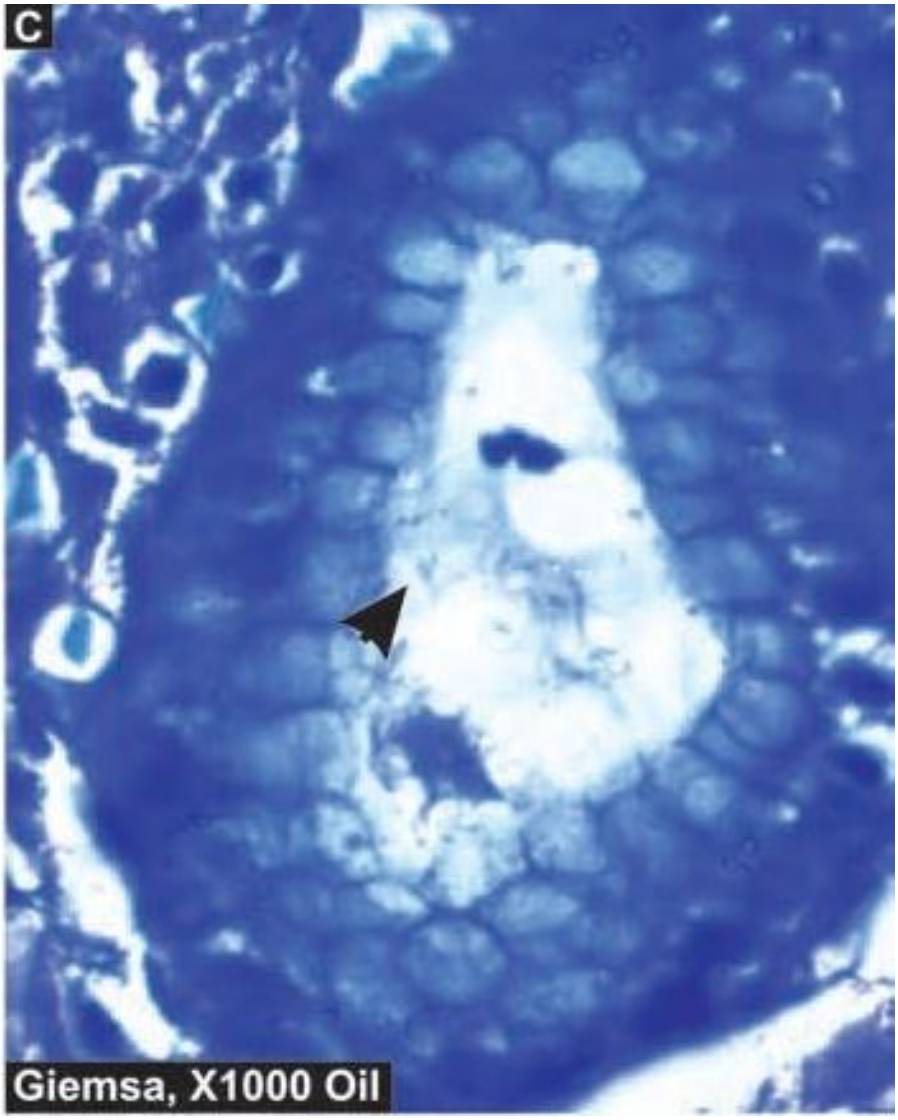
iv) Perforation may extend to involve the *adjacent organs* e.g. the liver and pancreas.

4. Malignant transformation. The dictum *'cancers ulcerate but ulcers rarely cancerate'* holds true for most peptic ulcers. A chronic duodenal ulcer never turns malignant, while less than 1% of chronic gastric ulcers may transform into carcinoma.


H. pylori infection
After months
Chronic H. pylori infection
Asymptomatic infection
After years
Antral/atrophic gastritis
Atrophic gastritis
Nonatrophic pangastritis
Intestinal metaplasia/dysplasia
Lymphoproliferative disorders
Duodenal/gastric ulcer 3-6 times
Gastric carcinoma 3-6 times
MALT lymphoma 6-50 times



H.PYLORI IN H&E STAIN



H.PYLORI IN GIEMSA STAIN

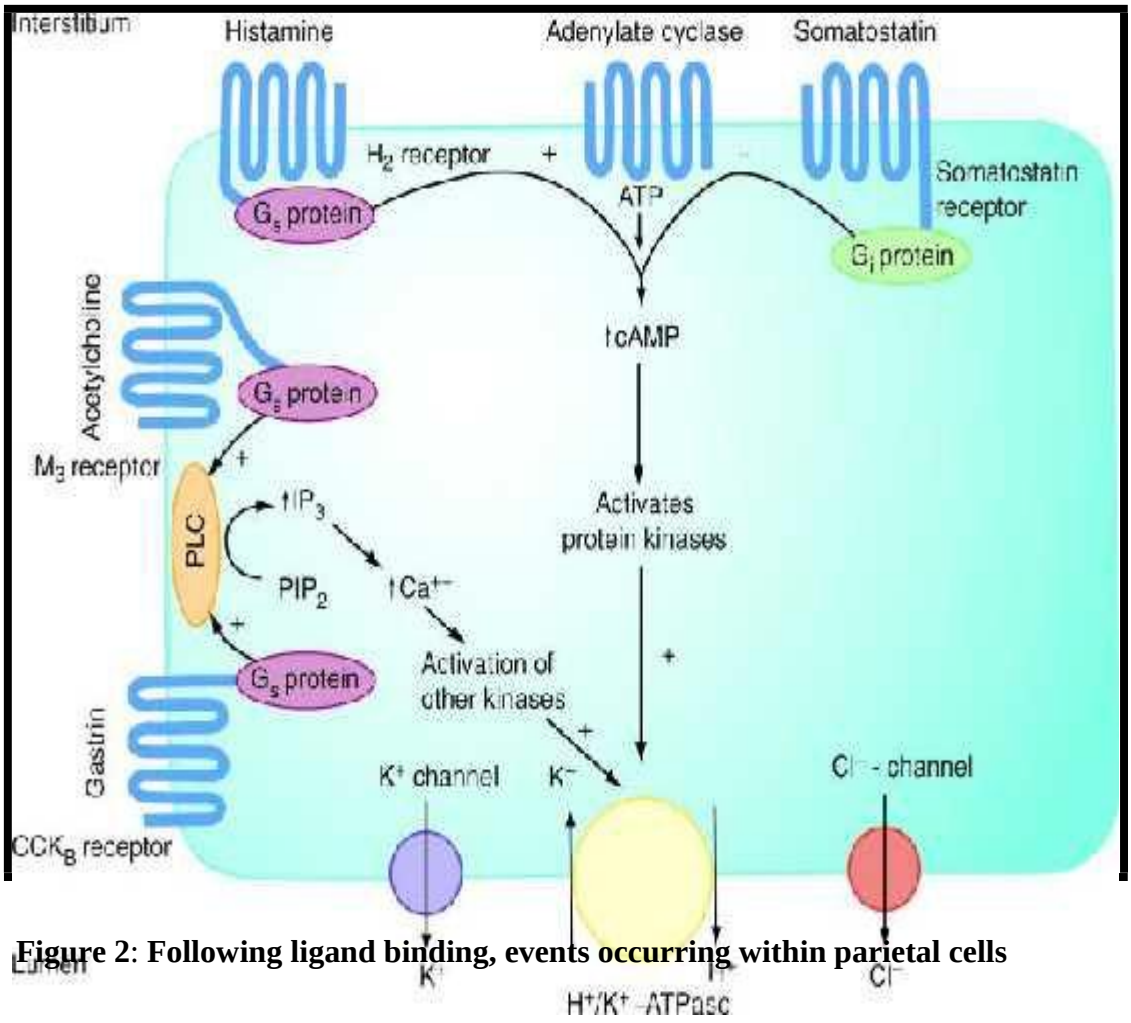


Figure 2: Following ligand binding, events occurring within parietal cells

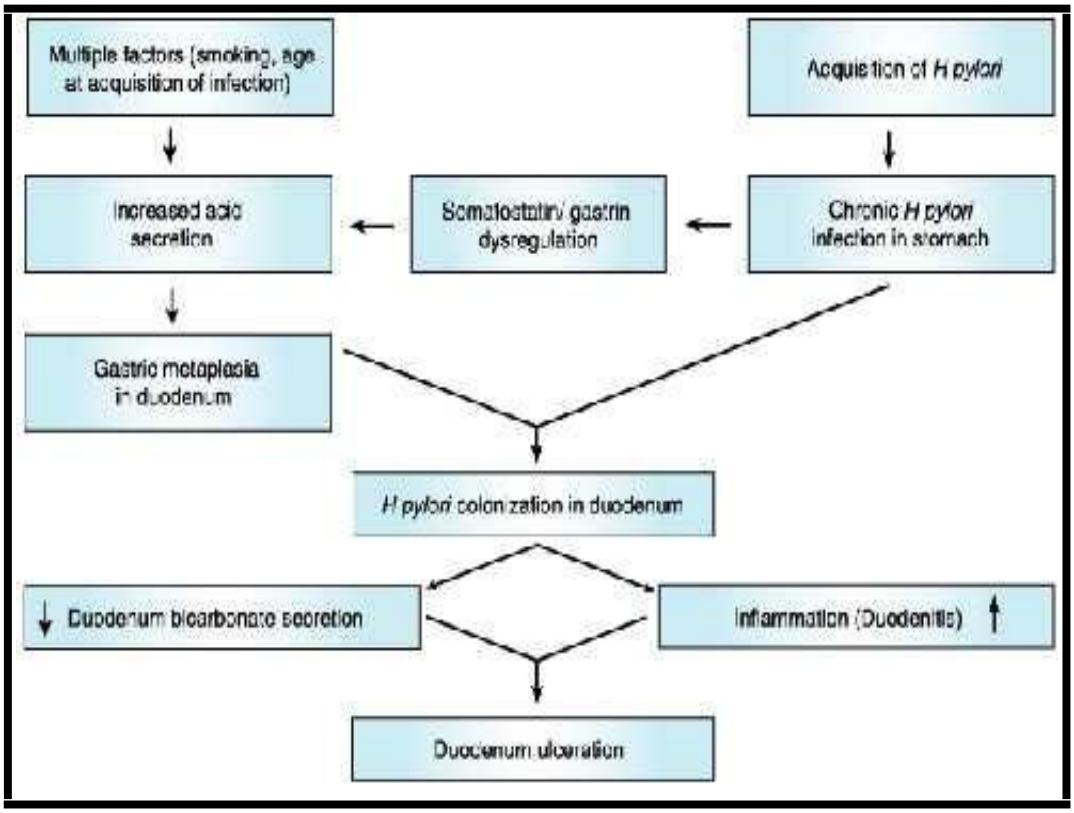


Figure 3: Model of H. pylori related pathophysiology in duodenal ulcer.

Gastric ulcer:

The pathogenesis of gastric ulcer is mainly explained on the basis of impaired gastric mucosal defences against acid-pepsin secretions. Some other features in the pathogenesis of gastric ulcer are as follows:

1. Hyperacidity may occur in gastric ulcer due to *increased serum gastrin* levels in response to ingested food in an atonic stomach.

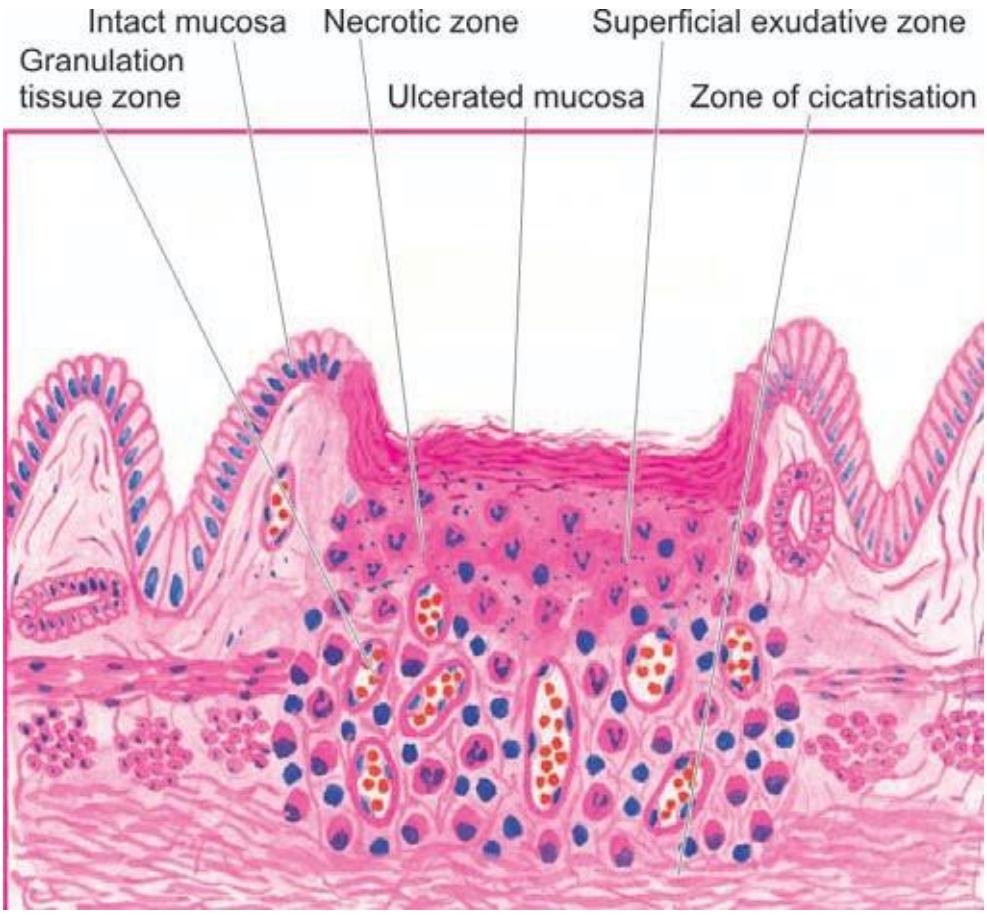
2 However, many patients of gastric ulcer have low-tonormal gastric acid levels. Ulcerogenesis in such patients is explained on the basis of damaging influence of *other factors* such as gastritis, bile reflux, cigarette smoke etc.

3. The normally protective *gastric mucus 'barrier'* against acid-pepsin is deranged in gastric ulcer. There is depletion in the quantity as well as quality of gastric mucus. One of the mechanisms for its depletion is colonisation of the gastric mucosa by *H. pylori* seen in 75-80% patients of gastric ulcer.

Peptic ulcers are remitting and relapsing lesions.

Their chronic and recurrent behaviour is summed up the saying: *'once a peptic ulcer patient, always a peptic ulcer patient.'* The two major forms of chronic peptic ulcers show variations in clinical features which are as follows:

1. **Age.** The peak incidence of duodenal ulcer is in 5^{th} decade while that for gastric ulcer is a decade later.
2. **People at risk.** Duodenal ulcer occurs more commonly in people faced with more stress and strain of life (e.g. executives, leaders), while gastric ulcer is seen more often in labouring groups.
3. **Periodicity.** The attacks in gastric ulcers last from 2-6 weeks, with interval of freedom from 1-6 months. The attacks of duodenal ulcer, are classically worsened by *'work, worry and weather.'*
4. **Pain.** In gastric ulcer, epigastric pain occurs immediately or within 2 hours after food and never occurs at night. In duodenal ulcer, pain is severe, occurs late at night ('hunger pain') and is usually relieved by food.



Peptic ulcer histology

AETIOLOGY

“The perforation into peritoneal cavity transforms a peptic ulcer from a grievous but tolerable burden to a dire calamity” – Illingworth (1944).

Genetic and blood group

There is definitive evidence that peptic ulcer occurs in families. Moreover blood group ‘O’ are about three times more likely to develop a peptic ulcer, ABO genes may modify the size the parietal cell mass[18].

Neurogenic theory

Stress and strain leads to stimulation of vagus results in gastric hypersecretion and hypermotility[18].

Infection

H. pylori is a spirocheatal bacterium that exists in the antrum and duodenum deep to the mucosal layer. It causes local rise in pH leading to epithelial cellular damage and ulceration[18].

Endocrine

The effects of emotional as well as physical stress are hormonally transmitted to the stomach via the pituitary adrenocortical axis. Specific endocrine disorders associated with ulceration are

(1) Zollinger-Ellison syndrome,

(2) multiple adenoma syndrome and (3) hyperparathyroidism[18].

Accessory causes

- Inadequate mastication.
- Alcohol
- Irregular meals
- Excessive smoking
- Vitamin deficiency[18].

Drugs

Non-steroidal anti-inflammatory drugs and steroids are most responsible for development of peptic ulceration[18].

Other causes

Conditions like burns, head injury, septicemia, multiorgonfailure and immunocompromised status etc[18].

Predisposing factors

Age

Perforation can occur at any time during adult life, but is most common between the ages of second to fifth decades.

Sex

Perforation of ulcer is more common in men than women. The male-to-female ratio ranges from 2.5 to 1 to 10 to 1[18].

Seasonal Incidence

Incidence is more common in the spring and in the autumn.

Occupation

Perforation is more likely to occur in those engaged in heavy manual work and those working under stress.

Weekly Incidence

Throughout the world, perforations occur most commonly on Friday and least commonly on Sunday[19].

Periodicity

Perforations occur most frequently in the late afternoon and fewer in the night[19].

Relation to Food

Most of the perforations occur a few hours after food, and whether gastric hypersecretion is important is unknown[19].

Relation to H.Pylori

Its direct relation to perforation is not yet proved.

CLINICAL FEATURES

Acute perforation of a peptic ulcer may be a life-threatening abdominal catastrophe that in usual circumstances can be easily diagnosed and treated.

Age:

Perforation can occur at any time during adult life, but is most common between the ages of second to fifth decades[18].

Sex:

Perforation of ulcer is more common in men than women.

History of present illness:

Pain:

- Severe epigastric or upper abdominal pain gradually preads all over the abdomen
- Sudden in onset[18]
- It occurs most commonly in the late afternoon[14]
- It may be reffered to tip of the shoulder, due to diaphragmatic irritation
- Back pain is uncommon[18].

Nausea and vomiting:

Initially reflex vomiting occurs due to irritation of nerves in the peritoneum and mesentary. In the later stages vomiting is due to toxin acting at the medullary centers and causing paralytic ileus[18].

Fever may be absent initially, but later it may appear due to bacterial peritonitis

History of peptic ulceration will be present in 80% of cases and recurrent perforation in 59% of cases.

Physical examination: General appearance:

Patient will be pale, anxious and loath to move. The patient appears severely distressed.

Decubitus:

Patient lie quietly with knees drawn up and breathing shallowly to minimize abdominal motion.

Pulse:

Initially it will be normal, increases when peritonitis sets in and becomes thready when patient is in shock.

Temperature:

Initially it will be normal, rises with onset of peritonitis.

Tongue:

Initially moist, becomes dry and brown when the peritonitis sets in.

Examination of abdomen:

Abdomen will be held still, moving little or not at all with respiration. Abdomen will be flat in initial stages later it becomes distended in diffuse peritonitis. Whole abdomen will be rigid with board like rigidity. Liver dullness will be obliterated in mid axillary line.

Rectal examination:

There may be fullness in the rectovaginal or rectovesical pouch.

Clinical features vary with the stage of perforation. The clinical course of the disease is divided into three stages, each of variable duration[18].

1. **Early stage of peritoneal irritation**: in this stage patient will be[18]
    - Pale, anxious and loath to move.
    - Temperature may be subnormal with raised pulse rate.
    - Abdomen is held still, moving little or not at all with respiration.
    - The whole abdomen is tender with board like rigidity.
    - Abdomen is dull to percussion.

- Obliteration of liver dullness.
- Pelvic tenderness on rectal examination.

2. Stage of peritoneal reaction[18]

- It starts after 3 hours to 6 hours
- The pain, tenderness and rigidity may lessen.
- The temperature rises to normal or higher with high pulse rate.
- Bowel sounds will be absent.
- This period is also called the 'period of illusion'

3. Stage of diffuse bacterial peritonitis[18]**.**

- It starts after 6 hours of onset.
- There will be silent abdominal distension.
- Enough free fluid may have collected to be clinically detectable.
- The rising pulse rate marks the progressive deterioration in the patient's condition with each hour that passes without operative treatment.

INVESTIGATIONS

Roentgenogram:

Three roentgenograms may be helpful. These are (1) a left lateral decubitus film, (2) an erect chest film and (3) a supine view of the abdomen. Using these views pneumoperitoneum will be detected in 60 to 82 per cent of patients. The left lateral decubitus view may demonstrate as little as 1cc of free air. The erect chest film must be taken after the patient has been in an upright position for 10 to 20 minutes. If roentgenographic diagnosis is uncertain, air or gastrograffin can be injected into a nasogastric tube to increase the amount of intraperitoneal air or identify the site of perforation[19].

Subphrenic gas is absent in cases of:

- Dry perforation
- Patient is not kept in sitting posture prior to X-ray
- Patient is not able to hold the breath at the time of taking X-ray[19].

Gastroduodenogram

It is performed after instilling 60-80ml gastrograffin through asogastric tube. Advantages: it demonstrates

- Site and size of perforation
- Evidence of chronicity and associated gastric ulcer
- Second posterior ulcer associated with perforated anterior ulcer
- Leaking versus sealed ulcer
- Indicated particularly in:
- Suspected perforation with free air
- Free air present but diagnosis is doubtful
- When conservative line of treatment is contemplated

Findings on X-ray if perforation is leaking:

- Diffuse spillage into peritoneal cavity
- Shunting into right lower quadrant
- Localized sub hepatic spill

Ultrasound

Ultrasonography of the abdomen performed using multifrequency probe (3.5-5 MHz). Evidence of intraperitoneal free fluid and of reduced intestinal peristalsis was considered as indirect sign of gastroduodenal perforation[20].

Computerised tomographic examination

CT examination of the abdomen and pelvis performed after intravenous contrast medium administration, no oral or rectal

contrast medium administered. This evidence of free peritoneal gas was considered as a direct evidence of gastrointestinal perforation[20].

Serum amylase

In perforation serum amylase level will be increased. Normal value of serum amylase is 80-180 somogyi units. Above 200 somogyi units is considered pathological. 200-500 somogyi units will be present in other than acute pancreatitis. Mortality rate is high for gastric and duodenal perforation with high serum amylase.

DIAGNOSIS OF HELICOBACTER PYLORI INFECTION

Diagnosis of H.Pylori infection[12]

1. **Non-invasive**

   I. Serology – ELISA II. Urea breath tests

2. **Invasive**

   I. Rapid urease test e.g. Eco, pyloritek

   II. Histology

   III. Culture

A. Serology: Serological tests can be done for detection of IgM, IgG or IgA antibodies. The systemic IgG response is the most commonly used parameter for this infection. ELISA, using a commercial kit, has high sensitivity (100%) and specificity (upto 95%) [12].

B. Urea breath test:.

The patient ingests a solution of urea containing a labelled non-radioactive ^{13}C or radioactive ^{14}C. The appearance of labelled carbon dioxide in the breath indicates the presence of infection[12].

C. Rapid urease test: This test depends on the ability of H.Pylori to produce the enzyme urease, which hydrolyse urea to produce carbon dioxide and ammonium ions, which change the colour of the pH indicator phenol-red from yellow red indicating positive result[12].

D. Histology: H.Pylori can be identified on haemotoxylin and eosin, modified Giemsa and Ethin-stony silver stains[12].

E. Culture: This is the most difficult method for diagnosing the H.Pylori infection.

Abdominal paracentesis

Diagnostic peritoneal tapping is a simple procedure, which can be done quickly in cases of suspicious hollow viscus perforations. Four quadrant abdominal paracentesis has to be done.

DIAGNOSIS

Diagnosis is easy in patients with past history of ulcer, who present with sudden onset of pain abdomen. In the early phase, the diagnosis may be missed unless the signs are carefully elicited. In the later stages, classical features of diffuse peritonitis or paralytic ileus marks it and ultimately the diagnosis of perforation may be missed unless the diminished liver dullness is elicited and subphrenic gas shadow is demonstrated by X-ray.

Differential diagnosis

Duodenal perforation has to be differentiated from the following conditions. These can be divided into intra abdominal and extra abdominal[21].

Intra abdominal conditions[21]**:**

- Acute gastritis
- Acute cholecystitis
- Acute appendicitis with perforation
- Acute pancreatitis
- Acute intestinal obstruction
- Ureteric colic
- Mesenteric vascular occlusion
- Perforated typhoid ulcer
- Diabetic gastric crisis

Extra abdominal conditions[21]**:**

- Coronary thrombosis
- Diaphragmatic pleurisy
- Herpes zoster

The above conditions can be differentiated by their other clinical symptoms, signs and corresponding laboratory investigations.

TREATMENT

Being the common problem, the treatment of perforated pyloroduodenal and gastric ulcer demand thorough planning (R.K.Sen 1959). The most important and immediate step in the management after patients admission to the hospital is adequate resuscitation.

The methods of treatment available are[12]

I.Non-surgical or conservative management

II. Surgical management

A. Open surgery

i. Simple closure of perforation

ii. Closure of perforation with definitive surgery

a. Truncalvagotomy and gastrojejunostomy

b. Antrectomy and vagotomy

c. Pyloroplasty and vagotomy

d. Highly selective vagotomy

B. Laparoscopic surgery.

Non-operative or conservative management:

The advent of powerful and suppressing agents has reawakened interest in the conservative management of perforated peptic ulcer. In majority of patients surgery remains the treatment of choice. In certain situations, conservative management should be considered[22].

It is indicated in:

- When the risks of a general anesthesia are considered too great.

E.g.: The patients who suffers a perforation within hours of an acute myocardial infarction or who has lobar pneumonia

- When appropriate surgical and anesthetic skills or equipment is not available.

- Patients who at presentation have clinically sealed of perforation, whose signs are localized to the epigastrium and in whom the gastrograffin swallow shows no leakage of contrast.

Conservative management consists of

- Continued nasogastric aspiration
- Nil by mouth

- Intravenous fluids.
- Administration of an H2 receptor antagonist intravenously.
- Appropriate sedation.
- Antibiotic.

Advantages:

a. Operation can be avoided.

b. A percentage of patients do not need any future definitive operation, in such patients, unnecessary operation can be avoided.

c. In a few patients, perforation found to be sealed and such patients would be benefited.

Disadvantages:

a. The site of perforation usually remains in doubt.

b. The nature of underlying condition (benign or malignant) remains uncertain.

c. The underlying ulcer diathesis is not treated.

d. Recurrence of ulcer symptoms (Illingworth 1946 e. Recurrence of perforation (2.5%)

f. Risk of deterioration.

g. Strain of patients subjecting themselves for second surgery.

Surgical management should be advocated in patients under conservative treatment when:

1) General condition of the patients starts deteriorating.
2) Persistence of pain after 6 hours of vigorous nasogastric aspiration.
3) Increasing tenderness and guarding of abdomen.
4) Doubtful diagnosis.
5) Associated hemorrhage / suspicious of malignancy.
6) Lack of full – fledged facilities and skill for conservative treatment.

Surgical management:

Perforated peptic ulcer is usually treated by surgery. The risk of operation is definite. The hazard is immeasurable (Moynihan).

Pre operative (treatment) preparation[9]:

1) Resuscitation of patients with intravenous fluid.
2) Nasogastric aspiration: A nasogastric tube is passed and the stomach is kept empty by nasogastric aspiration. To prevent the further contamination of peritoneal cavity.

- To prevent aspiration of gastric contents in to lungs.
- To decompress the stomach.

3) Antibiotics: Patients should be given broad –spectrum antibiotic with antibiotic against anaerobic organism. Third generation cephalosporin's with metronidazole are preferred.

4) Bladder catheterization: Bladder catheterized for all patients to monitor urinary output.

5) H2 blockers: The installations of H2 blockers therapy in preoperative period may not be important in view of decreased gastric acid secretion and motility due to peritonitis.

6) Investigations : Complete blood haemogram, blood glucose, blood urea, serum cretonne, serum electrolytes, HBs Ag, HIV are done ECG is mandatory in patients more than 40 years of age.

7) Preparation of abdomen to be done.

8) Discussion about operation with patient and attenders, written consent is must.

Anesthesia: Spinal or General Anesthesia

Position of the Patient: Supine.

Incision: Upper right paramedian or upper midline.

Procedure:

The abdomen is opened in layers. Bailey points out that in 10% of the cased, a muffled pop of escaping gas can be heard on opening the peritoneum. The free fluid is sucked and mopped with moist packs. The stomach is held near the greater curvature with a moist pack and search for perforations.

Methods of closure of perforation:

1. GRAHAMS live Omental Patch:

It was first described by Roscoe Graham in 1938. Laparotomy pads are placed around the perforation to contain any further spill while the sutures are being placed. After placing three or four sutures, a vascularized (pedicled) tongue of omentum is mobilized and brought superiorly to close the defect[23].

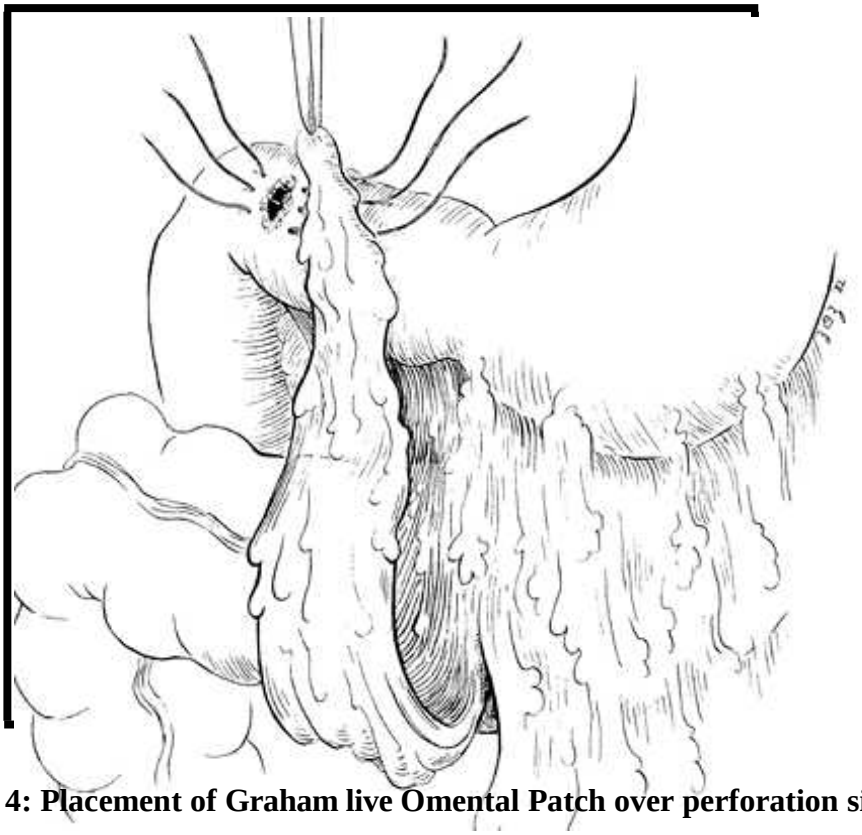


Figure 4: Placement of Graham live Omental Patch over perforation site

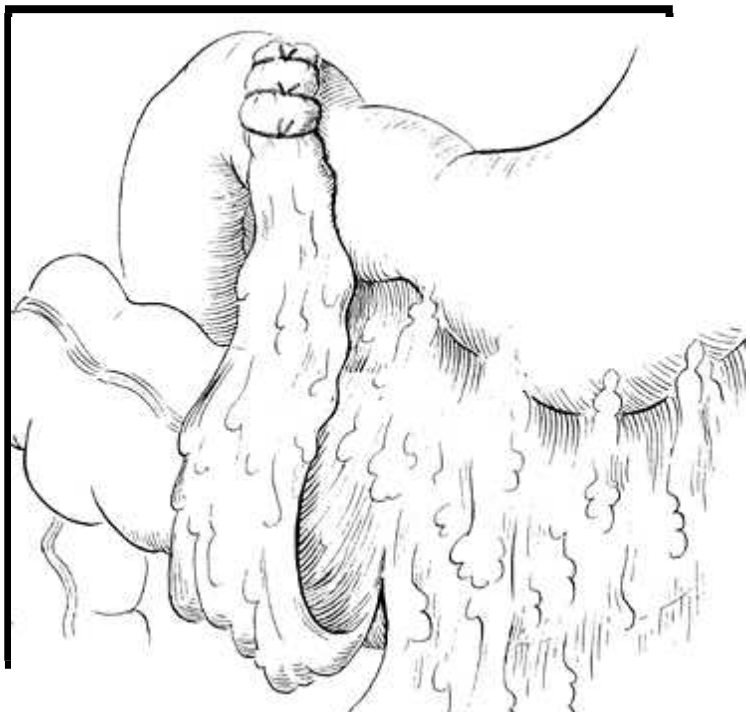


Figure 5: Sealed perforation with Pedicled Omental Patch.

2. Free Omental Patch:

After confirming the site of perforation, a healthy piece of Omentum is harvested. This free piece of omentum, devoid of any vascular pedicle is then fixed to the perforation site by 3 to 4 interrupted sutures of 2.0 mersilk taken between the omentum and the healthy duodenum about 3-4mm away from the margins of the perforation.

On completion of the procedure using one of the above mentioned methods, 30Fr. Malecot catheters were placed in the Morrison's pouch and in the pelvis to act as drains. The abdominal incision is then closed in layers[24].

Eradication of H.pylori infection:

The H. Pylori treatment has become a key success factor and widely advocated in managing peptic ulcer disease, but the ideal regimen has not been achieve. Current regimens for H.Pylori eradication are quite diverse, not only in the combination of agents used but – also in dosage and duration of the treatment[25].

Treatment should be associated with high cure rates, low side effects, a simple regimen, good compliance, efficacy in all subjects including those with strains resistant to antimicrobial agents and low acquisition of acquired resistance if therapy fails. Furthermore, the costs of these regimens should be considered[25].

There is a multitude of regimens against H. Pylori[25]

A. Dual drug therapy

   a. Proton pump inhibitor + Clarithromycin/amoxycillin.
   b. Ranitidine + Clarithromycin for 14 days. Not recommended because of its sub optimal results.

B. Triple drug therapy.

Table 2: Triple drug therapy

1.	Omeprazole 40 mg OD + Clarithromycin 500mg BID + metronidazole 400 mg BID.	For 7 days.
2.	Omeprazole 40 mg OD + Amoxycillin 500 mg BID + Clarithromycin 00 mg BID	7 days.
3.	Omeprazole 40 mg OD + Amoxycillin 500 mg BID + Metronidazole 400 mg BID	7-10 days.
4.	Colloidal Bismuth Subcitrate 125 mg QID + Amoxycillin 500 mg BID + Metronidazole 400 mg BID.	14 days.

C. Quadruple drug therapy.

Table 3: Quadruple drug therapy

1.	Omeprazole 40 mg OD + Colloidal Bismuth subcitrate 125 mg 40 OD + Tetracycline 500 mg TID + Metronidazole 400 mg TID. For 7 days.	

Prognosis

We speak of mortality of only one percent, but may be one in some ones hundred – MOYNIHAN.

The higher mortality rates reported in literature in early part of the century have been reduced to an insignificant rate.

Mortality depends upon the following factors[26]:

a. Age Sex

b. Duration of perforation.

c. General conditions of the patient.

d. Presentation with shock.

e. Presentation with rental failure.

f. Ulcer history.

g. Concomitant second complication

h. Concomitant medical / surgical illness

i. Site and size of perforation.

j. Extent of contamination of peritoneum

k. Perforation of malignant ulcer.

l. Postoperative complication.

MATERIALS AND METHODS

A study of 60 patients admitted with duodenal perforations at Govt. Stanley medical college, Chennai was undertaken from November 2013 to November 2014. These 60 cases were studied thoroughly according to the proforma. The details of 60 patients were sorted in a master chart for convenience of analaysis and presentation. The patients suspected of duodenal perforations undergoing emergency laparotomy were divided into 2 groups of 30 patients each based on the technique of Simple Randomization.

Group A: Grahams live Omental Graft

Group B: Free Omental Graft

Inclusion criteria :

All patients diagnosed with perforated duodenal ulcer who are fit to undergo surgery.

Exclusion Criteria :

1) Very large perforations exceeding 20 mm in size
2) Patients having severe co-morbidities i.e. failure of other organ systems, recent MI, malignancy

3) Patients in whom malignant duodenal ulcer is suspected
4) Patients having multiple perforations
5) Patients who have undergone GI surgeries in the past

The patients with duodenal perforations admitted to our hospital were treated as follows:

A detailed history of patient was taken when the condition of the patient is fair. When the patients present with shock,after stabilisation,detailed history was taken.

The hospital records were also reviewed to have appropriate information of age, sex, occupation, and presentation, chronology of symptoms, any past history, investigations and mode of treatment. During admission this data was essential to evaluate the condition, duration between perforation and surgery, mode of treatment patient received and post surgical morbidity and mortality and regular follow up of the patients for the period of 1 month.

The data was also essential to evaluate the efficacy of Pedicled Omental patch against Free Omental Patch in closure of duodenal perforations. Patients were also followed up to know whether they develop recurrence of ulcer symptoms in order to know the

effectiveness of operation. The data was also compared with other series to see their conclusion were also true in our patients.

Examination:

All the patients with suspected duodenal perforations were examined thoroughly and base line findings are recorded, repeated examination of the patients was done during resuscitation and till the diagnosis is confirmed.

Investigations:

Plain x-ray of abdomen (Erect), blood grouping and Rh typing, Hb%, TC, DC, ESR, Blood urea, serum creatinine, blood sugar, HBsAg, HIV and urine routine. In plain x-ray erect abdomen, air under diaphragm indicated hollow viscus perforation.

I have done four quadrant abdominal paracentesis in all patients. Fluid drawn was found to be turbid and bile stained indicating upper G.I.T. perforation. A dry tap will not rule out perforation. The variables studied and analysed are:

1. Age
2. Sex
3. Duration of (problem prior to admission) perforation.

4. General condition of the patient at the time of admission.
5. Site& Size of perforation
6. Type of surgery
7. Post-operative complications
8. Duration of hospital stay
9. Outcome of the patient

RESULTS

From November 2013 to November 2014 a total of 60 patients with duodenal perforations were studied from surgical units of Govt.Stanley medical college,Chennai.

Age: There is a gradual increase in incidence of duodenal perforation in old age group.

Table 4: The age incidence:

Age group (in years)	No of cases	Percentage
11 – 20	3	5
21 – 30	10	16.66
31 – 40	13	21.66
41 – 50	10	16.66
51 – 60	7	11.66
> 60	17	28.33

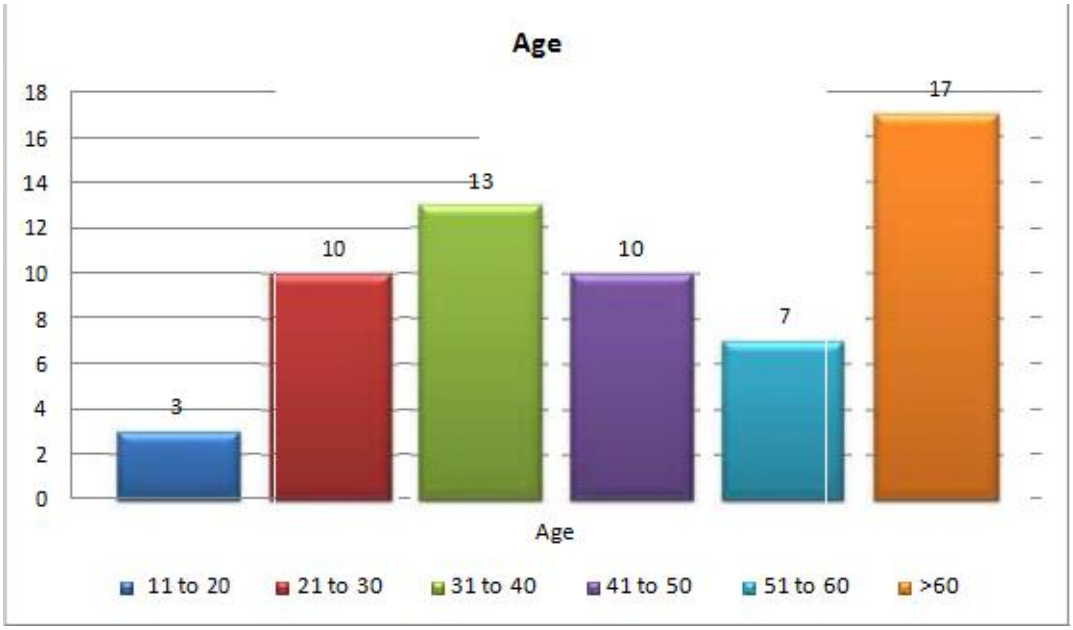
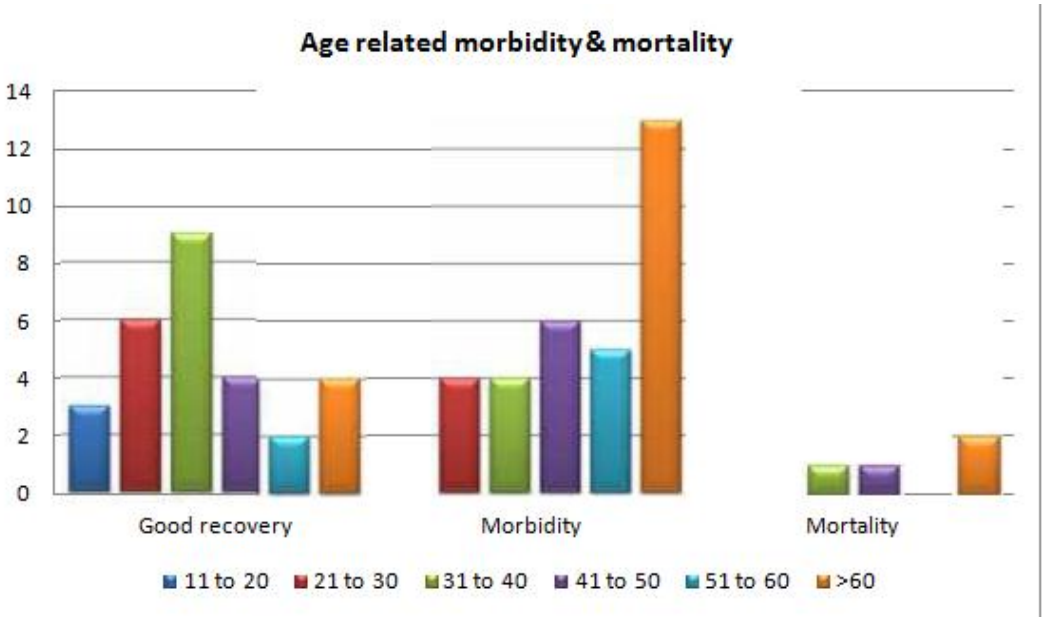


Table 5: Age related morbidity and mortality

Age group (in years)	No. of case	Good recovery	Morbidity	Mortality
11 – 20	3	3	--	--
21 – 30	10	6	4	--
31 – 40	13	9	4	1
41 – 50	10	4	6	1
51 – 60	7	2	5	--
>60	17	4	13	2

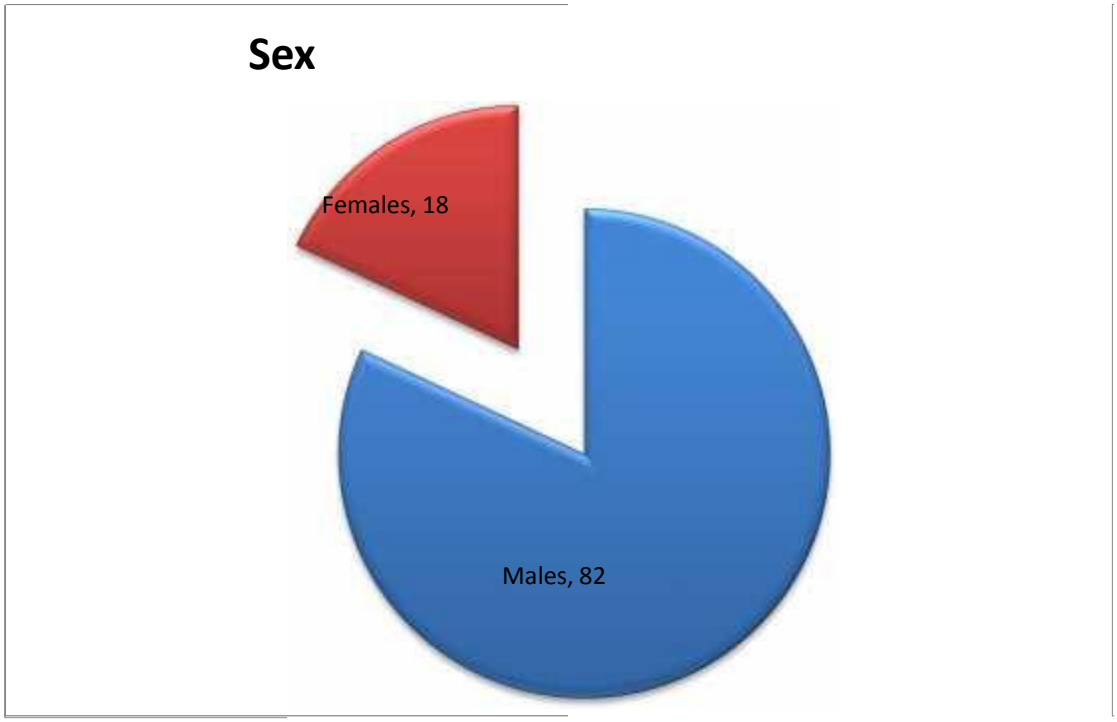


Sex:

Perforation is more common in males with Male: Female ratio in present study is 4.45:1.

Table 6: Sex incidence of duodenal perforation

Sex	No. of cases
Males	49
Females	11



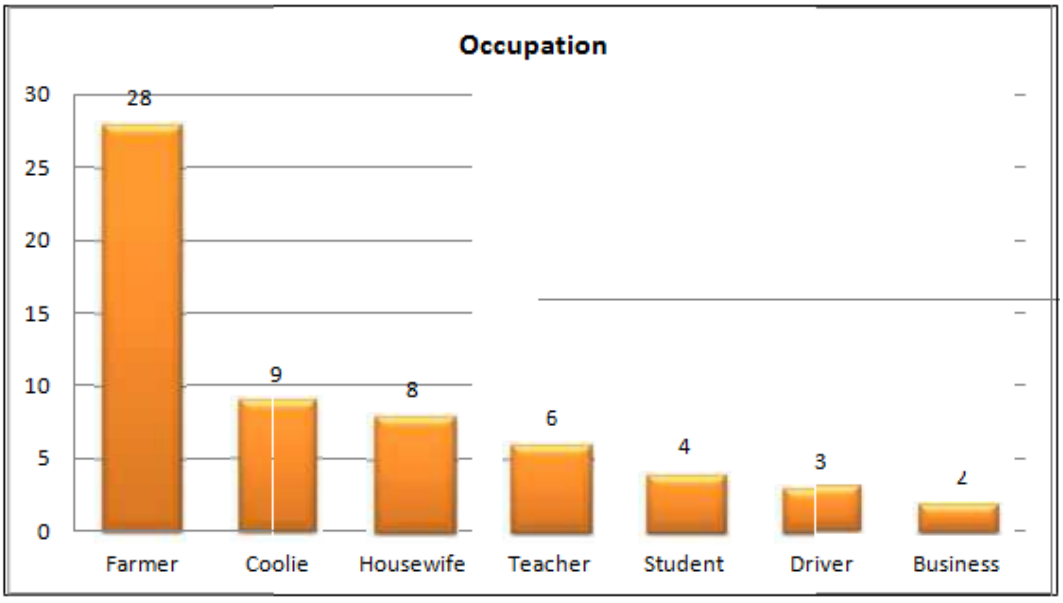
Graph 3: Sex incidence of duodenal perforation

Occupation:

Perforation is more common in farmers in present study.

Table 7: Occupation incidence

Occupation	No. of patients	Perc ntage
Farmer	28	47.54
Coolie	9	14.75
Housewife	8	13.11
Teacher	6	9.84
Student	4	6.56
Driver	3	4.92
Business	2	3.28



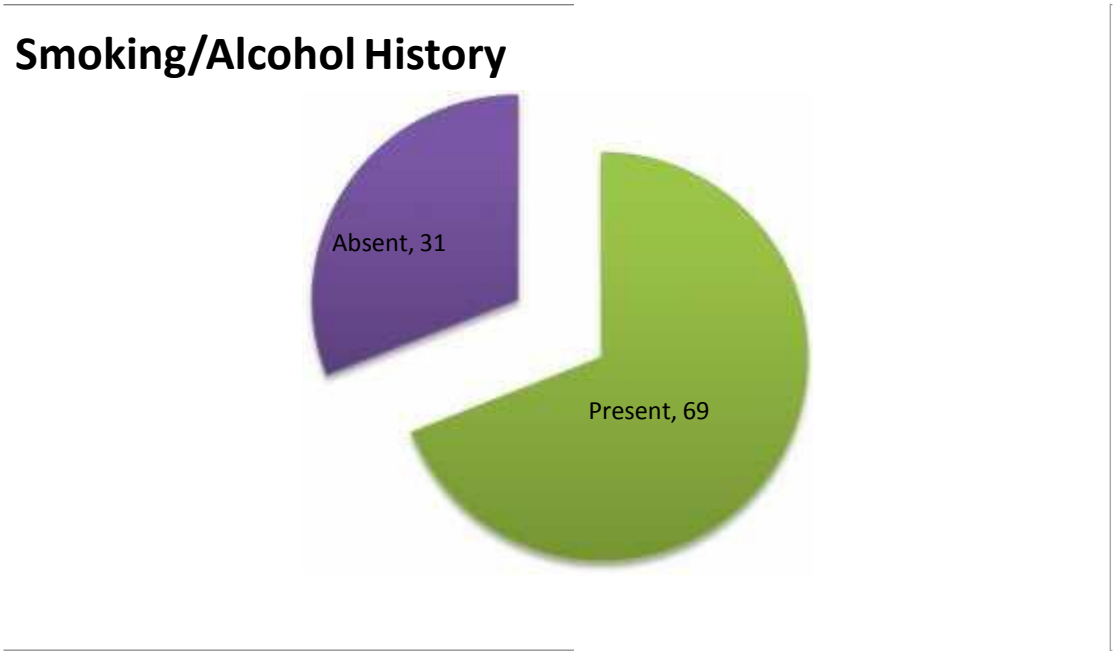
Graph 4: Occupation incidence

Relation with smoking and alcohol:

In this series of study, there was an obvious relationship between the alcohol and tobacco, when compared with non-smokers and non-alcoholics, the incidence is convincingly high in case of smokers and alcoholics.

Table 8: Relation of smoking to incidence of perforation

H/o smoking and alcohol	No. of cases
Present	41
Absent	19



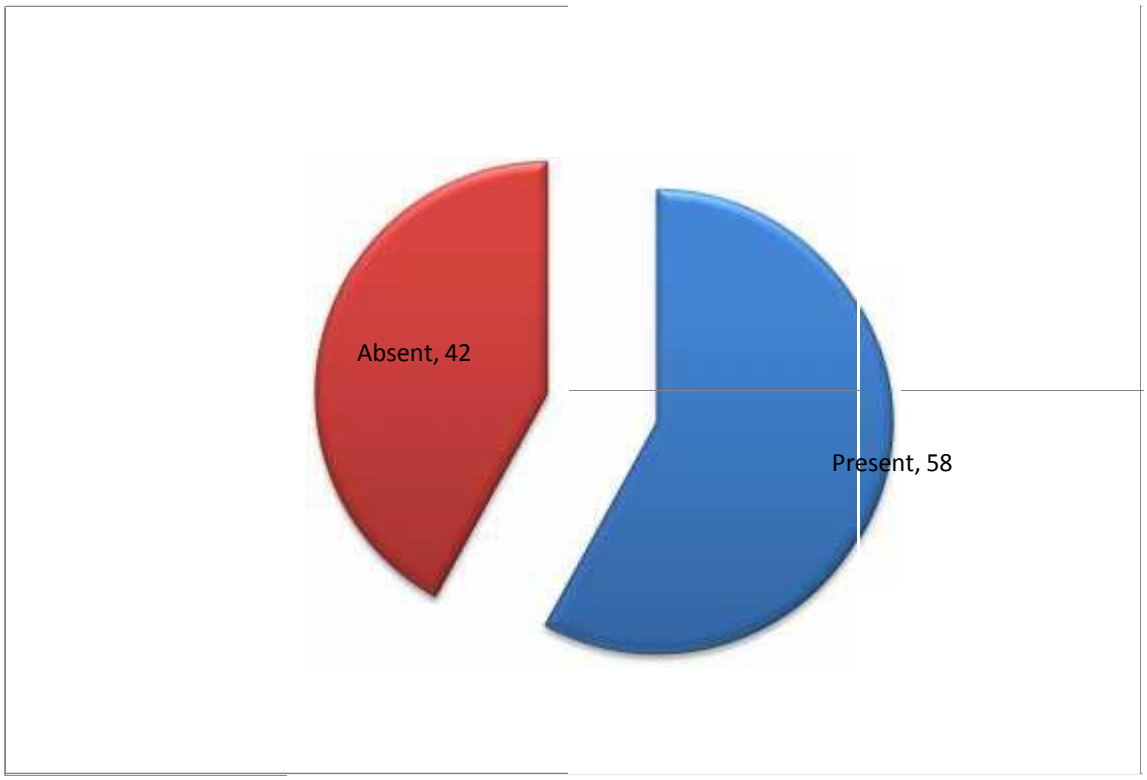
Graph 5: Relation of smoking to incidence of perforation

Previous history of peptic ulcer:

In our series s 58.33% of patients had previous history of peptic ulceration symptoms.

Table 9: Previous history of peptic ulcer

Previous history of peptic ulcer	No. of cases
Present	35
Absent	25



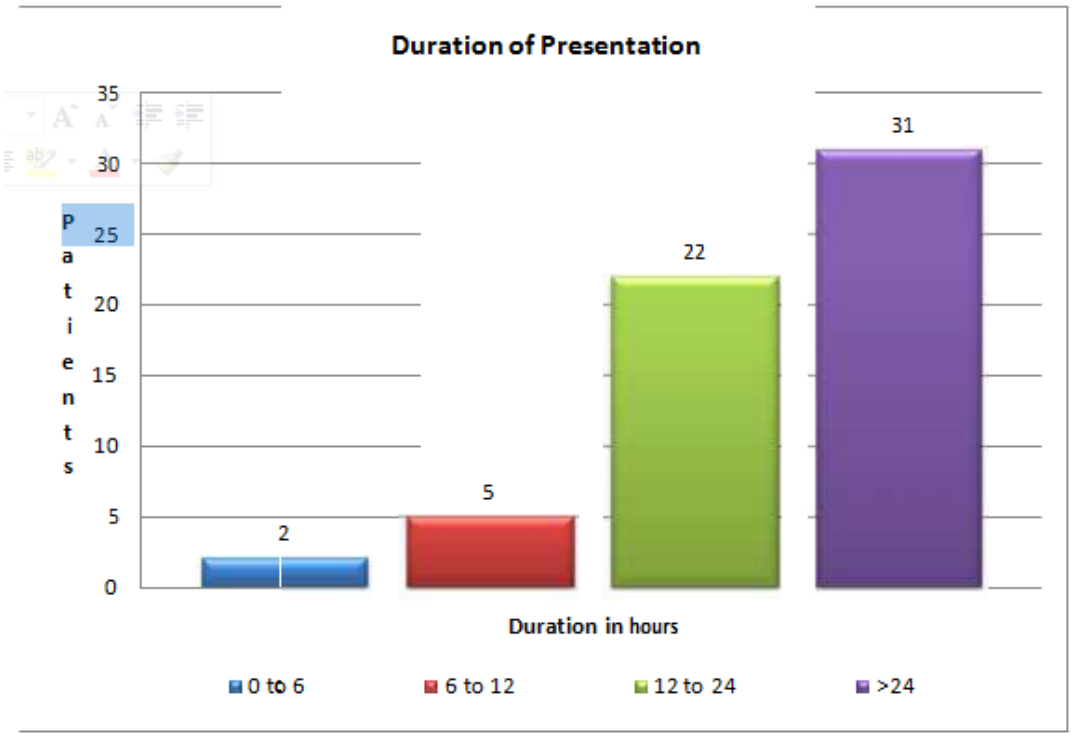
Graph 6: Previous history of peptic ulcer

Duration of symptoms before presentation:

Large group of patients had delayed presentations.

Table 10: Duration of presentation

Duration (in hrs)	No. of patients
0-6	2
6-12	5
12-24	22
> 24 hours	31

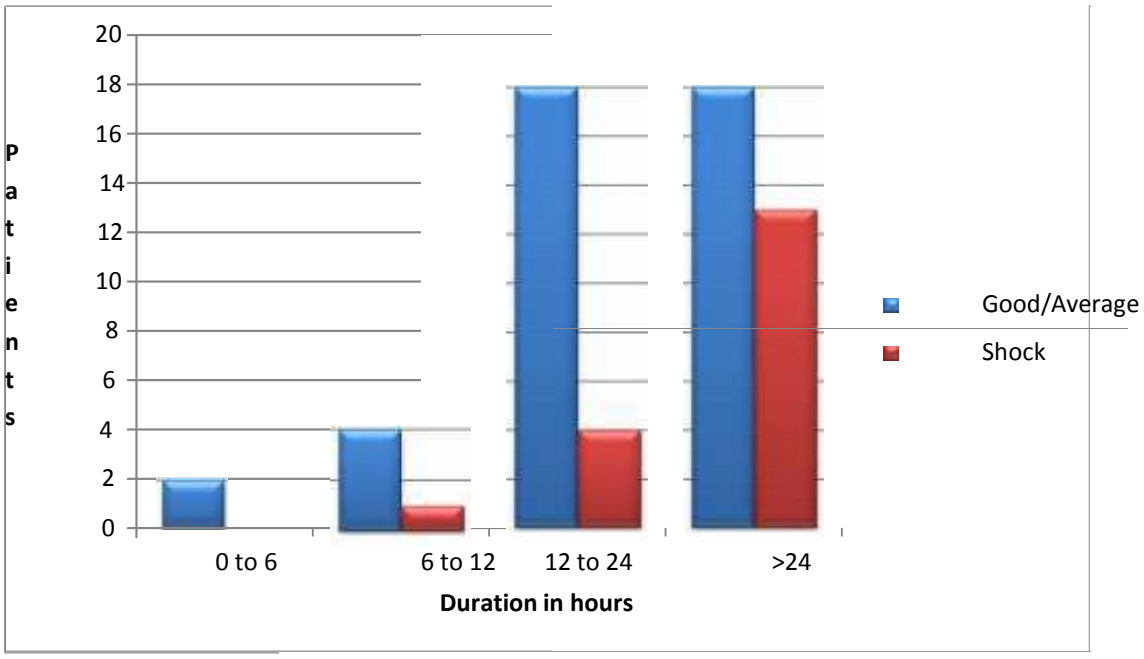


Graph 7: Duration of presentation

Table 11: Impact of duration on the general condition

Duration (in hrs)	No of cases	dition of the patient on admission	
		Good / Average	Shock
0 – 6	2	2	--
6 – 12	5	4	1
12 – 24	22	18	4
> 24 hours	31	18	13

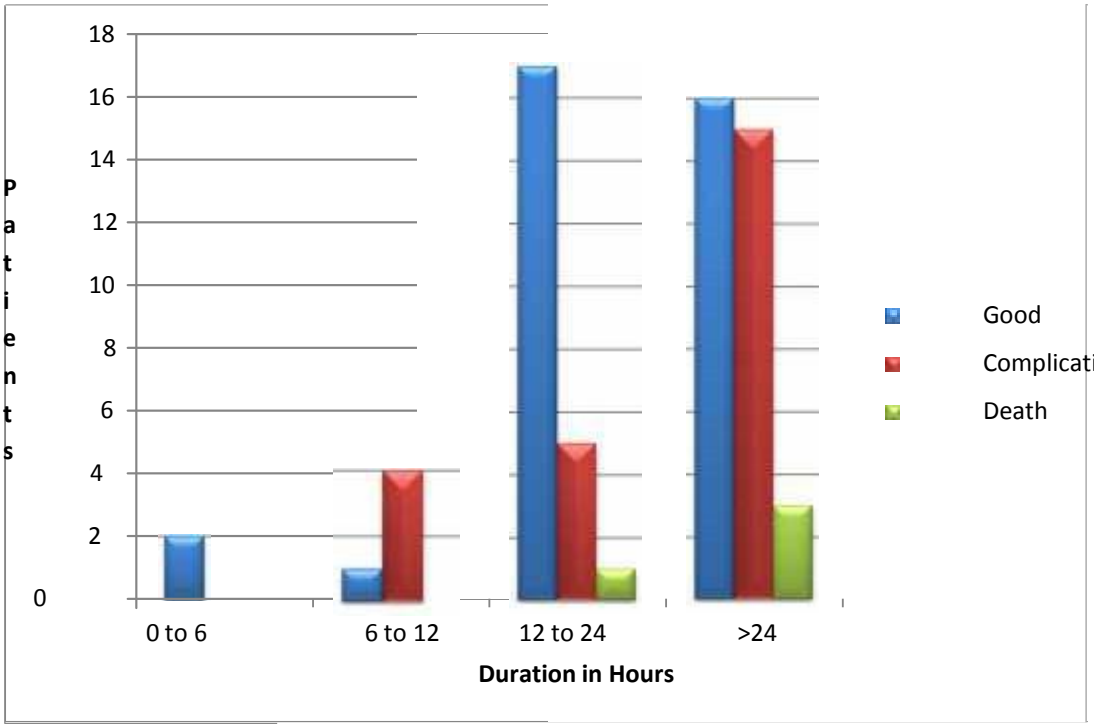
The poorer the general condition of the patient, poor is the final outcome of the patient.



Graph 8: Impact of duration on the general condition

Table 12: Impact of duration on the outcome

Duration (in hrs)	No of cases	Reco ery		
		Good	Complication	Death
0 – 6	2	2	--	--
6 – 12	5	1	4	--
12 – 24	22	17	5	1
> 24 hours	31	16	15	3



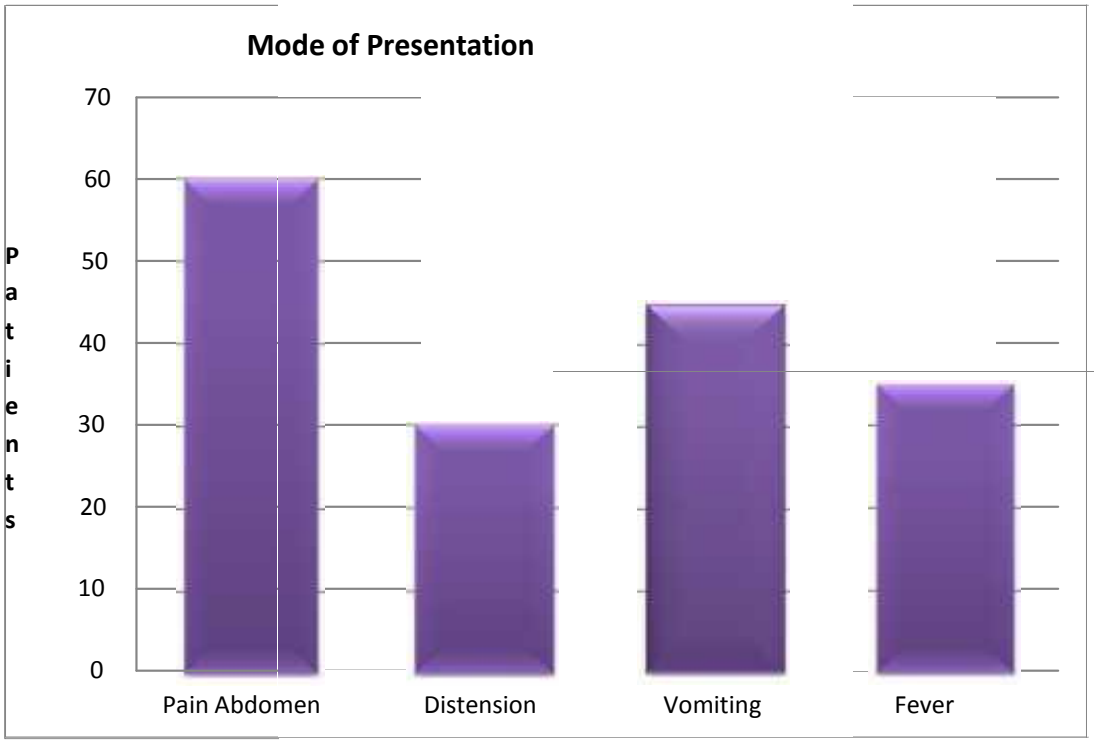
Graph 9: Impact of duration on the outcome

Mode of Presentation:

The common mode of presentation of these patients was abdominal pain, vomiting, distension, fever and shock.

Table 13: Mode of presentation

Symptoms	No of Cases
Pain abdomen	60
Distension of abdomen	30
Vomiting	45
Fever	35

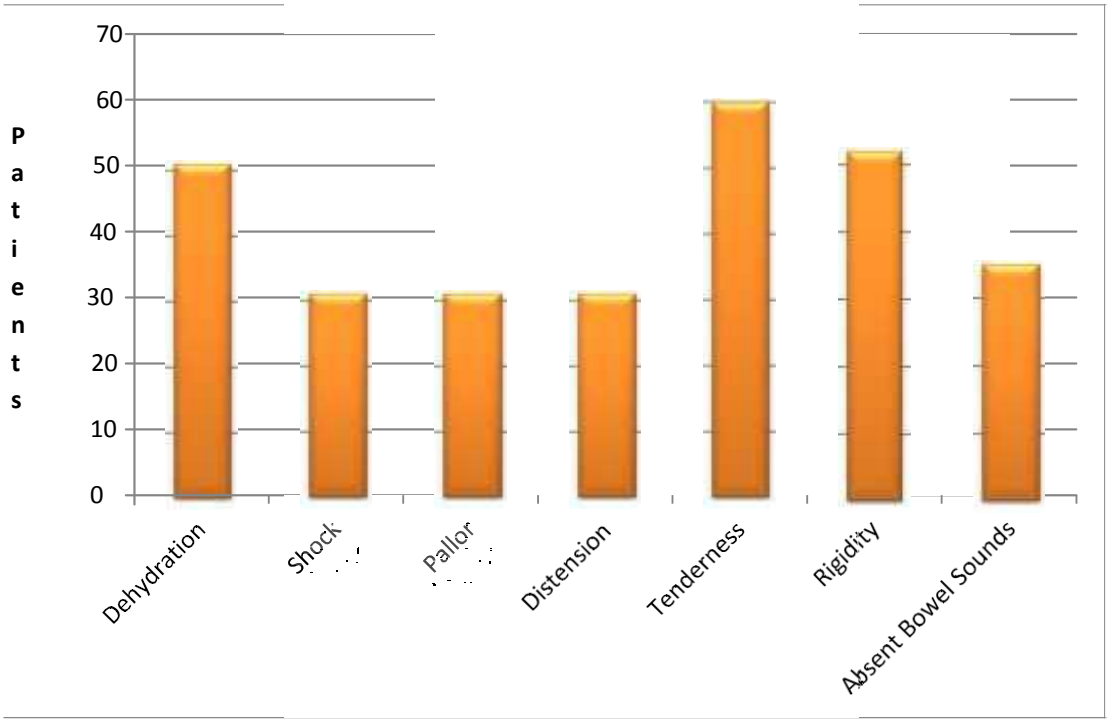


Graph 10: Mode of presentation

Clinical Findings:

Table 14: Clinical findings at the time of admission.

Signs	No. of cases
Dehydration	51
Shock	31
Pallor	31
Distension	31
Tenderness	60
Rigidity	52
Absent bowel sounds	35



Graph 11: Clinical findings at the time of admission

Radiological Investigations:

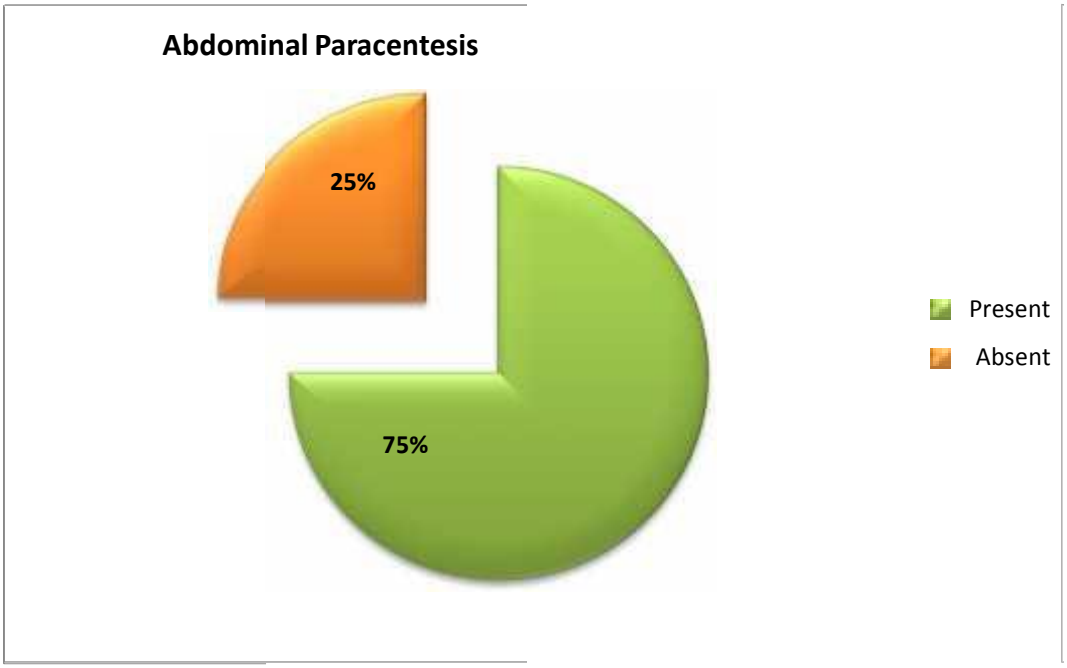
Plain X-ray abdomen in erect position was done in all 60 patients and pneumo-peritoneum (gas under the diaphragm) was found in 60 cases.

Abdominal Paracentesis:

Four quadrant abdominal paracentesis was done in all 60 cases and in 45 cases it revealed bile stained turbid fluid and in 15 cases it was a dry tap. In the present series, the accuracy is about 73.77%.

Table 15: Abdominal Paracentesis

Turbid bile stained fluid	No. of cases
Present	**45**
Absent	**15**



Graph 12: Abdominal Paracentesis

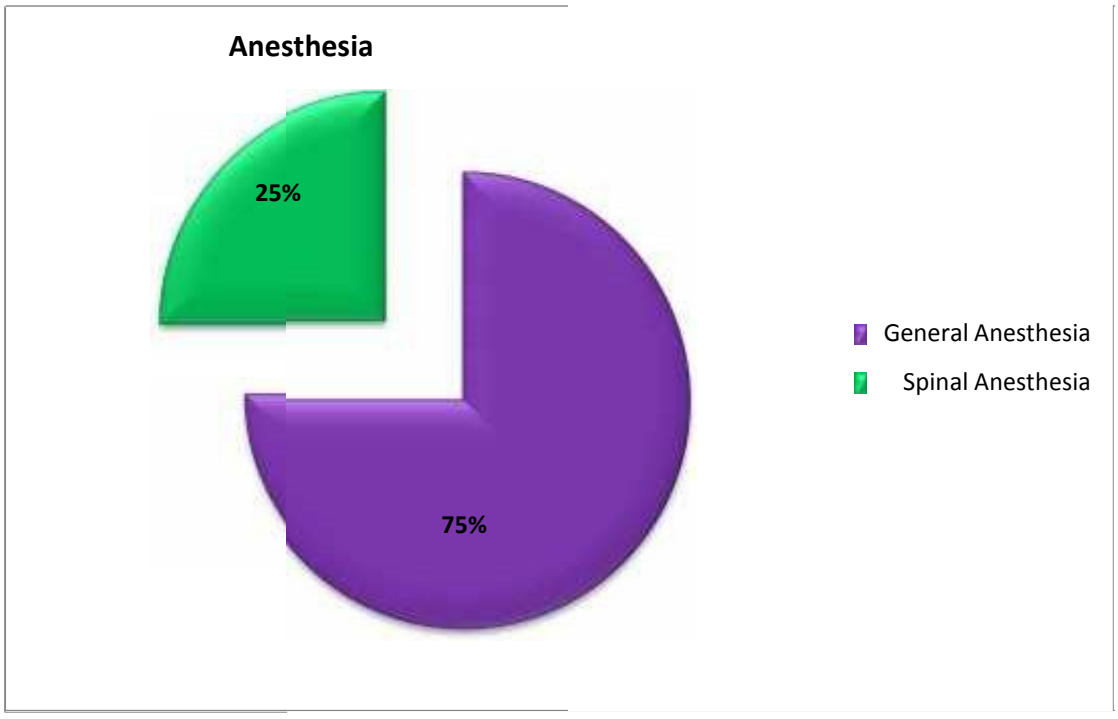
All the patients were operated as early as possible after resuscitation and stabilization.

Anesthesia:

General anesthesia was most commonly employed. General anesthesia was used in 45 patients and Spinal anesthesia in 15 patients.

Table 16: Type of anesthesia used

Anesthesia	No. of cases
General anesthesia	45
Spinal anesthesia(Epidural)	15



Graph 13: Type of Anesthesia

Site of perforation:

In this series, all perforations were found on the anterior aspect of the first part of duodenum.

Size of the perforation:

The size of the perforation is directly proportional to quantity of peritoneal fluid. This finding is directly related to presentation with shock at the time of admission.

Table 17: Relation of size of perforation to quantity of peritoneal contamination

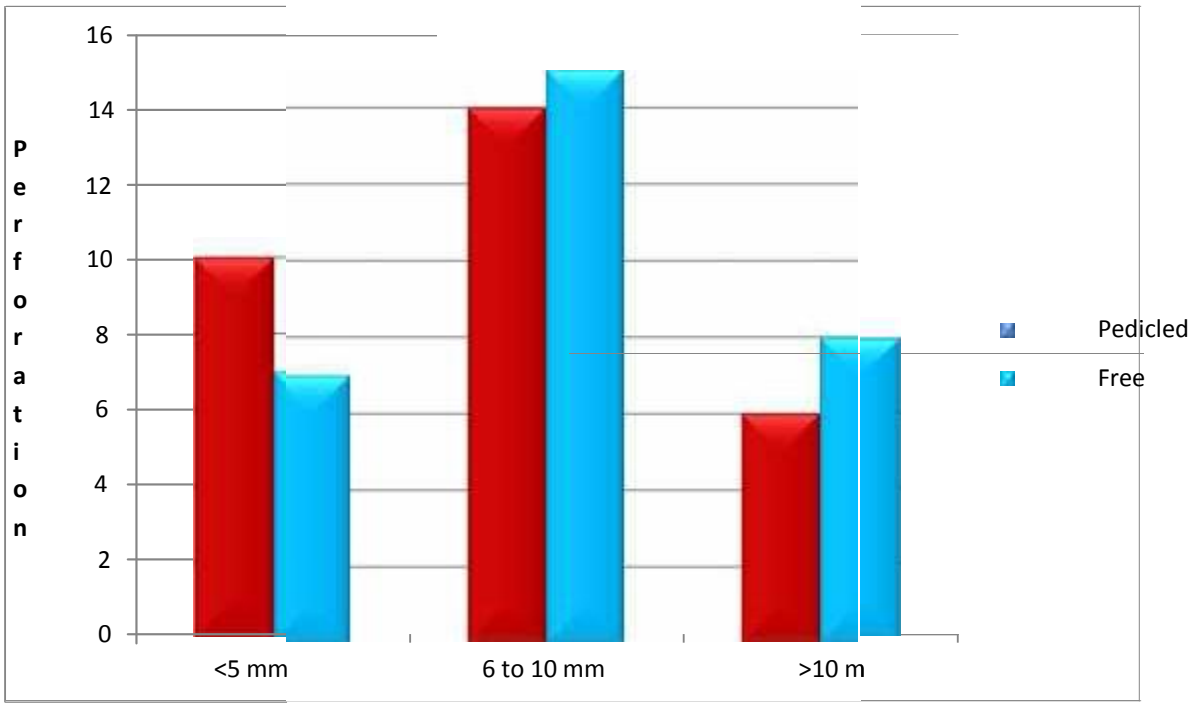
Size	Total cases	Peritoneal fluid		shock
		< 2 liters	> 2 liters	
<0.5 cm	4	2	2	--
0.6-1.0 cm	37	19	18	4
> 1.0 cm	19	6	13	12

Treatment:

Two groups of 30/30 each selected on random basis, one group treated with Grahams live Omental Patch & another with Free Omental Patch.

Table 18: Distribution of patients according to the size of the perforation and type of repair

Size	Total Cases	Pedicled Omental patch	Free Omental Patch
< 0.5 cm	17	10	7
0.6-1 cm	29	14	15
> 1.0 cm	14	6	8



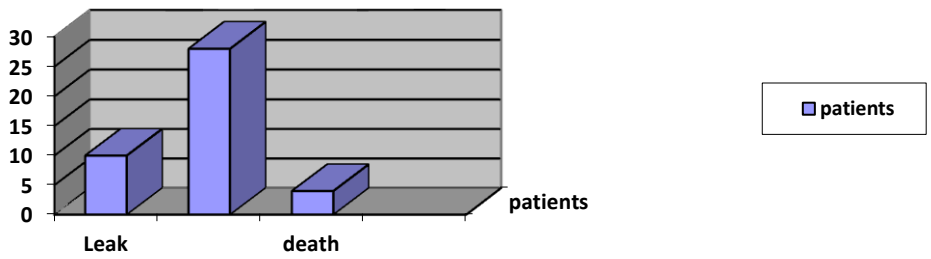
Graph 14: Distribution of patients according to the size of the perforation and type of repair

Table 19: Postoperative Complications

Post -Operative Complications:

In this study series, 34 patients had smooth recovery and 22 patients had suffered from various complications of which 4 patients had expired. The most common postoperative complication was wound infection in about 21 cases , which was one of definitive reasons for prolonged hospital stay

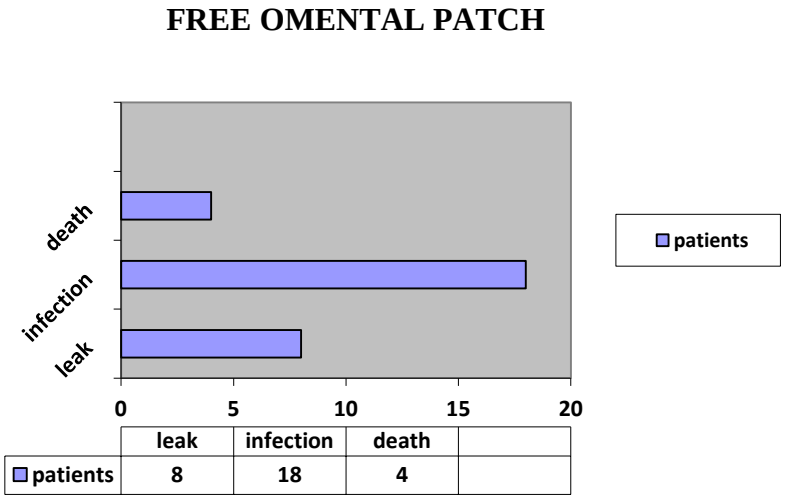
Complications	No. of cases	Percentage
Leak	10	16%
Wound infection	28	46%
Death	4	6.66%

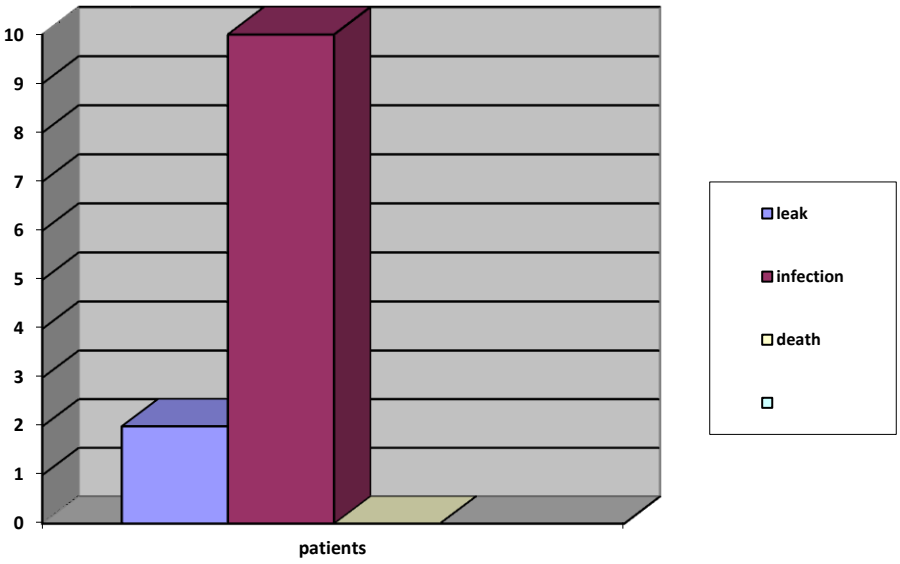


Graph 15: Postoperative complications

In 4 patients two died 3rd to 5th postoperative period, two patients died after 14th postoperative day. These patients presented with severe shock and septicaemia and died due to multiorgan failure.

Graph 16: Type of surgery with its postoperative complications





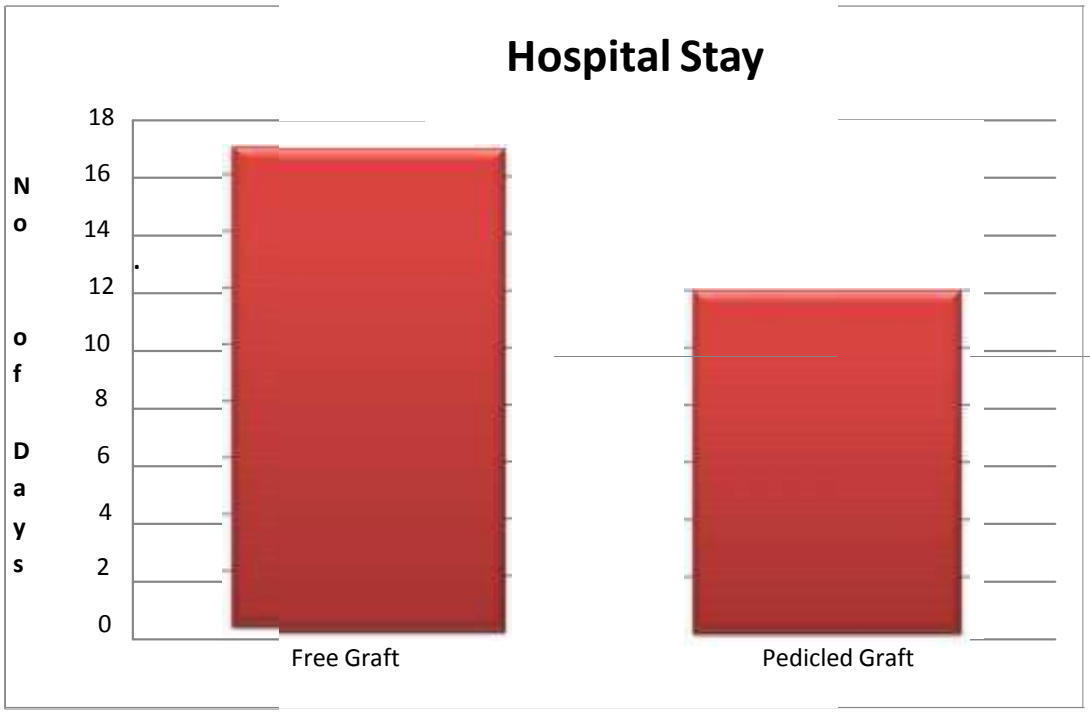
GRAHAMS LIVE OMENTAL PATCH

Duration of Post-Op Hospital Stay:

The average Post-op hospital stay in our series was 11.93 days for Pedicled Omental Patch and 17.03 days for Free Omental Patch with a 'p' value of 0.0001.

Table 21: Duration of hospitalization

Method of treatment	ospitalizati n (in days)
Free Omental Patch	17.03
Pedicled Omental Patch	11.93



Graph 17: Duration of hospitalization

In this present study of 60 patients, 4 patients died and follow up was done in 56 patients for 1 month. These patients were advised with proton pump inhibitor with anti H. pylori therapy.

DISCUSSION

Duodenal perforation is the most common surgical emergency needs admission and aggressive intervention .

Age

Duodenal perforation is common in the age group of >60 yrs in my study, but it can occur at any age.

Table 22: Chart showing peak age incidence by various authors

Author	Peak age in years
M.C.Dandpat et al (1991) [27]	20-40
Samuel J et al (1953) [28]	30-60
Ramesh C et al (1995) [29]	30-50
KalpeshJani et al (2006)[9]	30-50
Present series	>60

Sex:

Perforation incidence is more in males when compared with females. My present study series, the male: female ratio is 4.45:1. The explanation for this high incidence in the male was, that they were subjected to more stress and strain of life and female sex harmone offers some security with them against perforation as claimed by Debakey[10] (1940).

The incidence of smoking and alcohol association also may be contributory factor for males.

Table 23: Table showing sex incidence by various authors

Author	Male: Female Ratio
M.C.Dandpat et al (1991) [27]	10.3:1
Samuel J et al (1953) [28]	13:1
Ramesh C et al (1995) [29]	24:1
KalpeshJani et al (2006)[9]	8.1:1
ɔse n John (Bailey & Love, 2004)[30]	2:1
Present series (2014)	4.45:1

Table 24: time before admission to hospital

ration (in hours)	De Bakey Series (1940) [31]	Bharti C Ramesh et al (1996) [29]		Present series
0-6	50.83%		12.00%	12.00%
6-12	13.02%			14.00%
12-24	4.73%	24.00%		34.00%
> 24	13.60%	64.00%		40.00%

Tsugawa K et al reviewed those three adverse factors: shock at presentation, delayed surgical intervention over 24 hours and associated co morbidities, was shown by the progressive rise in the mortality rate with the increasing number of risk factors (Hepatogastroenterology,2001)[32].

In my study,we reported that the age, site ,size ,duration of perforation, shock at presentation are the Adverse factors for the outcome of perforated peptic ulcers.

In the presence of gross contamination, late exploration (after 48 hours) carried a high mortality i.e. 50% (Boey John et al, 1982) [34]. The importance of the peritoneal soilage and duration of perforation is mentioned as a risk in the outcome of the perforation of duodenal ulcer (Donaldson, 1970) [35]. Bharti C Ramesh et al reported that 12% of patients reached the hospital within 12 hours, 40% reached hospital within 25-48 hours and 24% after 48 hours [29]. Barazynski M et al reported that 48.15% patients presented to hospital after 2 hours of perforation[33]. Fombellid`s J Dens et al (1998) revealed three risk factors of immediate mortality in old age, elapsed time (>24 hours), and the existence of a situation of preoperative hemodynamic shock[36]. Lawel OO et al revealed 20% mortality rate in patients of late presentation and the presence of bacterial peritonitis at admission (1998)[37]. In the present series, 52.5% patients presented to hospital after 24 hours and the mortality in patients who presented to hospital after 24 hours is found to be 9.4%.

Surgical Management:

For perforated duodenal ulcers, two commonly performed procedures are Pedicled Omental grafting (GRAHAMS LIVE PATCH) and Free omental grafting.

The studies done in the past to determine the superior technique were inconclusive because the study results were highly controversial. While most surgeons prefer to use Pedicled Omental graft to preserve vascularity, some studies have proved otherwise. According to a study conducted in 2006, the post-operative leak rates were as higher as 12% in Pedicled Omental graft as compared to 0% in free omentalgraft[9] whereas another study conducted by Chaudhary A, Bose SM et al had proved Pedicled Omental grafting as a superior technique[38].

In the present study, we have done closure of duodenal perforation with Pedicled Omental Patch in 30 patients and Free Omental Patch in 30 patients. We found 13.33% mortality in patients treated with Free Omental Patch and no mortality in patients treated with Pedicled Omental Patch.

Mortality:

Svanes C said that the mortality and morbidity is more in the older[39]. Wysochi A et al[40] reported that the age of a patient, rather than a type surgery, influences the mortality rate in a perforated duodenal ulcer and he reported the mortality rate of 0.6% in <50 years age group, 15% in 50-60 years age group and 45.2% in >60 years age group (1998), in the present series (2014), the mortality in >50 years group is 16.67%.

CONCLUSION

Pedicled Omental Patching was found to be a superior surgical technique over Free Omental Patching for the closure of duodenal perforations measuring upto 20 mm.

- The Post-operative leak rate was significantly lower in patients who underwent Pedicled Omental grafting.

- The mortality rate was lower in patients who underwent Pedicled Omental Grafting even though not statistically significant.

- The average hospital stay was significantly lower in patients who underwent Pedicled Omental grafting.

SUMMARY

A series of 60 cases of duodenal perforations were studied and analyzed at Govt.stanley medical college,chennai. Among them 30 patients underwent closure of duodenal perforation by Pedicled Omental Patching and 30 patients underwent Free Omental Patching. The cases were followed for 1 month. The following observations were made:

1) The peptic perforations more common in the age group of more than 60 years.

2) Male: Female ratio is 4.45:1 (49 male, 11 female patients)

3) Most of the patients were farmers with history of smoking, chewing tobacco and alcohol consumption.

4) In the present series we found 26.66% of post-operative leak (8 patients),

60% of wound infection (18 patients) in patients treated with Free Omental Patch and 6.66% of post-operative leak (2 patients), 33.33% wound infection (10 patients) in patients treated with Pedicled Omental Patch.

5) In the present series we found 13.33% mortality in patients treated with Free Omental Patch and no mortality in patients treated with Pedicled Omental Patch. However the mortality rate was statistically insignificant.(P = 0.04)

6) The average hospital stay in our series was 11.93 days for Pedicled Omental Patch and 17.03 days for Free Omental patch.

In our study, Grahams live Omental Patching was found to be a superior surgical technique over Free Omental Patching for the closure of duodenal perforations measuring upto 20 mm.

BIBLIOGRAPHY

1. Walter E. Dickinson, R. Stuart Ford. Perforated Ulcers. In: Donald E.Fry, editor. Surgical Infections. First edition. Little, Brown and Company; 1973.p.273-277.

2. Bhattacharya Kaushik et al: Peptic ulcer surgery – A historical review. Gastroenterology Today, 2002; 6: 38-40.

3. N. Ananthakrishnan, Rabindra B. Shrivastava. Stomach and duodenum. In: Ahmad A. Hai, editors. ASI Textbook of Surgery, first edition. TATA McGRAW HILL; 2003.p. 400-407.

4. Cellan-Jones CJ, Rapid method of treatment in perforated duodenal ulcer. BMJ 1929; 1: 1076.

5. Graham RR. Treatment of perforated duodenal ulcers. SurgGynecolObstet 1937; 64: 235-238.

6. Raj BR, Subbu K, Manoharan G. Omental plug of large duodenal defects – an experimental study. Trop Gastro 1997; 18(4): 180-182.

7. Sharma D, Saxena A, Rahman H, Free omental plug: a nostalgic look at an old and dependable technique for giant peptic perforations. Dig Surg 2000; 17: 216-218.

8. Lam PWF, Lam MCS, Hui EAL, Mok FPT. Laparoscopic repair of perforated duodenal ulcer: the ‘three stitch’ Graham’s patch technique. SurgEndosc. 2005;

19(12): 1627-30.

9. Jani K, Saxena AK, Vaghasia R. Omental plugging for large sized duodenal peptic perforations: A prospective randomized study of 100 patients. Southern Medical Journal 2006; 99(5):467-471.

10. Neil R. Borley. Duodenum. In: Susan Standring, editor. Gray's anatomy. 39th edition. ELSEVIER CHURCHILL LIVINGSTONE; 2005.p.1163-1166.

11. Vicky B. Tola, David I. Soybel. Anatomy and physiology of the duodenum.In: George D. Zuidema, Charles J. Yeo, editors. Shackelford's Surgery of the Alimentary Tract. Volume No.II, 5th edition. W. B. Saunders Company;2002.p.16-33.

12. David W Mercer, Emily K Robinson. Stomach. In: Courtney M T, editor. Sabiston Textbook of Surgery. Volume no.1, 17th edition. Saunders: Elsevier; 2005.p. 1265-1302.

13. Daniel T. Dempsey. Stomach. In : F. Charles Brunicardi, Dana K. Andersen, Timothy R. Billiar, David L. Dunn, John G. Hunter, Raphael E. Pollock, editors. Schwartz's Principles of Surgery. Eighth edition. Mc GRAW HILL; 2005.p.933-996.

14. John Del Valle. Peptic ulcer disease and related disorders. In: Dennis L. Kasper et al, editors. Harrison's Principles of Internal Medicine. Volume No.II, 16th Edition. Mc GRAW HILL; 2005. p.1746-1762.

15. James M. Crawford. The gastrointestinal tract. In: Ramzi S. Cotran, Vinay Kumar, Tucker Collins, editors. Robbins pathologic basis of disease. 6th Edition. W. B. Saunders Company; 2000. P. 793-796.

16. Peek RM Jr., Blaser MJ: Pathophysiology of Helicobacter pylori-induced gastritis and peptic ulcer disease. Am J Med 1997; 102:200.

17. Kaufman GL: AdvSurg 2000; 34:121.

18. Christopher Wastell. The stomach and duodenum. In: Charles V. Mann, R.C.G. Russel, Norman S. Williams, editors. Bailey & love's short practice of surgery. 22nd edition page. ELBS with Chapman & Hall; 1995. p. 669-700.

19. Ezra Steiger, Avram M. Cooperman: considerations in the management of perforated peptic ulcers, surgical clinics of North America. dec1976; 56(6), p.1395-1401.

20. Sawyers JL, Harrington JL, Jr. et al; Acute perforated duodenal ulcer, Arch Surg 1975; 110:527-530.

21. K. R. Palmer, I. D. Penman. Diseases of the alimentary tract and pancreas. In: Christopher Haslett, Edwin R. Chilvers, John A. A. Hunter, Nicholas A. Boon, editors. Davidson's principles and practice of medicine. 18th edition. CHURCHILL LIVINGSTONE; 2001. p. 599-682.

22. Svanes C: Trends in perforated peptic ulcer: Incidence, etiology and prognosis. World Jr. Surg. 2000; 24(3):277-283.

23. Robert J. Baker. Perforated Duodenal Ulcer. In: Josef E. Fischer, editor.

Mastery of Surgery. Volume No. II. 5th Edition, Lippincott Williams & Wilkins; 2007. p. 892-902.

24. Karanjia ND, Shanahan DJ, Knight MJ: Omental patching of a large perforated duodenal ulcer: a new method. Br J Surg. Jan-1993; 80(1):65.

25. KachintornUdom: Recommended regimens for H. Pylori eradication. Gastroenterology Today, 2000; 4: 160-162.

26. John Boey, John Wong, Ong GB. A prospective study of operative risk factors in Perforated Duodenal Ulcers. Ann Surg 1982; 195: 265-269.

27. M. C. Dandapat, L. M. Mukherjee, S. B. Mishra, P.C. Howlader: Gastro-intestinal perforations, Indian Journal of Surgery 1991; 53(5): 189-93.

28. Samuel J. Stabins, M.D, Rochester, N.Y: The aftermath of perforated duodenal ulcer, Surgery Sept-1953; 34(3):614-620.

29. Ramesh C. Bharti et al: Immediate definitive surgery in perforated duodenal ulcer: A comparative study between definitive surgery and simple closure, IJS Oct-1996; 275-79.

30. John N. Primrose. The stomach and duodenum. In: R.C.G. Russel, Norman S. Williams, Christopher J. K. Bulstrode, editors. Bailey & love's short practice of surgery. 24nd edition. Hodder Arnold; 2004. p. 1026-1046.

31. Debakey ME: Acute perforated gastroduodenal ulceration: A statistical analysis and review of the literature, Surgery, 1940; 8: 852-884.

32. Tsugwa K et al: The therapeutic strategies in performing emergency surgery for gastroduodenal ulcer perforation in 130 patients over 70 years of age. Hepatogastroenterology, 2001; 48(37): 156-162.

33. Barazynski M et al: Perioperative mortality for perforated duodenal and gastric ulcer- Analysis of 226 patients. Przegl. Lek, 1999; 56(3): 192-197.

34. Boey John et al: A prospective study of operative risk factors in perforated duodenal ulcers. Ann. Surg. 1982; 195: 265-269.

35. Donaldson GA et al: Perforated gastroduodenal ulcer disease at the Massachusetts General Hospital from 1952 to 1970. Am. J. Surg. 1970; 120: 306-311.

36. Fombellids J Deus et al: Risk factors in the surgical management of perforated duodeno-pyloric ulcer. Rev. Esp. Enferm. Dig., 1998; 90(7): 502-513.

37. Lawel OO et al: Clinical pattern of perforated pre-pyloric and duodenal ulcer at Ile-Ife, Nigeria. Trop. Doct., 1998; 28(3): 152-155.

38. Chaudhary A, Bose SM, Gupta NM, Wig JD, Khanna SK. Giant perforation of duodenal ulcer. Indian J Gastroenterol1991;10:14-5.

39. Svanes C: Trends in perforated peptic ulcer: incidence, etiology, treatment and prognosis, World Jr. Surg., 2000; 24(3):277-83.

40. Wysocki A et al: Mortality in perforated duodenal ulcer in 35 years of experience, Pr. Eg. Lek. 1998; 55(3): 120-23.

ANNEXURE – I

Informed Consent

Name: Age/ Sex: IP:

I herewith declare that I have been explained in a language fully understood by me regarding the purpose of this study, methodology, proposed intervention, plausible side effects, if any and sequelae.

I have been given an opportunity to discuss my doubts and I have received the appropriate explanation.

I understand that my participation in this study is completely voluntary and that I am free to withdraw from this study at anytime without any prior notice &/ or without having my medical or legal rights affected.

I permit the author and the research team full access to all my records at any point, even if I have withdrawn from the study. However my identity will not be revealed to any third party or publication.

I herewith permit the author and the research team to use the results and conclusions arising from this study for any academic purpose, including but not limited to dissertation/ thesis or publication or presentation in any level.

Therefore, in my full conscience, I give consent to be included in the study and to undergo any investigation or any intervention therein.

Patient's Sign Investigator's Sign

(Dr.RENGANATHAN.M)

Information Module

You are being invited to be a subject in this study.

Before you participate in this study, I am giving you the following details about this trial, which includes the aims, methodology, intervention, possible side effects, if any and outcomes:

I request you to volunteer for this study.

Thanking You,

(Dr.RENGANATHAN.M) Name:

ANNEXURE – II

PROFORMA

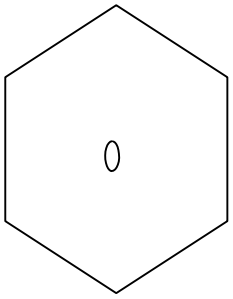
Patient Details	
Name:	**Age:**
Sex: M / F	**Registration no:**
Date of Admission:	**Address:**
Date of Discharge:	

Brief History
Chief Complaints:
1)
2)
3)
Other relevant history:

Examination		
General Examination:	**Vitals**	
	Pulse rate	
	BP	
	Temp	
	Respiratory Rate	

Systemic Examination

P/A:



CVS:

RS:

Other relevant examination:

Provisional Diagnosis:	

Investigations	
CBC	
USG	
Abdomen X-Ray	
Other	

Operation Details	
Name of Surgery:	Exploratory Laparotomy
Date of Surgery:	
Anaesthesia:	
Intra-Op findings:	Site of Perforation: Size(in mm): Nature of free fluid: Other findings:
Type of Omentopexy:	Free Omental Graft / Pedicled Omental Graft

Drain Chart		
Post-Op day	**Quantity (in ml)**	**Nature of Drain**
Day 1		
Day 2		
Day 3		
Day 4		
Day 5		
Further days		

Post-Operative Details	
No. of days of drain requirement	
Total Drain Quantity (in ml)	
No. of days of Post-Op hospital stay	
Persistent symptoms, if any	
Other complications, if any	

ANNEXURE -III

KEY TO MASTER CHART

IP	-	In Patient
M	-	Male
F	-	Female
OCC	-	Occupation
SOP	-	Size of Perforation
DR	-	Drain Requirement (in days)
DQ	-	Drain Quantity (in ml)
WI	-	Wound Infection
POHS	-	Post-Op Hospital Stay

MASTERCHART

FREE OMENTAL GROUP

Sl. No	Name	IP No.	Age	Sex	OCC	SOP	DR	DQ	Leak	WI	Death	POHS
1	Manoharan	46333	19	M	Farmer	3	5	170	No	No	No	11
2	Kumar	46853	20	M	Coolie	4	5	160	No	No	No	22
3	Dass	46895	21	M	Driver	4	4	125	No	No	No	9
4	Karthika	46317	23	F	Student	5	4	105	No	No	No	10
5	Rajendran	47138	27	M	Farmer	6	4	185	No	No	No	14
6	Maniram	47404	29	M	Farmer	6	5	235	No	No	No	12
7	Bharathy	47472	30	F	Teacher	6	4	100	No	No	No	20
8	Manikandan	47500	31	M	Farmer	6	8	490	No	No	Yes	24
9	Jayapaul	48712	33	M	Coolie	7	3	65	No	No	No	10
10	Jafer	48875	34	M	Coolie	7	4	115	No	No	No	16
11	Deepika	47015	34	F	Housewife	7	5	250	No	No	No	15
12	Karupasamy	49012	36	M	Coolie	8	6	340	No	No	No	10
13	Shankar	49104	37	M	Farmer	8	4	205	Yes	Yes	No	11
14	Gajendran	49401	44	M	Farmer	11	3	60	Yes	Yes	No	24
15	Murugesan	49422	45	M	Farmer	11	6	175	Yes	Yes	No	10
16	Indran	49581	45	M	Farmer	11	6	400	Yes	Yes	No	19
17	Kumar	49660	47	M	Farmer	11	5	370	No	Yes	No	18
18	Sikendar	50705	48	M	Farmer	12	4	65	No	Yes	No	18
19	Marimuthu	51420	48	M	Farmer	12	4	125	No	Yes	No	17
20	Divakar	51781	52	M	Farmer	12	4	225	No	Yes	No	19
21	Suman	52833	56	M	Farmer	3	3	140	Yes	Yes	Yes	19
22	Gnanavel	52284	58	M	Business	6	5	100	Yes	Yes	No	19
23	Rajendran	53635	64	M	Farmer	8	6	345	Yes	Yes	Yes	22
24	Moorthy	54183	65	M	Farmer	8	5	220	Yes	Yes	No	26
25	Ameen	542709	65	M	Driver	10	6	270	No	Yes	No	20
26	Hajifathima	47287	65	F	Housewife	11	4	125	No	Yes	Yes	16
27	Jayalakshmi	49818	65	F	Housewife	3	4	110	No	Yes	No	19
28	Manokaran	54842	68	M	Coolie	5	3	60	No	Yes	No	22
29	Abbas	55639	68	M	Farmer	6	4	210	No	Yes	No	18
30	Angamma	52912	70	F	Housewife	8	6	385	No	Yes	No	21

PEDICLED OMENTAL GROUP

Sl. No	Name	IP No.	Age	Sex	OCC	SOP	DR	DQ	Leak	WI	Death	POHS
1	Shanmugam	55225	19	M	Driver	2	3	45	No	No	No	13
2	Venkatesan	1401161	21	M	Student	4	4	120	No	No	No	11
3	Mariyammal	54288	22	F	Student	4	6	150	No	No	No	11
4	Mani	140835	22	M	Coolie	4	4	155	No	No	No	8
5	Lakshmanan	1401686	25	M	Farmer	5	2	40	No	No	No	10
6	Vasanth	1401702	26	M	Farmer	5	5	140	No	No	No	17
7	Gopi	1401685	32	M	Student	6	3	110	No	No	No	12
8	Shankar	1401725	32	M	Business	6	8	220	No	No	No	14
9	Elumalai	1401754	35	M	Coolie	7	8	140	No	No	No	14
10	Mohana	1401766	35	F	Housewife	7	4	110	No	No	No	13
11	Padmanaban	1401816	36	M	Farmer	7	4	140	No	No	No	15
12	Dhamodharan	1401896	38	M	Farmer	8	3	140	No	No	No	12
13	Dhanalakshmi	1401897	39	F	Housewife	8	4	90	No	No	No	10
14	Gurusamy	1401947	41	M	Farmer	8	8	155	No	No	No	11
15	Prakash	1401977	43	M	Farmer	9	4	130	No	No	No	7
16	Veeramani	51816	44	M	Farmer	10	6	155	No	No	No	16
17	Karthick	1402020	48	M	Farmer	11	4	120	No	Yes	No	8
18	Suresh	1401982	52	M	Coolie	12	6	405	No	Yes	No	11
19	Rajendran	1402035	53	M	Farmer	12	5	100	No	Yes	No	11
20	Raman	1402051	53	M	Farmer	14	4	100	No	No	No	11
21	Sagadevan	52548	55	M	Farmer	16	7	255	No	Yes	No	14
22	Anand	1402228	61	M	Coolie	6	3	200	Yes	Yes	No	9
23	Ramesh	1401706	64	M	Farmer	6	6	160	Yes	No	No	16
24	Manjula	1402010	65	F	Housewife	3	5	80	No	Yes	No	14
25	Sekar	1402636	66	M	Farmer	3	5	110	No	No	No	16
26	Balan	1402722	66	M	Farmer	4	4	160	No	Yes	No	8
27	Vellasamy	1402339	68	M	Farmer	4	6	245	No	No	No	11
28	Narayanan	1402732	70	M	Farmer	6	4	105	No	Yes	No	12
29	Sathish	1407741	72	M	Farmer	9	5	280	No	Yes	No	12
30	Jayalakshmi	1402885	74	F	Housewife	13	7	145	No	Yes	No	11