"A STUDY TO COMPARE THE EFFICACY OF FREE OMENTAL GRAFT AGAINST PEDICLED OMENTAL GRAFT IN PREVENTION OF POST OPERATIVE LEAK IN PATIENTS WITH DUODENAL PERFORATIONS"

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This is to certify that the dissertation entitled "**A STUDY TO COMPARE THE EFFICACY OF FREE OMENTAL GRAFT AGAINST PEDICLED OMENTAL GRAFT IN PREVENTION OF POST OPERATIVE LEAK IN PATIENTS WITH DUODENAL PERFORATIONS**" is a bonafide research work done by **Registration No. BH0108006.**

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ABSTRACT

Background:

Duodenal ulcer perforations are a common cause of peritonitis. For almost a century, duodenal perforations have been closed by Omentopexy. In this, a strand of omentum is drawn over the perforation and held in place by full thickness sutures placed on either side of the perforation, and this procedure has become the "gold standard" for the treatment of such perforations. However, occasionally large perforations of the duodenum may be encountered in which there exists the threat of post-operative leakage following closure by this simple method. Commonly, duodenal ulcer perforations are less than 1 cm in greatest diameter, and as such, are amenable to closure by omentopexy. It is generally felt by many surgeons that mobilization of the omentum on its pedicle from the colon, and placement of sutures into the normal duodenum away from the perforation makes the performance 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 Pedicled Omental Graft in preventing post-operative leak 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 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 in terms of post-operative leak rates, post-operative hospital stay, complications & mortality.

Results:

In the present series we found 33.33% of post-operative leak (10 patients), 30% of duodenal fistula (9 patients), 60% of wound infection (18 patients) in patients treated with Free Omental Patch and 6.66% of post-operative leak (2 patients), 3.33% of duodenal fistula (1 patient), 9.68% wound infection (3 patients) in patients treated with Pedicled Omental Patch. 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. The average hospital stay in our series was 11.93 days for Pedicled Omental Patch and 17.03 days for Free Omental patch.

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.

Key words: Duodenal perforation, Pedicled Omental graft, Free Omental graft.

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INTRODUCTION

During the nineteenth century peptic ulcer perforation was a rare disease that occurred mainly in young women, with perforations located near the cardia of the stomach. During the first decades of the twentieth century ulcer perforation incidence increased greatly, and there was an epidemic of ulcer perforations situated in the duodenum of middle aged men. Today ulcer perforation incidence is stable or tends to decline, and most patients with ulcer perforations are elderly men and women. Ulcer perforation was a lethal disease until surgical treatment was introduced at the turn of the century. Mikulicz sutured a perforated gastric ulcer for the first time in 1880, and suture is still the most common treatment for ulcer perforation.

The revolution in ulcer treatment that occurred with discovery of the role of H.Pylori has not yet led to any detectable changes in incidence or treatment of ulcer perforations. Thus ulcer perforation is still a surgical disease for which the possibility for improvement in prognosis lies with the general advances of acute surgery.

Many operations have been proposed to compensate for duodenal perforation/tissue loss. None have gained wide acceptance nor appear to be the best solution to the problem. The reasons cited for disruption of duodenal closures are (1) high intraluminal pressure, (2) the tendency of duodenal mucosa to extrude through closures, adding to the leakage, and (3) breakdown from autodigestive enzymes of pancreas and bile.

Perforation is one of the most catastrophic complications of peptic ulcer. In spite of modern advances in surgical, anesthetic and ancillary facilities, it is still assumes life threatening dimensions. A variety of surgical techniques have been advocated for the management of peptic perforation. However, these techniques are

not without the draw backs, especially while managing large perforations, delayed 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 preventing post-operative leak 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:

It is 5cm long starts as continuation of the duodenal end of the pylorus. The proximal half is mobile; the distal half is fixed. The duodenum passes upward from the pylorus to the neck of the gallbladder. It is related

1. Posteriorly to the common bile duct, portal vein, inferior vena cava, and gastroduodenal artery
2. Anteriorly to the quadrate lobe of the liver
3. Superiorly to the epiploic foramen
4. Inferiorly to the head of the pancreas

The initial 2.5 cm is freely movable and is covered by the peritoneum. The hepatoduodenal portion of the lesser omentum attaches to the superior border of the duodenum. The greater omentum attaches to its inferior border. The distal 2.5 cm is covered with peritoneum only on the anterior surface of the organ, so that the posterior surface is in intimate contact with the bile duct, the portal vein, and the gastroduodenal artery. The duodenum is separated from the inferior vena cava by a small amount of connective tissue[10].

Second (descending) part:

It is 8cm-10cm long, extends from the neck of the gallbladder to the upper border of L4 on a gentle curve. This part of the duodenum is crossed by the transverse colon and the mesocolon and consists, therefore, of a supramesocolic portion and an inframesocolic portion. The parts above and below the attachment of the transverse colon are covered with visceral peritoneum. The first and second parts of the duodenum join behind the costal margin a little above and medial to the tip of the ninth costal cartilage and on the right side of the first lumbar vertebra. The second part of the duodenum forms an acute angle with the first part, and descends from the neck of the gallbladder anterior to the hilum of the right kidney, the right ureter, the right renal vessels, the psoas major, and the edge of the inferior vena cava. It is related anteriorly to the right lobe of the liver, the transverse colon, and the jejunum. At about

the midpoint of the second part of the duodenum, the pancreaticobiliary tract opens into its concave posteromedial side. The right side is related to the ascending colon and the right colic flexure[10].

Third (horizontal) part:

It starts at the inferior duodenal flexure and is 10cm long. It extends from the right side of L3 or L4, across to the left, anterior to the inferior vena cava, and ends in continuity with the fourth part infront of the aorta. It lies posterior to the transverse mesocolon, the origin of the small bowel mesentery and the superior mesenteric vessels. It is anterior to the ureter, the right gonadal vessels, the psoas muscle, the inferior vena cava, the lumbar vertebral column, and the aorta.Inframesocolic portion of the duodenum is covered anteriorly by the peritoneum. The third part is related superiorly to the head and uncinate process of the pancreas. The inferior pancreaticoduodenal artery lies in a groove at the interface of the pancreas and the duodenum. Anteriorly and inferiorly, this part of the duodenum is related to the small bowel, primarily to the jejunum[10].

Fourth (ascending) part:

It is 2.5 cm long. It extends from the left side of the aorta to the left upper border of L2. It is directed obliquely upward. It ends at the duodenojejunal junction to the left and at the level of the second lumbar vertebra at the root of the transverse mesocolon. This junction occurs at about 4 cm below and medial to the tip of the ninth costal cartilage. It is related posteriorly to the left sympathetic trunk, the psoas muscle, and the left renal and gonadal vessels. Its termination is very close to the terminal part of the inferior mesenteric vein, to the left ureter, and to the left kidney. The duodenojejunal junction is suspended by the ligament of Treitz, a remnant of the

dorsal mesentery, which extends from the duodenojejunal flexure to the right crus of the diaphragm[10].

Blood supply:

Gastro-duodenal artery is a branch of common hepatic artery, before its division into the right gastro-epiploic and superior pancreatico-duodenal branches it gives retroduodenal branches that supply first and proximal portion of the second part. It also gives supraduodenal artery supplies superior aspect of the first part.

Superior pancreatico-duodenal artery supplies branches to the first and second part, and anastomosis with the anterior division of inferior pancreatico-duodenal artery.

Inferior pancreatico-duodenal artery arises from superior mesenteric artery. It directly divides into anterior and posterior branch. It supplies the second and third part.

First jejunal branch of the superior mesenteric artery supply the fourth part and anastomosis with inferior pancreatico-duodenal artery[10].

Venous drainage:

Duodenal veins drains into the portal vein through the pancreatico-duodenal veins accompanying corresponding named arteries[10].

Lymphatic drainage:

Duodenal lymphatics run to anterior and posterior pancreatic nodes that lie in the anterior and posterior grooves between the pancreatic head and the duodenum. These drain into the suprapyloric, infrapyloric, hepatoduodenal, common hepatic and superior mesenteric nodes[10].

Nerve supply:

Sympathetic fibres originate from the neurons in the 5th to 12th thoracic spinal segments. These travel via the greater and lesser splanchnic nerves to the coeliac plexus where they synapse on neurons in the celiac ganglion. Postganglionic axons are distributed via periarterial plexus on the branches of coeliac axis and superior mesenteric artery. Parasympathetic supply is from the vagus nerve via branches from coeliac plexus[10].

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:

The greater omentum is nothing but a large fold of peritoneum that hangs down from the greater curvature of the stomach like an apron to a variable extent over the intestinal loops. It appears as a thin fenestrated membrane with varying amount of fat deposition. Apart from serving as a storehouse of fat, it protects the peritoneal cavity from infections by virtue of its "milky spots," which are nothing but collections of macrophages. It is also said to limit the spread of intraperitoneal infections by

moving to the concerned site and sealing it from the nearby area, a function that has made the British surgeon Rutherford Morrison to rightly name it as "The policeman of the abdomen" in the early 20th century. In spite of all these valuable functions, the omentum is often forgotten.

In the past, the omentum was considered to be an inert tissue without much biological significance. But since the beginning of the last century, innumerable studies and trials have been conducted by surgeons and scientists all over the world, which have proven that the omentum is a unique, physiologically dynamic tissue with immense therapeutic potential.

It is a highly vascular organ with a rich source of angiogenic factors that promote the growth of blood vessels into whatever tissue it is placed close to. The potent lymphatic system of the omentum can absorb enormous amounts of edema fluids and remove metabolic wastes and toxic substances. Recent studies have revealed that the omentum, apart from being a great source of various growth factors, neurotransmitters, neurotrophic factors and inflammatory mediators, also contains omnipotent stem cells that can differentiate into a variety of cell types. It provides an excellent plastic material against inflammation and irradiation.

Anatomically, the attachments of the omentum and the arrangement of the vascular arcades are in such a way that it can be lengthened to an unbelievable extent at a vascular pedicle, exteriorized or detached using microvascular techniques for various intra-abdominal and extra-abdominal procedures. The arterial and venous supply to the omentum are derived from the branches of the right and left gastroepiploic vessels that course along the greater curvature of the stomach. Division of the right or left gastroepiploic artery and the vasa recta along the greater curvature

of the stomach with mobilization of the omentum from the transverse colon allows the development of a vascularized omental pedicle flap (Cellan-Jones graft).

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].

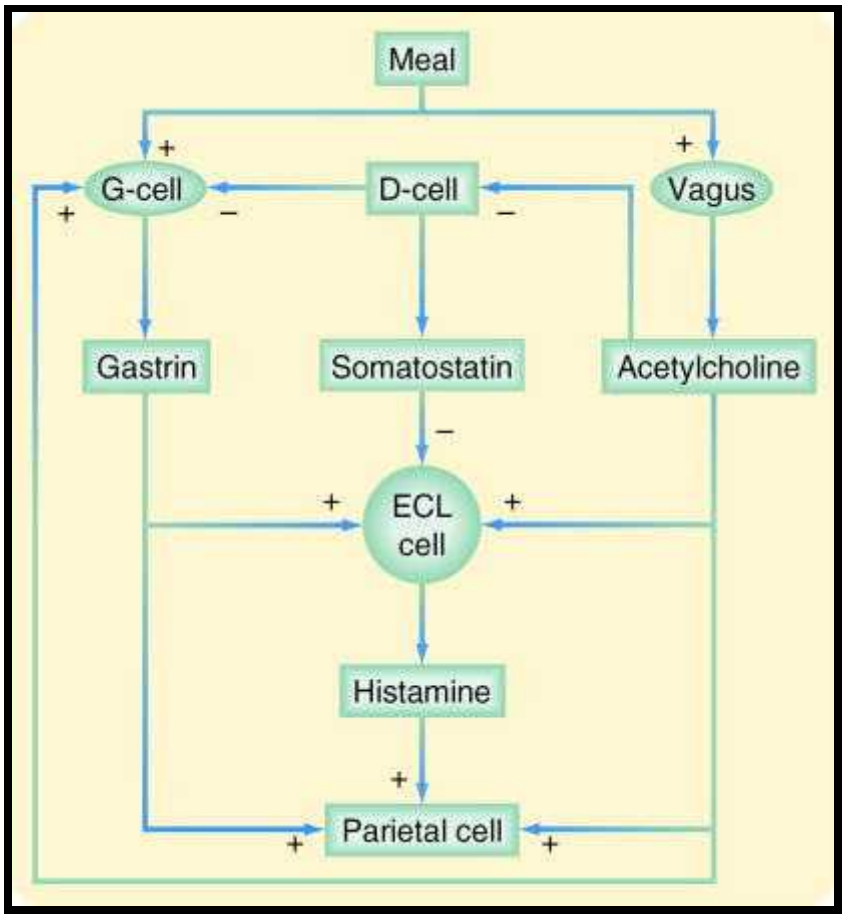


Figure 1: **Role of ECL cell in regulation of acid secretion by the parietal cell[12].**

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, there is always a basal level of acid secretion that is roughly 10% of maximal acid output (MAO). Basal acid secretion also exhibits a circadian variation, with night-time acid secretion being greater than daytime secretion. Under basal conditions, 1 to 5 mmol of hydrochloric acid is secreted, and this is reduced by 75% to 90% following vagotomy or administration of atropine. However, H2 - receptor blockade diminishes the magnitude of acid secretion by 90%. Thus, it appears likely that basal acid secretion is due to a combination of cholinergic and histaminergic input[12].

Stimulated Acid Secretion

Three phases of the acid secretory response to a meal have been described: cephalic, gastric, and intestinal. These three phases are inter-related and occur concurrently, not consecutively[12].

Cephalic phase

The cephalic phase originates with the sight, smell, thought, or taste of food, which stimulates neural centers in the cortex and hypothalamus. These higher centers transmit signals to the stomach by the vagus nerves, which release acetylcholine that in turn activates muscarinic receptors located on target cells. Acetylcholine directly increases acid secretion by the parietal cell and can both inhibit and stimulate gastrin release, the net effect being a slight increase in gastrin levels. It accounts for only 20% to 30% of the total volume of gastric acid produced in response to a meal in humans because of the short duration of the cephalic phase[12].

Gastric phase

The gastric phase of acid secretion begins when food enters the gastric lumen. Digestion products of ingested food interact with microvilli of antral G cells to stimulate gastrin release. Food stimulates acid secretion by causing mechanical distention of the stomach. Gastric distention activates stretch receptors in the stomach to elicit the long vagovagal reflex arc, independent of changes in serum gastrin levels. However, antraldistention also causes gastrin release in humans, and this reflex has been called the pyloro-oxyntic reflex. In humans, mechanical distention of the stomach accounts for about 30% to 40% of the maximal acid secretory response to a peptone meal, with the remainder due to gastrin release. The entire gastric phase accounts for most (60% to 70%) of meal-stimulated acid output, because it lasts until the stomach is empty[12].

Intestinal phase

The intestinal phase of gastric secretion remains poorly understood but appears to be initiated by entry of chyme into the small intestine. It occurs after gastric emptying and lasts as long as partially digested food components remain within the proximal small bowel. It accounts for only 10% of the acid secretory response to a meal and does not appear to be mediated by serum gastrin levels. It is hypothesized that a distinct acid-stimulatory peptide hormone (entero-oxyntin), which is released from small bowel mucosa, may mediate the intestinal phase of acid secretion[12].

CELLULAR BASIS OF ACID SECRETION

Gastrin receptors

These receptors are members of the classic G-protein–coupled 7-transmembrane-spanning receptor family and are classified as either type A or type B CCK receptors. The gastrin or CCK-B receptor has high affinity for both gastrin and CCK, whereas the type A CCK receptors have affinity for sulfated CCK analogues and a low affinity for gastrin. Binding of gastrin to the CCK-B receptor is coupled to the calcium signaling pathway[12].

Muscarinic receptors

Acetylcholine exerts its effect on the parietal cell through interactions with the M3 subtype of the muscarinic receptor family. This receptor is coupled to increased levels of intracellular calcium, mediated by phospholipase-induced production of inositol trisphosphate[12].

Histamine receptors

Histamine receptors are members of the family of G-protein–coupled 7-transmembrane-spanning receptors. On the parietal cell, the H2 subtype binds histamine to activate adenylatecyclase, which in turn leads to an increase in intracellular cyclic adenosine monophosphate (AMP) levels[12].

Somatostatin receptors

Somatostatin receptors are also 7-transmembrane-spanning receptors; there are at least five different types. Binding of somatostatin with its receptors is coupled to one or more inhibitory guanine nucleotide–binding proteins. Somatostatin can inhibit

parietal cell secretion via both G-protein– dependent and G-protein–independent mechanisms[12].

Second messengers

The two second messengers principally involved in stimulation of acid secretion by parietal cells are intracellular cyclic AMP and calcium[12].

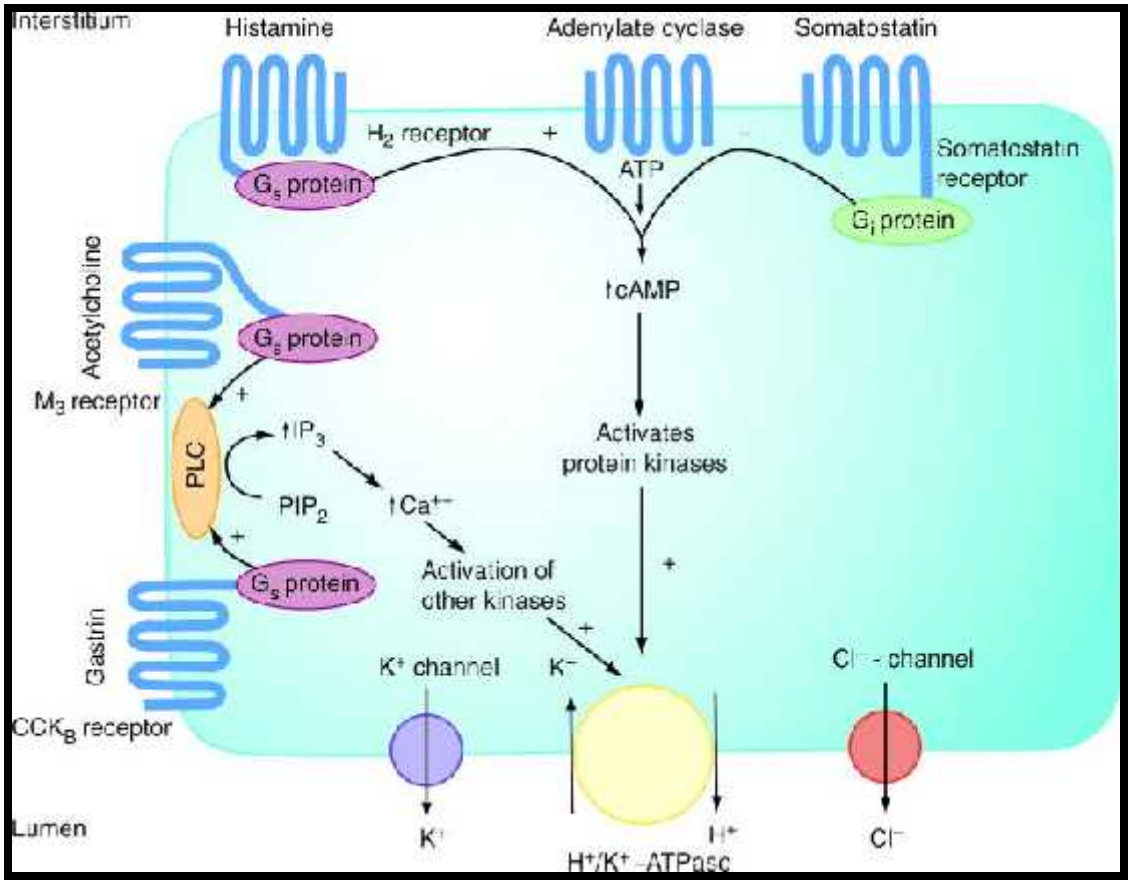


Figure 2: **The intracellular events following ligand binding to the parietal cell**

PEPTIC ULCER DISEASE

Peptic ulcers are focal defects in the gastric or duodenal mucosa which extend into the submucosa or deeper. They may be acute or chronic, and ultimately caused by an imbalance between the action of peptic acid and mucosal defences[13].

Epidemiology:

Annual incidence of active ulcer in United States is 1.8%. It is a chronic and recurrent disease hence the prevalence is always considerably higher than the incidence of new cases. Peptic ulcer disease is increased in women and decreased in men, due to increase in smoking and NSAID ingestion. H.pylori represents the most drastic change in our understanding of peptic ulcer disease and has led many experts to conclude that peptic ulcer disease is in reality an infectious disease. Human gastric bacteria were first discovered in early 1900s. In 1970s, gastric bacteria were rediscovered and found to be associated with inflammation. 12years later, the first successful culture of the organism was accomplished by Marshall and Warren who named it Campylobacter pyloridis. In 1987, eradication of the organism reduced duodenal ulcer recurrence. In 1989 it is reclassified into H.pylori. Over the last 2 decades, there has been an increase in emergency operations performed for complications of duodenal peptic ulcers. While the number of operations for an elective indication has decreased markedly[12]. In 1998, the crude mortality rate for peptic ulcer was 1.7/100,000 individuals. Recent studies have shown an increase in the rates of hospitalization and mortality in elderly patients for the peptic ulcer complication of bleeding and perforation[13]. Duodenal ulcers are estimated to occur in 6-15% of western population. Gastric ulcers occur later in life than duodenal lesions with peak incidence in 6th decade[14]. Male to female ratio for duodenal ulcer is about 3:1 and gastric ulcer is 1:5 to 2:1. Women most often affected at or after menopause[15].

Location and Type of Ulcer

It is divided depending on the site into gastric and duodenal ulcers, depending on the duration into acute and chronic ulcers. Duodenal ulcer disease is a disease of multiple etiologies. The only absolute requirements are secretion of acid and pepsin in conjunction with either H. pylori infection or ingestion of NSAIDs[12].

Gastric ulcer present in four forms[12].

- Type 1 gastric ulcers are most common, accounting for about 60% to 70% of the total. Typically, they are located on the lesser curvature at or proximal to the incisura. Most are associated with diffuse antral gastritis or multifocal atrophic gastritis.
- Type 2 gastric ulcers (15 %) occur in the same location as the type 1 lesions but are associated with either active are chronic duodenal ulcer disease.
- Type 3 gastric ulcers (20%) are typically located within 2 cm of the pylorus (pyloric channel ulcer).
- Type 4 gastric ulcer is located in the proximal stomach or in the gastric cardia and is rare.

Types 2 and 3 gastric ulcers appear to behave more like duodenal ulcers and are associated with excess acid, whereas types 1 and 4 gastric ulcers are not[12].

PATHOGENESIS

Helicobacter pylori Infection

It is associated with 90% of duodenal ulcers and 75% of gastric ulcers. It is spiral or helical gram negative rod with 4 to 6 flagella that resided in gastric-type epithelium. Its shape and flagella aided its movement through the mucus layer, and it was also found to produce a variety of enzymes that helped it adapt to a hostile environment. It is a most potent producer of urease of any bacteria. Splitting urea into ammonia and bicarbonate leads to an alkaline microenvironment in the setting of an acidic gastric milieu. The organism is microaerophilic, and the optimal temperature for isolation is 35°C to 37°C, with growth occurring after 2 to 5 days. It lives only in gastric epithelium because gastric epithelium expresses specific adherence receptors in vivo that can be recognized by the organism. Thus, it can also be found in heterotopic gastric mucosa in proximal esophagus, Barrett's esophagus, gastric metaplasia in the duodenum, Meckel's diverticulum, and heterotopic gastric mucosa in the rectum[12].

Three potential mechanisms for H. pylori–induced gastrointestinal injury are[12]

1. Production of toxic products to cause local tissue injury
2. Induction of a local mucosal immune response
3. Increased gastrin levels with a resultant increase in acid secretion.

Some of the locally produced toxic mediators include breakdown products from urease activity (i.e., ammonia); cytotoxins; a mucinase that degrades mucus and glycoproteins; phospholipases that damage epithelial cells and mucus cells; and

platelet-activating factor, which is known to cause mucosal injury and thrombosis in the microcirculation[12].

H.pylori is known to cause a local inflammatory reaction in the gastric mucosa and to produce chemotactic factors that attract neutrophils and monocytes. Activated monocytes and neutrophils in turn produce a number of proinflammatory cytokines and reactive oxygen metabolites. H.pylori appears to directly induce production of interleukin 8 by gastric epithelial cells, which is known to be proinflammatory. Subsequent stimulation of CD4+ T cells also leads to the production of a variety of cytokines. These cells can further stimulate B cells to differentiate into specific antibody-producing cells, and IgM, IgG, and secretory IgA production follows. In addition, H.pylori causes a gastric mucosal neutrophil and mononuclear cell infiltration that leads to epithelial damage and lymphoid follicle formation, which is not normally present in the gastric mucosa. H. pylori infection causes reduction of antral D cells leads to a reduction in somatostatin levels, which leads to an increase in serum gastrin levels due to disinhibition of the G cells with a resultant increase in serum gastrin and antral gastrin levels. H. pylori represents a chronic infection found worldwide. Once a person is infected, usually in childhood, it is probably for life because spontaneous remission is rare. There tends to be an inverse relationship between infection and socioeconomic status. The reasons for this remain poorly understood but may be due to factors such as sanitary conditions, familial clustering, and crowding[12].

NSAIDs

The prevalence of peptic ulcer in chronic NSAID users is about 25% (15% gastric and 10% duodenal). 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 over age 60. 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. Factors that clearly put patients at increased risk for NSAID-induced GI complications include age over 60, prior GI event, high NSAID dose, concurrent steroid intake, and concurrent anticoagulant intake[13]. After H. pylori infection, ingestion of NSAIDs is the most common cause of peptic ulcer disease. NSAIDs increase the risk of gastrointestinal complications approximately 2- to 10-fold. The acute gastroduodenal lesions typically appear within 1 to 2 weeks of ingestion of the NSAIDs and range from mucosal hyperemia to superficial gastric erosions. In contrast, chronic injury typically occurs after 1 month and may be seen in the stomach as erosions or ulcerations in the gastric antrum or in the duodenum. NSAID-induced ulcers are more frequently found in the stomach[12].

Smoking, Stress, and Other Factors

Epidemiologic studies suggest that smokers are about twice as likely to develop peptic ulcer disease as nonsmokers. Smoking increases gastric acid secretion and duodenogastric reflux. Smoking decreases both gastroduodenal prostaglandin production and pancreaticoduodenal bicarbonate production. 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 ulcer) [13].

Duodenal Ulcer Pathophysiology

Duodenal ulcer is a disease of multiple etiologies.Frequency of secretory abnormalities is more in duodenal ulcer patients. Many secretory abnormalities are

found in patients with duodenal ulcer disease and clearly not every patient has the same secretory abnormalities[12].

- Decreased duodenal bicarbonate secretion: 70%
- Increased nocturnal acid secretion: 70%
- Increased duodenal acid load: 65%
- Increased daytime acid secretion: 50%
- Increased pentagastrin-stimulated MAO: 40%
- Increased sensitivity to gastrin: 35%
- Increased basal gastrin: 35%
- Increased gastric emptying: 30%
- Decreased pH inhibition of gastrin release: 25%
- Increased postprandial gastrin release: 25%[12]

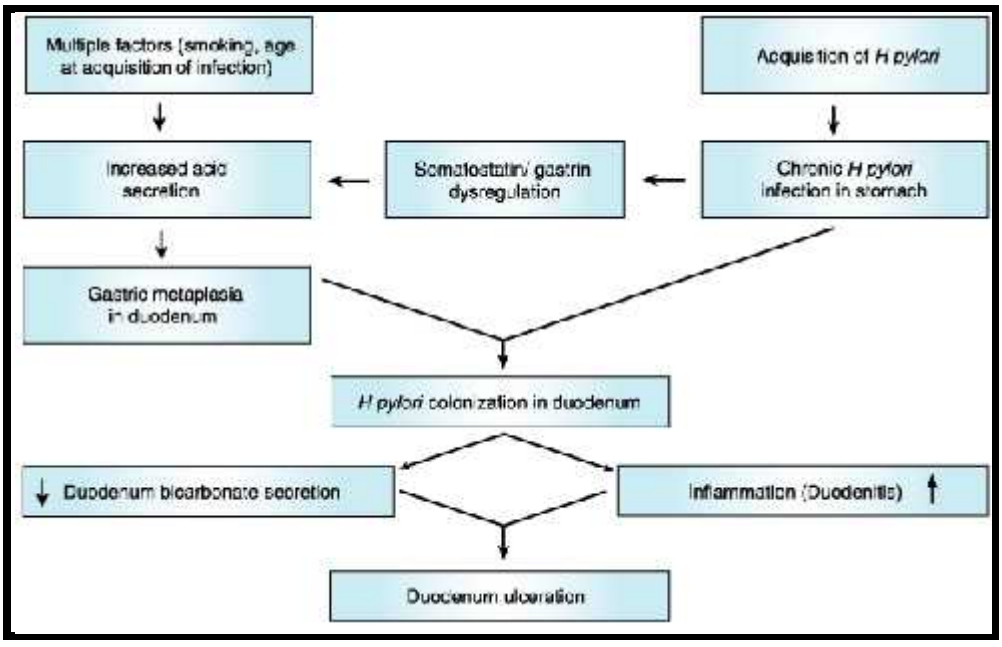


Figure 3: Model of *H. pylori*–induced effects on duodenal ulcer pathogenesis[16].

Gastric Ulcer Pathophysiology

Conditions that may predispose to gastric ulceration are age older than 40 years, sex (female:male, 2:1), ingestion of barrier-breaking drugs such as aspirin or NSAIDs, abnormalities in acid and pepsin secretion, gastric stasis through delayed gastric emptying, coexisting duodenal ulcer, duodenal gastric reflux of bile, gastritis, and infection with H. pylori. Clinical conditions that may predispose to gastric ulceration include chronic alcohol intake, smoking, long-term corticosteroid therapy, infection, and intra-arterial therapy. With regard to acid and pepsin secretion, the presence of acid appears to be essential to the production of gastric ulcer; however, the total secretory output appears to be less important[12].

Mechanism of gastric mucosal damage by H.pylori

- Local effects
    - Elaboration of toxins
        - VacA
        - CagA
- Effect on immune response
    - Elaboration of cytokines
    - Elaboration of interleukin 8
    - Recruitment of inflammatory cells
    - Release of inflammatory mediators
    - Production of immunoglobulins

- Effect on acid secretion
    - Initial hypochlorhydria
    - Subsequent hyperchlorhydria
    - Elevated serum gastric levels
    - Reduced gastric antralsomatostatin levels
    - Increased levels of gastric fundic N-methylhistamine
    - Hypergastrinemia may contribute to greater parietal cell mass
- Effect on duodenal bicarbonate secretion
    - Reduced secretion of duodenal bicarbonate in patients colonized with *H. pylori*

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 spreads 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 nasogastric 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 H.PYLORI INFECTION

Following are the different methods for the 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**: This test is based on the production of urease by H.Pylori. 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. **Pedicled 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].

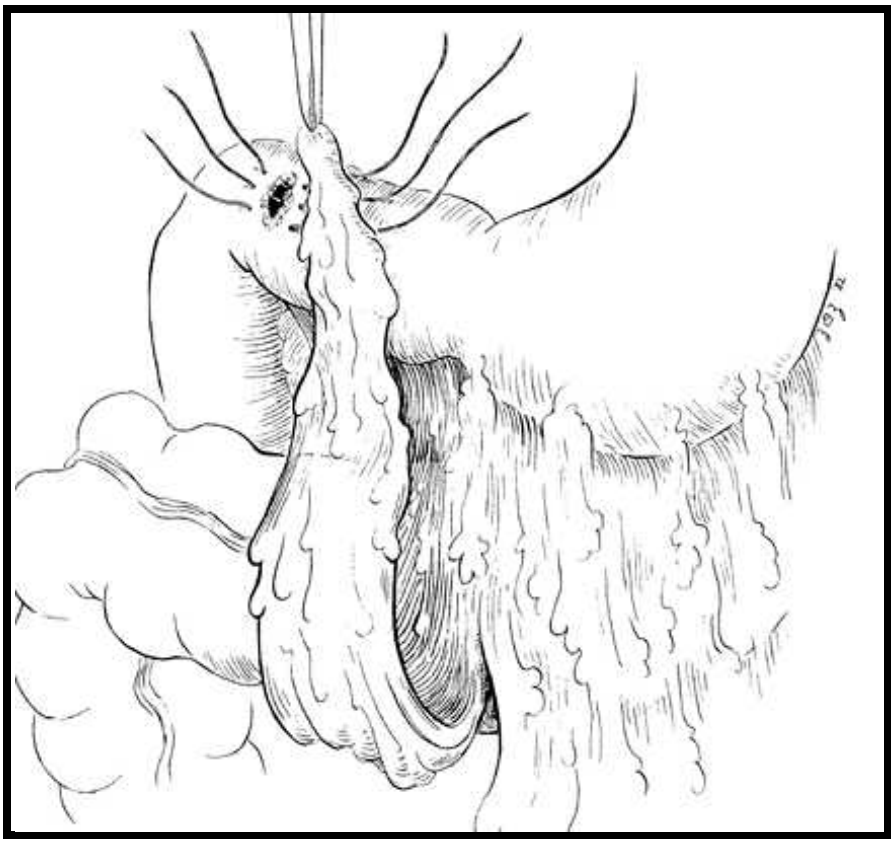


Figure 4: Placement of Pedicled Omental Patch over perforation site

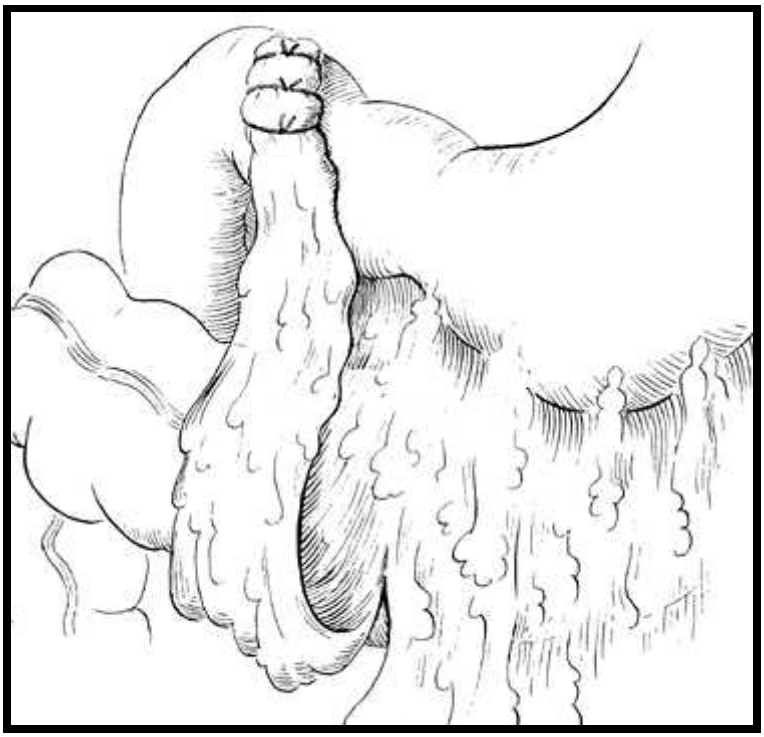


Figure 5: Sealed perforation with Pedicled Omental Patch.

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

b. Sex

c. Duration of perforation.

d. General conditions of the patient.

e. Presentation with shock.

f. Presentation with rental failure.

g. Ulcer history.

h. Concomitant second complication

i. Concomitant medical / surgical illness

j. Site and size of perforation.

k. Extent of contamination of peritoneum

l. Perforation of malignant ulcer.

m. Postoperative complication.

MATERIALS AND METHODS

A study of 60 patients admitted with duodenal perforations at KLES Dr. Prabhakar Kore Hospital & Medical Research Center, Belgaum was undertaken from December 2008 to December 2009. 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& 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: Pedicled 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 stable. In critically ill patients, the patients were resuscitated and history was taken after the patient is stabilized.

The hospital records were also reviewed to obtain appropriate epidemiological information regarding age, sex, occupation, and clinical presentation, duration of symptoms, past history of chronic duodenal ulcer, investigations and mode of treatment. This data was essential to evaluate the condition of the patient at the time of admission, duration between perforation and surgery, mode of treatment patient received and post-operative complications and 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 abdomen of erect posture, gas 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

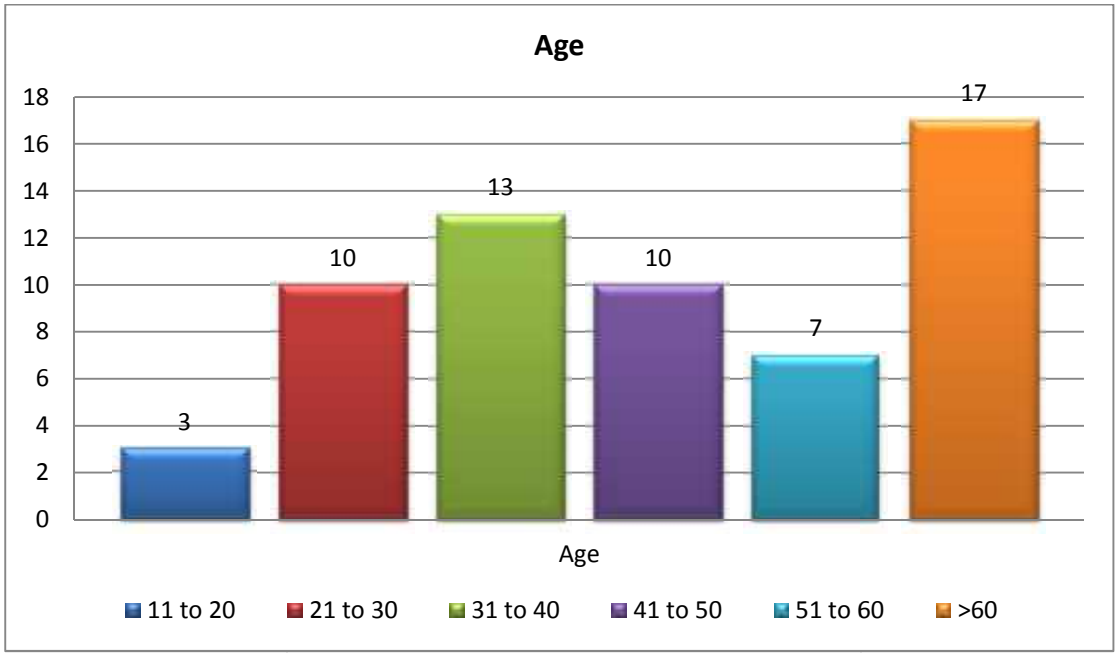
RESULTS

From December 2008 to December 2009 a total of 60 patients with duodenal perforations were studied from surgical units of KLES Dr. Prabhakar Kore Hospital & Medical Research Center, Belgaum.

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

Table 4: The age incidence:

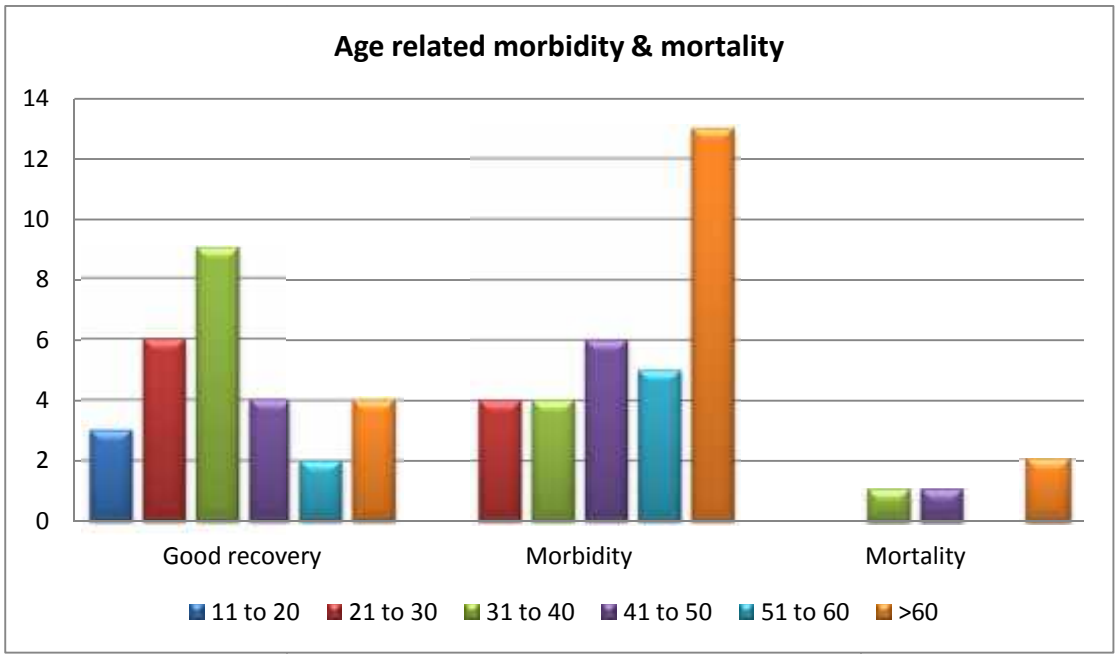
Age group (in years)	No of cases	Percentage
11 – 20	3	5
21 – 30	10	16.66
31 – 40	13	21.66
41 – 50	10	16.66
51 – 60	7	11.66
> 60	17	28.33



Graph 1: The age incidence

Table 5: Age related morbidity and mortality

Age group (in years)	No. of cases	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



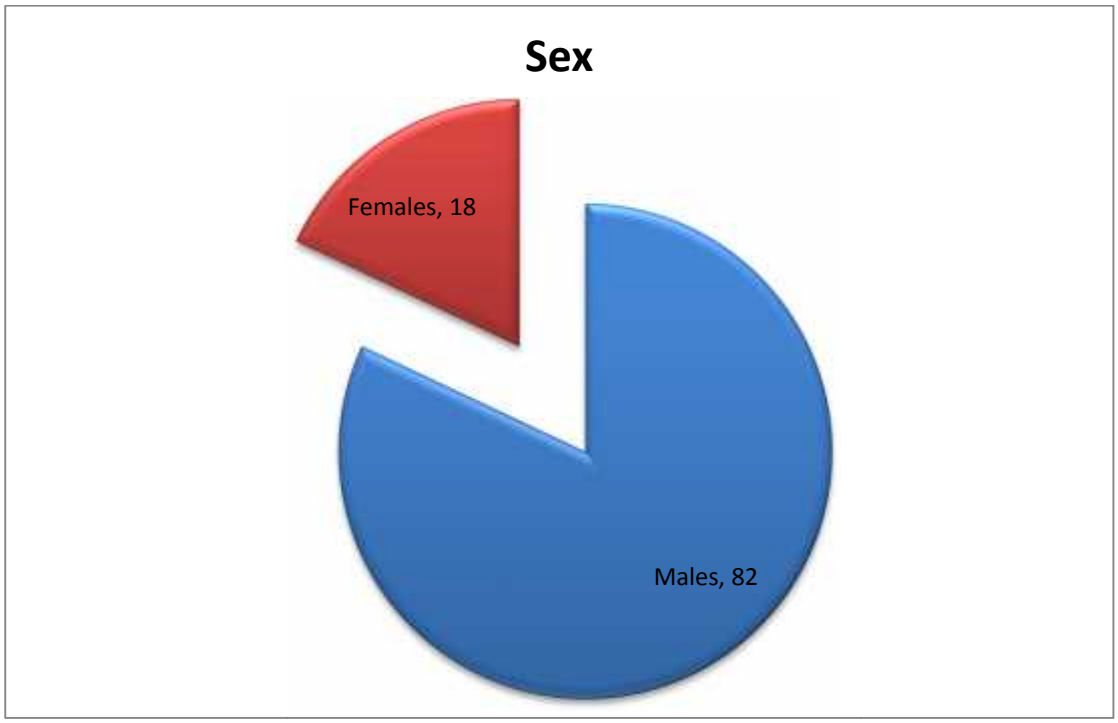
Graph 2: Age related morbidity and mortality

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



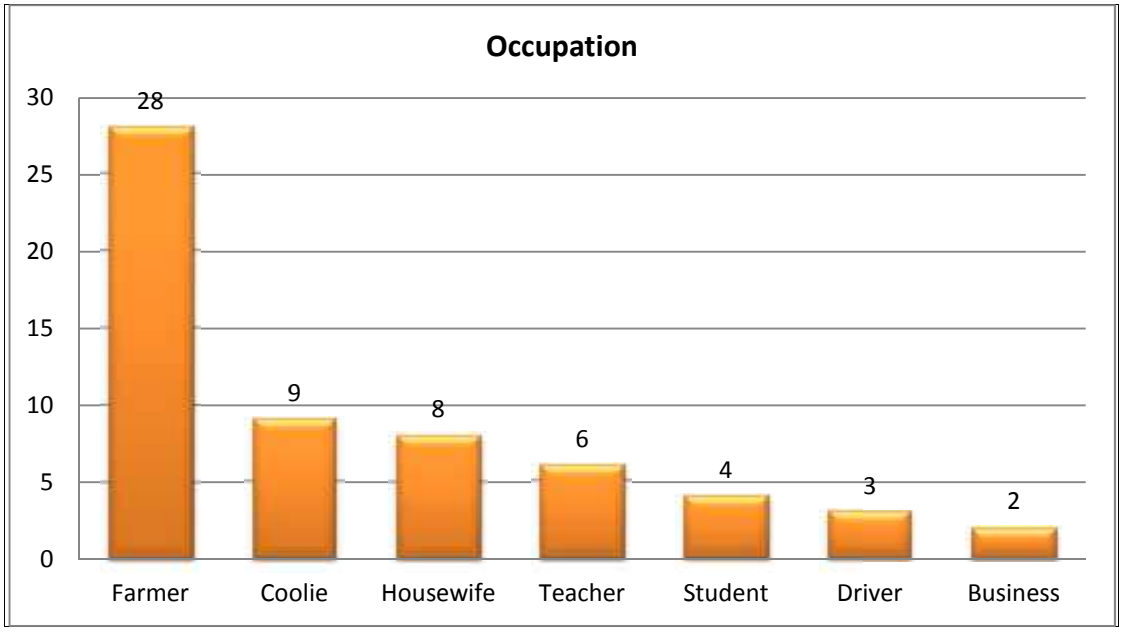
Graph 3: Sex incidence of duodenal perforation

Occupation:

Perforation is more common in farmers in present study.

Table 7: Occupation incidence

Occupation	No. of patients	Percentage
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



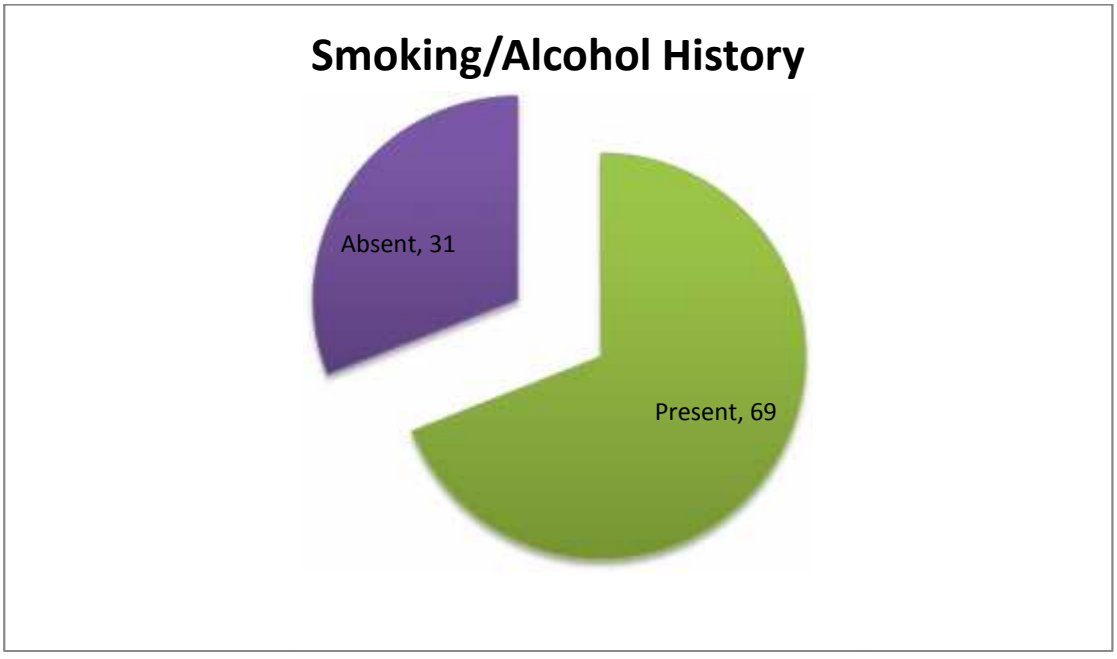
Graph 4: Occupation incidence

Relation with smoking and alcohol:

In this series of study, there was an obvious relationship between the smoking, 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



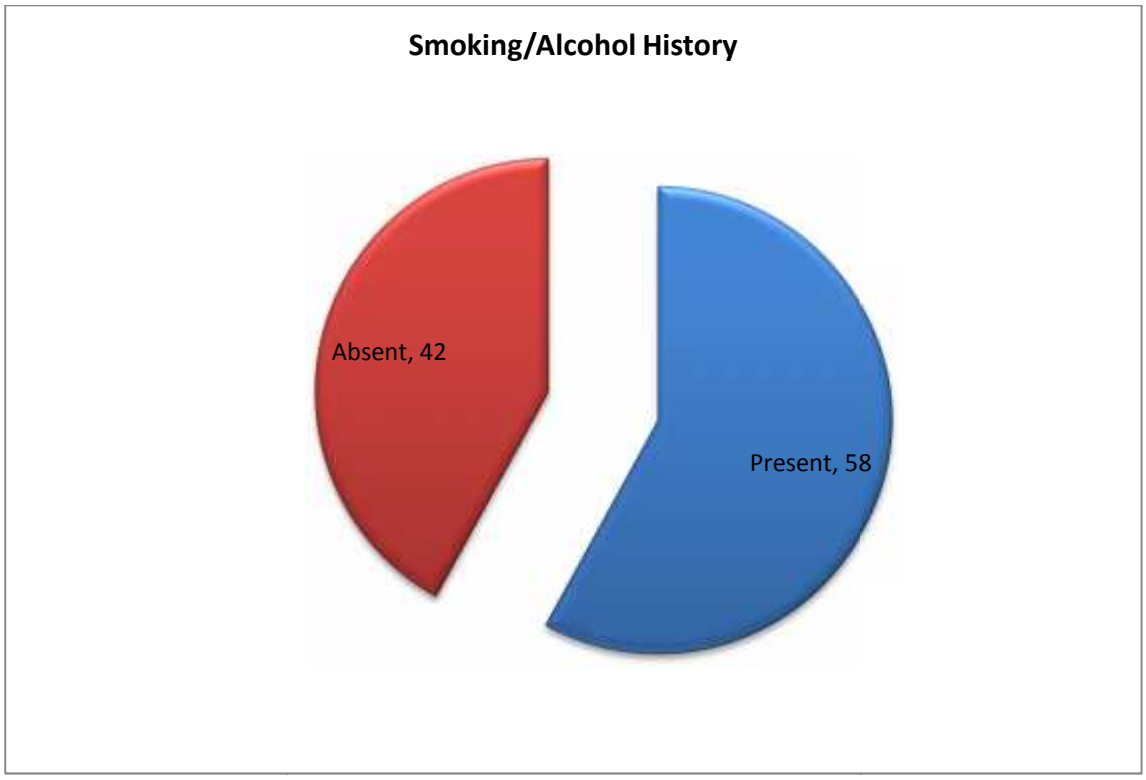
Graph 5: Relation of smoking to incidence of perforation

Previous history of peptic ulcer:

In our series 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



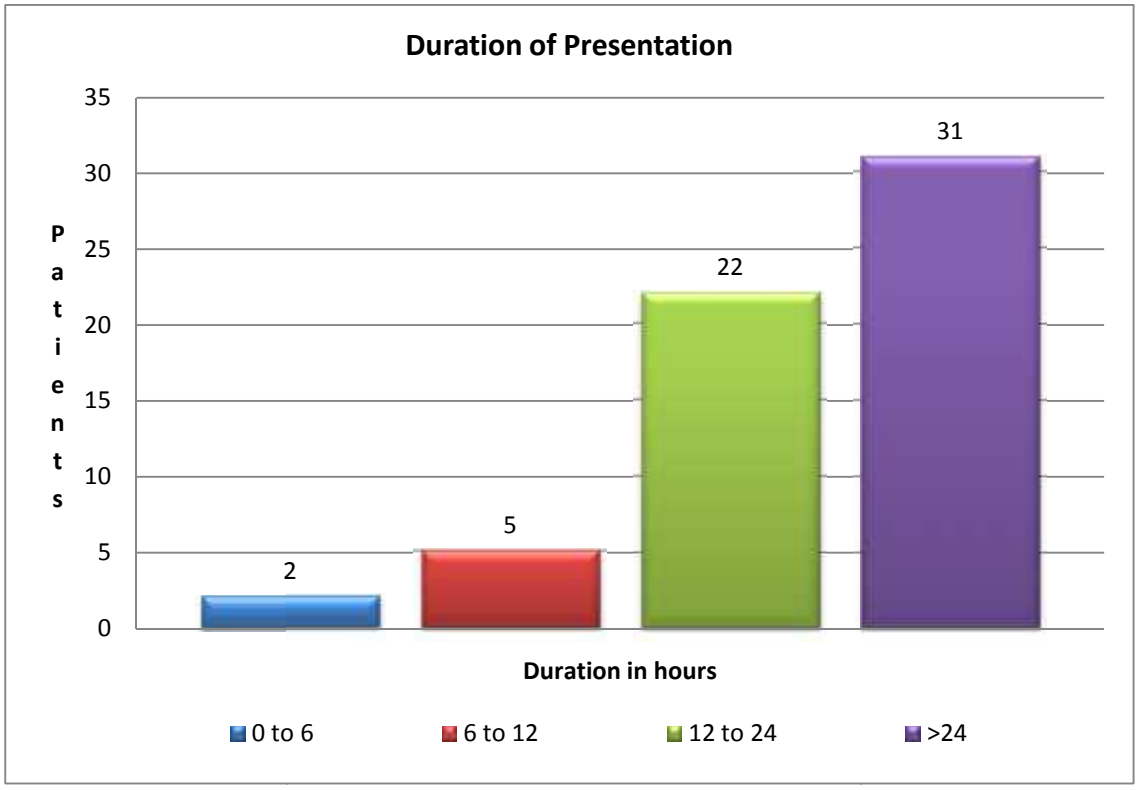
Graph 6: Previous history of peptic ulcer

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



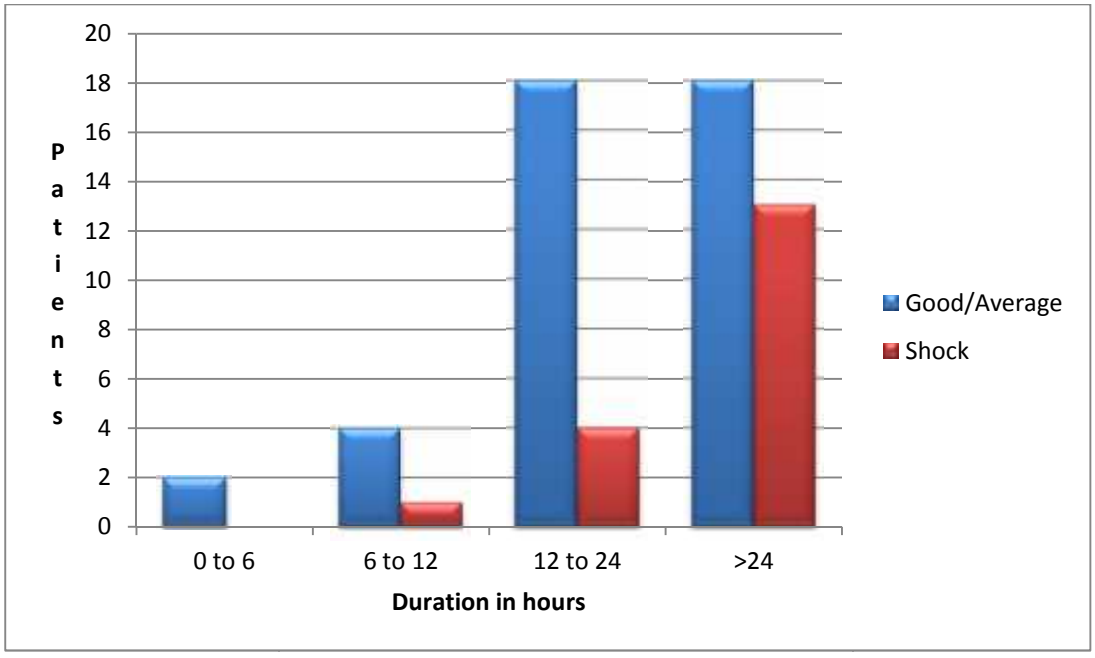
Graph 7: Duration of presentation

The delayed presentation had significant impact on increased morbidity/ mortality of the patients.

Table 11: Impact of duration on the general condition

Duration (in hrs)	No of cases	General condition 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

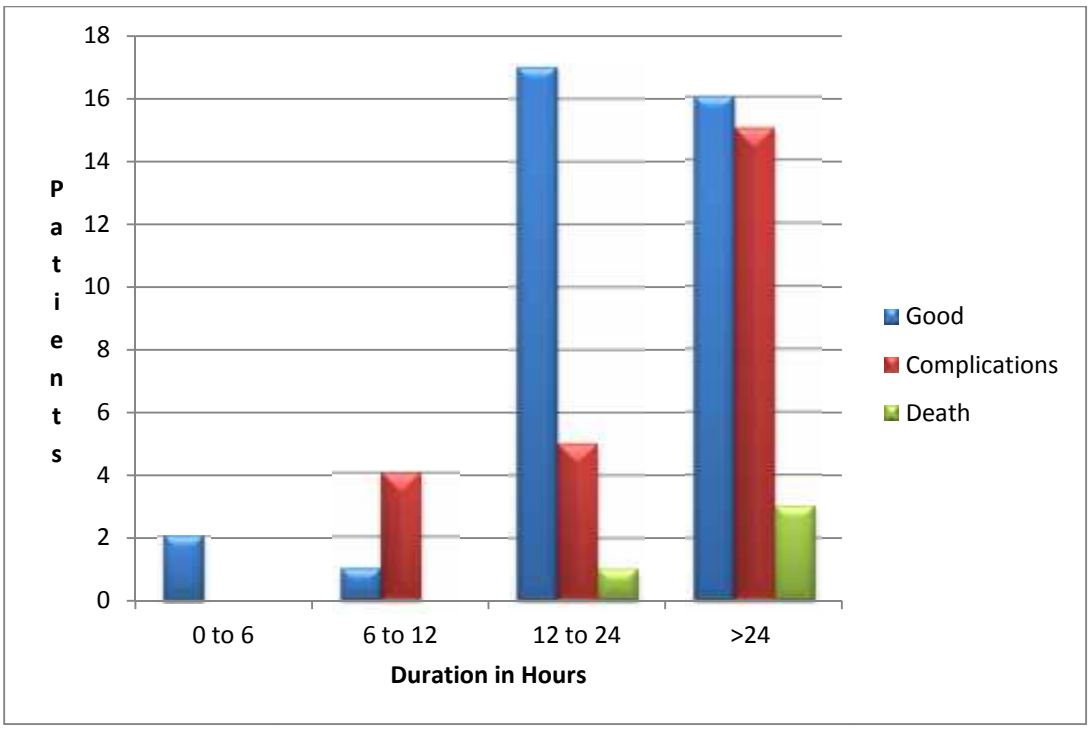
The poorer the general condition of the patient, poor is the final outcome of the patient.



Graph 8: Impact of duration on the general condition

Table 12: Impact of duration on the outcome

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



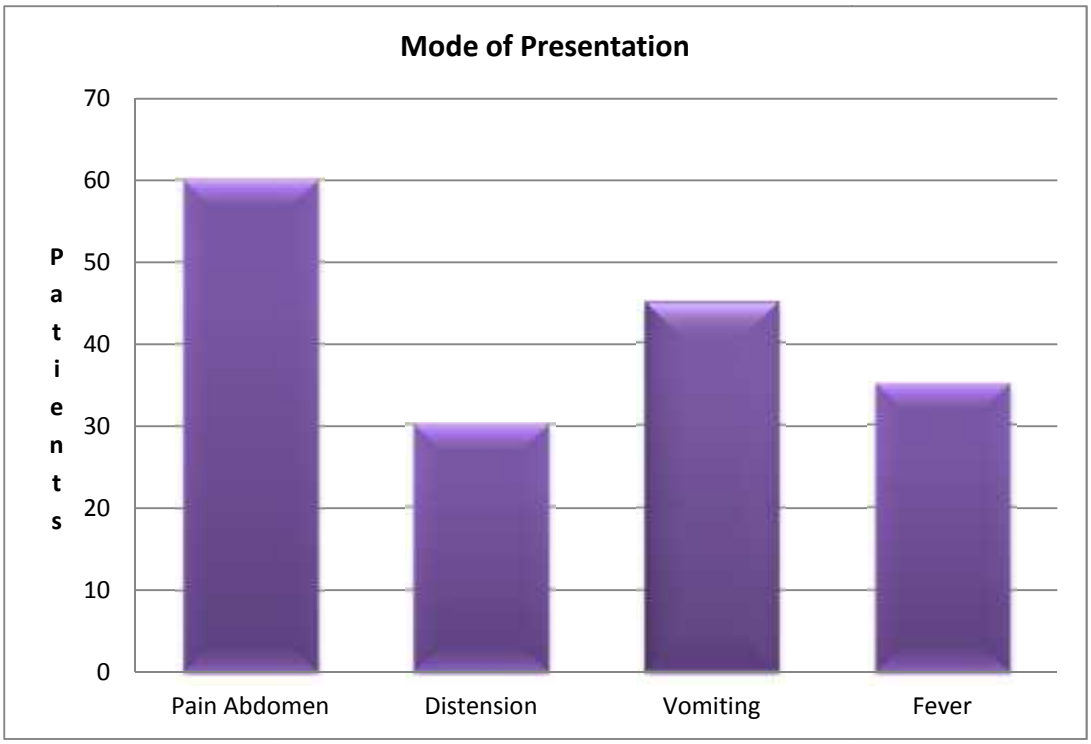
Graph 9: Impact of duration on the outcome

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

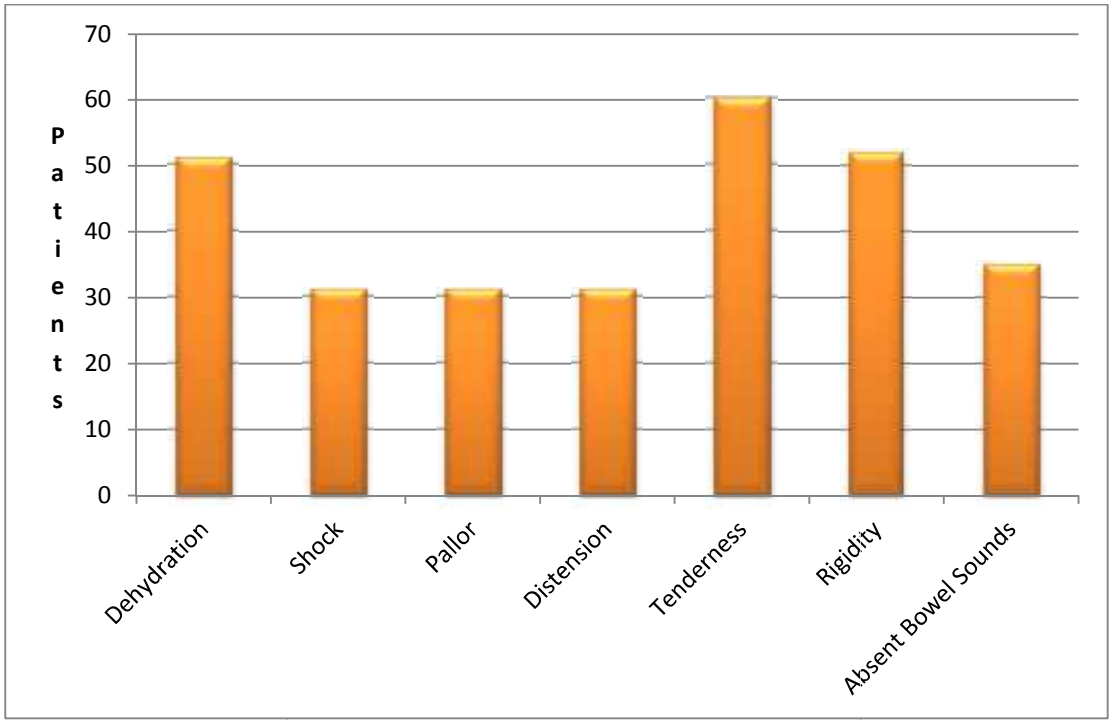


Graph 10: Mode of presentation

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:

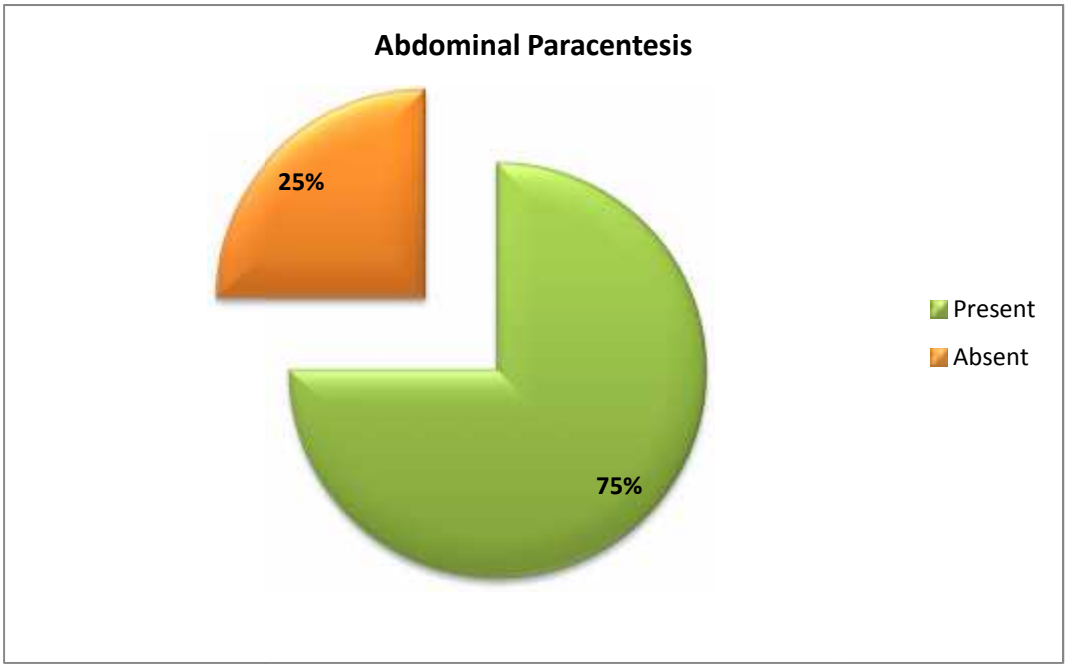
Plain X-ray abdomen in erect position was done in all 61 patients and pneumo-peritoneum (gas under the diaphragm) was found in 61 cases.

Abdominal Paracentesis:

Four quadrant abdominal paracentesis was done in all 61 cases and in 45 cases it revealed bile stained turbid fluid and in 16 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

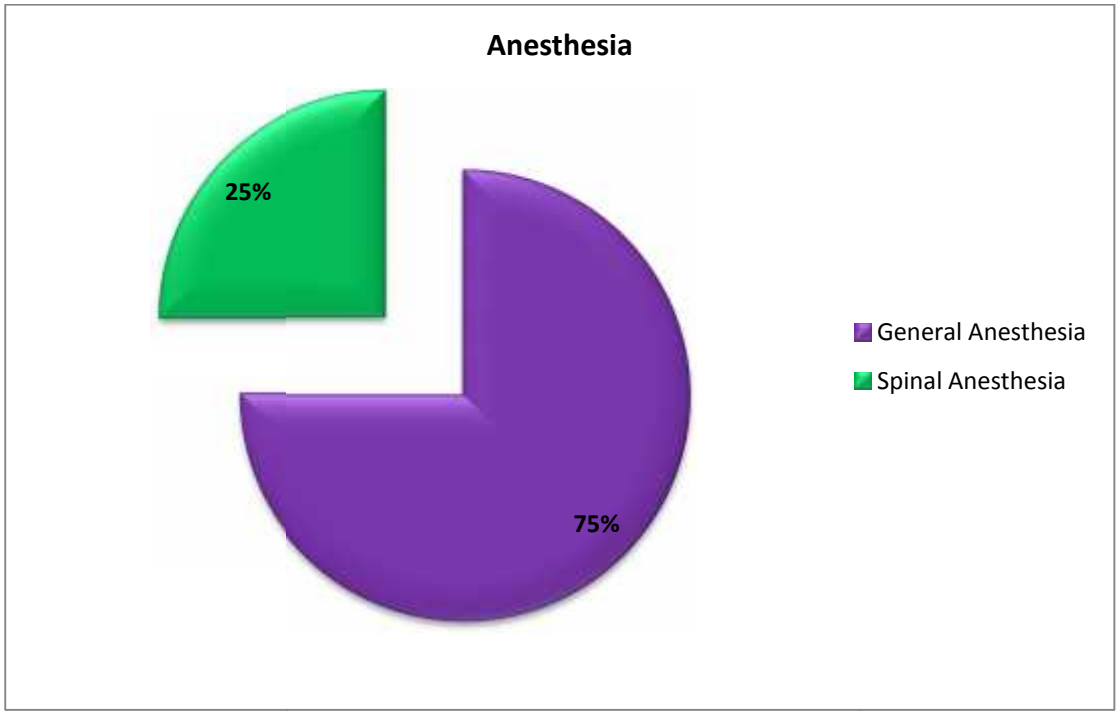
All the patients were operated as early as possible after resuscitation and stabilization.

Anesthesia:

General anesthesia was most commonly employed. General anesthesia was used in 46 patients and Spinal anesthesia in 15 patients.

Table 16: Type of anesthesia used

Anesthesia	No. of cases
Generalanesthesia	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

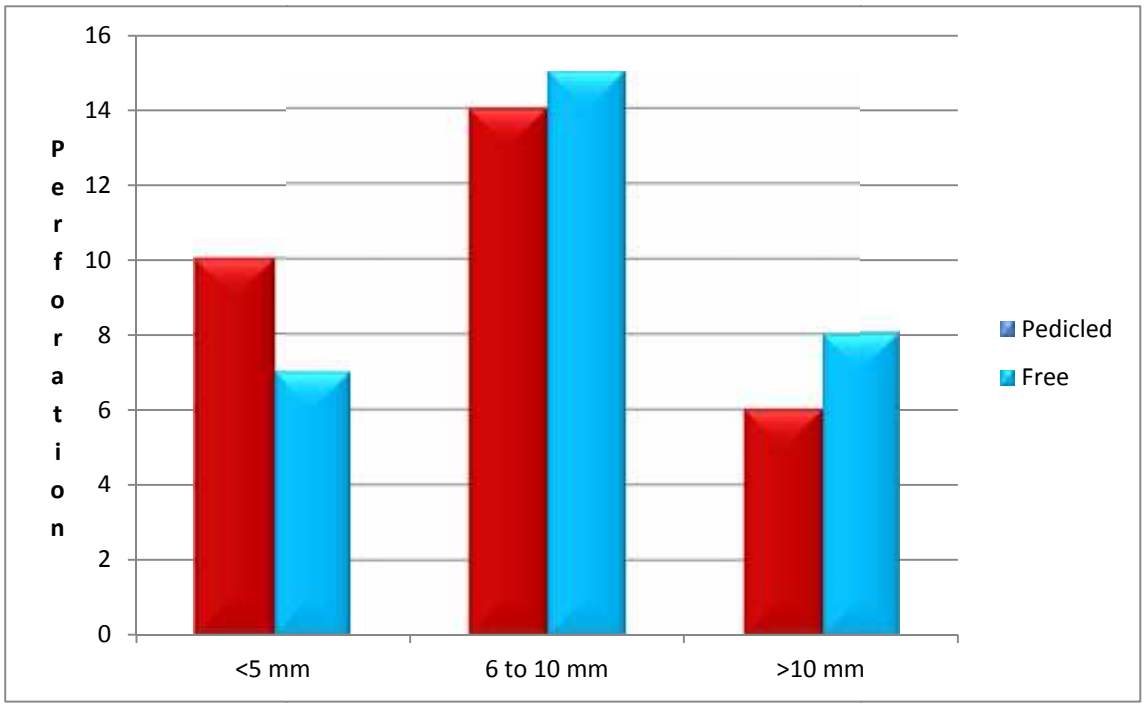
Size	Total cases	Peritoneal fluid		In shock
		< 2 liters	> 2 liters	
<0.5 cm	4	2	2	--
0.6-1.0 cm	37	19	18	4
> 1.0 cm	19	6	13	12

Treatment:

Two groups of 30/30 each selected on random basis, one group treated with Pedicled 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



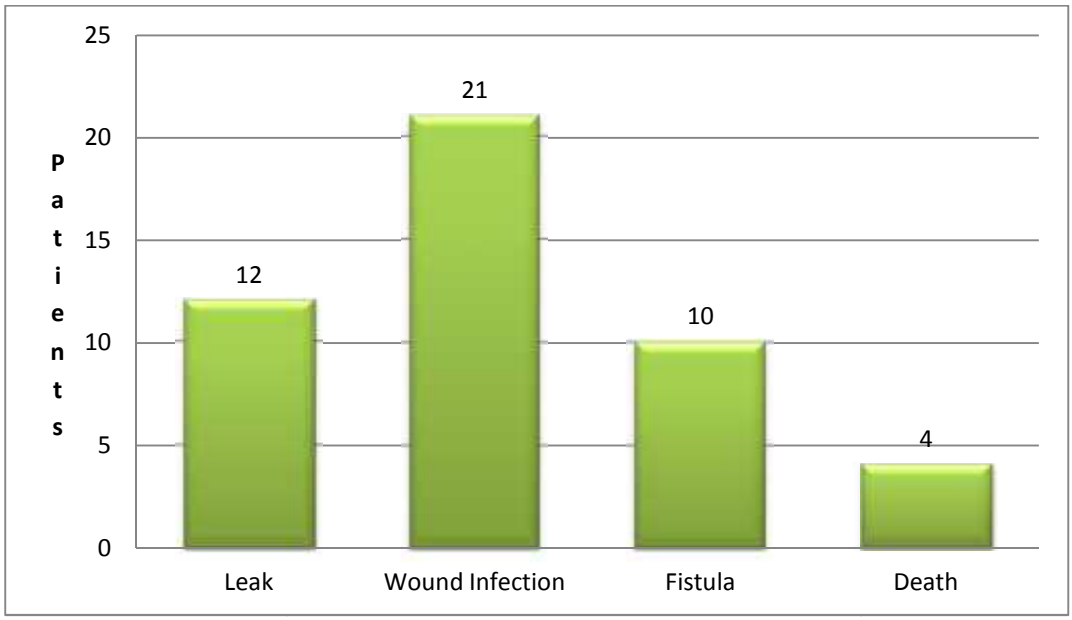
Graph 14: Distribution of patients according to the size of the perforation and type of repair

Post -Operative Complications:

In this study series, 38 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 and biliary fistula in 10 cases, which was one of definitive reasons for prolonged hospital stay.

Table 19: Postoperative Complications

Complications	No. of cases	Percentage
Leak	12	20%
Wound infection	21	35%
Fistula	10	16.66%
Death	4	6.66%

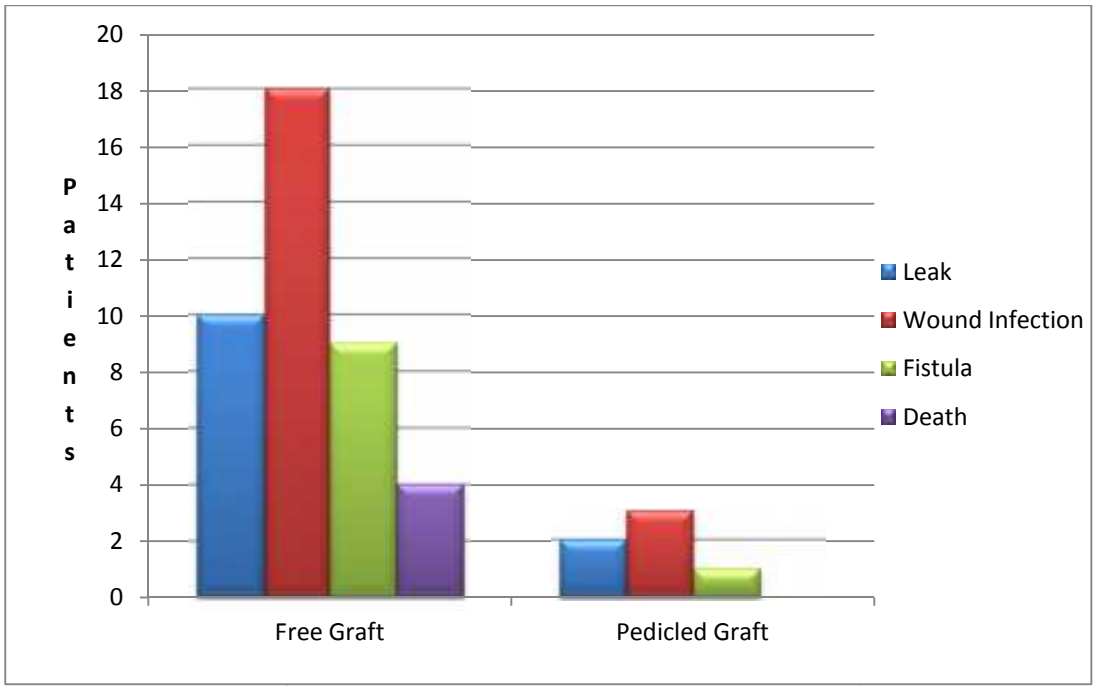


Graph 15: Postoperative complications

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

Table 20: Type of surgery with its postoperative complications

Complications	FREE GRAFT		PEDICLED GRAFT		P value
	No of Cases	PERCENTAGE (%)	No of Cases	PERCENTAGE (%)	
Leak	10	33.33	2	6.66	0.00925
Wound infection	18	60	3	9.68	0.00001
Fistula	9	30	1	3.23	0.00001
Death	4	13.33	0	0	0.07



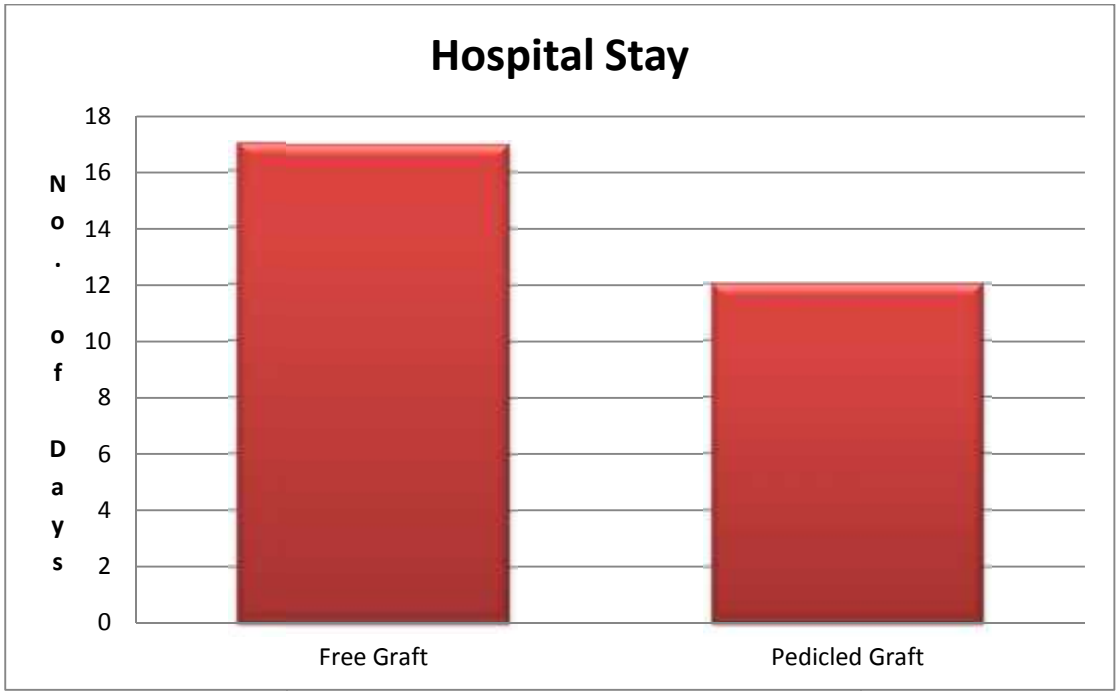
Graph 16: Type of surgery with its postoperative complications

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	Average hospitalization (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 one of the commonest surgical emergencies requiring hospitalization and early management.

Although perforated duodenal ulcer remains a dramatic surgical emergency, nowadays it seldom results in death. The surgical mortality has decreased steadily and is about 5% (Sawyers et al, 1976) [19].

Age:

Duodenal perforation is common in the age group of more than 60 years in our study, but the age is no bar for perforation to occur.

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 is more common in males than females. In the present 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
Primrose n John (Bailey & Love, 2004)[30]	2:1
Present series (2004)	4.45:1

Duration of symptom before presentation to hospital:

Table 24: Duration of symptoms before presentation to hospital

Duration (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 risk factors: pre-operative shock, delay to surgery over 24 hours and medical illness, was shown by the progressive rise in the mortality rate with the increasing number of risk factors (Hepatogastroenterology, 2001)[32].

Barazynski M et al reported age, presence of three or more co-existing diseases, delay in surgical treatment over 24 hours and septic shock as risk factors for the outcome of the perforated duodenal ulcer (1992) [33]. Boey John et al revealed concurrent medical illness, pre-operative shock and delayed operation (more than 48 hours) as significant risk factors that increase mortality in patients with perforated duodenal ulcers (1982)[34]. in the present study we reported that the age, site of perforation ,size of perforation, duration of perforation, pre-operative shock are the risk 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 (Cellan-Jones patch) and Freeomental grafting (Graham's patch). The omental plug can be reliably used for occluding large duodenal defects, wherein it promotes healing through a combined process of inflammation, granulation, vascularization and fibrosis, eventually providing a normal duodenal mucosal cover to the perforation site.

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 has reported that the lethality is higher in the elderly[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 (2009), 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.0
- 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 KLES Dr. Prabhakar Kore Hospital & Medical Research Center, Belgaum. 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 33.33% of post-operative leak (10 patients), 30% of duodenal fistula (9 patients), 60% of wound infection (18 patients) in patients treated with Free Omental Patch and 6.66% of post-operative leak (2 patients), 3.33% of duodenal fistula (1 patient), 9.68% wound infection (3 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 = 0.07)
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, 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.

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ANNEXURE – I

CONSENT FOR PARTICIPATION IN RESEARCH

Mr./Mrs._____ we are requesting you to enroll yourself in a study titled **"A STUDY TO COMPARE THE EFFICACY OF FREE OMENTAL GRAFT AGAINST PEDICLED OMENTAL GRAFT IN PREVENTION OF POST OPERATIVE LEAK IN PATIENTS WITH DUODENAL PERFORATIONS"** conducted by **Dr. P. UMASHANKAR**, postgraduate student in MS GENERAL SURGERY under the guidance of **DR. SHASHI M. UPPIN, MS,** FICS, J. N. Medical College, Belgaum.

Objective / purpose of this study:

You have been requested to participate in research because we find your profile matching with our study group.

Your participation in the research is absolutely voluntary. Your decision to participate in the study or otherwise will not affect your relationship with J.N.M.C. If you decide not to participate, you are free to withdraw at any time.

This study is being done to compare two techniques used in management of perforated duodenal ulcers which are widely practised all over the world. Patients who are provisionally diagnosed pre-operatively as perforated duodenal ulcers are recruited for this study to learn about the post-operative outcome in terms of post-operative leakage.

Procedure:

Two commonly performed techniques in management of duodenal perforations i.e. Pedicled Omental Graft (Cellan-Jones patch) and Free OmentalGraft (Graham's patch) are compared in this study.

Risk and benefits:

There is no increased risk involved in becoming a part of this study and the complications are those which are normally anticipated. This study will help us to estimate the incidence of post-operative leakage in comparison with the two techniques involved. The results derived at the end of study will benefit all similar patients admitted in this hospital.

Withdrawing / Removal from the study:

The participant has full freedom to withdraw from the study whenever he/she wishes and without prior notice. . Even if you decline to participate, there will not be any change in the line of your management or the relationship with your doctor. You will be told about all the new information that may affect your decision to participate in the study. The Investigator may also exclude a participant from the study at any time.

Privacy and Confidentiality:

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

1. In emergency to protect your rights and welfare.
2. If required by law.

Institutional / Sponsors policy:

If any unforeseen complications or injury occurs during the period of study the participant will be given the best available treatment within the limitations of KLE's Prabhakar Kore Hospital General ward.

Financial incentives for participation:

The participant will neither get any financial incentives during the period of study nor will be asked to pay for the purpose of this study.

Contact details :

In case you have any questions related to the study, please feel free to contact the Principal investigator **Dr. P. UMASHANKAR** at any time (Phone no. 9731095741).

Authorisation to Publish Results:

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

Consent Statement:

I, the undersigned, have been explained in my own vernacular language about the study. I am aware that my participation in this study is voluntary and I could withdraw at any time. Also I had been given enough time to comprehend and clarify my doubts about the study and my rights as a study participant.

Signature or the left thumb impression of the participant or legally authorized representative

Participant's name_____ Signature_____

Witness's name _____ Signature_____

Investigator's name _____ Signature_____

Place _____

Date _____

ANNEXURE – II

PROFORMA

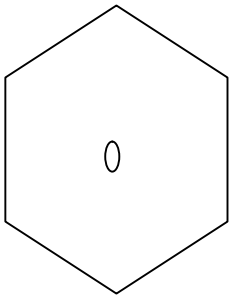
Patient Details	
Name:	**Age:**
Sex: M / F	**Registration no:**
Date of Admission:	**Address:**
Date of Discharge:	

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	Fistula	Death	POHS
1	KK	291039	19	M	Farmer	3	5	170	No	No	No	No	11
2	SK	308513	20	M	Coolie	4	5	160	No	No	No	No	22
3	SK	327441	21	M	Driver	4	4	125	No	No	No	No	9
4	SY	331348	23	F	Student	5	4	105	No	No	No	No	10
5	KV	298437	27	M	Farmer	6	4	185	No	No	No	No	14
6	IN	329789	29	M	Farmer	6	5	235	No	No	No	No	12
7	MP	300135	30	F	Teacher	6	4	100	No	No	No	No	20
8	MP	348120	31	M	Farmer	6	8	490	No	No	Yes	Yes	24
9	SP	350302	33	M	Coolie	7	3	65	No	No	No	No	10
10	MA	345657	34	M	Coolie	7	4	115	No	No	No	No	16
11	AP	279548	34	F	Housewife	7	5	250	No	No	No	No	15
12	SP	309460	36	M	Coolie	8	6	340	No	No	No	No	10
13	SM	359704	37	M	Farmer	8	4	205	Yes	Yes	No	No	11
14	BI	302211	44	M	Farmer	11	3	60	Yes	Yes	Yes	No	24
15	NV	330168	45	M	Farmer	11	6	175	Yes	Yes	Yes	No	10
16	SH	357047	45	M	Farmer	11	6	400	Yes	Yes	Yes	No	19
17	SS	284917	47	M	Farmer	11	5	370	No	Yes	Yes	No	18
18	RK	333612	48	M	Farmer	12	4	65	No	Yes	Yes	No	18
19	KQ	331494	48	M	Farmer	12	4	125	No	Yes	No	No	17
20	RM	323642	52	M	Farmer	12	4	225	No	Yes	No	No	19
21	DP	314012	56	M	Farmer	3	3	140	Yes	Yes	No	Yes	19
22	BH	336412	58	M	Business	6	5	100	Yes	Yes	No	No	19
23	DR	331304	64	M	Farmer	8	6	345	Yes	Yes	No	Yes	22
24	SP	345107	65	M	Farmer	8	5	220	Yes	Yes	No	No	26
25	YP	346412	65	M	Driver	10	6	270	Yes	Yes	No	No	20
26	AP	327697	65	F	Housewife	11	4	125	Yes	Yes	No	Yes	16
27	RG	321461	65	F	Housewife	3	4	110	No	Yes	No	No	19
28	GM	343290	68	M	Coolie	5	3	60	No	Yes	Yes	No	22
29	MA	348604	68	M	Farmer	6	4	210	No	Yes	Yes	No	18
30	TR	298176	70	F	Housewife	8	6	385	No	Yes	Yes	No	21

PEDICLED OMENTAL GROUP

Sl. No	Name	IP No.	Age	Sex	OCC	SOP	DR	DQ	Leak	WI	Fistula	Death	POHS
1	KK	287072	19	M	Driver	2	3	45	No	No	No	No	13
2	BB	330795	21	M	Student	4	4	120	No	No	No	No	11
3	RP	321641	22	F	Student	4	6	150	No	No	No	No	11
4	HR	296898	22	M	Coolie	4	4	155	No	No	No	No	8
5	MM	324716	25	M	Farmer	5	2	40	No	No	No	No	10
6	SK	320192	26	M	Farmer	5	5	140	No	No	No	No	17
7	PM	314804	32	M	Student	6	3	110	No	No	No	No	12
8	BH	334500	32	M	Business	6	8	220	No	No	No	No	14
9	BP	317124	35	M	Coolie	7	8	140	No	No	No	No	14
10	SB	331803	35	F	Housewife	7	4	110	No	No	No	No	13
11	RS	320796	36	M	Farmer	7	4	140	No	No	No	No	15
12	PG	326814	38	M	Farmer	8	3	140	No	No	No	No	12
13	MC	298462	39	F	Housewife	8	4	90	No	No	No	No	10
14	AC	345406	41	M	Farmer	8	8	155	No	No	No	No	11
15	GG	345608	43	M	Farmer	9	4	130	No	No	No	No	7
16	GM	337649	44	M	Farmer	10	6	155	No	No	No	No	16
17	SG	353195	48	M	Farmer	11	4	120	No	No	No	No	8
18	NA	336465	52	M	Coolie	12	6	405	No	No	No	No	11
19	SK	344757	53	M	Farmer	12	5	100	No	No	No	No	11
20	BB	348179	53	M	Farmer	14	4	100	No	No	No	No	11
21	SM	342271	55	M	Farmer	16	7	255	No	No	No	No	14
22	SB	285490	61	M	Coolie	6	3	200	Yes	Yes	No	No	9
23	BI	317388	64	M	Farmer	6	6	160	Yes	No	No	No	16
24	SS	312141	65	F	Housewife	3	5	80	No	No	No	No	14
25	NG	345839	66	M	Farmer	3	5	110	No	No	No	No	16
26	MN	284620	66	M	Farmer	4	4	160	No	Yes	No	No	8
27	RS	324799	68	M	Farmer	4	6	245	No	No	No	No	11
28	SS	312690	70	M	Farmer	6	4	105	No	No	Yes	No	12
29	KH	336002	72	M	Farmer	9	5	280	No	No	No	No	12
30	DB	317207	74	F	Housewife	13	7	145	No	Yes	No	No	11