A COMPARATIVE STUDY TO ASSESS THE OUTCOME AND COMPLICATIONS OF GRAHAMS OMENTAL PATCH CLOSURE VERSUS MODIFIED GRAHAMS OMENTAL PATCH CLOSURE IN PERFORATED DUODENAL ULCER AMONG PATIENTS ADMITTED IN GENERAL SURGERY DEPARTMENT,

M.S. DEGREE EXAMINATION

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

CHENNAI, INDIA.

CERTIFICATE

This is to certify that this dissertation titled **"A COMPARATIVE STUDY TO ASSESS THE OUTCOME AND COMPLICATIONS OF GRAHAMS OMENTAL PATCH CLOSURE VERSUS MODIFIED GRAHAMS OMENTAL PATCH CLOSURE IN PERFORATED DUODENAL ULCER AMONG PATIENTS ADMITTED IN GENERAL SURGERY DEPARTMENT, GRH, MADURAI"** submitted by **Dr.VEENA R UNNI** to the faculty of General Surgery, The Tamil Nadu Dr. M.G.R. Medical University, Chennai in partial fulfilment of the requirement for the award of MS Degree Branch I General Surgery, is a bonafide research work carried out by her under our direct supervision and guidance from May 2018 to September 2018.

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DECLARATION BY THE CANDIDATE

I **Dr.VEENA R UNNI** hereby solemnly declare that this dissertation entitled **"A COMPARATIVE STUDY TO ASSESS THE OUTCOME AND COMPLICATIONS OF GRAHAMS OMENTAL PATCH CLOSURE VERSUS MODIFIED GRAHAMS OMENTAL PATCH CLOSURE IN PERFORATED DUODENAL ULCER AMONG PATIENTS ADMITTED IN GENERAL SURGERY DEPARTMENT, GRH, MADURAI "** is a bonafide and genuine research work carried out by me. This is submitted to the TamilNadu Dr. M.G.R. Medical University, Chennai, in partial fulfilment of the regulations for the award of M.S. degree (Branch I) General Surgery.

PLACE: Madurai

DATE: Dr.VEENA R UNNI

ACKNOWLEGMENT

First I would like to give thanks to Lord God Almighty whose blessings made this study possible

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CONTENTS

INTRODUCTION

Duodenal ulcer perforation is one of the manifestation of Peptic ulcer disease. Duodenal ulcer represent almost 2/3rd of all peptic ulcer diseases. Perforated duodenal ulcer remains a major health problem world wide. Peptic ulcer disease is primarily associated with H-pylori infection and excessive use of NSAIDs. Since the burden of peptic ulcer disease and its complications are significant worldwide, it is important to conduct a study based on peptic ulcer disease.

Life prevalence of duodenal ulcer has found to be 11-14% for males and 8-10% for women . Male to female ratio of duodenal ulcer have reduced from 10:1 to 1.5:1.

Duodenal ulcer have been characterized by the presence of a well demarcated break in the mucosa that may extend into muscularis propriya of the duodenum.

Duodenal perforation is the second most common complication of PUD and occurs in as many as 10% of patients with PUD

My study compares the outcome and complications of two surgical procedures done for perforated duodenal ulcer namely GRAHAMS OMENTAL PATCH REPAIR and MODIFIED GRAHAMS OMENTAL PATCH CLOSURE.

AIMS AND OBJECTIVES

AIM

The aim of this study is to compare outcome and complications of GRAHAM'S OMENTAL PATCH CLOSURE versus MODIFIED GRAHAM'S OMENTAL PATCH CLOSURE for treating perforated duodenal ulcers.

OBJECTIVES

To compare outcome and complications of GRAHAM'S OMENTAL PATCH CLOSURE versus MODIFIED GRAHAM'S OMENTAL PATCH closure for treating perforated duodenal ulcers

STUDY DESIGN

PERIOD OF STUDY:

6 months (April 2018– September 2018)

COLLABORATING DEPARTMENT:

None

PLACE OF STUDY:

Government Rajaji Hospital,Madurai.

SELECTION OF STUDY SUBJECTS :

All patients diagnosed with peritonitis secondary to hollow viscus perforation who are willing for definitive surgery.

SAMPLE SIZE:

60 patients

DATA COLLECTION:

Data regarding history, clinical examination,laboratory values & postoperative analysis.

METHODS:

Prospective comparitative study.

ETHICAL CLEARANCE:

Approved by the Institute of Ethical Committee, Madurai Medical College.

CONSENT :

Informed and written consent from all patients

ANALYSIS:

Data analysis was done with the help of computer using SPSS 16 and Sigma Stat 3.5 version.

Using this software range, frequencies, percentages, means, standard deviations, chi square and 'p' values were calculated by One way ANOVA and Chi-square test was used to test the significance of difference between quantitative variables.

CONFLICT OF INTEREST:

None

FINANCIAL SUPPORT :

Nil from the institution

ELIGIBILITY CRITERIA:

A.INCLUSION CRITERIA:

All patients with perforated duodenal ulcer size < 20 mm who are admitted in general surgery department GRH, Madurai.

B.EXCLUSION CRITERIA:

Duodenal perforation of other origin such as traumatic and neoplasia.

Large duodenal perforation >20 mm

Posterior duodenal perforation

Sealed duodenal perforation

Patient who expired before definitive surgery.

Patient not willing for definitive surgery

Patient not willing for the study

REVIEW OF LITERATURE

- The knowledge of perforation dates back to over 2000 years remote past

when "**Sushrutha**", the great surgeon of India described it as*"Parinamashula"* giving the relation of the pain ,vomiting, and food.

- The history of peptic ulcer dates back since 1500 B.C. when Hemorrhage and peptic ulcer was noted from **Egyptian Papyri**.
- The acute pathological condition of abdomen "Hippocratic Facies" that represents the terminal stage of perforative peritonitis was recognized by **Hippocrates.**
- The symptoms which are caused by peptic ulcer disease were described by **Diokles** (350-325 B.C).
- The 1st illustration of gastric ulcer is credited to Italian physician **Marcello Donati** in the year 1586 and the 1st case of perforated gastric ulcer was declared by **Christopher Rawlinson** in England 1727.
- Duodenal ulcer was 1st described by **Georg Hamberger** in Germany in 1746, and **Jacopo Penada** from Italy recorded a perforated duodenal ulcer in 1793.
- In 1881, **Theodor Billroth**, Father of Surgical Audit and Father of Abdominal surgery, excised the distal part of the stomach with an anastomosis of the gastric stump to the duodenum (Billroth I Surgery).
- **Mikulicz** was first to suture a perforated gastric ulcer in the year 1885.
- **Bennett** demonstrated sealing a large perforation with omentum in the year 1896.
- **Keetley** of London in 1902 did the 1st partial gastrectomy for a perforated ulcer.

- In 1938 **Graham** popularized the simple closure of perforated duodenal ulcer.
- **Wangensteen** in I935 first advocated non operative treatment for duodenal perforation.
- **Mathur S.N., Khandelwal R.** (1991): In a study of 43 cases of perforated peptic is a safe procedure in all ulcer patients. Definitive ulcer healing operation may be done in selected cases of perforated chronic duodenal ulcers.
- **Siu WT et al.** (2004) demonstrated the laparoscopic mode of repair of perforated duodenal peptic ulcer as a safe emergency procedure as a routine practice for patients with perforated duodenal ulcer.

SURGICAL ANATOMY OF DUODENUM

Anatomy

The adult duodenum is approximately 25 cm long and is the shortest, widest and most predictably placed part of the small intestine. The proximal 2.5 cm is intra peritoneal and the remainder is retroperitoneal. The duodenum forms an elongated ‘C’ that lies between the level of the first and third lumbar vertebrae in the supine position. The lower ‘limb’ of the C extends further to the left of the midline than the upper limb. The head and uncinate process of the pancreas lie within the concavity of the duodenum, which is ‘draped’ over the prominence formed by the lumbar spine; the duodenum therefore curves in an antero-posterior direction as well as forming a ‘C’. The duodenum lies entirely above the level of the umbilicus. It is described as having four parts.

FIRST (SUPERIOR) PART

The first, and most mobile, part of the duodenum is about 5 cm long. It starts at the duodenal end of the pylorus and ends at the superior duodenal flexure. The proximal 2.5 cm is intraperitoneal while the distal 2.5 cm is covered by peritoneum on its anterior and superior surfaces and forms the inferior boundary of the epiploic foramen.

The lesser omentum is attached to its upper border and the greater omentum to its lower border. The first 2–3 cm of the duodenum is lined by relatively smooth mucosa and readily distends on insufflation during endoscopy. This part is

frequently referred to as the duodenal ‘cap’. During contrast radiology, it shows a few longitudinal folds continuous with the pylorus (Mather Cordiner and Calthrop 1936) and has a triangular appearance; it is often visible on plain radiographs of the abdomen as an isolated triangular gas shadow to the right of the first or second lumbar vertebra. The first part of the duodenum passes superiorly, posteriorly and laterally for 5 cm before curving sharply inferiorly at the superior duodenal flexure. It becomes more retroperitoneal during this part of its course, until peritoneum only covers its anterior aspect. The section from the duodenal cap to the superior duodenal flexure lies posterior and inferior to the quadrate lobe of the liver. Beyond the duodenal cap, the internal appearance is characterized by circumferential mucosal folds that remain pronounced, even during endoscopic insufflation. The first part of the duodenum lies anterior to the gastroduodenal artery, common bile duct and portal vein, and anterosuperior to the head and neck of the pancreas. The gastroduodenal artery lies immediately behind the posterior wall of the duodenum; a penetrating peptic ulcer on the posterior wall may erode into the gastroduodenal artery or one its branches and cause dramatic haemorrhage. A penetrating peptic ulcer on the anterior wall may perforate into the peritoneal cavity because the anterior surface of the first part is covered only by peritoneum. The common hepatic and hepatoduodenal lymph nodes lie close to the first part of the duodenum and can be visualized using endoscopic ultrasound; this may be important in the staging of gastric, pancreatic or bile duct tumours. The proximity of the common bile duct to the

first part of the duodenum allows endoscopic ultrasound examination of the distal common bile duct and the formation of a surgical anastomosis between bile duct and duodenum (choledochoduodenostomy) when required. The junction of the first and second parts of the duodenum lies posterior to the neck of the gallbladder.

SECOND (DESCENDING) PART

The second part of the duodenum is approximately 8 cm long. It starts at the superior duodenal flexure and runs inferiorly in a gentle curve, convex to the right side of the vertebral column and extending to the lower border of the third lumbar vertebral body. It then turns sharply medially at the inferior duodenal flexure, which marks its junction with the third part of the duodenum. It is covered by peritoneum only on its upper anterior surface, lies posterior to the gallbladder and the right lobe of the liver at its start, and is crossed anteriorly by the transverse colon. The right end of the gastrocolic omentum and the origin of the transverse mesocolon are attached to the anterior surface of the duodenum by loose connective tissue. Below the attachment of the transverse mesocolon, the connective tissue and vessels forming the mesentery of the upper ascending colon and hepatic flexure are loosely attached to its anterior surface. This part of duodenum is at risk of injury during surgical mobilization of the ascending colon and hepatic flexure. The second part lies anterior to the hilum of the right kidney, the right renal vessels, the lateral edge of the inferior vena cava and the

right psoas major . The head of the pancreas and the common bile duct are medial and the hepatic flexure is above and lateral. Part of the pancreatic head is sometimes embedded in the medial duodenal wall, and pancreatic ‘rests’ in the duodenal wall may produce small filling defects on contrast radiology. The internal appearance is similar to that of the distal portion of the first part of the duodenum, with pronounced mucosal folds. The common bile duct and pancreatic duct enter the medial wall, where they usually unite to form a common channel, which frequently contains a dilated segment known as the hepatopancreatic ampulla (of Vater) . The narrow distal end of this channel opens on the summit of the major duodenal papilla, a mucosal elevation situated on the posteromedial wall of the second part, 8–10 cm distal to the pylorus. A duodenal mucosal fold often partially encircles the major papilla, forming a hood (Horiguchi and Kamisawa 2010). A second, accessory pancreatic duct is sometimes present and opens about 2 cm proximal to the major papilla on a minor duodenal papilla (Suda 2010, Kamisawa et al 2010).

THIRD (HORIZONTAL) PART

The third part of the duodenum starts at the inferior duodenal flexure and is approximately 10 cm long. It runs from the right side of the lower border of the third lumbar vertebra, and passes to the left and slightly superiorly, anterior to the inferior vena cava and abdominal aorta, becoming continuous with the ascending fourth part. It lies posterior to the transverse mesocolon, and is crossed anteriorly by the origin of the small bowel mesentery and the superior

mesenteric vessels. The lower portion of its anterior aspect is covered by peritoneum, which is reflected anteriorly to form the posterior layer of the root of the small bowel mesentery. The anterior surface of the duodenum close to the junction with the fourth part, is also covered by peritoneum. The third part lies anterior to the right ureter, right psoas major, right gonadal vessels, inferior vena cava and abdominal aorta (at the origin of the inferior mesenteric artery), and inferior to the head of the pancreas. Anteroinferiorly, loops of jejunum lie in the right and left infracolic compartments. The mid portion of the third part lies in the angle between the superior mesenteric artery anteriorly and the abdominal aorta posteriorly; narrowing of this angle may occur from loss of perivascular adipose tissue or spinal straightening and is a rare cause of duodenal obstruction (Merrett et al 2009).

FOURTH (ASCENDING) PART

The fourth part of the duodenum is 2.5 cm long. It starts just to the left of the aorta, runs superiorly and laterally to the level of the upper border of the second lumbar vertebra, then turns sharply anteroinferiorly at the duodenojejunal flexure to become continuous with the jejunum. The inferior mesenteric vein lies either posterior to the duodenojejunal flexure or at its lateral margin beneath a peritoneal fold. The duodenojejunal flexure is a useful landmark to locate the vein radiologically or surgically. The aorta, left sympathetic trunk, left psoas major, left renal and left gonadal vessels are all posterior relations,

the left kidney and left ureter are posterolateral, and the transverse colon and mesocolon are anterior, separating it from the stomach . The inferior border of the body of the pancreas is superior. The peritoneum of the root of the small bowel mesentery descends over its anterior surface. At its left lateral limit, the fourth part becomes progressively invested in peritoneum, such that the duodenojejunal flexure is suspended from the retroperitoneum by a double fold of peritoneum, the suspensory ligament of the duodenum (or ligament of Treitz). The ligament of Treitz is in two parts; the first part may contain skeletal muscle fibers and runs from the right crus of the diaphragm to connective tissue around the coeliac trunk, and the second part contains smooth muscle and descends from connective tissue around the coeliac trunk to the duodenum, passing behind the pancreas anterior to the left renal vein. The ligament is often absent or rudimentary in adults and its function is unknown (Kim et al 2008). The ligament of Treitz is avascular; the vascular supply to the fourth part of the duodenum enters its wall from the posteromedial aspect. The duodenojejunal flexure is an important landmark in the radiological diagnosis of incomplete rotation and malrotation of the small intestine

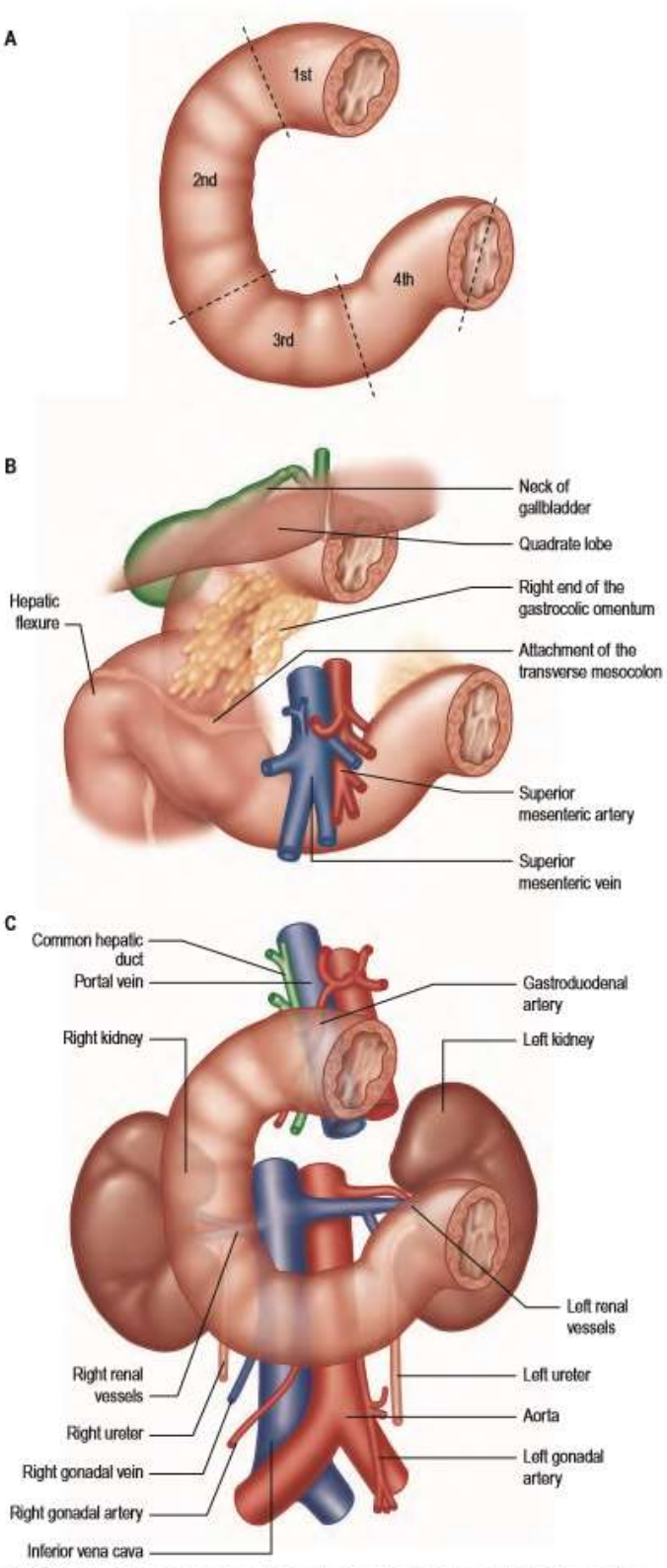
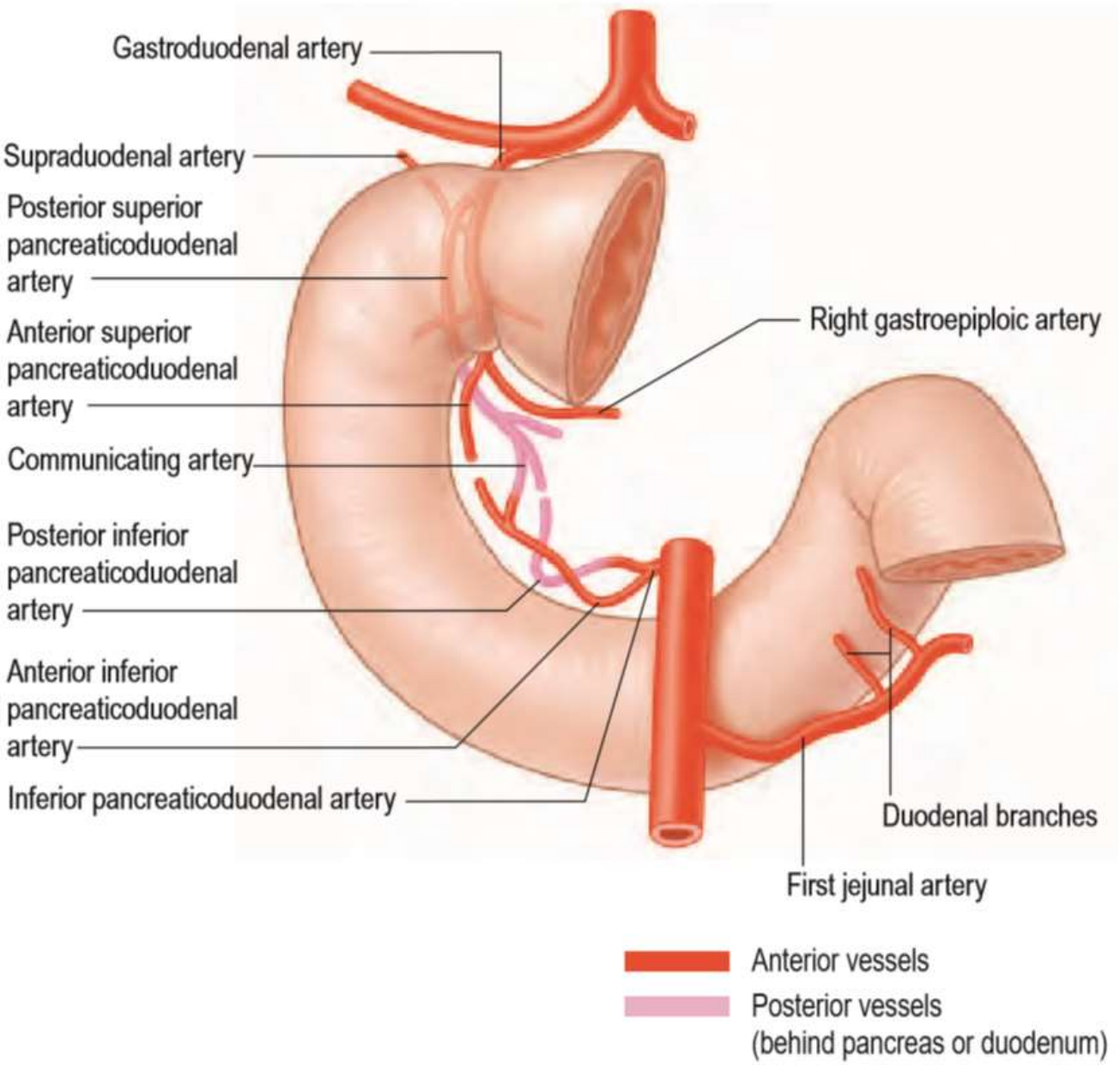


Fig. 65.1 **A**, The four parts of the duodenum. **B**, Anterior relations of the duodenum. **C**, Posterior relations of the duodenum.

ARTERIAL SUPPLY OF DUODENUM



VASCULAR SUPPLY AND LYMPHATIC DRAINAGE

Arteries

The main vessels supplying the duodenum are the superior and inferior pancreaticoduodenal arteries. The first and second parts also receive contributions from other sources, including the right gastric, supraduodenal, right gastroepiploic, hepatic and gastroduodenal arteries (Fig. 65.4). Branches of the superior pancreaticoduodenal artery may contribute to the supply of the pyloric canal, anastomosing to a minor extent with the gastric arteries within the muscular layer of the pyloroduodenal junction. Gastroduodenal artery The gastroduodenal artery usually arises from the common hepatic artery behind or above the first part of the duodenum. It descends behind the retroperitoneal portion of the first part of the duodenum to the left of the common bile duct. At the lower border of the first part of the duodenum, it is commonly described as dividing into the right gastroepiploic and superior pancreaticoduodenal arteries but this anatomical arrangement is rare (Bradley 1973, Bertelli et al 1995, Bertelli et al 1996) and its usual branching pattern is as follows. As it descends behind the first part of the duodenum, it usually gives off the posterior superior pancreaticoduodenal artery, several retroduodenal branches that supply the first part and proximal portion of the second part of the duodenum, and a supraduodenal artery that supplies the anterosuperior part of the proximal duodenum (Bianchi and Albanèse 1989). As the gastroduodenal artery emerges below the first part of the duodenum, it usually gives off the right gastroepiploic

artery and several pyloric branches. It then descends on the anterior surface of the pancreas, where it divides into the anterior superior pancreaticoduodenal artery and pancreatic branches. Although the gastroduodenal artery usually branches from the common hepatic artery, it may occasionally originate from other sources, including: as a trifurcation with the right and left hepatic arteries; the coeliac trunk; the superior mesenteric artery; or from the right or left branch of the hepatic artery. The gastroduodenal artery or one of its branches may be a source of haemorrhage from a penetrating posterior duodenal ulcer (see above) or it may be the site of aneurysm or pseudoaneurysm formation; for these reasons, it is an important vessel for interventional radiologists. Superior pancreaticoduodenal arteries There are usually two superior pancreaticoduodenal arteries: a posterior and anterior. The posterior superior pancreaticoduodenal artery is usually a separate branch of the gastroduodenal artery and is given off behind the upper border of the first part of the duodenum. It descends to the right, anterior to the portal vein and common bile duct, where the latter lies behind the first part of the duodenum. It then spirals around the right side of the bile duct to run behind the head of the pancreas, crosses posterior to the retropancreatic segment of the common bile duct (which is embedded, to a variable degree, in the head of the pancreas), and anastomoses with the posterior division of the inferior pancreaticoduodenal artery (Bertelli et al 1996). The posterior artery supplies branches to the head of the pancreas, the first and second parts of the duodenum, and several branches to the lowest part

of the common bile duct. The anterior superior pancreaticoduodenal artery is usually a terminal branch of the gastroduodenal artery and descends in the anterior groove between the second part of the duodenum and the head of the pancreas or on the anterior surface of the gland parallel to the groove (Bertelli et al 1995). It supplies branches to the first and second parts of the duodenum and to the head of the pancreas, and then passes posteriorly to anastomose with the anterior division of the inferior pancreaticoduodenal artery. inferior pancreaticoduodenal artery The inferior pancreaticoduodenal artery usually arises from the superior mesenteric artery or its first jejunal branch, near the superior border of the third part of the duodenum (Bertelli et al 1996). It crosses behind the superior mesenteric vein and passes behind the uncinate process of the pancreas, where it divides into anterior and posterior branches. The anterior branch passes to the right, immediately inferior and then anterior to the lower border of the head of the pancreas, and runs superiorly to anastomose with the anterior superior pancreaticoduodenal artery. The posterior branch runs posteriorly to the right behind the head of the pancreas, and anastomoses with the posterior superior pancreaticoduodenal artery (Bertelli et al 1997). Both branches supply the pancreatic head, its uncinate process, and the second and third parts of the duodenum. Occasionally, the anterior and posterior branches arise separately from the superior mesenteric or first jejunal artery. Jejunal artery branches Branches from the first jejunal branch of the superior mesenteric artery supply the fourth part of the duodenum and frequently anastomose with a

terminal branch of the anterior superior pancreaticoduodenal artery. The fourth part of the duodenum therefore receives a potential collateral supply from the coeliac trunk and superior mesenteric artery, which means that it is not commonly affected by ischaemia. Veins Submucosal and intramural veins give rise to small veins that accompany corresponding named arteries. The venous anatomy of this region is variable and not well characterized. The superior pancreaticoduodenal vein runs superiorly on the posterior surface of the head of the pancreas, posterior to the distal common bile duct, and usually drains into the portal vein. The inferior pancreaticoduodenal vein runs inferiorly and usually drains into the superior mesenteric vein or its first jejunal tributary. Small veins from the first and upper second parts of the duodenum drain directly into the portal vein, and veins from the third and fourth parts may drain directly into the superior mesenteric vein. Numerous small anastomoses are present between veins of the second and third parts of the duodenum and retroperitoneal veins (Murakami et al 1999).

LYMPHATIC DRAINAGE

Duodenal lymphatics run to superior and inferior pancreaticoduodenal lymph nodes, and from there to supra- and infrapyloric, hepatoduodenal, common hepatic, coeliac and superior mesenteric nodes.

Lymphatic drainage to para-aortic nodes has also been described (Hirai et al 2001).

INNERVATION

The duodenum is innervated by both parasympathetic and sympathetic neurones. Preganglionic sympathetic neurones have their cell bodies in the intermediolateral columns of the grey matter in the fifth to the twelfth thoracic spinal segments. Their fibres travel via the greater and lesser splanchnic nerves to the coeliac plexus and synapse in the coeliac and superior mesenteric ganglia; postganglionic axons are distributed to the duodenal wall via peri-arterial plexuses on the branches of the coeliac trunk and superior mesenteric artery. The sympathetic nerves are vasoconstrictor to the duodenal vasculature and inhibitory to duodenal musculature. The preganglionic parasympathetic supply is carried by vagal fibres that travel from the coeliac plexus and synapse on neurones in the duodenal wall. The parasympathetic supply is secretomotor to the duodenal mucosa and motor to the duodenal musculature. Referred pain In common with other structures derived from the foregut, pain arising from the proximal duodenum is poorly localized and referred to the epigastrium. It is mediated by afferent fibres that accompany the sympathetic neurons.

HISTOLOGY.

The duodenum is lined by a mucus-secreting columnar epithelium. In addition, Brunner's glands lie beneath the mucosa and are similar to the pyloric glands in the pyloric part of the stomach. Endocrine cells in the duodenum produce cholecystokinin and secretin.

PHYSIOLOGY

GASRTIC SECRETION:

The stomach secretes water and electrolytes, primarily in the form of acid and small amount of bicarbonates, enzymes such as pepsin, glycoprotein such as intrinsic factor and mucous. Gastric juice also contains small amounts of calcium, magnesium and trace amount of zinc, copper and iron.

I. ACID SECRETION:

Human stomach secretes about 2-5 mEq/hour of HCL in the fasting state, constituting basal acid secretion. After a mixed meal, the amount of acid secretion increases to 15-25mEq/hour. Acid is secreted by the parietal cells situated in the glands of the fundus and body of the stomach. Regulation of acid secretion is a very complex process involving endocrine, neural, paracrine and even autocrine mechanisms.

There are three phases in gastric secretions

i.Cephalic phase - is stimulated by the sight of smell of chewing of food.

ii.Gastric phase - is stimulated by the presence of food in the stomach

iii. Intestinal phase- is stimulated by presence of food in small intestine.

i. Cephalic phase- Cephalic phase stimuli (sight or smell of food) presumably activate the vagal nuclei in the medulla. Impulses traverse the peripheral vagi and terminate in the gastric mucosa with the release of acetylcholine from vagal nerve endings. Release of acetylcholine in the fundic mucosa directly, stimulates and secretion by the parietal cell and release of pepsinogen by chief cells.

Acetylcholine release in the antral mucosa may cause discharge of the antral hormone gastrin. Distension of stomach excites vaso-vagal reflex that also results in the release of acetylcholine in the fundic and antral mucosa.

ii. Gastric phase- This phase is initiated by the entry of food into the stomach. Food that enters the stomach buffers acid, raises pH and allows other stimuli to release acid. Through this gastrin is liberated from the gastric mucosa either due to antral distension and when it reaches 1.5, gastrin output is absolutely stopped. So this is a feed back mechanism in which production of gastrin is inhibited by the presence of acid in the antrum of stomach. The most remarkable action of gastrin is its ability to stimulate gastric acid secretion. It is 30 times more potent than histamine. Beside its action on acid secretion, it stimulates pepsin secretion and increases gastric mucosal blood flow. It also stimulates pancreatic enzyme secretion in man.

iii. Intestinal phase- The intestinal phase of secretion begins as chyme begins to empty from the stomach into the duodenum. Distension of jejunum will also stimulate secretion. The cholecystokinin, the duodenal hormone which acts to stimulate secretion of pancreatic enzymes an stimulate contraction of gall bladder, also acts like gastrin.

II. PEPSIN SECRETION:

It is influenced by hydrogen ion secretion, cholinergic stimuli and by polypeptide hormones. Increased H+ secretion causes increased pepsin secretion.

III. GASTRIC MUCUS SECRETION:

This is secreted by gastric mucosa serves the function of the lubrication, protecting the mucosa from mechanical damage and gastric acids. Its secretion is evoked by vagal stimulation, on feeding and gastric irritation. Its pH is alkaline, the mucous barrier by bile acids refluxed through the pylorus and drugs.

Duodenal Exocrine Secretion:

It is alkaline in nature, secreted by Brunner's gland into the crypts of Lieberkuhn, the amount of secretion is related to the acid delivered through the pylorus.

GASTRIC & DUODENAL ENDOCRINE SECRETION

From the Stomach: Gastrin is secreted by the 'G' cells of the antrum, exists as **G**-17 gastrin predominantly found in circulation.

Functions:

- Stimulates HCl secretions from the parietal cells. Pepsinogen is converted into pepsin in the presence of HCl.

- Stimulates pancreatic enzyme and bicarbonate secretion.

- Stimulates bile secretion from the liver and promotes gall bladder contraction.

-Gastrin secretion is inhibited by fall of gastric pH below 3 and by somatostatin.

From the Duodenum: The presence of acid chyme in the duodenum stimulates the secretion secretin, Cholecystokinin-pancreozymin (CCK-PZ), enteroglucagon and enterogastrone hormones.

Functions of secretin:

a. Inhibits acid secretion from parietal cells.

b. Stimulates bile secretion from the liver.

c. Stimulate bicarbonate secretion.

ETIOPATHOGENESIS

PEPTIC ULCER DISEASES

Peptic ulcer is a term used to refer to a group of ulcerative disorders of the gastrointestinal tract, involving principally the most proximal position of duodenum, the stomach, the lower end of the oesophagus, the jejunum after surgical anastamosis to stomach or rarely the ileum adjacent to the Meckel's diverticulum due to ectopic gastric epithelium. Approximately 98 - 99% of peptic ulcers occur in the first portion of duodenum or in the stomach, in a ratio of about 4%. About 5% of individuals with gastric ulcer develop duodenal ulcers, but 20% of those with duodenal ulcers develop gastric lesions. The pyloric channel, which is 1-2cms in length, is the narrowest portion of the gastric outlet. Because of their gastric acid secretory characteristics and clinical features, pyloric channel are classified with duodenal rather than gastric ulcer. Ulcers in this location often produce symptoms similar to those of duodenal ulcer. In-patients with pyloric channel ulcers, food may accentuate rather than relive ulcer pain.

EPIDEMOLOGY

Peptic ulcers are remitting relapsing lesions, at one time duodenal ulcers were much more common than gastric ulcer, but their incidence and prevalence are now approaching those of gastric ulcers. Most often diagnosed in middle aged to older

adults, but may first become evidence in young adult life. Male to female ratio for duodenal ulcer is about 3:1 and for gastric ulcers around 1.5:2.

Genetic influence plays some role in predisposition to both forms of ulcers, but more clear cut with the duodenal ulcers. Duodenal ulcers are three times more common in the first-degree relatives of ulcer patients than in general population. A 50% concurrence of duodenal ulcers in monzygotic twins, but only 14% in dizygotic twins. An increased incidence of HLA-B5 antigen has also been identified in white males with duodenal ulcers. Individuals with blood group 'O' are about 30% more likely to develop duodenal ulcer than those with other blood group. Increased use of NSAIDS and corticosteroids in one of the common cause in producing duodenal ulcer. Environmental factors: Duodenal ulcer is more frequent in patients with Alcoholic cirrhosis, chronic renal failure, chronic obstructive pulmonary disease and hyperparathyroidism.

Role of H. Pylori Infection in peptic ulcer

The word "No acid-No ulcer" does not hold good now a days, because peptic ulcer is considered now more an infective disease, caused by Helicobacter Pylori. In 1983, Warren and Marshall first reported isolation of Helicobacter Pylori from the mucosal biopsy specimen of patient with peptic ulcer diseases.

Helicobacter pylori are a small spirally curved, gram negative, micro aerophilic rod with multiple pollar flagellae. 80-90% of population are affected with infection. The incidence of infection within a population increases along with age. The infection is inversely propotional to socioeconomic group. H.pylori infection is the major cause of peptic ulcer not associated with the use of Non steroidal anti inflammatory drugs. Human are the major reservoirs of Helicobacter pylori. The organism colonizes in the

stomach, lodging most frequently in the antrum. The route of transmission of Helicobacter pylori infection is mainly by faeco-oral route and oro-oral route.

Pathogenesis of Duodenal Ulcer due to H. Pylori

Helicobacter pylori infection invariably results in chronic gastritis. Helicobacter pylori colonizes in the gastric epithelium causing Type-B gastritis by which it reduces the resistance of gastric mucosa to attack by acid and pepsin resulting in gastric ulcer. Although, Helicobacter pylori normally reside in stomach, it also leads to causation of duodenal ulcers. This can be explained by the fact that antral Helicobacter pylori infection impairs the inhibitory feedback control of acid secretion, thus promoting duodenal ulcerogenesis by increasing duodenal acid load, resulting into duodinitis which leads to local inflammation, mucosal injury and eventually ulcer formation through the following mechanism:

1. By increasing acid secretion: The organism produces urease enzymes, which hydrolyzes urea, resulting in production of ammonia, a strong alkali. Ammonia generated causes the release of gastrin (hypergastrinaemia) from antral G-Cells, which in turn leads to gastric acid hypersecretion.

2. By distrupting the gastric mucous barrier.

3. By secretion of number of enzymes and chemicals, urease, catalase, mucinlipase, phospholipase, porins, protease's, hemolysins and alkaline phosphatase.

4. By inducing inflammation in gastric epithelium (Wyatt and Dixon): The organism causes inflammation, which causes migration and degeneration of acute inflammatory cells, such as neutrophils and accumulation of chronic inflammatory cells, such as macrophages and lymphocytes.Overall, Helicobacter pylori are undoubtedly the dominant factor in the pathogenesis of peptic ulcer disease. There is,

however, a small minority of duodenal ulcers where Helicobacter pylori has no effect, such as ulcers related to use of NSAIDs, Crohn's disease and in Zolinger Ellison Syndrome.

Helicobacter pylori infection has also been implicated as a risk factor for carcinoma and low-grade gastric lymphoma of mucosa associated with lymphoid tissue (Malt).

PATHOGENESIS OF PEPTIC ULCER

All peptic ulceration probably arises because of an imbalance between aggressive action of acid pepsin secretion and the normal defenses of the gastroduodenal mucosa. For duodenal ulcer, the major causal influence appears to be exposure of the duodenal mucosa to excess amount of acid and pepsin. For gastric ulcer, the major causal influence appears to be some breakdown in the gastric mucosal defenses against acid and pepsin. The hypersecretion is related to an abnormally large total mass of parietal cells in the gastric mucosa, perhaps to either increased responsiveness of the parietal cells to secretory stimuli of the parietal cells to secretory stimuli or lack of normal regulatory controls. Increased levels of gastric or unusual sensitivity of the parietal Cells into gastrin stimulation may be involved. Individual with achlorhydria never develops a duodenal ulcer. Defect in the defense mechanism includes deficiencies in mucosal cell removal, in mucous production in elaboration of bicarbonate and in production of prostaglandin. Irrespective of treatment, peptic ulcer takes one of the courses :

- Healing
- Chronicity

The **complications** of peptic ulcer :

1. Haemorrhage
2. Perforation
3. Cicatrical contraction
4. Carcinomatous changes.

PATHOPHYSIOLOGY OF PEPTIC ULCER

"Perforation" is the natural termination of an ulcer, which continues to penetrate deeper tissues. Perforation of duodenal ulcer greatly out number gastric ulcer (Illingworth, 1975). The incidence of perforation 7- 10 cases per 100000 population/year. Perforation occurs in 10-15% of established cases of peptic ulcers and in about 2% of patients perforation is the first manifestation. Anterior ulcer tends to perforate because of the absence of protective viscera and major blood vessels, in contrast to the bleeding ulcers that are usually situated posteriorly in <10% of patients with high death rate. **Boyd** is of the opinion that perforation is more common in ulcers of short duration from few days to few weeks in which there is rapid penetration of deep layers. Ulcers of long duration with abundant scar tissue are less likely to perforate or penetrate. Ulcers with continuous symptoms are more harmful than ulcers with history of remissions.

In a study of 201 cases of perforation by John Gelmon in 1953, 119 (58%) were found to be acute and 82 (42%) were chronic ulcers. As judged by operation (Illingworth, 1975), in about 90% of cases the perforation has resulted from sloughing of the floor. In rest of the most careful questioning fails to elicit a previous history of peptic ulcer .It is also fallacious to conclude whether the ulcer is chronic or acute from the naked eye appearance of the perforation at the time of operation. Lawdon (1952) in a survey

of series treated by primary gastrectomy and subjected to histopathological examination concludes that perforation occurs more often in chronic ulcers. From those of 41 cases, 22 were undoubtedly chronic, 16 were grouped as subacute and remaining 3 as relatively acute.

PATHOGENESIS OF SEPSIS AND MULTIORGAN FAILURE SYNDROME

Various host defense mechanisms and bacterial virulence factors play important role in determining outcome of a patient with peritonitis. The significant and major intraperitoneal defence mechanisms already elucidated are:

1. Mechanical clearance of the bacteria via lymphatics.
2. Phagocytic killing of pathogens by immune cells.
3. Mechanical sequestration.

The bacteriological factors are:

Normal bowel flora

Even though anaerobic species make up for majority of normal colonic flora, they contribute very little to clinical intra-abdominal infections. The most commonly bacteria encountered in clinical infections are E.Coli and Enterobacter species, Klebsiella species and Pseudomonas species.

Synergestic interactions between anaerobes and endotoxic gram –ve organisms suppresses local defence mechanism and facilitates the establishment of severe infection. Aerobic bacteria lowers the oxidation reduction potential, favouring the growth of certain species of anaerobic bacteria.

Other factors

Some GIT secretions and body fluids act as adjuvant thereby increasing bacterial virulence or interfering with host defences.

Sequelae leading to multiorgan failure

Sepsis is the major risk factor in the development of multiorgan failure syndrome. Sequential pulmonary, hepatic, GIT and renal failure may be recognized as early as 12 hours beginning of sepsis in septic shock or as late as 7-10 days. The observation that MODS increases with severity and duration of shock highlights the importance of vigorous resuscitation and complete restoration of perfusion as rapidly as possible for better prognosis. Injury to micro vascular especially microvascular endothelium, is a factor common to ischaemia reperfusion injury and multiorgan failure syndrome. Neutrophils are potential mediators of micro vascular injury. These cells produce an assortment of agents.

Toxic neutrophil products

Proteases

Toxic Oxygen Products

Elastase OH+O2-

Collagenase -HO2

Cathepsin G HOC, H2O2

These products not only destroy bacteria, they also act in a non-specific fashion producing injury to normal microcirculation. The endothelial cell itself produces injury, when they are exposed to ischaemia, i.e. depression of ATP levels and increased xanthine oxidase. They produce free radical oxygen which causes

endothelial activation and injury directly through both membrane peroxidation and increased neutrophil adherence in chemotaxis. Considering importance of oxidant injury, as the main cause of MODS, several clinical trials are being conducted to evaluate oxidant scavengers, as a treatment modality to prevent MODS, NSAIDS that inhibit cyclooxygenase and prostanoid production may reduce pulmonary and myocardial injury in sepsis and ischaemia. Pentoxyphillin is an agent that may benefit patients with ischemic and septic injury through inhibition of neutrophil adherence. Anti capsule (LPS) antibodies are being tried to prevent gram -ve endotoxin damage.

Decisive Period

After understanding the sequence of perforation sepsis leading to MOFS, Miles and Burke brought a new concept of decisive time for the management of bacterial infection. This period refers to the time needed for bacterial numbers in peritoneal fluid or associated tissue to exceed a number greater than 105 / mm3 or (per gm of tissue) and establish an highly potent infection. Surgeons must be dealing with the infection before the bacterial count proliferates and reach these levels or remove the infective foci so that after operation, the residual numbers of bacteria are controlled and kept less than 105 / mm3.

DIAGNOSIS

The diagnosis can be divided into

1. Evaluation of perforation
2. Diagnosis of sepsis syndrome.

1. Evaluation of perforation

Clinical feature

Abdominal pain is almost universally the predominant presenting symptom. The historical characteristics of the abdominal pain can vary tremendously depending upon the ultimate cause. The pain of a fully established peritonitis is constant, burning and greatly aggravated by movement. Pain is usually localized to that dermatome distribution of the diseased visceral organ. Visceral peritoneum irritation usually is from the distension of a hollow viscus, causes a dull, poorly localized, and very often periumbilical in location and often severe crampy type of pain. Most symptoms result from the inflammation of the visceral peritoneum, which receives numerous afferent innervation from the autonomic nervous system and is relatively quiet insensitive, visceral afferent nerves respond primarily only to traction or distension, but less well to pressure. Hence, stimuli are perceived as poorly localized dull discomfort. As inflammation spreads from visceral to parietal peritoneum, the somatic pathways of the parietal peritoneum becomes involved, the pain seems to "migrate" from the region of epigastrium or umbilicus, to that involved quadrant or to the entire abdomen, depending on the extent of inflammation. Patient may present with other signs and symptoms like nausea, vomiting, alteration in bowel habits and systemic features like fever, sweating, tachycardia depending on the extent of inflammation. They can be conveniently divided into localized peritonitis and diffuse (generalized) peritonitis.

1. Localised Peritonitis

Here, the signs and symptoms are intimately related to the origin of the condition. Patient present with abdominal pain and usually there is an associated vomiting. **The most important sign is guarding and rigidity of abdominal wall , which gets involved, with a very typical positive "release" sign.** The guarding may be severe to produce board like rigidity, (rebound tenderness). It may be associated with increased local temperature and increase in pulse rate, depending upon the inflammation, the features may either subside or progress to diffuse peritonitis.

2. Diffuse peritonitis

Diffuse peritonitis presents in different ways depending on the period of infection.

Early :

Abdominal pain is usually quiet severe and made worse on moving. Patient lies still in this case. Tenderness with rigidity and palpation are typically found features when the peritonitis affects the anterior abdominal wall. Patients with pelvic peritonitis may not have abdominal wall tenderness and may complain of urinary symptoms. Vomiting may occur. The pulse keeps rising continously, but if the peritoneum is filled with any irritant fluid, there is a sudden immediate rise. The Temperature changes can be variable and may be even subnormal.

Late :

If resolution or localization of generalized type of peritonitis does not follow, the abdomen keeps silent and increasingly gets distended. Circulatory failure can even ensue, with cold, clammy extremities, sunken eyes and tongue turns dry with pulse

being thready and drawn in anxious face(Hippocratic faces). The patient finally lapse into unconsciousness.

Percussion :

The liver dullness may be obliterated due to gas under diaphragm. There may be hyper-resonance due to distended bowel.

Ausculation :

It is performed how to determine whether the bowel sounds are diminished, normoactive and hyperactive. Any Hyperactive sounds are suggestive of features of some obstructive element either as a primary process or as a part of localized inflammatory process. Paralytic ileus results in silent abdomen later.

Radiological investigation

Plain radiograph of the abdomen is often the first investigation to be performed. It is usually done in erect position, it may reveal free air under the diaphragm. Patient should remain in an upright position for about 5 minutes or more the film is taken. If the patient cannot stand, a lateral decubitus film can be taken and air at the right lateral abdominal wall can be detected under the liver, with liver as contrast. Other informative features include paralytic ileus, inflammatory exudates and oedema of the intestines produced widening of the space between adjacent loops on a plain film. Peritoneal fat lines and the retroperitoneal psoas shadow can get obliterated, because of the associated edema. The fat lines of the pelvis may get obliterated suggesting the fluid collection in the pelvis. A new clinical test by Khanna was devised which uses air insufflation test, with radiography reported high accuracy of 95%. This test is best utilized for preoperative detection of site of perforation in peritonitis. Water contrast

upper GI studies can be done to detect site of perforation but is best reserved for early cases.

Peritoneal diagnostic aspiration

Four quadrant aspiration using needle or intraveneous needle attached to syringe into which free fluid is obtained.Bile stain fluid indicates that a perforated ulcer or gall bladder perforation, the presence of pus and this indicates bacterial peritonitis.The fluid can be sent for culture. Ultra sound and CT scanning are mainly utilized for detection of subsequent complications like intraabdominal abscesses.

Diagnosis of sepsis syndrome

Diagnosis of sepsis and sepsis syndrome hinges on understanding and identifying at an early stage the existence of a generalized inflammatory state.The systemic responses include hyperpyrexia, tachycardia, tachypnoea, decreased urine output, leucocytosis.While the presence of fever, leucocytosis, hypotension and hypermetabolic state are suggestive of sepsis, overwhelming may also result in leucopenia, cardiac suppression and shock.

Feature of Sepsis

Temperature > 101oF (or <96oF as is frequently encountered in elderly)

Heart rate > 100 / minute

Respiratory rate > 20 / minute

Leukocytosis (> 12,000 / mm3 or < 4000 / mm3)

Manifestation of inadequate organ perfusion / Diminished mental status

Acidosis -plasma lactate with >3.0nmol / L

Urine output < 30 ml / hour or 0.5 ml / kg /hour.

Hypoxemia (Pa O2 < 70 on room air in the absence of underlying pulmonary disease)

The most important complication of sepsis is MODS. There have been efforts to quantitate MODS using practical bedside information. According to Fry, the criteria for failure were:

Pulmonary failure in this system was defined as 5 or more consecutive days of need for ventilator support at an FIO2 of 0.4 or greater.

Hepatic - Bilirubin > 2 gm / dl, SGOT / LDH > twice normal. The inclusion of enzyme data was designated to exclude transient hyperbillirubinemia that might be associated with retroperitoneal hematoma, pelvic fracture or potential icterus from an incompatible unit of blood.

Renal failure – S. creatinine> 2 mg /dl.

GIT failure - UGI haemorrhage

The above criteria are mainly for the use of clinical trails and epidemiological studies. In clinical practice, the above criteria should be correlated with clinical findings as most of the criteria are hypothetical and further clinico-pathological studies are needed to confirm its validity.

PATHOLOGY:

At the onset of perforation there is sudden spillage of gut contents into general peritoneal cavity and results in peritonitis.Most commonly secondary to Perforation of peptic ulcer , which may be classified as follows:

1. Acute perforation
2. Subacute perforation
3. Chronic perforation

4. Perforation associated with haemorrhage

5. Perforation of intrathoracic gastric ulceration

6.Pseudoperforation

1. Acute perforation: The ulcer perforates and the general peritoneal cavity become flooded with gastric and duodenal contents, causing chemical peritonitis. The clinical features vary according to the stage of perforation. The clinical course can be divided into three stages, each of variable duration.

a. Primary stage or the stage of peritonism:

The patient feels acute agonizing pain in the epigastrium or right hypochondrium, which usually becomes generalized. The symptoms are due to the intense irritation of peritoneum by the gastric and duodenal contents. Pulse rate is normal or raised. Respiration is shallow with increased respiration rate. On inspection, the abdomen will be seen to be immobile with no movement with respiration; the muscles are rigid and board like. On auscultation bowel sounds are absent. This stage lasts for 3- 6 hours.

b. Secondary stage or the stage of peritoneal reaction:

In this stage the spontaneous sealing of perforation may occur. If there is gross leakage of gastric contents, the patient may pass onto the stage of septic peritonitis. The length of this stage rarely exceeds 6 hours. During this stage the pain is lessened markedly. There would be general improvement in the patients' condition. For this reason this stage of reaction has sometimes been called stage of delusion.

c. Tertiary stage or the stage of bacterial peritonitis:

This is the stage of diffuse peritonitis, begins about 12 hours after perforation and lasts for about 24 hours until it passes on to the final stage of paralytic intestinal obstruction. Pathogenic organism multiplies rapidly. Peritoneal fluid becomes more purulent. Intestinal movements diminish and finally disappear with the onset of paralytic ileus. The patient drifts into toxemia, dehydration and circulatory failure. Death usually takes place 4-5 days after perforation.

2. Sub acute Perforation:

An ulcer may perforate and the perforation may seal rapidly before there is spillage of gastric and duodenal contents, into general peritoneal cavity. There is sudden onset of abdominal pain, more severe to the right upper quadrant. On examination, there isa local tenderness and rigidity, but rest of the abdomen will be soft to palpate and non-tender. Unusualy and X-ray film reveal a small amount of gas under the diaphragm. After an hour or two, the pain will usually subside. Rarely tenderness and rigidity may extend and the signs of an acute perforation develop.

3. Chronic Perforation:

When an ulcer perforates into an area that is walled off by adhesions or by adjacent viscera such as liver, colon or greater omentum or when gastric ulcer perforates into omental sac, a chronic abscess may develop and will give rise to considerable confusion in diagnosis. As these patients do not present with signs and symptoms of peritonitis, they are seldom diagnosed as having perforated peptic ulcer. An X-ray of abdomen may show subphrenic abscess, containing gas, and diaphragm is raised and fixed on the right side. USG of abdomen is the most reliable investigation on diagnosing intraperitoneal abscess.

4. Perforation associated with Hemorrhage:

The association of a perforation with massive haemorrhage is grave but the association of a perforation with massive haemorrhage is grave but fortunately one of the rare complication. It may present the three ways:

a. Haemorrhage and perforation occurring concommitantly.

b. Haemorrhage following a recently sutured perforation.

c. Perforation occurring during the medical treatment of haemorrhage.

The clinical features are that of perforated peptic ulcer with signs of haemorrhage.

5.**Perforation of an Intrathoracic gastric ulcer:**

This is a rare variety of perforations. The ulcer is in hiatus hernia, which is fixed in the mediastinum. Unless existence of Hiatus hernia is known it is extremely difficult to make a correct diagnosis.

Rare type of perforated peptic ulcer:

A peptic ulcer in a Meckel's diverticulum, in intestinal duplication occasionally perforates. Multiple simultaneous perforations occur in less than one percent of all cases.

CLINICAL FEATURES

Age and sex: It is common in 30-40 years age group and common in males than females.

History of Present illness:

Time of onset: Very often the patient tells the exact time of onset of perforation, common particularly after an exertion in the evening.

Mode of onset: Sudden in onset, at times the patient may wake up from the sleep, due to onset of pain.

Pain: Pain is intense in the epigastrium then spreads all over the abdomen.

Shifting of pain: The pain shifts to right iliac fossa as the collected fluid flows along the paracolic gutter to settle in right iliac fossa, thus mimicking appendicitis.

Referred pain: Pain gets referred to the tip of the shoulder.

Nausea: Present in some cases.

Vomiting: Initially reflex vomiting occur due to irritation of nerves in the peritoneum and mesentery. In the later stages the vomiting is due to toxin action at the medullary centre's and causing paralytic ileus. The vomiting then contains undigested food material and occasionally blood when hemorrhage is present.

Bowels: In the later stage, there may be desire to defecate due to irritation of retrovesical pouch. Malaena occurs when the hemorrhage is associated.

Micturition: Oliguria is present if the patient is in shock.

Past History:

In 80% of patients, there is a past history of dyspepsia of variable duration. In the rest of the cases, the perforation may be the early clinical manifestation of a silent peptic ulcer.

Physical Examination:

General Appearance: In the initial stage, the face is pale livid with sweating.

Decubitus: The patient lies in a characteristic posture of supine, rigid and immovable, refusal of any attempt to shift his postures.

Pulse: Initially it is normal, rapid when peritonitis sets.

Respiration: It becomes rapid and shallow when peritonitis sets in.

Temperature: Initially normal; rises with the onset of peritonitis.

Examination of Abdomen:

Respiratory movements: Thoracic movement predominates over the abdominal movement with respiration. The abdomen does not move with respiration.

Rigidity of abdomen: The abdomen exhibits a board-like rigidity. Rigidity of abdomen is constant, continuous and characteristic board like. It is due to reflex contraction of the abdomen with predominance in the epigastrium and right hypochondrium.

Liver dullness: Obliteration of liver dullness elicited in front and in midaxillary line, is characteristic of this abdominal catastrophe in the second stage.

Free fluid: Free fluid is present in variable degree in many acute abdominal conditions. When internal hemorrhage is excluded, fluid of appreciable amount points out the provisional diagnosis of perforation in acute abdomen.

Rectal examination: There may be fullness in rectovesical or rectovaginal pouch.

INVESTIGATIONS

1. Plain x-ray: The "gold standard" for diagnosis remains the finding of pneumoperitoneum, which can be seen on an upright anteroposterior radiograph of the chest or the left lateral decubitus view of the abdomen. If the radiograph is taken with the patient in sitting posture and the patient has been in the upright position for 5 to 10 minutes, as little as 5 mL of free air can be seen under one or the other hemidiaphragm. With the left lateral decubitus position, the patient should be lying on the left side, and the first film should be taken with the patient on the cart in that position so that even a very small amount of air will become visible with, again, 5 to 10 minutes in the indicated position.

2. Gastroduodenogram: Some clinics have used X-ray pictures of abdomen following injection of 60ml of 50% gastrograffin through nasogastric tube. The dye escapes through perforation, thus enabling to demonstrate the site and size of perforation, evidence of chronicity, associated gastric ulcer and if any second ulcer present. Use of barium for contrast radiography is contraindicated.

3. Ultrasound Examination: Ultrasonography of abdomen is done using a convex multi frequency probe (3.5-5 MHz). The Evidence of free fluid present intraperitoneum and of reduced intestinal peristalsis was considered as an indirect evidence of perforation. Ultrasound will also demonstrate the free air and occasionally a "fish-eye sign" when the anterior wall of the duodenum is perforated.

4. Computerized Tomographic Examination: Computed tomography (CT) is not often necessary, although it can be used when free air is not detected on conventional films or ultrasound; it is highly accurate in detecting even very small amounts of extra luminal free air.

5. Helicobacter Pylori diagnosis:

I. Non invasive -a) Serology- ELISA.

b) Urea breath test.

II. Invasive – a) Rapid urease test e.g. Eco, Pyloritek

b) Histology.

c) Culture.--Noninvasive tests do not require endoscopy, whereas invasive tests do.

TREATMENT

Immediate management (Resuscitation):

1. Patient is kept to be nil per oral.

2. Treatment begins with insertion of a NG tube to decompress the stomach and limit additional peritoneal soilage.

3. A Foley's catheter is inserted to monitor urine output and direct resuscitation.

4. The patient is resuscitated aggressively by administration of intravenous crystalloid.

5. Intravenous broad-spectrum antibiotics are also administered.

6. Invasive hemodynamic monitors (e.g., central venous, arterial, and pulmonary artery catheters) are inserted as clinical status and comorbid medical conditions dictate.

7. Associated medical illnesses such as respiratory disease should be treated quickly and effectively so as to minimize complications.

8. Informed consent should be taken.

Operative treatment is generally advocated as the best option, but conservative treatment is an alternative in carefully selected patients.

NON OPERATIVE / CONSERVATIVE TREATMENT:

It can be considered in

•In patients who do not have generalized peritonitis,

•In patients who are hemodynamic stable,

•In patients whom a water-soluble contrast study has confirmed that the ulcer is sealed with no extravasation of contrast into the peritoneal cavity.

The patient can be treated expectantly with nasogastric suction, intravenous fluids, antibiotics, and bed rest. If at any time during conservative management the patient deteriorates, an operation is indicated. Conservatively managed patients often develop intra-abdominal abscesses, especially in the subhepatic or subdiaphragmatic locations, and these abscesses usually can be managed percutaneously.

In the largest published series of patients with perforated duodenal ulcer who were managed conservatively, patients who were 70 years of and older were much more likely to require operative therapy and had a higher mortality rate.

OPERATIVE TREATMENT:

In perforated duodenal ulcers options

•SIMPLE CLOSURE WITH AN OMENTAL ONLAY REINFORCEMENT OR PATCH.

Simple patch closure is appropriate in patients who have not been previously managed for peptic ulcer disease and who take Proton pump inhibitors and antibiotics for *H. pylori* infection.

•Perforated duodenal ulcer patch closure has, at least in North America, come to be called the Graham closure, first described by Roscoe Graham in 1938.

•The operation is usually done through an upper midline incision. After clearing the peritoneal cavity of purulent and/or bilious fluid, visual inspection and palpation direct the surgeon to the site of perforation. The point of perforation

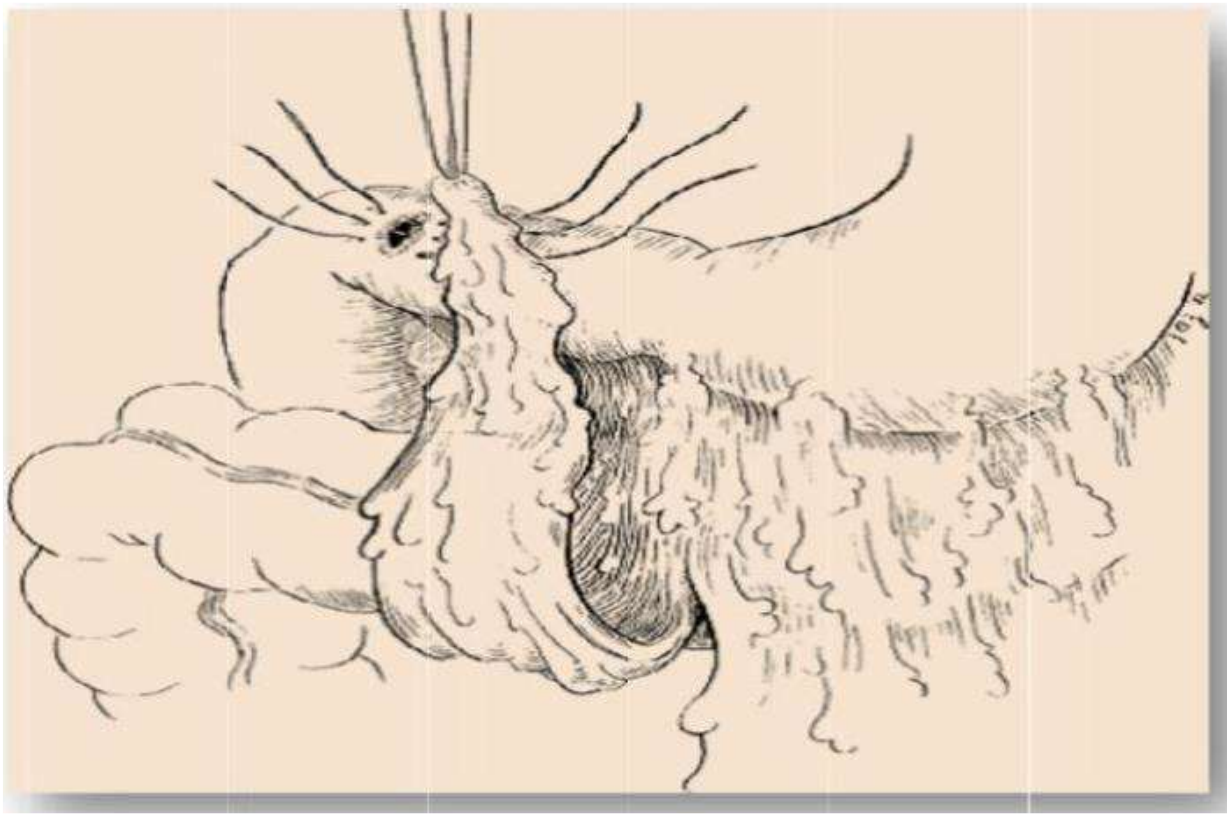
usually is recognized easily in the proximal anterior duodenum. If not apparent, exploration of the remainder of the duodenum, the anterior and posterior gastric walls, and the jejunum is undertaken.

•Three, sometimes four, sutures are used, preferably of nonabsorbable material.

•The sutures are placed at perforation site before any are tied. Adjacent omentum is brought up to the perforation with the sutures untied and laid out on the anterior surface of the duodenum, and are successively tied from the superior to the inferior side, so as to tampon the perforation with the vascularized omental pedicle graft. Care should be exercised to be sure that the sutures are tied sufficiently snugly to hold the omentum in place, but the tension exerted by the tied sutures on the omentum should be such that the blood supply to the omentum is not impaired. The patch must be a living omental patch, and the omentum should not be strangulated.

•Some surgeons have modified this technique in which the three or four sutures are placed and are then tied to close the ulcer. The omental patch is placed on the tied sutures, and another set of knots is tied to hold the omentum in place over the duodenal ulcer perforation closure.

• After proceeding with closure of the perforation, irrigation of the peritoneal cavity with warm normal saline solution should be carried out and abdomen is then closed in standard fashion.



After placing three or four sutures, a vascularized tongue of omentum is mobilized and brought superiorly to close the defect. It is not necessary to push the end of the omentum into the defect like an obturator, but rather use the omentum as an external patch. When the sutures are tied loosely enough so that the blood supply to the omentum is not compromised, the seal is complete, even with larger perforations.

Modified Graham patch repair (MGPR),

In which the three or four sutures are placed as described above and are then tied to close the ulcer. The omental patch placed on the tied suture, and another set of knots are tied to hold the omentum in place over the duodenal perforation closure

2. Simple closure with proximal gastric vagotomy

In patients with perforated duodenal ulcers, proximal gastric vagotomy has been used in conjunction with patching the perforation. The acidproducing parietal cells are selectively denervated while smooth muscle innervation to the antrum is preserved. So a drainage procedure is not required.

3. A truncal vagotomy & drainage procedure

Definitive surgery in the form of vagotomy and pyloroplasty or antrectomy should be considered in patients who will require continuednonselective NSAIDs, who fail with optimal medical treatment or in whom the compliance with *H. pylori* therapy is doubtful.The choice between pyloroplasty and gastroenterostomy is dictated by the conditions that prevail in the pyloroduodenal area. Where

possible, a perforation is incorporated in a pyloroplasty but significant stenosis may mean that simple closure of the perforation and a gastroenterostomy is preferable.A gastrojejunostomy may also be required when the repair of a very scarred duodenal ulcer might produce some gastric-outlet obstruction.

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LAPAROSCOPIC REPAIR of perforated ulcer:

•This is becoming popular in patients who are haemodynamically stable and have no evidence of sepsis.

•The principles are very muchsimilar to that of an open technique. The falciform ligament can be sutured over the ulcer as a good alternative procedure to the classical Graham patch. This repair issimpler to do than a laparoscopic omental patch repair type.

•If a naturally occurring omental plug is found, a simple peritoneal toilet alone may be tried. This laparoscopic approach helps to get better access and vision Especially peritoneal Lavage in the subphrenic and pelvic spaces.

•The laparoscopic approach usually offers comparable efficacy, morbidity and mortality to open surgery with a shorter hospital stay, lower analgesic needs, and a shorterrecovery time to daily chores. The laparoscopic repair is associated with shorter operative period.

The outcome of patients with a perforated ulcer depends on the following:

1. Delay from initial evaluation to treatment: recent data suggest increasing delay until surgical treatment.

2. Site of perforation: gastric perforations are associated with a poorer prognosis.

3. Patient's age: elderly patients, who often have associated comorbid conditions, have a worse outcome.

4. Presence of hypotension at initial evaluation (systolic blood pressure <100 mm Hg).

Post operative complications:

Complications are likely to happen in higher risk patients. The common Complications includes:

Paralytic Ileus

Wound Infection

Intraperitoneal abscess,usually subphrenic or pelvic peritonitis

Respiratory complications like atelectasis, pneumonia and pleural effusion and respiratory failure

Gastric and duodenal fistulae

Renal failure

Mediastinitis

MATERIALS AND METHODS

AIM:

The aim of this study is to compare outcome and complications of Graham's omental patch closure versus Modified Graham's omental patch closure for treating perforated duodenal ulcers.

MATERIALS USED:

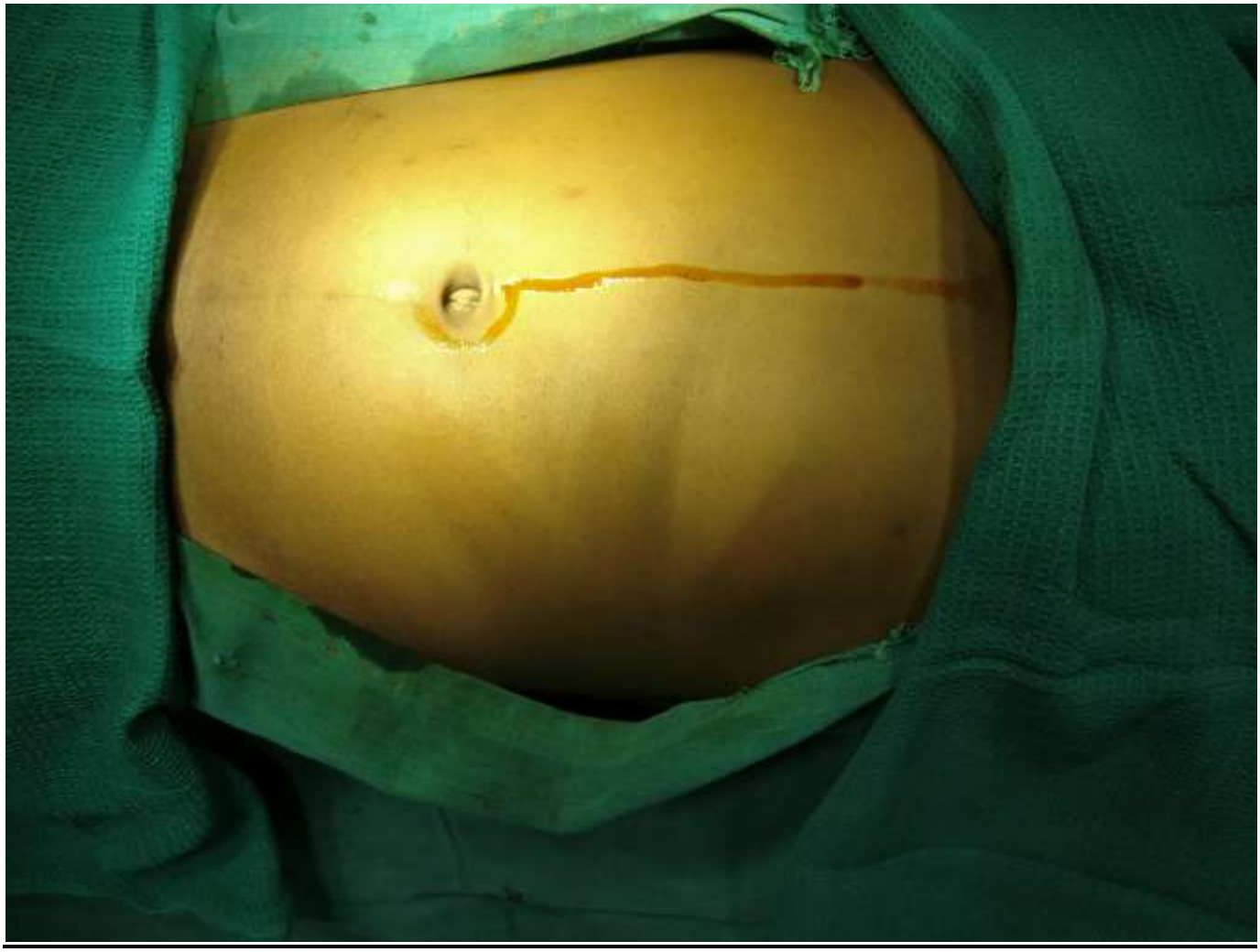
Proforma containing patient history, clinical examination ,Informed consent forms.

METHODOLOGY:

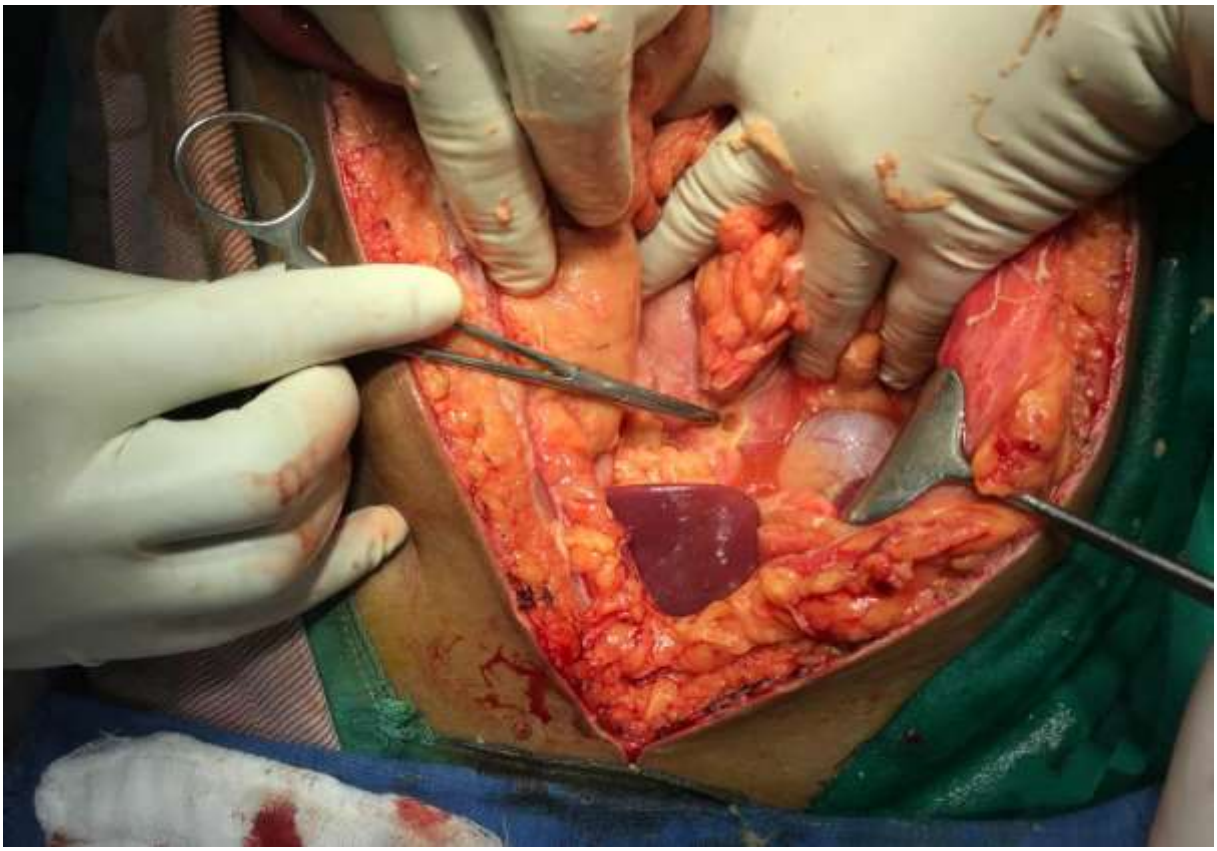
After obtaining clearance and approval from the institutional ethical committee and patients fulfilling the inclusion / exclusion criteria were included in the study after obtaining informed consent.

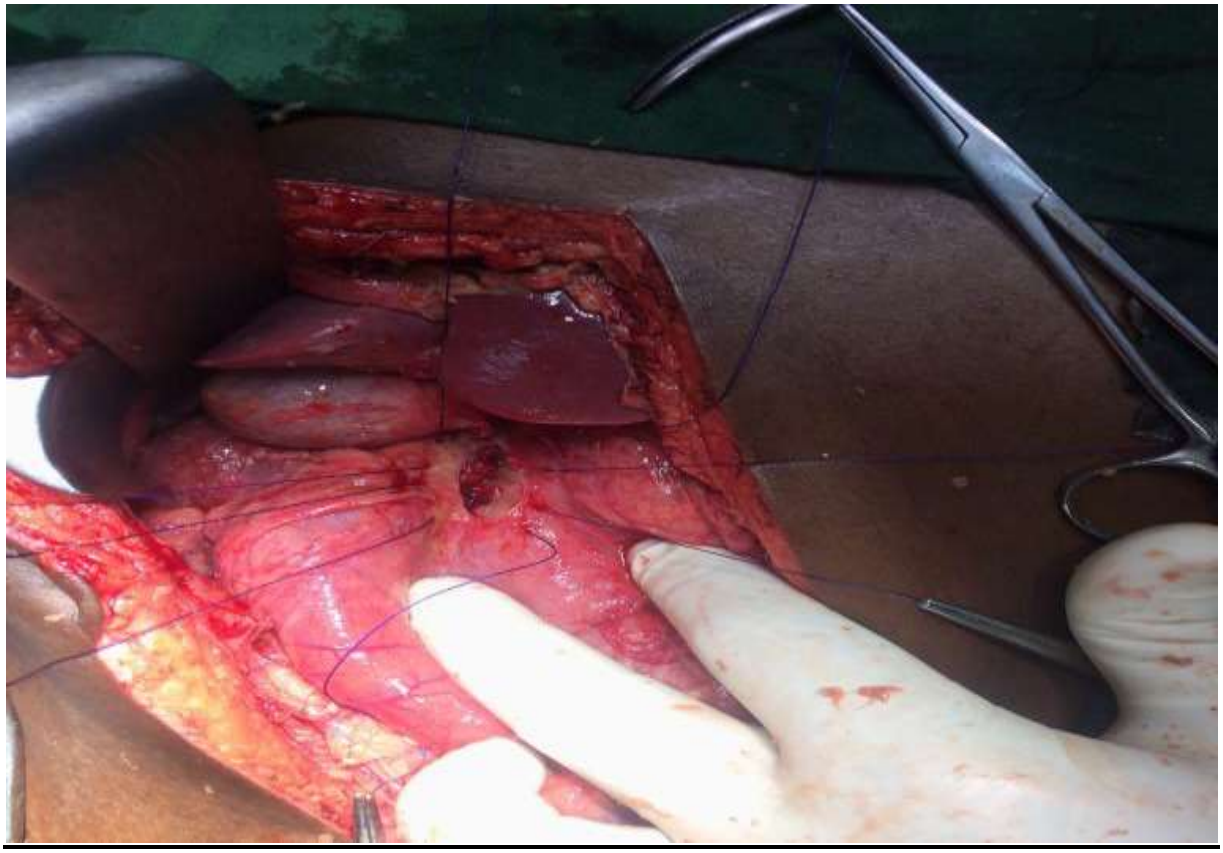
- Patients admitted in surgery department who are diagnosed with perforated duodenal ulcer are grouped into group 1 and group 2.
- Non probability purposive sampling technique was used to allocate the subjects into group 1(Grahams omental patch closure)and group 2 (Modified Graham's omental patch closure).
- Initial preoperative work up and resuscitation with intravenous fluids, antibiotics, analgesics, nasogastric decompression was done in all the cases.

- **GRAHAMS OMENTAL PATCH REPAIR**



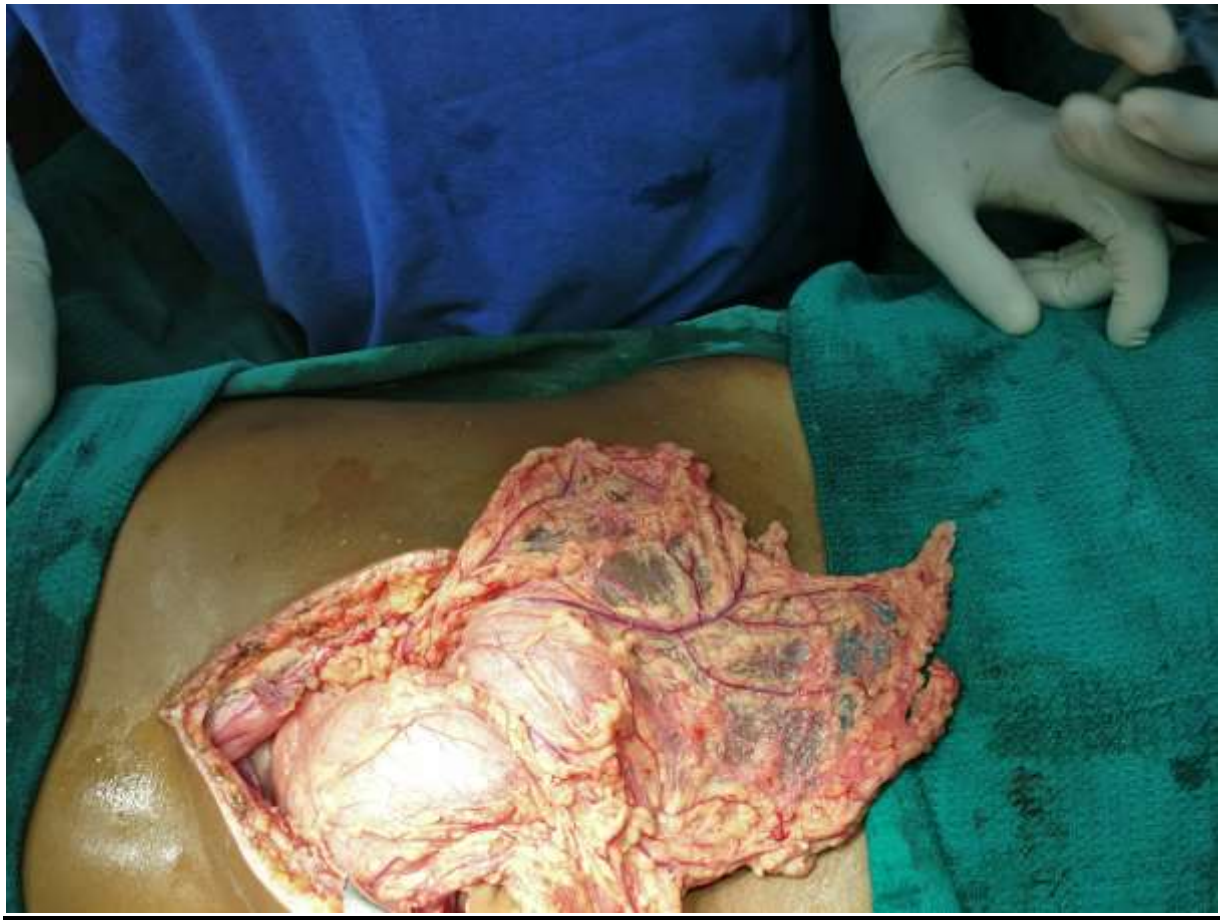
- Under ETGA, under strict aseptic precautions, parts painted and draped.
- Midline laparotomy incision made. Incision deepened
- Peritoneal cavity opened
- After confirming the diagnosis of perforation , suctioning of peritoneal toxic fluid done.
- After laparotomy, packs are placed around the perforation to contain any further spill.





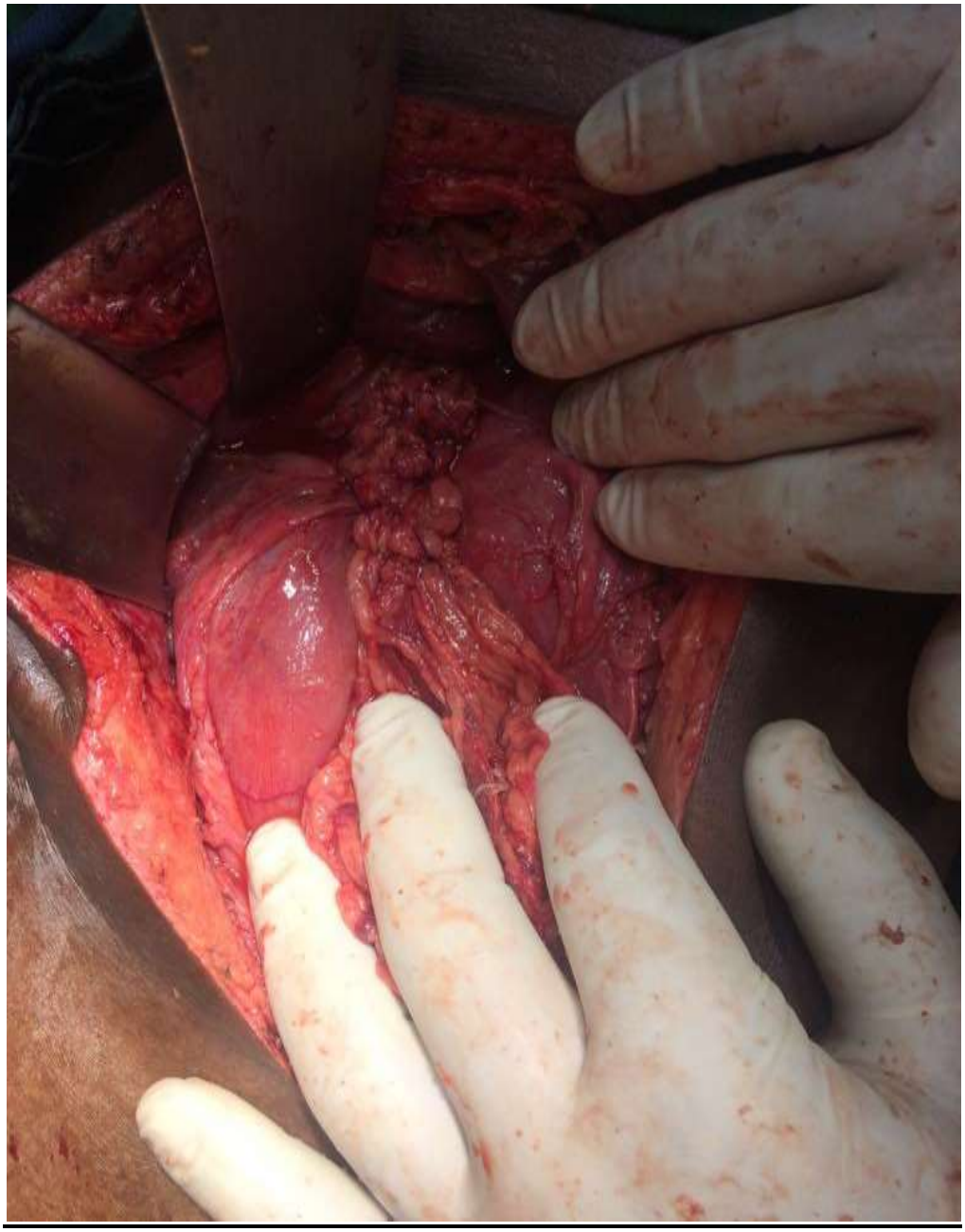
. Three or four sutures are used preferably of non absorbable material. If the needle is introduced, with care being taken to avoid the posterior duodenal mucosa and the needle is passed parallel to the anterior wall of duodenum

.Before sutures are tied, the adjacent omentum is brought up to the perforation with the sutures untied and laid out on the anterior surface of the duodenum,



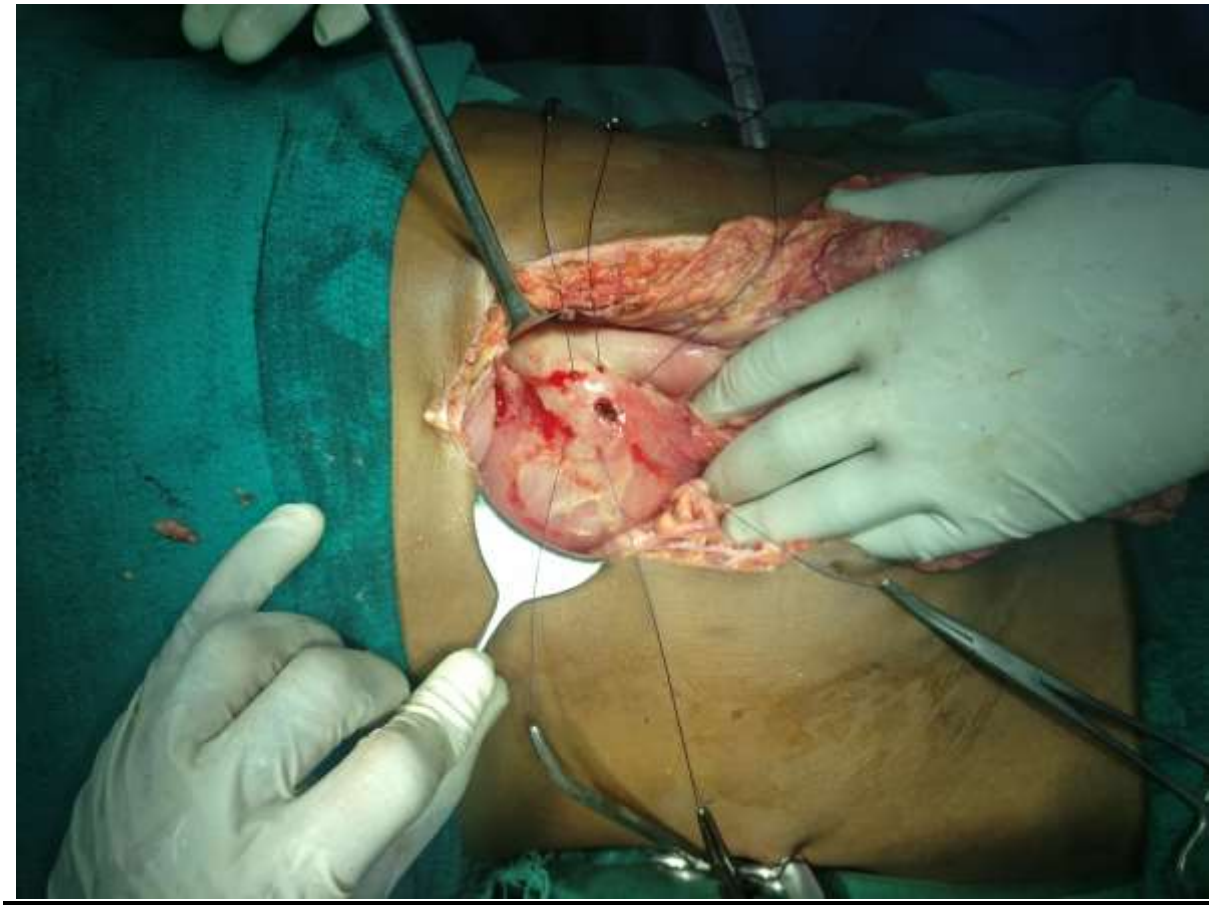
- Sutures are then successively tied from the superior to inferior side, so as to tampon the perforation with the vascularised omental pedicle graft.

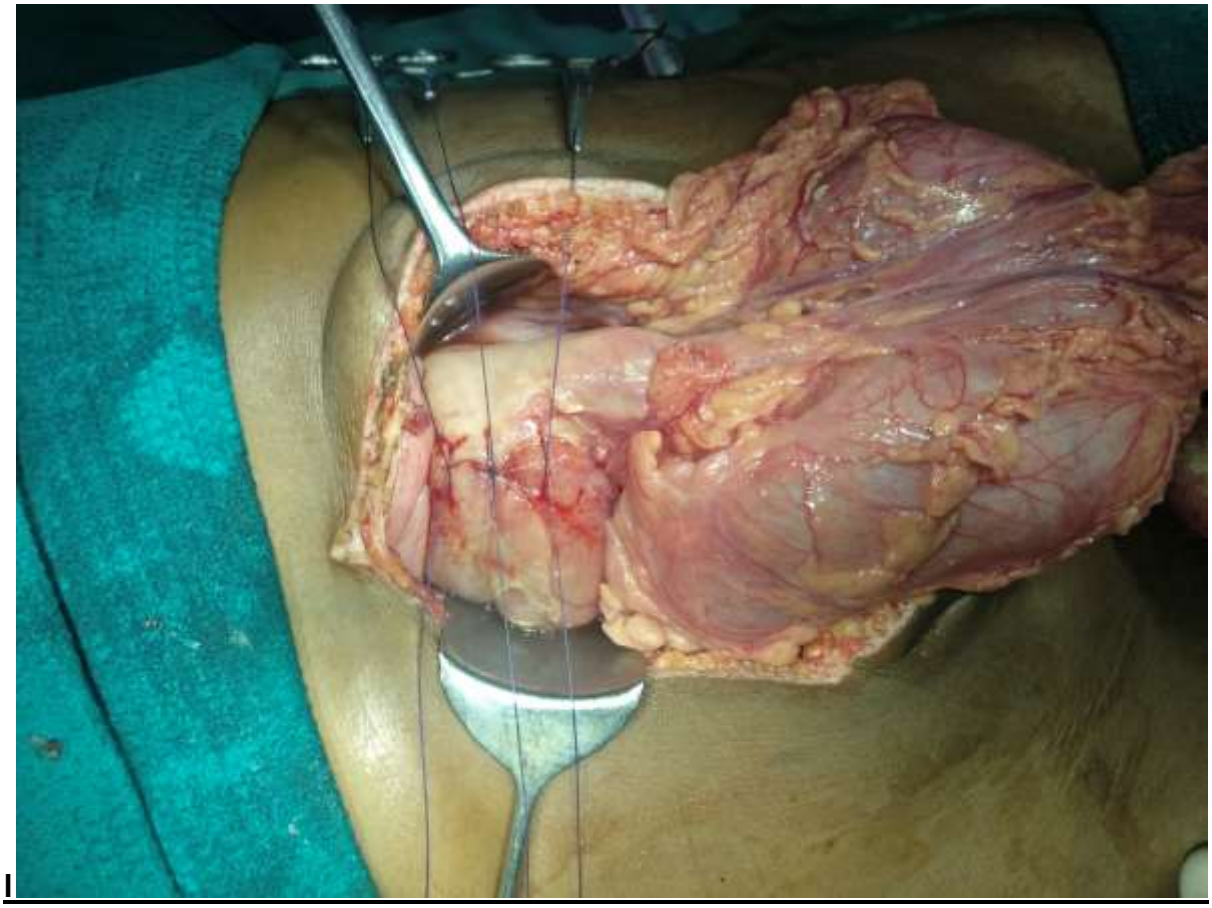
- Care should be exercised to be sure that the suture are tied sufficiently snugly to hold the omentum in place, but the tension exerted by the tied suture on the omentum should be such that the blood supply to the omentum is not impaired.
- The patch must be a living omental patch, and the omentum should not be strangulated

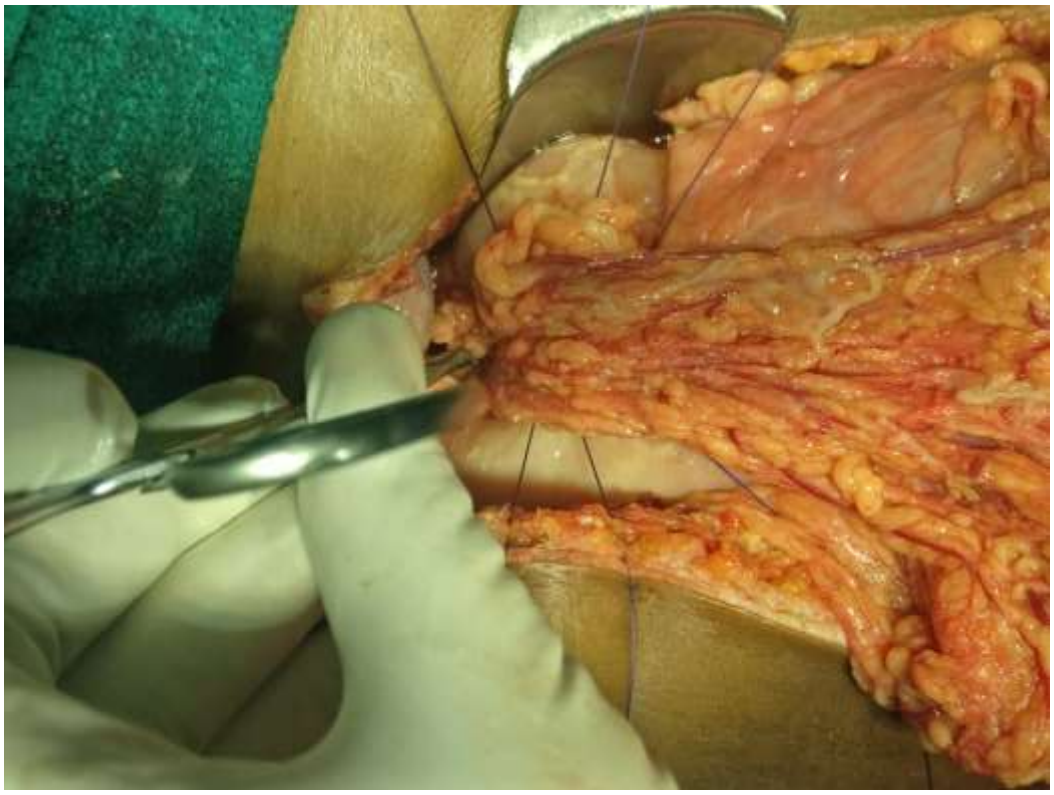


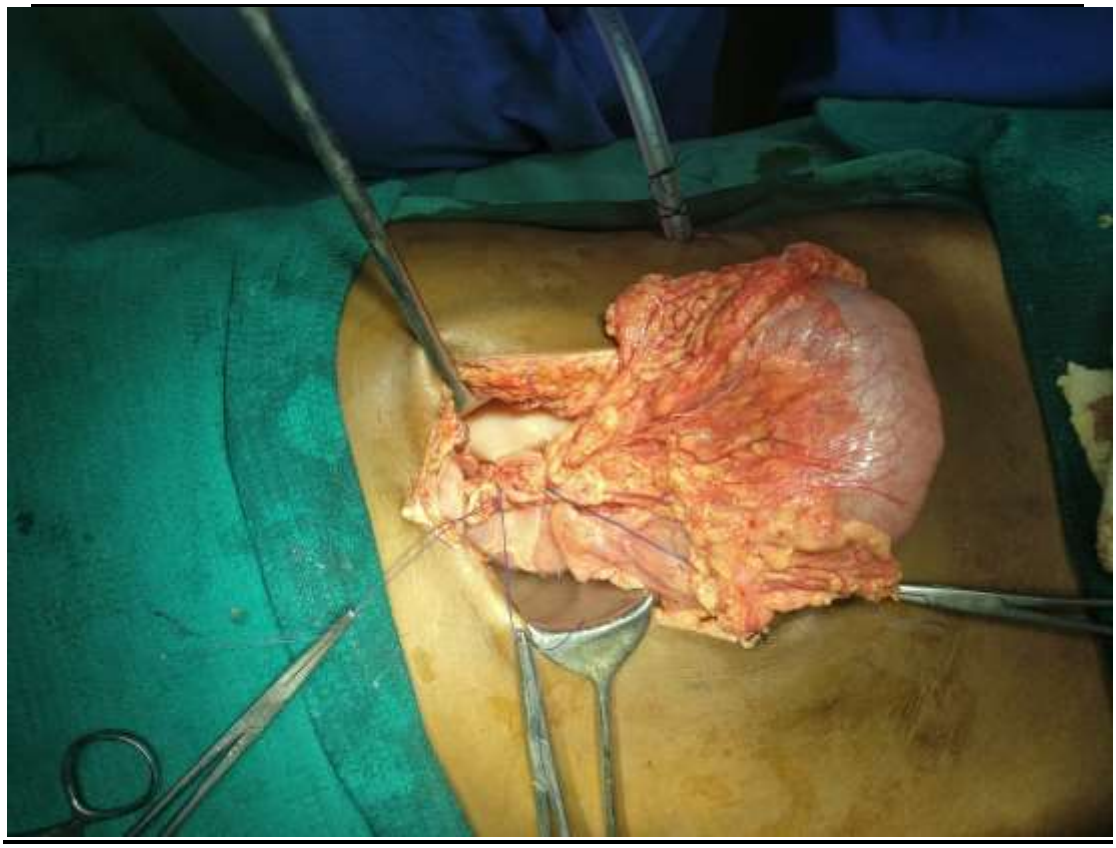
Modified Graham patch repair (MGPR)

- Under ETGA, under strict aseptic precautions, parts painted and draped.
- Midline laparotomy incision made. Incision deepened
- Peritoneal cavity opened
- After confirming the diagnosis of perforation , suctioning of peritoneal toxic fluid done
- Packs are placed around the perforation to contain any further spill. .
- Three or four sutures are used preferably of non absorbable material. If the needle is introduced, with care being taken to avoid the posterior duodenal mucosa and the needle is passed parallel to the anterior wall of duodenum
- In this surgery, the three or four sutures are placed as shown in picure and are then tied to close the ulcer
- The omental patch placed on the tied suture, and another set of knots are tied to hold the omentum in place over the duodenal perforation closure.









- Further resuscitation and ICU care was continued as and when necessary
- Assessment of patients 48 hrs after surgery & Postoperative complications.
- Outcome of the study was evaluated.

POSTOPERATIVE ANALYSIS

- **Assessment of patients 48 hrs after surgery**
    - Pulse rate
    - Systolic BP
    - Diastolic BP
    - Temperature
    - Respiratory rate
    - spO2
- **Postoperative complications**
    - Surgical site infection
    - Return of bowel function
    - Duration of ventilatory support
    - Duration of hospital stay
    - Death

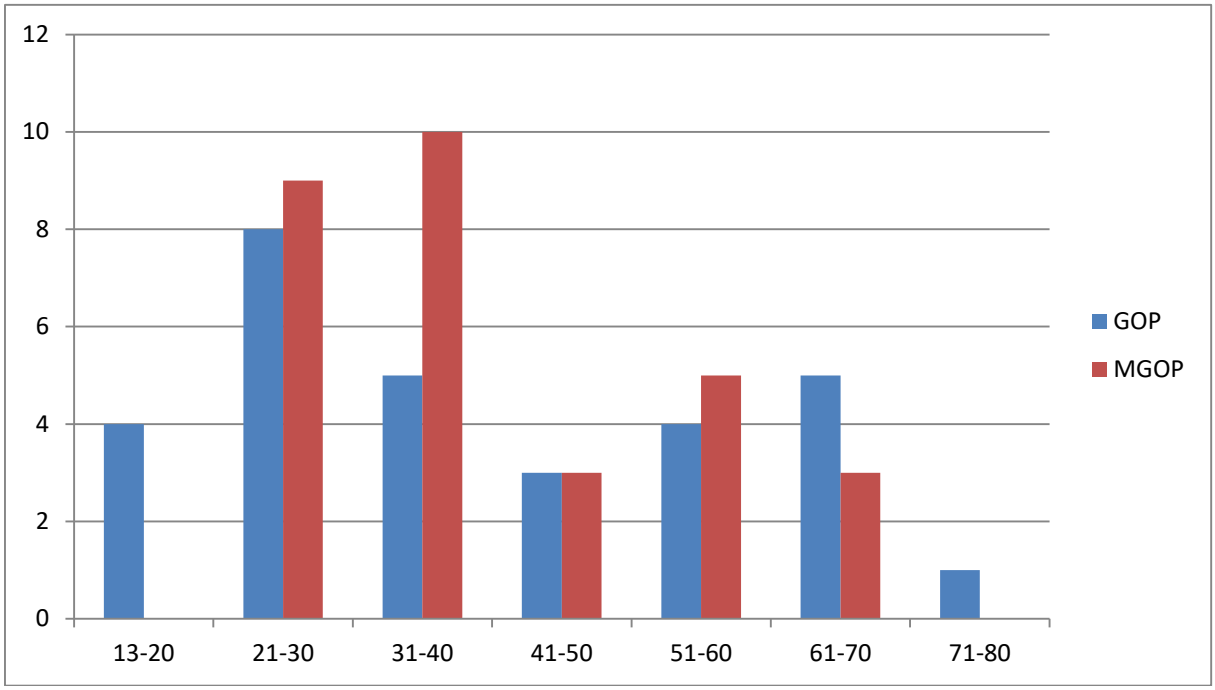
To determine and compare the accuracy of the MPI score and WSES prognostic score in predicting mortality,

The information collected regarding all the selected cases were recorded in a Master Chart. Data analysis was done with the help of computer using SPSS 16 and Sigma Stat 3.5 version.

Using this software range, frequencies, percentages, means, standard deviations, chi square and 'p' values were calculated by One way ANOVA and Chi-square test was used to test the significance of difference between quantitative variables.

A 'p' value less than 0.05 is taken to denote significant relationship.

OBSERVATION AND RESULTS

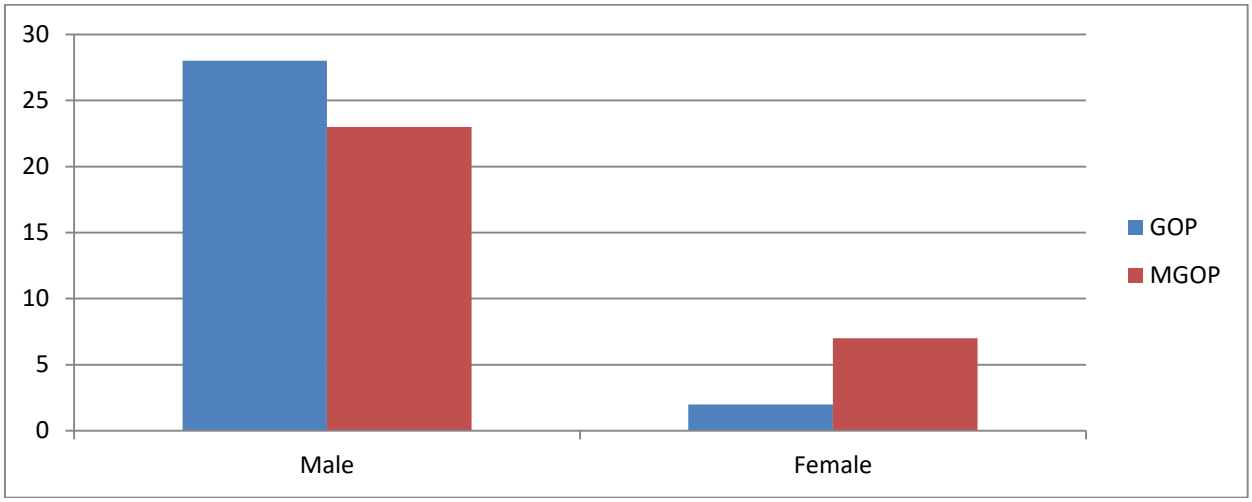


AGE WISE COMPARISON			
Sl No	Age group	GOP	MGOP
1	13-20	4	0
2	21-30	8	9
3	31-40	5	10
4	41-50	3	3
5	51-60	4	5
6	61-70	5	3
7	71-80	1	0

Age distribution of the patients affected ranged from 13-70 years in the present study. The maximum number of cases studied were in the age group of 31 to 40(25%) .

In group 1 mean age was 40.7 (SD – 15.020). In group 2 the mean age was 40.83 (SD-23.181). the two tailed P value equals 0.9795. this difference is considered to be statistically not significant.

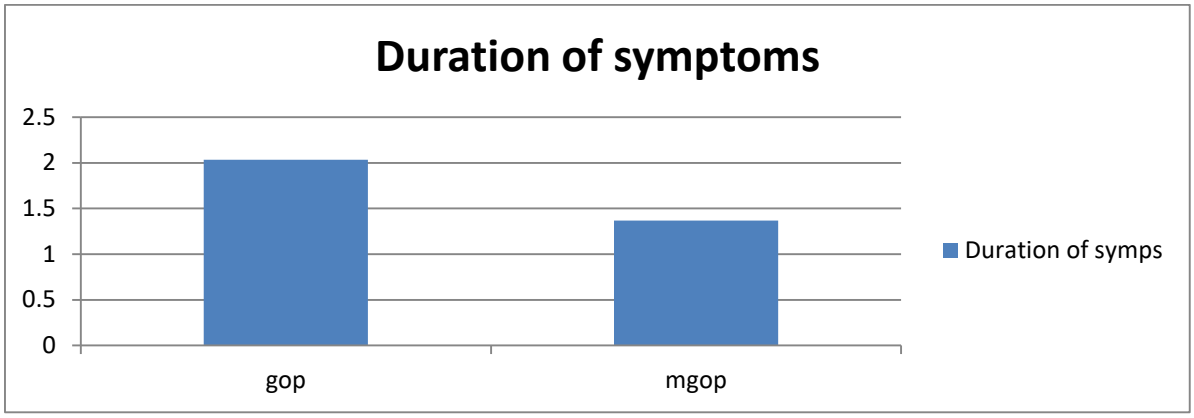
SEX WISE COMPARISON



SEX WISE COMPARISON		
	Male	Female
GOP	28	2
MGOP	23	7

Most of the patients were males (males 51%). Male female ratio was 51:9.

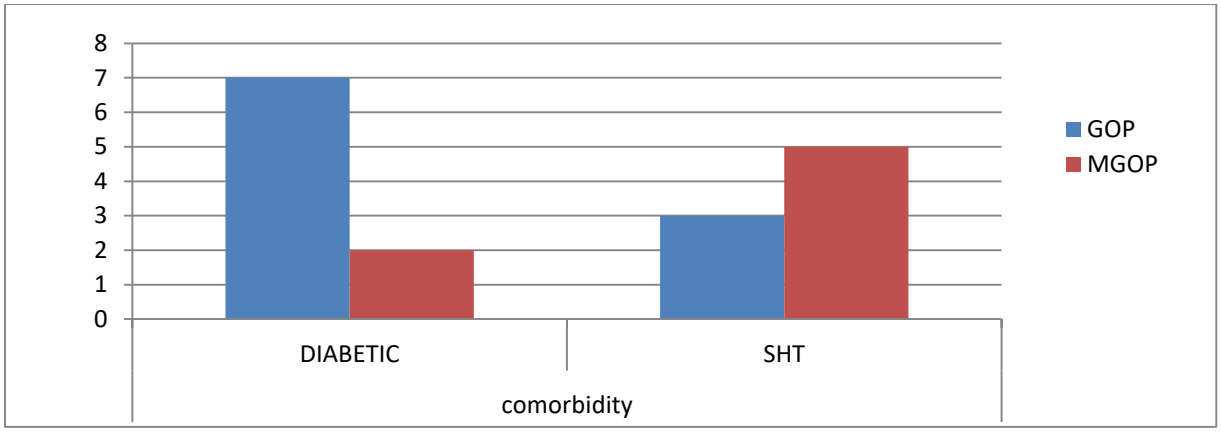
Comparing the two groups chi square statistics is 3.268. the p value is 0.70645. this result is not significant at $p< 0.05$.



Majority of patients presented with symptoms of abdominal pain for 1 to 2 days.

(mean days of presentation- 1.68 days).

COMORBIDITIES



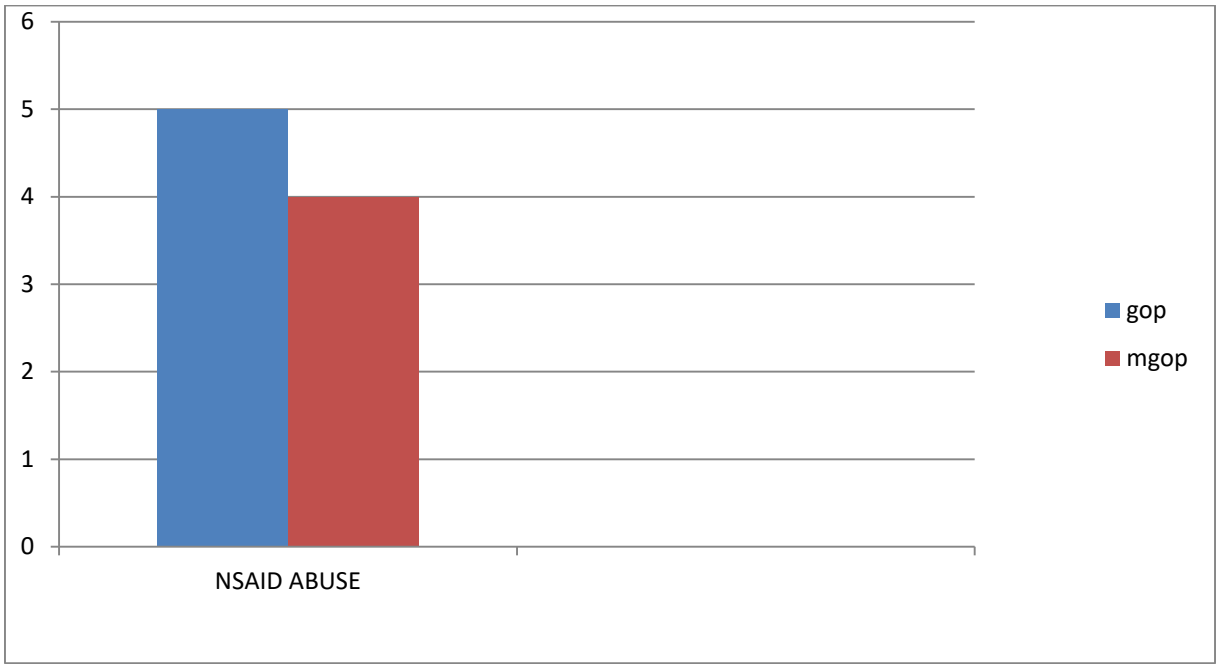
TYPE OF SURGERY	DIABETIC	SHT
GRAHAMS OMENTAL PATCH CLOSURE	7	3
MODIFIED GRAHAMS OMENTAL PATCH CLOSURE	2	5

9 out of 60 patients were diabetic,

3 out of 60 patients were hypertensive.

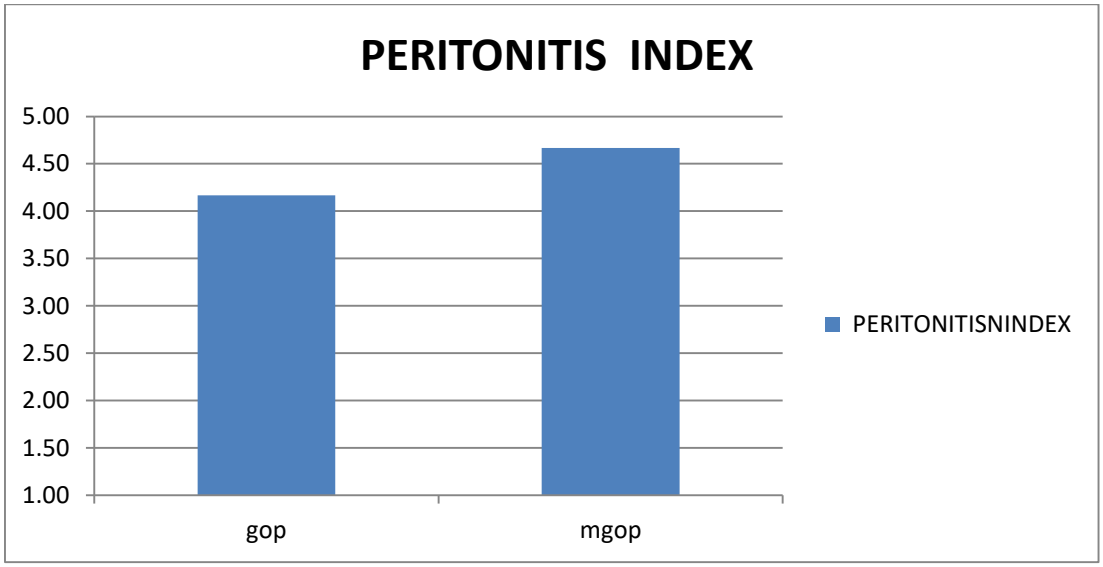
SUBSTANCE ABUSE

TYPE OF SURGERY	NSAID USAGE	ALCOHOL / SMOKING
GRAHAMS OMENTAL PATCH CLOSURE	5 (16.6%)	21 (7O%)
MODIFIED GRAHAMS OMENTAL PATCH CLOSURE	4 (13.3%)	20 (66%)



41 patients out of 60 patients studied had history of smoking (68.3%)

9 out of 60 patients had history of NSAID abuse (15%).

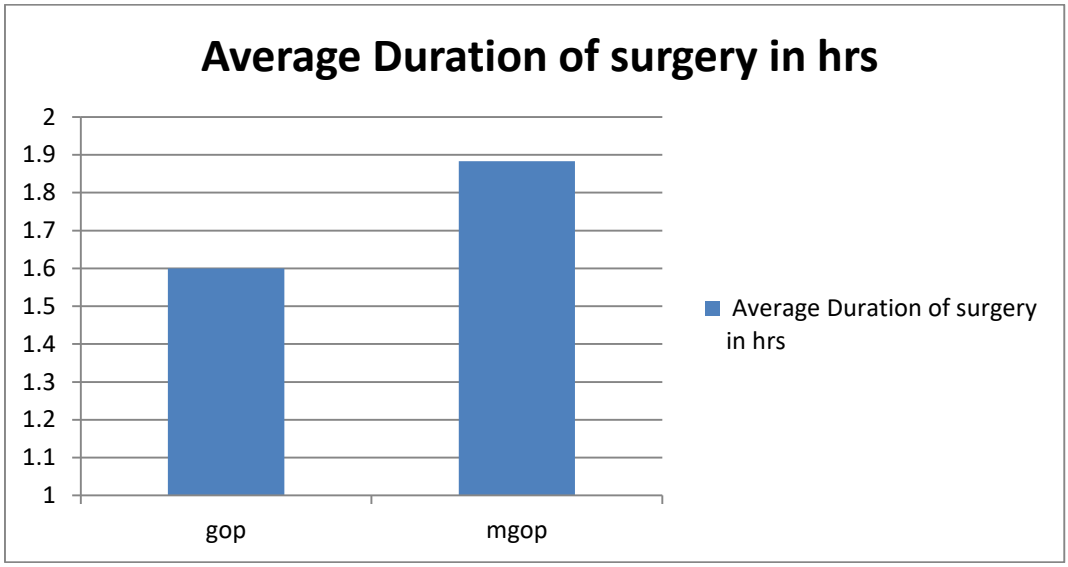


TYPE OF SURGERY	PERITONITIS INDEX
GOP	**4.17**
MGOP	**4.67**

The average peritoinitis index for group 1 was 4.17

The average peritonitis index for group 2 was 4.67

Comparing the two groups the p value was <0.05 which was statistically insignificant

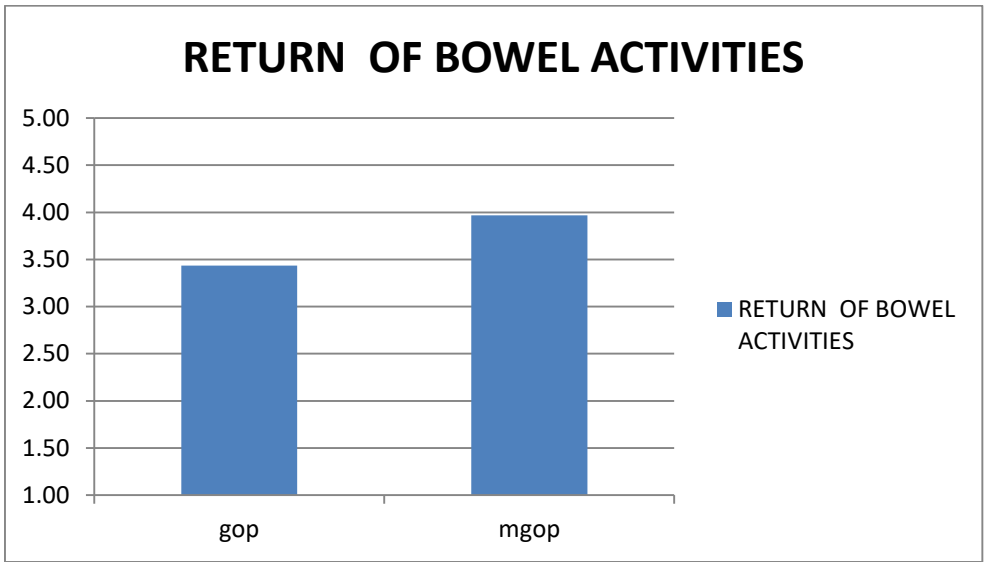


TYPE OF SURGERY	AVERAGE DURATION OF SURGERY IN HRS
GOP	1.6
MGOP	1.883333333

The mean duration of surgery for GRAHAMS OMENTAL PATCH REPAIR was 1 hr 36 minutes.

The mean duration of surgery for MODIFIED GRAHAMS OMENTAL PATCH REPAIR was 1 hr 53 minutes.

Comparing the two groups chi square statistics is the p value is 0.70645. This result is not significant at $p< 0.05$.



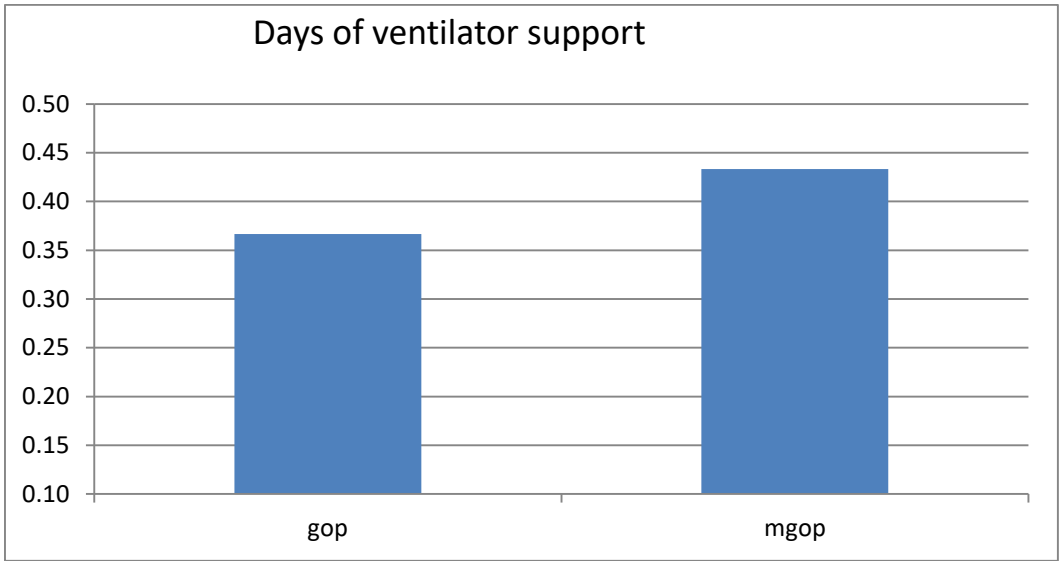
TYPE OF SURGERY	RETURN OF BOWEL ACTIVITIES IN DAYS AVERAGE
GRAHAMS OMENTAL PATCH REPAIR	3.43
MODIFIED GRAHAMS OMENTAL PATCH REPAIR	3.97

Patients in group 1 have return of bowel activity in an average of 3.43 days.

Patients in group 1 have return of bowel activitiy in an average of 3.966 days.

Comparing the two groups the two tailed p value is 0.3788. This result is not statistically significant at $p < 0.05$.

VENTILATOR SUPPORT

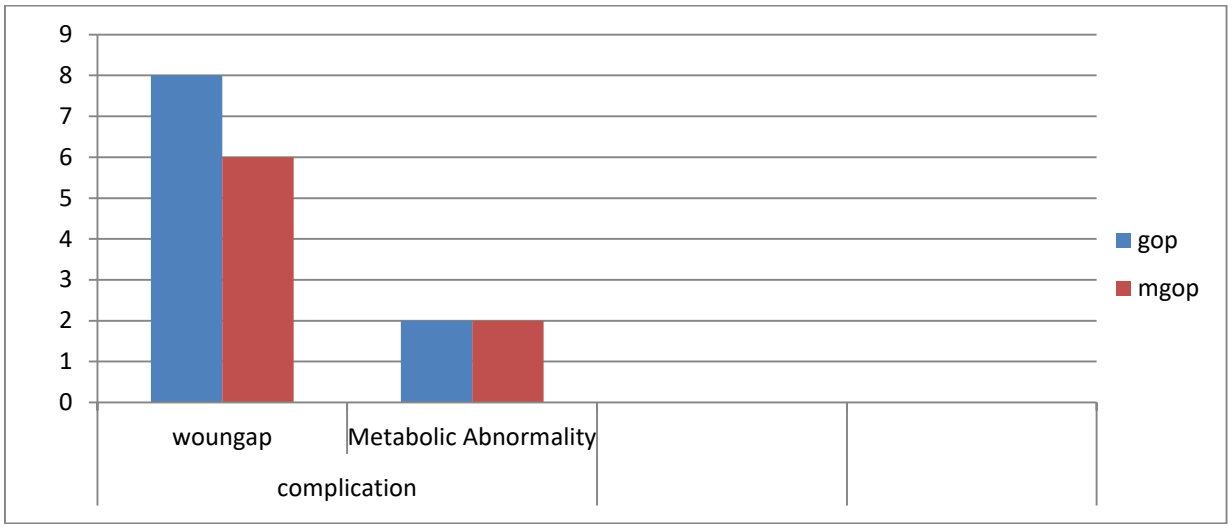


TYPE OF SURGERY	DAYS OF VENTILATOR SUPPORT
GOP	0.37
MGOP	0.43

The mean duration of ventilator support was 0.3667 days in group 1 and 0.43333 days in group 2.

Comparing the two groups the two tailed p value is 0.9409. This result is not statistically significant at $p < 0.05$.

POST OPERATIVE COMPLICATIONS



TYPE OF SURGERY	COMPLICATIONS	
	SURGICAL SITE INFECTION	METABOLIC ABNORMALITY
GRAHAMS OMENTAL PATCH CLOSURE	8	2
MODIFIED GRAHAMS OMENTAL PATCH CLOSURE	6	2

8 out of 30 patients in group 1 developed surgical site infection, while 6 out of 30 patients in group 2 developed surgical site infection.

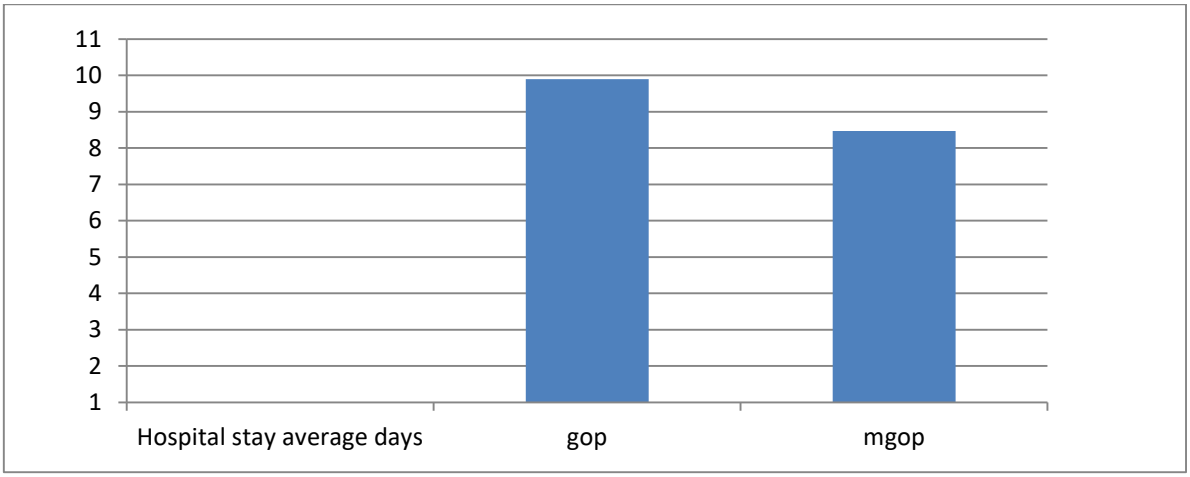
Comparing the two groups chi square statistics is 0.3727 the p value is 0.541552. This result is not statistically significant at $p< 0.05$.

2 out of 30 patients in group 1 developed metabolic abnormalities post operatively .

2 out of 30 patients in group 2 developed metabolic abnormalities post operatively

Comparing the two groups chi square statistics is 0 .the p value is 1 . This result is not statistically significant at $p< 0.05$.

AVERAGE DAYS OF HOSPITAL STAY

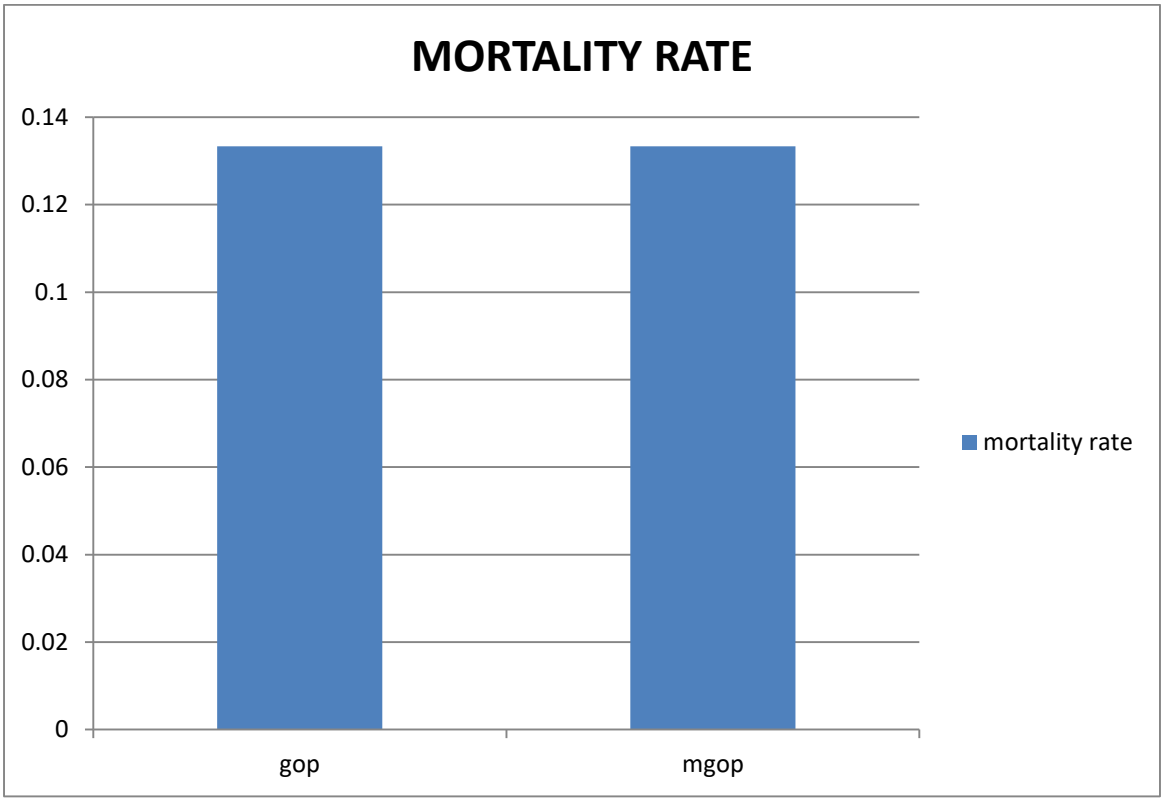


Hospital stay average days	
GOP	9.9
MGOP	8.466667

Average days of hospital stay in group 1 is 9.9 days

Average days of hospital stay in group 2 is 8.46 days

Comparing the two groups.the two tailed p value is 0.7840 . This result is not statistically significant at $p< 0.05$.



TYPE OF SURGERY	DISCHARGED	DEATH (MORTALITY RATE)
GRAHAMS OMENTAL PATCH REPAIR	26	4(0.133)
MODIFIED GRAHAMS OMENTAL PATCH REPAIR	26	4 (0.133)

4 out of 30 patients in group 1 died during hospital stay.

4 out of 30 patients in group 2 died during hospital stay.

Comparing the two groups chi square statistics is 0 .the p value is 1 . This result is not statistically significant at $p< 0.05$.

COMPARISON CHART OF GOP AND MGOP IN VARIOUS PARAMETERS

Type of surgery	sample	Age	female	Duration of symps	Smoking alcohol	NSAID ABUSE	RETURN OF BOWEL ACTIVITIES	PERITONITIS INDEX	Complication		Comorbidity		Days of ventilator support	Hospital stay	Death	Duration of surgery in hrs
	Number	Average	%	Average	Number	Number	Average	Average	Surgical site infection	Metabolic Abnormality	DIABETIC	SHT	Average	Average	Number	Average
GOPR	30	40.7 SD(15.020)	2	2.033333 SD(0.8899)	21	5	3.43333333 SD(1.2544)	4.166667 SD(3.806)	8	2	7	3	0.36667 SD(3.5382)	9.9 SD(20.5601)	4	1.6 SD(22.313)
MGOPR	30	40.83 SD(23.181)	7	1.366667 SD(0.8088)	20	4	3.96666667 SD(3.0454)	4.666667 SD(3.461)	6	2	2	4	0.43333 SD(3.3907)	8.46667 SD(19.7625)	4	1.883333 SD(21.503)

DISCUSSION

In our study we included 60 patients.

30 patients were under group 1 (GRAHAMS OMENTAL PATCH REPAIR) and 30 patients under group 2 (MODIFIED GRAHAMS OMENTAL PATCH REPAIR).

Age distribution of the patients affected ranged from 13-70 years in the present study. The maximum number of cases studied were in the age group of 31 to 40(25%) .

In group 1 mean age was 40.7 (SD – 15.020). In group 2 the mean age was 40.83 (SD-23.181). the two tailed P value equals 0.9795. this difference is considered to be statistically not significant.

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9 out of 60 patients were diabetic, 3 out of 60 patients were hypertensive.

41 patients out of 60 patients studied had history of smoking (68.3%)

9 out of 60 patients had history of NSAID abuse (15%).

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Comparing the two groups chi square statistics is 0 .the p value is 1 . This result is not statistically significant at p< 0.05.

Average days of hospital stay in group 1 is 9.9 days

Average days of hospital stay in group 2 is 8.46 days

Comparing the two groups.the two tailed p value is 0.7840 . This result is not statistically significant at p< 0.05.

4 out of 30 patients in group 1 died during hospital stay.

4 out of 30 patients in group 2 died during hospital stay.

Comparing the two groups chi square statistics is 0 .the p value is 1 . This result is not statistically significant at p< 0.05.

CONCLUSION

The outcome and complication of both surgeries for perforated duodenal ulcer , that is GRAHAMS OMENTAL PATCH REPAIR and MODIFIED GRAHAMS OMENTAL PATCH REPAIR are independent of the method of surgery done.

Thus it is the surgeons choice to select one of the two methods. The major contributing factor for outcome and complication of surgery are the age, day of presentation, peritonitis index and comorbidities of patient rather than the type of surgery done.

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PROFORMA

PROFORMA

Name :- I. P. No

Age :- Unit

Sex :- D.O.A

Occupation :- D.O.D

Address :-
Phone no :

DIAGNOSIS:

PRESENTING COMPLAINTS

1) Abdominal pain
2) Abdominal distension
3) co existing co morbidities
4) Duration of disease
5) Treatment history

GENERAL PHYSICAL EXAMINATION

1. General survey

2. Body build and nourishment

3. Appearance

4. Attitude: Restless/ Quiet

5. Dehydration: Mild/ Moderate/ Severe/ Nil

6. Anaemia/ Jaundice/ Clubbing/ Cyanosis/ Lymphadenopathy/ Pedal edema

7. Pulse

8. Temperature

9. Respiratory rate

10. Blood pressure

ABDOMEN EXAMINATION

1.Inspection
2.Palpation
1. Percussion
2. Auscultation

SYSTEMIC EXAMINATION

- Cardiovascular system
- Respiratory system
- Central nervous system
- Genito-urinary system

Procedure & Intraop findings : D.O.S:

Type of Anaesthesia :

Vital signs 48 hrs after surgery	
Pulse rate	
Systolic BP	
Diastolic BP	
Temperature	
Respiratory rate	
spO2	

Complications	
Surgical site infection	
Return of bowel function	
Duration of ventilatory support	
Duration of hospital stay	
Death	

ஆராய்ச்சிதகவல்அறிக்கை

மதுரை அரசு இராசாசி மருத்துவமனையில் வரும் நோயாளிக்குள்

ஏற்பட்டு உள்ளவர்களுக்கு ஒரு ஆராய்ச்சி இங்கு நடைபெற்றுவருகிறது. நீங்களும் இந்த ஆராய்ச்சியில் பங்கேற்க விரும்பிகிறோம் .

உங்களை சில சிறப்பு பரிசோதனைக்கு உட்படுத்தி அதன் தகவல்களை ஆராய்வோம். அதனால் தங்களது நோயின் ஆய்வரிகையோ அல்லது சிகிச்சையோ பாதிப்பு ஏற்படாது என்பதை தெரிவித்து கொள்கிறேன் .

முடிவுகளைவெளியிடும்போது அல்லது ஆராய்ச்சியின்போதோ தங்களது பெயரோ அல்லது அடையாளங்களோ வெளியிடமாட்டோம் என்பதை தெரிவித்து கொள்கிறோம்.

இந்த ஆராய்ச்சியில் பங்கேற்பது தங்களுடைய விருப்பத்தின் பேரில்தான் நடக்கும். . மேலும் நீங்கள் எந்நேரமும் இந்த ஆராய்ச்சியில் இருந்து பின்வாங்கலாம் என்பதையும் தெரிவித்துகொள்கிறோம்.

இந்த சிறப்பு பரிசோதனை முடிவுகளை ஆராய்ச்சியின்போது அல்லது ஆராய்ச்சியின் முடிவின்பொது தங்களுக்கு அறிவிப்போம் என்பதையும் தெரிவித்து கொள்கிறோம்.

ஆராய்ச்சியாளரின் கையொப்பம் **பங்கேற்பாளர் கையொப்பம்**

ABBREVIATIONS

SSIs - Surgical site infections

WSES - World Society of Emergency Surgery

GOPR-Grahams omental patch repair

MGOPR-Modified Grahams omental patch repair

CERTIFICATE – II

This is to certify that this dissertation work titled of the candidate **Dr.VEENA R UNNI** with registration Number **221611125** for the award of in the branch of M.S. degree General Surgery (Branch I) MAY 2019. I personally verified the urkund.com website for the purpose of plagiarism Check. I found that the uploaded thesis file contains from introduction to conclusion pages and result shows 7% percentage of plagiarism in the dissertation.

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age	sex	duration of symps	smoking alcohol	NSAID ABUSE		type of surgery	RETURN OF BOWEL ACTIVITIES	PERITONITISNINDEX	complication	comorbidity	days of ventillator support	hosital stay	death	duration of surgery in hrs
60	m	3	yes	yes		gop	3	5	no	DIABETIC	0	10	NO	2
67	m	2	yes	no		gop	4	5	wound gap	SHT	0	21	NO	1.5
58	m	1	yes	no		gop	3	6	no	DIABETIC	0	16	yes	2
15	m	2	no	no		gop	2	3	NO	NO	0	7	NO	1
76	m	4	yes	no		gop	3	5	wound gap	NO	0	14	NO	1.5
33	m	2	yes	no		gop	4	5	no	NO	0	12	NO	1
27	m	3	no	no		gop	5	3	NO	NO	0	7	NO	2
50	m	2	yes	no		gop	4	5	wound gap	DIABETIC	2	21	yes	1
34	m	2	yes	no		gop	4	6	no	NO	0	13	NO	1
70	m	1	no	yes		gop	5	5	no	SHT	1	7	yes	1.5
70	m	1	no	yes		gop	6	5	no	SHT	0	6	NO	1.5
48	m	1	yes	no		gop	4	6	wound gap	DIABETIC	0	7	NO	2
25	m	2	yes	no		gop	3	3	NO	NO	0	5	NO	2
26	m	1	yes	no		gop	3	3	wound gap	NO	0	13	NO	1.5
31	m	2	yes	no		gop	4	3	wound gap	NO	0	12	NO	1.5
18	m	1	no	yes		gop	2	4	no	NO	0	10	NO	1
36	m	4	yes	no		gop	2	4	METABOLIC ABNORMALITY	NO	4	8	yes	1
18	f	3	no	yes		gop	3	3	N0	NO	0	7	NO	2
60	f	2	no	no		gop	4	3	no	NO	0	7	NO	2
65	m	1	yes	no		gop	3	3	wound gap	NO	0	10	NO	1.5
22	m	1	yes	no		gop	3	4	NO	NO	0	9	NO	1.5
22	m	2	yes	no		gop	4	4	NO	NO	0	5	NO	2
27	m	2	yes	no		gop	3	4	NO	NO	0	7	NO	2
43	m	1	yes	no		gop	3	5	wound gap	DIABETIC	1	14	NO	3

20	m	3	no	no		gop	3	5	NO	DIABETIC	2	9	NO	1
63	m	2	yes	no		gop	4	6	METABOLIC ABNORMALITY	DIABETIC	0	7	NO	2
25	m	3	yes	no		gop	3	3	NO	NO	0	7	NO	1.5
25	m	2	yes	no		gop	2	3	NO	NO	0	14	NO	2
35	m	3	yes	no		gop	3	3	NO	NO	0	6	NO	1
52	m	2	yes	no		gop	4	3	no	NO	1	6	NO	1.5
22	f	1	no	yes		mgop	2	3	wound gap	NO	1	6	NO	1.5
22	m	1	no	no		mgop	3	3	NO	NO	0	6	NO	2
40	f	1	no	no		mgop	4	4	NO	NO	0	13	NO	1
47	m	2	yes	no		mgop	2	4	NO	NO	0	12	NO	1
58	m	2	yes	no		mgop	3	7	wound gap	DIABETIC	0	7	NO	2
27	m	1	no	no		mgop	4	8	no	NO	0	11	NO	1
21	m	1	no	no		mgop	2	3	NO	NO	0	5	yes	2
40	f	2	no	yes		mgop	3	4	NO	NO	2	7	NO	1.5
28	m	2	no	no		mgop	4	4	NO	NO	1	7	NO	1.5
24	m	2	yes	no		mgop	2	4	wound gap	NO	0	12	NO	1
48	f	3	no	no		mgop	34	7	no	NO	0	8	NO	2
39	f	1	no	yes		mgop	3	5	no	NO	0	8	NO	1
65	m	1	yes	no		mgop	2	5	no	NO	2	7	yes	2
37	m	2	yes	no		mgop	4	5	wound gap	NO	0	6	NO	2
24	f	2	no	no		mgop	4	6	no	NO	0	9	NO	1
70	m	1	no	yes		mgop	3	4	no	NO	0	9	NO	1.5
40	f	1	no	no		mgop	2	5	no	NO	0	8	NO	1.5
60	m	2	yes	no		mgop	2	5	wound gap	NO	0	30	NO	1.5
40	m	1	yes	no		mgop	3	6	wound gap	NO	1	6	NO	1
40	m	1	yes	no		mgop	4	6	no	NO	1	10	NO	2
29	m	1	yes	no		mgop	4	2	no	NO	1	9	NO	2
42	m	1	yes	no		mgop	3	2	no	NO	0	6	NO	13

56	m	1	yes	no		mgop	2	4	no	NO	0	8	NO	1
56	m	1	yes	no		mgop	2	4	no	DIABETIC	0	9	NO	1
40	m	1	yes	no		mgop	3	4	no	NO	0	6	NO	2
33	m	1	yes	no		mgop	4	4	NO	NO	0	7	NO	1.5
32	m	1	yes	no		mgop	3	4	NO	NO	0	6	NO	1
56	m	1	yes	no		mgop	2	6	METABOLIC ABNORMALITY	NO	2	5	NO	1
24	m	1	yes	no		mgop	4	6	METABOLIC ABNORMALITY	NO	1	5	yes	2
65	m	2	yes	no	m	mgop	2	6	NO	NO	1	6	yes	2

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ETHICS COMMITTEE CERTIFICATE

Name of the Candidate	:	Dr.Veena R Unni
Course	:	PG in MS., General Surgery
Period of Study	:	2016-2019
College	:	MADURAI MEDICAL COLLEGE
Research Topic	:	A comparative study to assess the outcome and complications of grahams patch closure versus modified graham patch closure in perforated duodenal ulcer among patients admitted in General Surgery Department
Ethical Committee as on	:	17.07.2018

The Ethics Committee, Madurai Medical College has decided to inform that your Research proposal is accepted.

Member Secretary | Chairman | Dean / Convenor

Prof Dr V Nagaraajan
M.D., MNAMS, D.M., Dsc.,(Neuro), Dsc (Hon)
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Urkund Analysis Result

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