"A CLINICO PATHOLOGICAL STUDY OF GASTRIC OUTLET OBSTRUCTION AND ITS SURGICAL OUTCOME IN TIRUNELVELI MEDICAL COLLEGE – A PROSPECTIVE STUDY"

A DISSERTATION SUBMITTED TO THE TAMILNADU

DR MGR MEDICAL UNIVERSITY

CHENNAI

In partial fulfillment of the requirement for the degree of

M.S. (GENERAL SURGERY)

BRANCH – I

Register No: 221711365

DEPARTMENT OF GENERAL SURGERY

TIRUNELVELI MEDICAL COLLEGE

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Dr. S.M. KANNAN M.S, M.Ch (Uro)

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PRINCIPAL INVESTIGATOR: Dr.PRAKASH.N, MBBS.,
DESIGNATION OF PRINCIPAL INVESTIGATOR: PG STUDENT
DEPARTMENT & INSTITUTION: TIRUNELVELI MEDICAL COLLEGE, TIRUNELVELI

Dear Dr.PRAKSH.N, MBBS, The Tirunelveli Medical College Institutional Ethics Committee (TIREC) reviewed and discussed your application during The IEC meeting held on 27.10.2017.

THE FOLLOWING DOCUMENTS WERE REVIEWED AND APPROVED

1.	TIREC Application Form	☐
2.	Study Protocol	☐
3.	Department Research Committee Approval	☐
4.	Patient Information Document and Consent Form in English and Vernacular Language	☐
5.	Investigator's Brochure	☐
6.	Proposed Methods for Patient Accrual Proposed	☐
7.	Curriculum Vitae of The Principal Investigator	☐
8.	Insurance /Compensation Policy	☐
9.	Investigator's Agreement with Sponsor	☐
10.	Investigator's Undertaking	☐
11.	DCGI/DGFT approval	☐
12.	Clinical Trial Agreement (CTA)	☐
13.	Memorandum of Understanding (MOU)/Material Transfer Agreement (MTA)	☐
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1. The approval is valid for a period of 2 year/s or duration of project whichever is later
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4. An annual status report should be submitted.
5. The TIREC will monitor The study
6. At The time of PI's retirement/leaving the institute, The study responsibility should be transferred to a person cleared by HOD
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This is to certify that this dissertation work title "**A CLINICO PATHOLOGICAL STUDY OF GASTRIC OUTLET OBSTRUCTION AND ITS SURGICAL OUTCOME IN TIRUNELVELI MEDICAL COLLEGE – A PROSPECTIVE STUDY**" of the candidate **Dr. PRAKASH N** with registration Number **221711365** for the award of M.S. Degree in the branch of **GENERAL SURGERY (I).** 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 page and result shows **1 percentage** of plagiarism in the dissertation.

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https://www.intechopen.com/books/gastric-carcinoma-new-insights-into-current-management/gastric-carcinoma-morphologic-classifications-and-molecular-changes
https://www.ijss-sn.com/uploads/2/0/1/5/20153321/ijss_sep_oa20_-_2016.pdf
https://www.slideshare.net/rajeshwarkamineni/gastric-outlet-obstruction-41939356
https://www.hopkinsmedicine.org/gastroenterology_hepatology/_pdfs/esophagus_stomach/gastric_cancer.pdf

Instances where selected sources appear:

LIST OF ABBERVATION

AIHPS	-	ADULT IDIOPATHIC HYPERTROPHIC PYLORIC STENOSIS
ALP	-	ALKALINE PHOSPHATESE
CDH GENE	-	CADHERIN-1
COX2	-	CYCLOOXYGENASE 2
EMR	-	ENDOSCOPIC MUCOSAL RESECTION
ERCP	-	ENDOSCOPIC RETROGRADE CHOLANGIOPANCREATOGRAPHY
ESD	-	ENDOSCOPIC SUBMUCOSAL DISSECTION
EUS	-	ENDOSCOPIC ULTRASOUND
FAP	-	*FAMILIAL ADENOMATOUS POLYPOSIS*
GI LYMPHOMA	-	GASTROINTESTINAL LYMPHOMA
GIST	-	GASTRO INTESTINAL STROMAL TUMOR
GOO	-	GASTRIC OUTLET OBSTRUCTION
H.PYLORI	-	HELICPBACTER PYLORI
HIPEC	-	HYPERTHERMIC INTRAPERITONEAL CHEMOTHERAPY
HPS	-	HYPERTROPHIC PYLORIC STENOSIS
JCGC	-	JAPANESE CLASSIFICATION OF GASTRIC CARCINOMA GRADING SYSTEM
LNS	-	LYMPH NODES
MALT	-	MUCOSA-ASSOCIATED LYMPHOID TISSUE
NBI	-	NARROW BAND IMAGING

NIIC	-	NORMOTHERMIC INTRA PERITONEAL CHEMOTHERAPY
NSAIDS	-	NON-STEROIDAL ANTI INFLAMMAORTY DRUGS
OG JUNCTION	-	OESOPHAGO- GASTRIC JUNCTION
POD	-	POSTOPERATIVE DAY
PUD	-	PEPTIC ULCER DISEASE
SMA	-	SUPERIOR MENSENTERIC ARTERY
TB	-	TUBERCULOSIS
TNM	-	TUMOR NODAL METASTASIS STAGING
TPN	-	TOTAL PARENTAL NUTRITION
UICC	-	UNION INTERNATIONALE CONTRELE CANCER
VEGFR	-	VASCULAR ENDOTHELIAL GROWTH FACTOR RECEPTOR
VGP	-	VISIBLE GASTRIC PERISTALSIS

CONTENTS

INTRODUCTION

Gastric outlet obstruction defined as intramural or extramural pathology causing complete or partial obstruction of distal stomach, pylorus, and duodenum. Modern day's gastric outlet obstruction is caused malignancy either by gastric carcinoma or peripancreatic malignancies. Gastric outlet obstruction due to benign etiology is in decreasing trend.[1, 2]

This may occur as an obstructing tumour in intraluminal causing narrowing or external compression causing luminal narrowing or chronic inflammation produces scarring and fibrosis or a combination of both.

Cause of Gastric outlet obstruction in adults is malignancy until otherwise proven. Malignant GOO cause includes gastric carcinoma, pancreatic malignancy, duodenal malignancies, and etc.

Helicobacter pylori infection association with duodenal ulcer is first discovered by marshall and warren in Australia in 1984. After discovering treatment philosophy has shifted to eradicate the H.pylori infection. H2 and proton pump inhibitor are introduced to treatment of peptic ulcer. Using the H2 and proton pump inhibitors decreases incidence of the peptic ulcer but it not decreased the complicated peptic ulcer like bleeding and perforation. But incidence of gastric carcinoma increased due to increased rate of early diagnosis by newer techniques.[3]

This study has been done to find the changes in presentation of gastric outlet obstruction in view of newer discoveries in the treatment because of new chemotherapy regimens and newer modalities like narrow band endoscopy, magnification endoscopy and etc. diagnostic criteria for gastric outlet obstruction is different in each centers, even though any one of following can be used to diagnose the gastric outlet obstruction.[4]

- Non bilious, projectile, undigested food particle in the vomiting
- Palpable mass or hypertrophy of stomach
- Visible gastric peristalsis
- Saline load test; residual volume more than 400ml
- OGD scope findings suggestive of GOO
- Intra operative findings suggestive of gastric outlet obstruction

Patients with symptoms and signs of gastric outlet obstruction are admitted and preoperatively electrolyte imbalance correction, correction of hydration status, nutritional improvement, and anaemia correction done to improve the postoperative results.

AIM AND OBJECTIVE OF THE STUDY

- To estimate the incidence of etiology causing gastric outlet obstruction and estimate the outcome of surgical procedures performed for Gastric outlet obstruction patients.

REVIEW OF LITERATURE

EMBRYOLOGY OF STOMACH AND DUODENUM[5]

Stomach is arising from foregut. Below the tracheal diverticulum foregut forms the esophagus and stomach formed dilation in foregut at 5th week of gestation. At 5th week of life the stomach is situated in c3 to c5 level. Stomach is descends to between T11 to L4 at 7th week of gestation and it due to elongated growth of esophagus.[5]

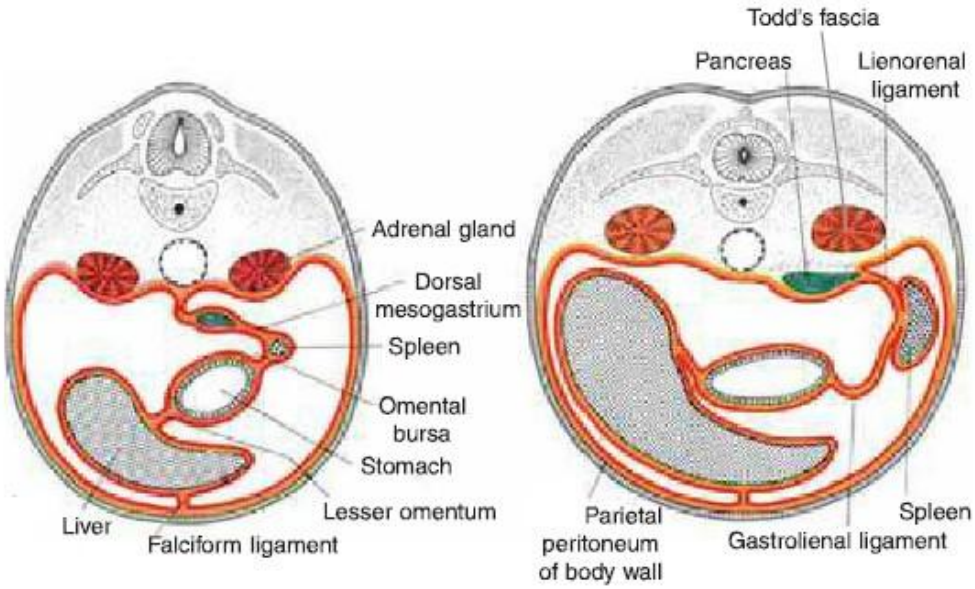

Fig-1: early developmental relation of viscera

Stomach and duodenum developed in between two mesentery, which are ventral mesentery and dorsal mesentery. Ventral mesentery is connecting the viscera to anterior abdominal wall and dorsal mesenteries connect the viscera to posterior abdominal wall. Viscera are arranged in order of liver, stomach, spleen, and pancreas. Liver connected anterior abdominal wall by falciform

ligament. Liver and stomach are attached through the lesser omentum. Stomach and spleen connected through the gastrolienal ligament. Spleen and pancreas connected via lienorenal ligament. Pancreas attached to posterior abdominal wall through the ligament and during development this ligament blend with posterior abdominal wall(todd's fascia) and pancreas positioned in retroperitoneum.

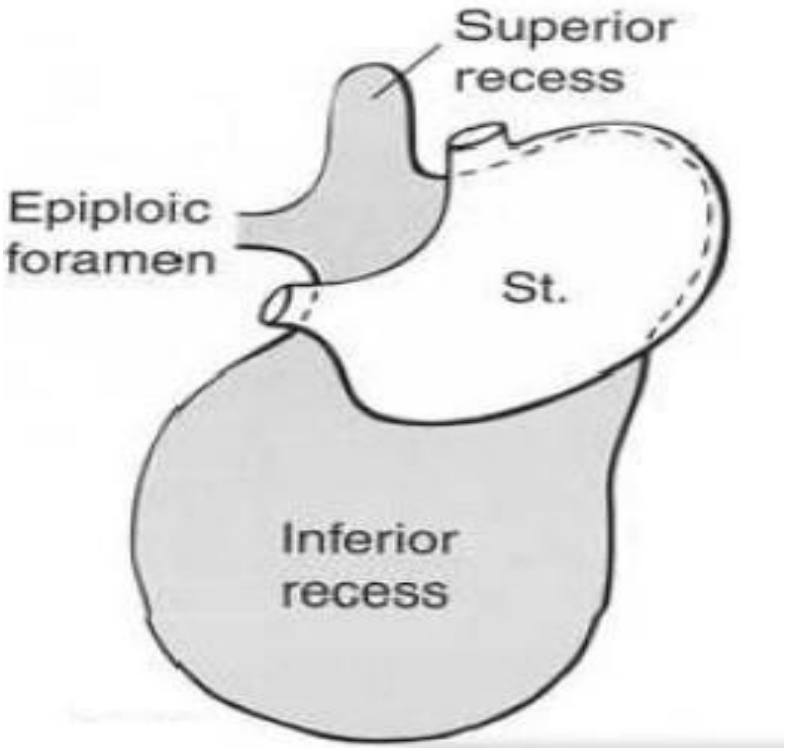

Fig-2: development of lesser sac

Behind the stomach,omental bursa forms by coalescence of smaller space and it forms right pneumatoenetric recess. Below this recess liver develops and recess associated with liver called hepatoenteric recess .lesser sac developed behind stomach, which is bounded by superiorly, superior recess behind the

liver and between below by two leaves of greater omentum. Throughout the development of lessar sac, it communication with rest of abdomen through the foramen of winslow`s .

During development of stomach, lesser curvature and greater curvature formed by differentital growth of left side wall of stomach. On developmental process of liver, it positioned into right posterior aspect of abdominal cavity. Pancreas positioned into retroperitoneum due to ligament attaching pancreas and posterior abdominal wall disappears and it forms avascular plane known as todd`s fascia. splenorenal ligament developed form ligament connecting spleen and pancreas.

EMBRYOLOGY OF OMENTUM

Increased growth of left side of stomach, also causes the downward elongation of the dorsal mesogastrium (gastrosplenic ligament) and forms omentum. Posterior Two layers of omentum fuse with the mesentry and transverse colon

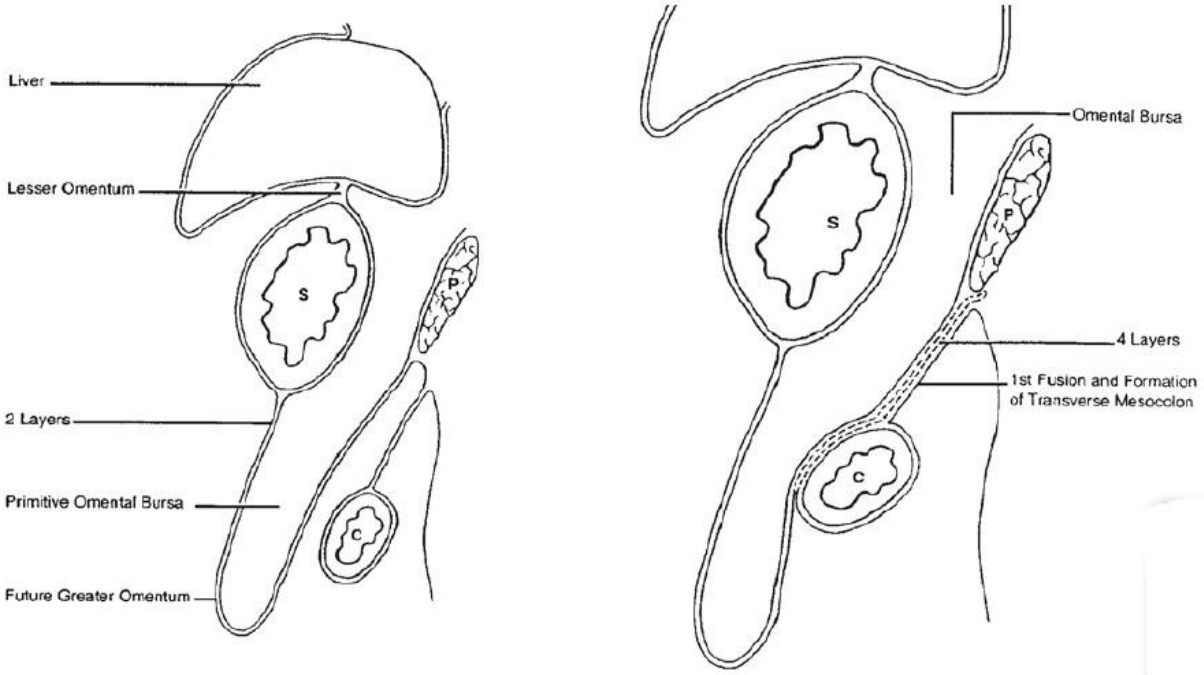

Fig-3: development of greater omentum

EMBRYOLOGY OF DUODENUM[5]

The duodenum corresponds to embryonic foregut and midgut, distal to stomach. At 5th week of gestation, the mid gut will undergo physiological herniation, through the umbilicus. 90 degree rotation occurs in the herniated intestine along axis of superior mesenteric artery and it brings colon above to small intestine. On 10th week of life the intestine returns into the abdominal cavity. Duodenum returns first and it occupying the position under the superior mensenteric artery. During returning of the intestine make another 180 degree rotation, brought the colon and distal ileum in front of SMA.

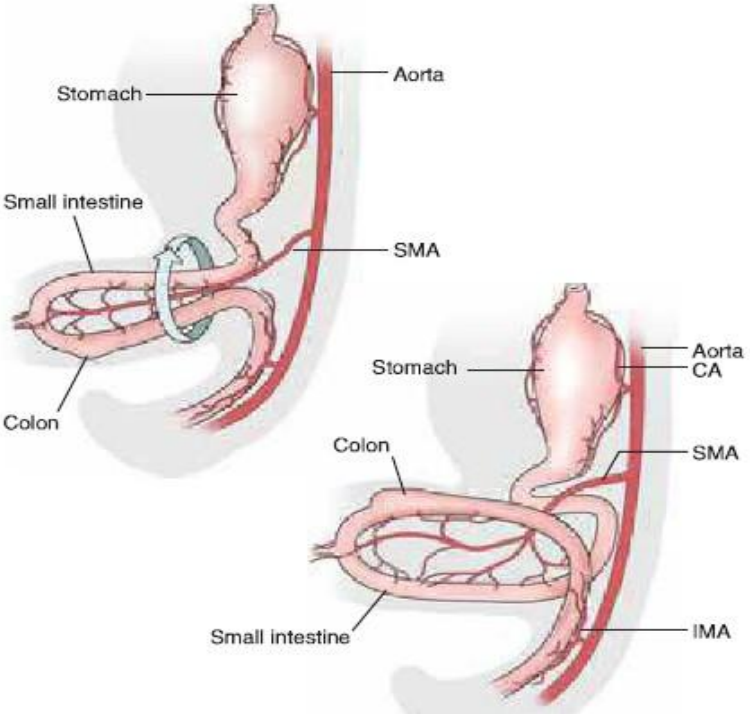

Fig-4:duodenal development and rotation of mid gut

The duodenum has mesentery in early developmental period. The ventral pancreas develops in dorsal portion of the mesodudenum. The Ventral mesentry disappears. Left turn of stomach occurs and it affects the lower part of duodenum, it causes joining of the ventral pancreas and dorsal pancreas. The right leaf of mesentry was positioned posteriorly, where this mensentry meet the parietal peritoneum of abdominal wall, it forms avascular plane. This avascular plane used in kocher`s maneuver during mobilization of head of pancreas along with duodenum.

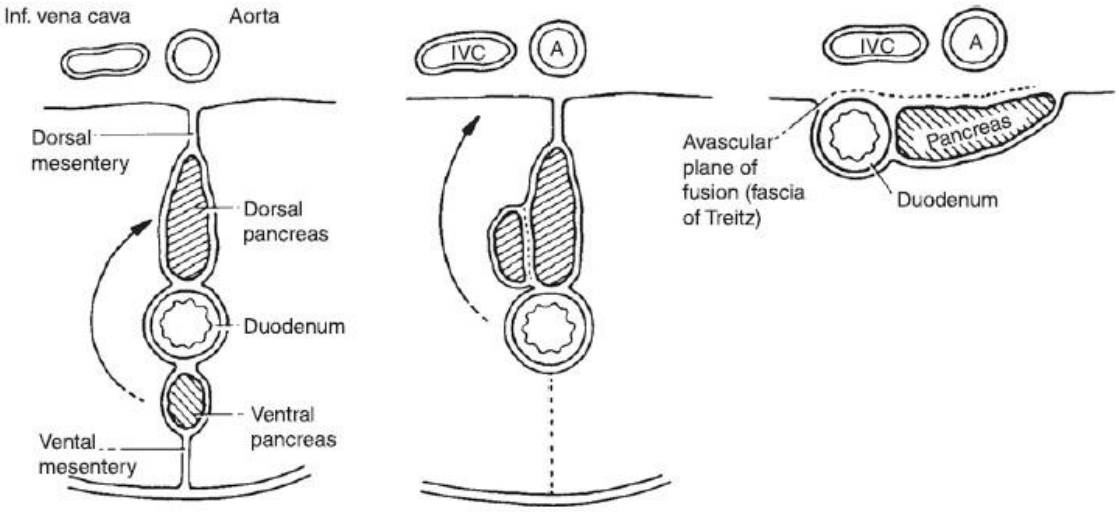

Fig-5: Development of pancreas

SURGICAL ANATOMY OF STOMACH

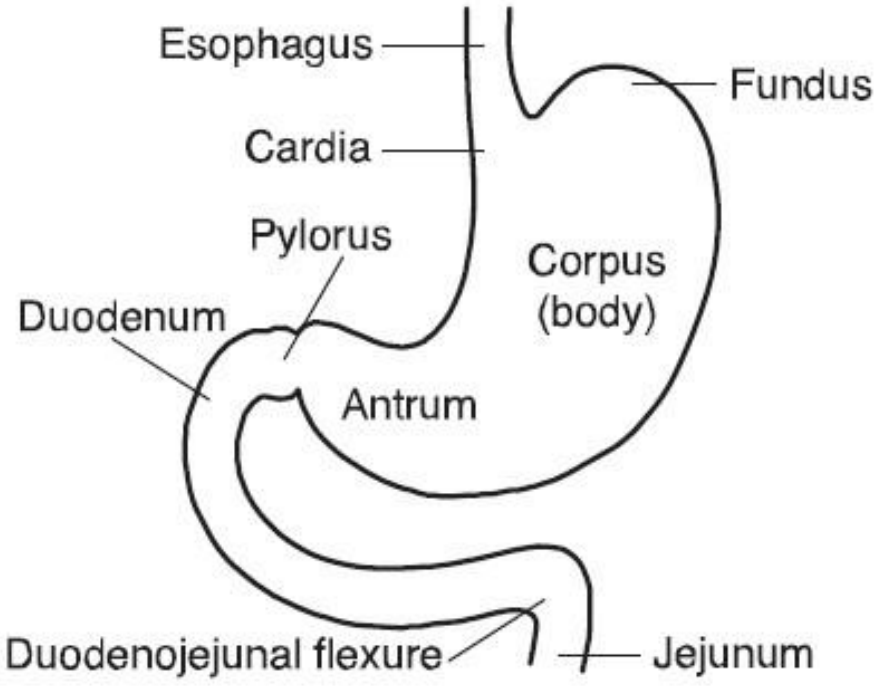

Fig-6: gross anatomy of stomach

Stomach anatomically divided into the cardia, fundus, body, pyloric antrum and pyloric canal. No well-defined border between the cardia and the fundus of stomach. Fundus is the part of stomach above the level of esophago gastric junction. The body of stomach located between the fundus and pyloric antrum and it has no external landmark. Pyloric antral region of stomach has proximally separated from the body of stomach by angular notch and distal border by constriction of pylorus near the duodenum. Angular notch found at junction between two-fifth of lesser curvature from pylorus and one eighth of greater curvature from pylorus. Pylorus and duodenum divided by constriction in the pylorus and pyloric vein of mayo consider as external landmark for junction between pylorus and duodenum.

The gastro-esophageal junction is present where the esophagus joins the proximal stomach. Histologically this area called as junctional epithelium, because it between true esophageal epithelium and true gastric epithelium.

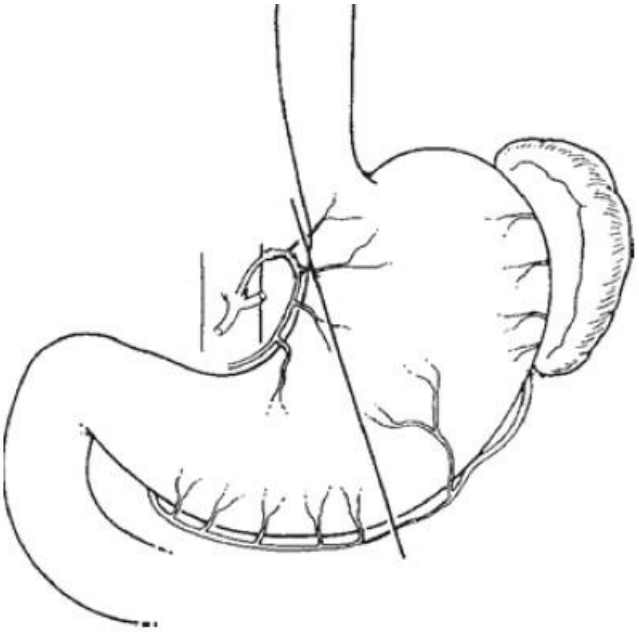

Fig -7: line divinding stomach into proximal and distal gastric unit

Surgically stomach divided into the proximal gastric unit and distal gastric unit and both gastric unit have its own special characters like anatomy, physiology, pathology. The Proximal gastric units include the esophagogastric junction, fundus, cardia, part of body. The distal gastric unit includes the part of body, pyloric antral region, first part of duodenum. Both gastric units separated by line from the lesser curvature to the greater curvature. A line draw from third vein from OG junction on lesser curvature to the greater curvature at the point where left gastro omental vessels come close to the gastric wall.

Gastroduodenal junction is a part of distal gastric unit which includes pylorus and first part of duodenum. Frist of duodenum is measuring as approximately 5cm in length. Proximal part of first of duodenum is mobile and

distal part is fixed. Histologically three different types of normal mucosa present in gastroduodenal junction, which are antral, transitional, jejunal.

BLOOD SUPPLY OF STOMACH

The stomach has rich arterial supply because of extensively interconnected that provide advance of ligating 3 out of 4 arteries without producing necrosis. Entire stomach receives blood supply from the celiac axis. Blood supply to stomach reach through the two mesenteric borders by the left gastric artery from celiac axis, the right gastric artery arise from the common hepatic artery. The left and right gastric artery enters into lesser omentum and runs adjacent to lesser curvature. Right epiploic arteries from the common hepatic artery, left epiploic arteries and short gastric arteries arise from the splenic artery are run within greater omentum near the greater curvature. These arteries have multiple anterior and posterior branches that pierce the muscular layer stomach and reach the submucosal layer. In submucosal layer, these branches forms extensive network of submucosal plexus which contains both arteries and their venous counterparts. Submucosal plexus present in all parts of stomach except in lesser curvature.[6]

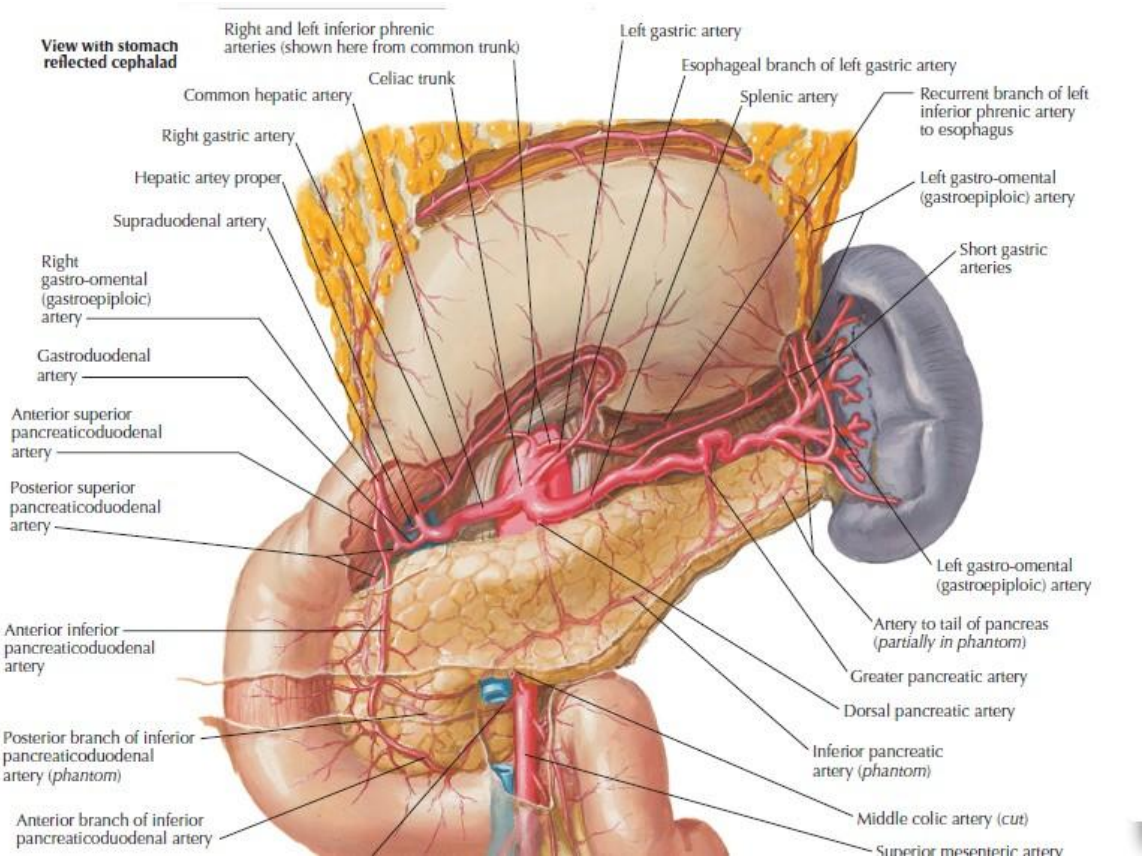

Fig-8: arterial supply of stomach

NERVE SUPPLY OF STOMACH

The stomach receives sympathetic supply from the celiac plexus. Pre-ganglionic efferent fibres originate from 5^{th} to 10^{th} thoracic segments these sympathetic fibres unite to join the greater splanchnic nerves and its cross the sympathetic ganglion without forming the synapses, then it reach the celiac ganglion, it synapses with postganglionic fibres. Postganglionic fibres travel along arteries supplying the stomach and duodenum.

The parasympathetic supply is formed by vagus nerve. Parasympathetic supply is secretomotor nerve to the stomach. Anterior vagus nerve gives three

branches to stomach are hepatic branch, the pyloric nerve of McCrea, nerve of latarjet.

The pyloric nerve runs along lesser curvature within the lesser omentum to distal antrum. Nerve of latajet is runs within lesser omentum to reach the pylorus. It terminal portion of nerve ends like crow foot.

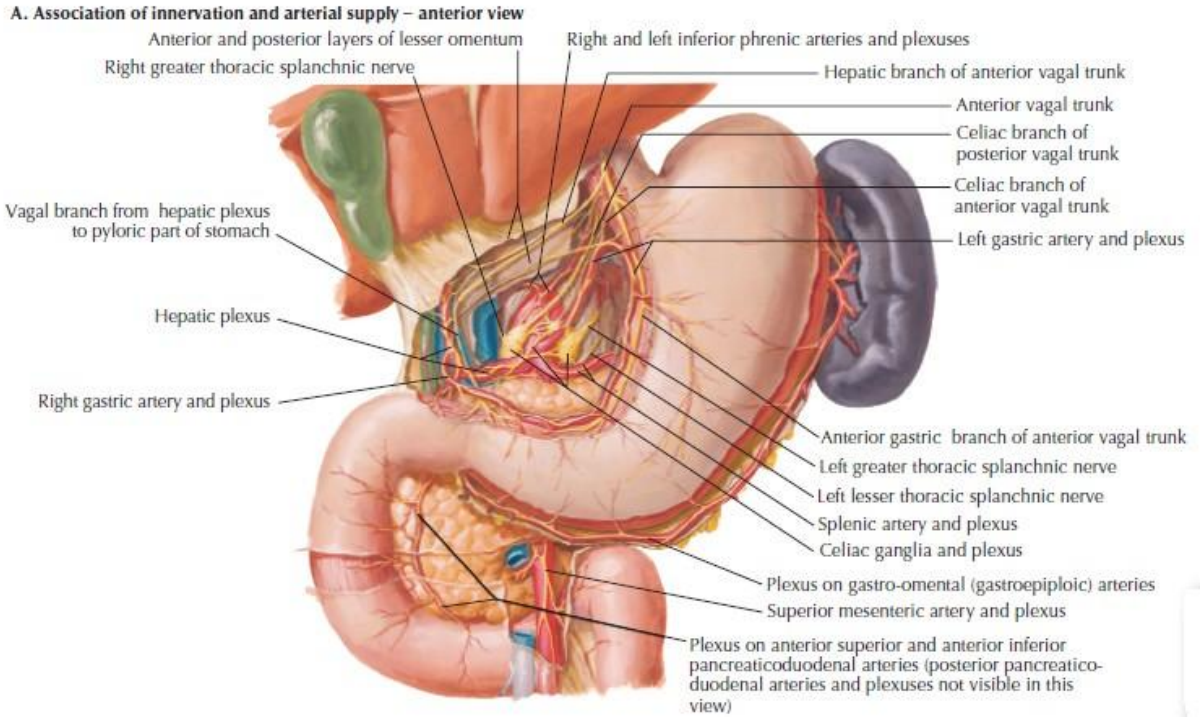

Fig-9: anterior vagus nerve supply to stomach and duodenum

The posterior vagus gives rise to two branches. Branches are posterior gastric branch and celiac branch.[6]

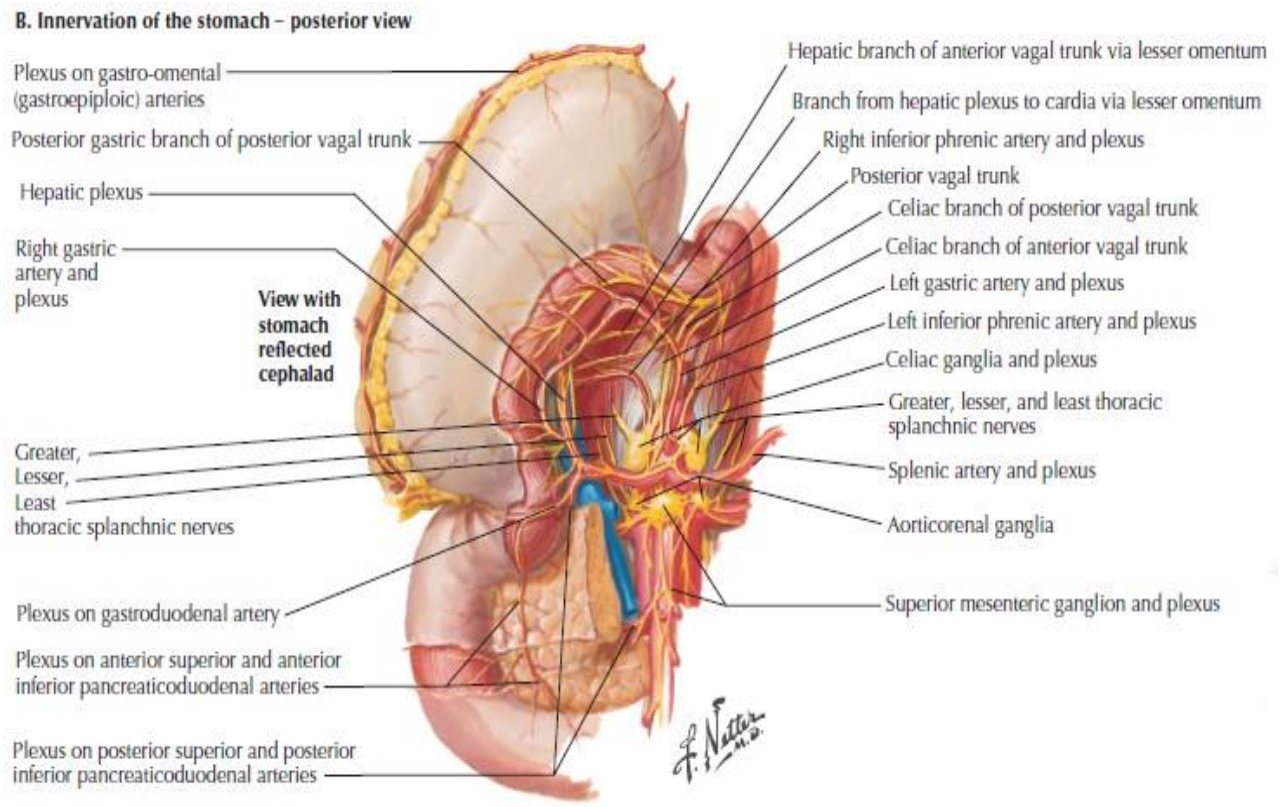

Fig -10: posterior vagus relation to stomach and duodenum

LYMPHATIC DRAINAGE

The lymph arises from mucosa and its drains into submucosal lymphatic plexus and submucosal plexus are connected to oesophageal submucosal lymphatic plexus proximally, but duodenum devoid of submucosal plexus. Submucosal plexus then drains into the subserosal plexus just below the visceral peritoneum. From here subserosal lymphatic vessels drains into extrinsic channels. Extrinsic channels are divided into four groups.

a) The subpyloric node drain the area supplied by right gastro epiploic arteries, then from subpyloric node to hepatic node along the hepatic artery and it drains into celiac nodes present in celiac axis

b) The left gastro epiploic node drains the area supplied by short gastric and left gastro epiploic artery. Its drains into pancreaticosplenic node from here along splenic artery it reaches the celiac node.

c) The left gastric node present in the lesser omentum its drains the area supplied by left gastric artery and then its drain into celiac node.

d) The right gastric node drains the area supplied by right gastric artery. Its drains into subpyloric nodes. subpyloric node drains into hepatic node it present along the hepatic artery and finally drains into celiac node.[6]

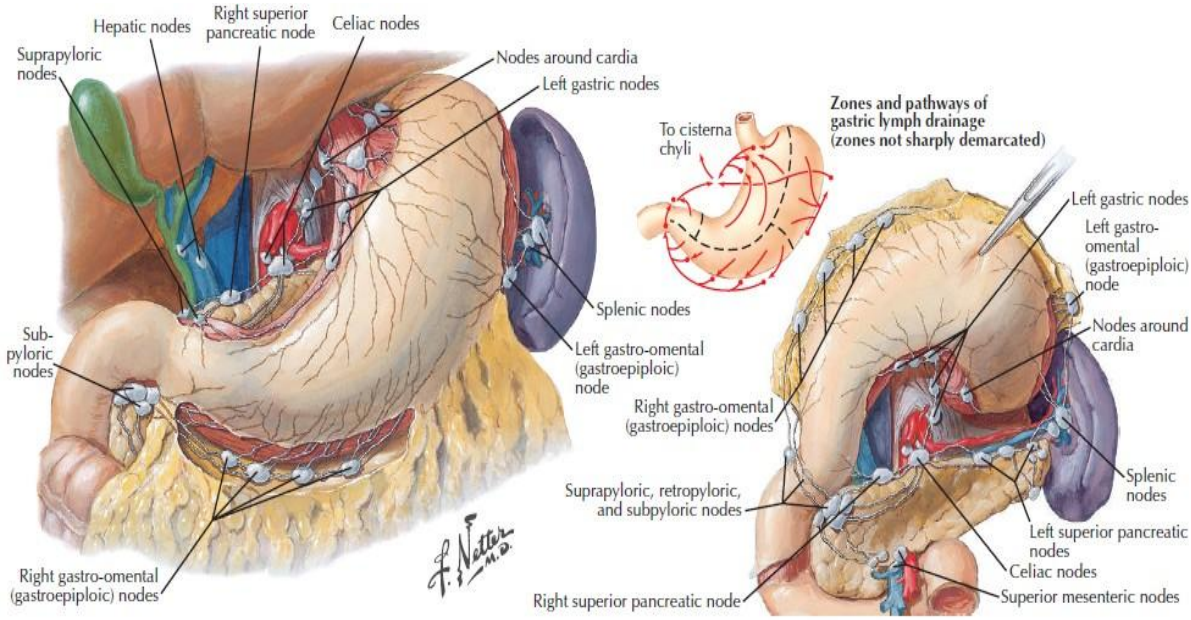

Fig-11: lymphatic drainage of stomach.

Gastric outlet obstruction is caused by conditions in and around the peripancreatic region. GOO is causative etiology divided into benign or malignant causes. Recent studies show that malignant outlet obstruction is the most common etiology cause. Benign Gastric outlet obstruction is in decreasing trend for last few decades, especially peptic ulcer related diseases.

AETIOLOGY OF BENIGN GASTRIC OUTLET OBSTRUCTION

- Peptic ulcer disease
- NSAID-associated stricture
- Caustic ingestion
- Postsurgical stricture or scarring
- Acute pancreatitis
- Pancreatic pseudocyst/Chronic pancreatitis
- Annular pancreas
- Radiation-induced stricture
- Bezoar or foreign body
- Crohns disease
- Eosinophilic gastroenteritis
- Tuberculosis
- Adult hypertrophic pyloric stenosis

- Amyloidosis
- Bouveret syndrome
- Gastric volvulus

AETIOLOGY OF MALIGNANT GASTRIC OUTLET OBSTRUCTION

- Gastric carcinoma
- Head of Pancreatic cancer
- Periampuallary carcinoma of pancreas
- Duodenal cancer
- Carcinoma gallbladder
- Cholangiocarcinoma
- Hepatocellular carcinoma
- Gastric lymphoma
- Retroperitoneal sarcoma
- Retroperitoneal lymphadenopathy
- Metastatic disease like colon,pancreas,lung

BENIGN GASTRIC OUTLET OBSTRUCTION

A.PEPTIC ULCER DISEASE

Peptic ulcer disease is still common diagnosis in outpatient department. Peptic ulcer disease defined as local breach or defect in duodenal or stomach mucosa it may extend beyond submucosa. PUD may present as acute or chronic

and it’s due to imbalance in protective mucosal defense and acid. Causes of peptic ulcer are H.pylori infection, NSAID induced, zollinger Ellison syndrome, antral G cell hyper functioning, trauma, physiological stress and smoking, psychological stress.

5 % of the patient with peptic ulcer disease may develop gastric outlet obstruction, either due to acute or chronic. Acute inflammation in duodenum or pyloric orifice produces the mechanical obstruction, with functional gastric outlet obstruction. Histologically acute ulcer confine to mucosa and submucosa. Chronic inflammation of duodenum and pylorus lead to repeated healing and ulcer followed by repair and scarring eventually leading to stenosis of lumen.

Histologically it shows destruction of epithelium, proliferation of margin, destruction of part of mucularis propria, Infiltration with inflammatory cells like mononuclear cell infiltration, endarteritis and fibrosis in the base.

In acute obstruction PUD manifests as delayed gastric emptying, anorexia, nausea, vomiting. Prolonged vomiting may leads to dehydration, electrolyte imbalance develop a hypochloremic hypokalemic metabolic alkalosis which is due to loss of gastric juice it contains hydrogen and chloride.

In chronic obstructive PUD manifests as painless large volume of non-billious vomiting, marked weight loss, dehydration and large dilated stomach. Electrolyte imbalance is similar to the acute obstructing PUD.

HELICOBACTER PYLORI INFECTION

H.Pylori infection has association with duodenal ulcer (80-90%) and gastric ulcer (75%). H.pylori infection is the most common causative organism for peptic ulcer disease. H.pylori has 4 to 6 flagella with spiral or helical shaped gram negative rod. H.pyIori uses the urease to create the alkaline environment in acidic environment of stomach by splitting urea into ammonia and bicarbonate. H.pylori organism can live only in gastric epithelium.

Physiology changes due to H.pylori infection in duodenum and stomach

- Toxic mediators like ammonia, mucinase, phospholipase, platelet activating factor are causes local tissue injury and promote inflammation.
- Activation of local mucosal inflammatory response by H.pylori
- H.pylori infection is significantly increases the basal and stimulated gastrin level. Gastrin increases the acid secretion with concomitant decrease in somatostatin level.
- Increased acid secretion causes the gastric metaplasia of duodenal epithelium as protective mechanism. Gastric metaplasia facilitates H.pylori colonization in duodenum.[7]

B.NSAIDS INDUCED STRICTURE

NSAIDs ingestion is rare cause of GOO. Mechanism of GOO caused by NSAID is unknown. Decreased levels of prostaglandin E2 may be related to pathogenesis of gastric outflow obstruction by causing pyloric edema and scarring. Decreased levels of prostaglandin also Increases histamine release leads to increased gastric secretion, reduction of mucosal absorption, and gastric motility disturbances. NSAIDS can cause stricture anywhere in alimentary tract. Small bowel strictures caused by NSAIDs are short (2-3 mm) web-like and common in jejunum and ileum.[8]

C. CAUSTIC INGESTION INDUCED STRICTURE

Caustic ingestion associated stricture are common in young age group, and it account for 80% of cases. Accidental ingestion of caustic agents is usually small in quantity. In adult, Intentional ingestion of large quantity of corrosive poisoning is lethal to life. When ingestion of acidic agent cause coagulative necrosis and forms the eschar, it limits the further damage. Acids commonly affect stomach more than oesophagus. Alkaline commonly produce injury to oesophagus and it doesn't form the eschar, so it continue the damage still diluted or neutralised. Usually GOO symptoms occur after 1 to 6 week of ingestion of corrosive agent due to stricture formation.[9]

D.POSTOPERATIVE ADHESION

Postoperative adhesion causing gastric outlet obstruction is rare entity. Adhesion usually found between the liver and gastroduodenal junction, and it causes the acute angulation of pyloric and duodenal region. Open cholecystectomy, hepatobiliary surgeries are cause for postoperative adhesion.[10]

E.GASTRIC OUTLET OBSTRUCTION DUE TO PRIMARY GASTRIC TUBERCULOSIS

About 1-3% account for abdominal tuberculosis and about 12% of extrapulmonary tuberculosis. Ileocecal region is common site for abdominal tuberculosis, but involvement of gastroduodenal region is very rare. Gastroduodenal TB usually present as gastric outlet obstruction. Acidic environment and rapid transit of food are produce the stomach and duodenum from gastroduodenal TB. Lesion commonly located in lesser curvature and ulcer is multiple and shallow or present as submucosal mass. Celiac lymph node may be the source of infection for gastroduodenal TB.[12]

F.PSEUDOCYST OF PANCREAS

Pseudocyst is most common cystic lesion of pancreas and it account for two thirds of all cystic lesion of pancreas. 90% of pseudocyst is single in number. Obstruction of stomach and biliary obstruction are relatively common due to compression. Large cyst in lesser sac can produce mechanical obstruction

to duodenum and pyloric antrum. Pseudocyst is usually resolves spontaneously. Severity of Symptoms may vary according to site and size of pseudocyst.[7, 10]

G.RADIATION INDUCED STRICTURE

Benign gastric outlet obstruction is usually obstructs the pylorus or duodenum due to process that induces inflammatory or fibrotic changes. Stomach has highly resistant to radiation. Radiation induced stricture usually presents as irregular contractions of the antrum. In duodenum it causes postbulbar mucosal thickening, ulceration and stricture. Radiation induced ulcer involves the mucosa with muscularis propria. Ulcer usually multiple in number, sharply demarcated, vary size from 1 to4 mm in diameter with surrounding induration and stomach wall thickening.[13, 14.]

CROHNS DISEASE

Isolated gastroduodenal crohns disease is very rare it only account for 0.07%. crohns disease is most commonly involves ileocolonic and colonic region and it account for 40%. Around 30% involves small intestine.[15]

ADULT IDIOPATHIC HYPERTROPHIC PYLORIC STENOSIS

AIHPS is rare aetiology causing gastric outlet obstruction. Aetiology of AIHPS is unknown, probably genetic and environmental factors involved. AIHPS may be persistence of juvenile form of HPS and both conditions have similar histological and anatomical changes. Late presentation of juvenile HPS

may be triggered by pyloric inflammation, spasm or edema. Histologically AIHPS have marked hypertrophy and hyperplasia of muscularis propria of pylorus without inflammatory cells.[16]

BEZOAR AND FOREIGN BODY

Bezoar disease classified into phytobezoars, trichobezoars, diospyrobezoars, pharmacobezoars and etc. bezoar disease is most commonly seen in young females, behavioural abnormalities and mentally retarded persons. Trichobezoars is most common type of bezoars and it is patients who chew and swallow their own hair. Phytobezoars (vegatable like fibres, seeds) usually occur in patients with delayed gastric emptying due to gastric bypass or partial gastrectomy with vagotomy, diabetic mellitus, and mixed connective tissue disease.[17]

BOUVERETS SYNDROME

Bouverets syndrome is gastric outlet obstruction caused by impacted gallstone in duodenum or in stomach. The gallstone reached stomach or duodenum through the biliary enteric fistula. It’s more prevalent in elderly and female. The formation of biliary enteric fistula is started when the walls of biliary system and the bowel are chronically inflamed and both walls are adherent. Increasing intrabiliary pressure caused by gallstone, it leads to wall ischemia and necrosis followed by perforation of intestine, and it allows the

gallstone to enter the duodenum or stomach. Usually less than 2.5 cm gallstone passes through the small intestine; large stone may cause gallstone ileus.[18-21]

WILKIES SYNDROME

It is also called duodenal ileus or superior mesenteric artery (SMA) syndrome. Wilkies syndrome has obstruction at the level of 3rd part of duodenum due to decreased angle between SMA and aorta. It may aggravated by plaster casts, lordosis, pancreatic tumour, enlarged lymph node in the 3rd part of duodenum.[7,22]

GASTRIC VOLVULUS

Gastric volvulus occurs between two fixed points of stomach (cardia and pylorus). Etiology of gastric volvulus is idiopathic or secondary to hiatus hernia, eventration, adhesions, and pyloric obstruction with dilated stomach. Stomach twist upwards between oesophagogastric and pyloroduodenal junction. It is associated with hiatal hernia or diaphragmatic eventration.[23]

BENIGN TUMOURS OF THE STOMACH

POLYPS

Benign gastric polyps are divided as neoplastic and nonneoplastic polyps.

Neoplastic polyps are fundic gland polyps, adenoma

Non neoplastic polyps are inflammatory, hyperplastic polyps, hamartomatous polyps

Inflammatory and hamartomatous polyps are not premalignant conditions. Hyperplastic polyps occur due to chronic inflammation. Hyperplastic polyps more than 2cm may shows dysplastic changes or carcinoma insitu. Gastric adenoma are premalignant condition, especially patient's with familial adenomatous polyposis and they have 10 times more risk of develop gastric carcinoma than the general population.[24]

MESENCHYMAL NEOPLASMS

These are fibromas, schwannomas, neurofibromas, lipomas , leiomyoma and etc. leiomyoma is commonest mesenchymal neoplasm. Leiomyoma are usually asymptomatic. Larger than 2 cm lesions may present as obstruction or bleeding. Histologically these lesions show the smooth muscle origin. Lipomas are submucosal fatty lesion and it is asymptomatic.

Gastric lymphomas

Gastric lymphomas are commonest site for extranodal non Hodgkin lymphoma and its account for 95% of GI Lymphomas and account for 4% of gastric malignancies. 50% of gastric lymphomas are high grade and another 50% are low grade. Most common cell type is B cell and it arises from the MALT. Chronic gastritis and H.pylori are the causative factors. In primary lymphomas are nodular, enlarged gastric folds. [7]

MALIGNANT GASTRIC OUTLET OBSTRUCTION

A.CARCINOMA STOMACH

Adenocarcinoma of stomach is the commonest primary gastric neoplasm and its accounts for 95%. Carcinoma stomach is the fourth most common type and second leading cause for cancer related death. Adenocarcinoma of stomach is more prevalent in Asia, Europe. For carcinoma stomach 5 year survival rate is 27%. The aetiological factors are divided into acquired, genetic, precursor lesions.

AETIOLOGICAL FACTORS

1. Acquired factors
    a. Nutritional factors
        i. Diet high in pickled, salted, smoked food
        ii. Dietary nitrates
        iii. Lack of refrigeration
        iv. Lack of dietary vitamins A and C
    b. Occupational factors
        i. Coal mine workers
        ii. Rubber factory workers
    c. Smoking
    d. Helicobacter pylori infection
    e. Epstein barr virus
    f. History of gastric surgery especially vagotomy with gastectomy

2. Genetic factors
    a. Sporadic gastric cancers have p53 and cox 2 gene mutation
    b. Hereditary diffuse gastric carcinoma – CDH gene encodes for E cadherin
    c. Menetrier`s disease
    d. Li fraumeni syndrome
    e. Familial adenomatous polyposis
3. Precursor lesions
    a. Adenomatous gastric polyps
    b. Chronic atrophic gastritis
    c. Large hyperplastic polyps
    d. Intestinal metaplasia

Chronic H.pylori infection is associated with three fold increase in risk of adenocarcinoma of stomach. Chronic superifical gastritis caused by H.pylori infection, it further progress to atrophic gastritis and intestinal metaplasia, finally dysplasia and cancer.

Regular aspirin has some protective effect on gastric cancer. Vegetables and high antioxidant including vitamins C and E are decrease the risk of gastric cancer.

MORPHOLOGICAL CLASSIFICATION

Morphologically gastric cancer divided into four subtypes

- Polypoid – intraluminal lesion
- Fungating – largely intraluminal with ulceration
- Ulcerative – confine to wall of the stomach
- Scirrhous – infiltrate full thickness of stomach with large area

Distribution of carcinoma stomach according to location is 40% distal, 30% middle, 30% proximal.

PATHOLOGICAL CLASSIFICATION

Most important prognostic factors are lymph node involvement and depth of invasion

TUMOUR GRADING

Tumour grading is also important in prognosis. Tumours are graded according to degree of differentiation like

Well differentiated

Moderately differentiated

Poorly differentiated

WHO HISTOLOGICAL CLASSIFICATION

- Adenocarcinoma
    - Papillary adenocarcinoma
    - Tubular adenocarcinoma
    - Mucinous adenocarcinoma
    - Signet ring adenocarcinoma
    - Adenosquamous carcinoma
- Squamous carcinoma
- Small cell carcinoma
- Undifferentiated carcinoma
- Others

LAURENS CLASSIFICATION

Lauren classified the gastric carcinoma into intestinal type and diffuse type

- The Intestinal type is less aggressive than the diffuse type. It associated with gastric atrophy and intestinal metaplasia and dysplasia. Men are common affected than woman. The intestinal type are typically well differentiated and tendency to form the glands. H.pylori infection play a role in development of intestinal type of gastric cancer

- The diffuse type is poorly differentiated adenocarcinoma and it lacks gland. It spread through the submucosal layer and metastatic spread by transmural extension and lymphatic invasion. Female are mostly affected by this type. Prognosis less favourable in diffuse type. It associated with hereditary diffuse gastric cancer.

BORRMANS CLASSIFICATION

The Borrman classified the advanced gastric carcinoma into five subtypes, based on macroscopic appearance

- Type I – single ,polypoid carcinoma
- Type II- ulcerated with elevated border
- Type III- ulcerated with infiltrating the gastric wall
- Type IV – diffusely infiltrating carcinoma
- Type V- unclassified

MINGS CLASSIFICATION

It based on histomorphologic staging

- Expanding type (67%)– it is uniformly polypoid , and it has favourable prognosis
- Infiltrative type (33%) – diffuse on gross appearance and it has poor prognosis

JAPANESE CLASSIFICATION

The Japanese classification is developed by the Japanese research society for early gastric cancer based on endoscopic findings.

- Type I- Protruded
- Type IIa- superficial elevated
- Type IIb- superficial flat
- Type IIc- superficial depressed
- Type III- excavated

MODE OF SPREAD

1. DIRECT; local extension occurs by radical intramural spread and also by deep invasion through the wall to involve adjacent structures. Local extension occurs through the serosa and also involvement of the omentum, spleen, adrenal gland, liver, and pancreas. Through the subserosal lymphatic plexus radial spread occurs. Direct infiltration occurs through the duodenum. Oesophageal spread occurs through the submucosal lymphatic plexus.
2. LYMPHATIC SPREAD; tumour spread through draining lymph node in adjacent structure. Sister mary josephs nodule or umbilical nodule is formed by lymphatic spread from the gastric malignancy via ligamentum teres. Left supraclavicular node spread occurs through the thoracic duct and it’s called troisier`s sign.

3. Transcoelomic spread; once tumour infiltrates the serosa there is no barrier to prevent the tumour spread. Tumour cells are freely enters the peritoneal cavity causing peritoneal deposits and ascites. Tumour cells deposit into pouch of douglas ,its palpated per rectally and it's called blummer`s shelf . krukenberg`s tumour occurs due to deposit of tumour cell into active ovarys in premenopausal women.
4. Haematogenous spread : haematogenous spread to liver, lungs, omentum, pancreas, bones and skin.[5,7,25]

DUODENAL MALIGNANCY

Primary malignant tumours of the duodenum are account for 0.3% of all gastro-intestinal tract tumors but up to 50% of small intestine malignancies. Tumours arises from Periampullary region like periampullary carcinoma, pancreatic carcinoma, cholangiocarcinoma must be ruled to confirm the diagnosis of primary duodenal malignancy. Adenocarcinoma is commonest tumour of duodenum. Other malignancies like lymphomas, leiomyosarcomas, carcinoid tumours, GIST occurs in duodenum. Duodenal polyps especially polps asscoiated with FAP or gardeners syndrome are premaglinant conditions. Most common location of primary duodenal malignancy is second part of duodenum.[26]

PANCREATIC MALIGNANCIES

Pancreatic malignancy is related 15 -20% of gastric outlet obstruction in western countries. Pancreatic tumour causing GOO is due to progressive tumour enlargement causing extrinsic compression on duodenum, and less common by the direct infiltration of tumour, lymphadenopathy.[27, 28]

CHOLANGIOCARCINOMA

Epithelial cells of intra or extra hepatic bile ducts are the cell of origin for cholangiocarcinoma and its account for only 3 % of all gastrointestinal tumours. Cholangiocarcinoma is rare with incidence of 1-2 cases per 100,000. Adenocarcinoma is most common type of malignancy and its subdivided into nodular, sclerosing and papillary. Distribution of cholangiocarcinoma according to location is 50% in perihillar disease, 40% in distal disease, 10% in intrahepatic disease. Invasion of the tumour into porta hepatis can lead to duodenal obstruction and it indicates unresectability.[29]

CLINICAL FEATURES

Clinical features common to all conditions causing gastric outlet obstruction can be considered as general and the symptoms pertaining to specific disease can be considered later.[7, 25, 30]

I. GENERAL CLINICAL FEATURES

SYMPTOMS

ABDOMINAL PAIN

Patient with GOO has upper abdominal pain, and its dull aching in type, and it constant in between meal time and it increases after food intake and relieved by vomiting.

VOMITING

Vomiting is the most common symptom of gastric outlet obstruction. If vomiting absent diagnosis of GOO must be reconsider. Vomiting is projectile, large amount of vomitus and which is contains undigested food particle. Vomiting occur at any time but usually takes several hours after meal.

LOSS OF APPETTITE

It is early feature of gastric malignancy and in peptic ulcer disease appetite never lost. Loss of appetite occurs in malignancy due to tumour releases varies substances like proinflammatory cytokines , parathormone related peptides or tumours causing dysphagia or altering gut function or psychological factors like depression.

Loss of weight

Weight loss occurs in Gastric outlet obstruction due to mechanical obstruction prevents food to enter into small bowel for further digestion and absorbtion or due to vomiting or tumour alters basic metabolic rate.

HAEMETAMESIS AND MELENA

Most common cause of haemetamesis is chronic peptic ulcer. It's also seen in chronic gastritis, oesophageal varices, bleeding tumour, and Mallory Weiss syndrome. Melena may occur in all cases of haemetamesis or slow bleeding ulcer or tumour in gastrointestinal tract.

CONSTIPATION

Constipation is due to repeated vomiting or melena or dehydration. Recent onset of constipation is complaint of patients with gastric outlet obstruction

BALL ROLLING MOVEMENT

Patients with GOO sometimes experience the gastric peristalsis and it usually occurs after food intake.

CLINICAL EXAMINATION

ANAEMIA

Anaemia occurs due to poor nutrition intake or chronic bleeding ulcer or malignancy or massive hematemesis and melena. In tumour release the pro inflammatory cytokines it suppresses the bone marrow.

DEHYDRATION

Dehydration occurs once body losses fluid more than 6% of body weight. In GOO dehydration due to repeated vomiting and decreased water absorption, because of complete obstruction in pylorus.

VISIBLE GASTRIC PERISTALSIS

VGP is the inspection finding of gastric movement seen in upper abdomen because of gastric peristalsis from left to right. It starts from left hypochondrium, crosses the midline and ends in the right hypochondrium. Make the patient to drink water to elicit the VGP.

PERCUSSION METHOD

In gastric outlet obstruction stomach is dilated due to increased volume of gastric content. Dilated stomach is percussed from central to periphery, and greater curvature is marked at the point of change of note.

AUSCULTOPERCUSSION METHOD

Bell of stethoscope is placed just below and abdomen is scraped toward outward from xiphisternum with a blunt object and note the point of change in sound and this point indicate that dilated greater curvature.

SUCCESSION SPLASH

Succession splash is done after four hours of nil per oral, diaphragm of stethoscope placed just below the xiphisternum and shaking the patient we can hear the gurgling sound of gastric stasis

SPECIFIC CLINICAL FEATURES

PEPTIC UCLER DISEASE

VOMITING

Painless Non bilious vomiting is seen chronic cicatrising peptic ulcer. Painful non bilious vomiting is seen in acute peptic ulcer due to inflammation, edema of pylorus and pyloric spasm. Vomiting occur more on evening.

PAIN

Dull aching pain is in the epigastric region and which is radiating to back, because ulcer may penetrate into deeper layer or pancreas. In acute gastric ulcer disease pain in epigastrium, pain increases after food intake inbetween period patient free of pain. Vomiting may relieve the pain.

PERIODICITY

Untreated peptic ulcer disease has periodicity. It's due to sponateous healing of the ulcer. Symptoms may disappear for weeks or months to return again.

FEATURES OF NUTRITIONAL DISTURBANCES

Vomiting and dehydration may cause loss of taste, phargitis, coated tongue, oral ulcers and charring of tooth. Patient may have typical ulcer facies, confused mental status and tetanus are due to loss of eletrolytes. Signs of Dehydration are dry, wrinkled skin and loss of skin turgor.

NSAIDS INDUCED STRICTURE

There will be history of long term use of the NSAIDS. Examine for the osteoarthritis and rheumatoid arthritis.

CASUATIVE INGESTION INDUCED STRICTURE

There is history of corrosive poisoning and history of mental retardation or borderline behavioural disease.

POSTOPERATIVE ADHESION

History of pervious upper abdominal surgeries, especially open cholecystectomy. Malignancy must be ruled out. Look for pervious history of attack of pancreatitis or cholecystitis. Look for Pervious surgical scar.

PRIMARY GASTRODUODENAL TUBERCULOSIS

Gleason et al study shows that most common presenting symptoms were pain (73 %) and vomiting (55 %), whereas GI bleeding was rare (16 %).long term H2 blocker associated with gastroduodenal TB due to decreased acidity of stomach.

CHRONIC PANCREATITIS OR PSEUDOCYST OF PANCREAS

In pseudocyst of pancreas patients may present with abdominal pain (76 -94%), early satiety, nausea, vomiting and weight loss. There will be previous hospitalisation for pancreatitis. Physical examination may reveal upper abdominal tenderness, fullness in epigastrium, vague mass may palpable.

RADIATION INDUCED STRICTURE

History of radiotherapy for hepatobiliary malignancies or non hodgkins lymphoma.

CROHNS SYNDROME

Isolated upper gastrointestinal crohns disease present as like as gastritis like epigastric pain over number of years. Patients are present with symptoms of gastric outlet obstruction like bloating, vomiting, postprandial pain, weight loss.

ADULT IDIOPATHIC HYPERTROPHIC PYLORIC STENOSIS

Symptoms of AIHPS are vomiting, postprandial nausea, early satiety, and epigastric pain. History of diabetic mellitus, peptic ulcer, and previous surgery must be asked to differentiate from primary AIHPS and secondary AIHPS. On physical examination abdominal mass rarely felt as compared to juvenile HPS

BEZOARS AND FOREIGN BODY

Gastric bezoars are usually asymptomatic. Common presentations of bezoars are abdominal pain, nausea, vomiting, anorexia, hematemesis, and melena. Changing the position of patient may relive the symptoms because larger foreign body in stomach acts as ball valve mechanism. There will be the history of mental retardation, psychological illness, and diabetic mellitus (diabetic gastropathy decrease the gastric motility it affect the grinding function of stomach). On physical examination abdominal mass may palpable in epigastrium.

BOUVERTS SYNDROME

Symptoms of patients with Bouveret's syndrome as nausea and vomiting (86%), and abdominal pain (71%), and hematemesis, weight loss, and anorexia are less commonly present. Past history of painful jaundice or elevated liver function may be present. On physical examination, abdominal tenderness in right hypochondrium and epigastrium, abdominal distension, and dehydration may present.

WILKIES SYNDROME

Wilkie’s syndrome clinically present with bilious vomiting, postprandial fullness, anorexia. Upper abdominal fullness, visible peristalsis and other signs of gastric outlet obstruction are present on physical examination.

GASTRIC VOLVULUS

Gastric volvulus present as acute epigastric pain, non-projectile violent vomiting, hematemesis and on examination of abdominal tenderness and inability to pass a nasogastric tube

POLYP

Atrophic gastritis is associated with hyperplastic polyps. Epithelial polyps presents with epigastric pain, haematemesis or malena. Gastric leiomyoma presents with hematemesis or malena. Bleeding from tumour may be massive or intermittent.

GASTRIC LYMPHOMA

Gastric lymphoma are present with abdominal pain occurs in more than 80% of patients and also associated with early satiety, nausea, night sweats, fever, and haemorrhage. On examination, a mass palpable in left upper abdomen and Splenomegaly may be present.

MALIGNANT GASTRIC OUTLET OBSTRUCTION

CARCINOMA STOMACH

Symptoms of carcinoma stomach are generally nonspecific, so at the time of diagnosis most of patients are in advanced stage of disease. Symptoms of the gastric carcinoma include epigastric pain, nonbilious vomiting, early satiety, and weight loss. These symptoms are frequently mistaken for more common benign disease like peptic ulcer disease. Pain in gastric cancer lost it periodicity and it more constant and non-radiating type of pain. Generally pain not relieved by eating. Anaemia seen in 40% of patients due to bleeding from tumour, its either present as melena or hematemesis or both. Frank hematemesis is only seen in 15% of patients. Early satiety is the earliest symptoms due to stomach loss its dispensability because of the infiltration tumour in stomach wall. Weight loss is commonest symptoms. Non bilious vomiting occurs in patients with pyloric-antral growth. Patients with jaundice, palpable mass, ascites, left supraclavicular node, umbilical node are inoperability and metastatic disease. Once tumour breaches the serosa of stomach wall there is barrier to prevent tumour cell to enter peritoneal cavity once it enters deposit in varies parts like

ovaries (krukenberg tumour), in pelvis it deposit in rectovesical pouch and it felt like firm shelf on per rectal examination(blumer shelf). Left supraclavicular node (virchow), subcutaneous deposit around umbilicus (sister mary joseph node), and left axillary node (irish node) are seen in advance stage of disease. Most common site of metastasis is liver.

STAGING

Most common used staging system at present time is the TNM staging created by AJCC. This system helps to stage the disease, to determine the treatment, and predict the prognosis. TNM staging based on the depth of tumour invasion (T), number of positive nodes (N), and presence or absence of distant metastasis (M). Previously N stage described by anatomical location of nodes with respect to primary tumour, but now number of positive lymph node used to determine the N staging. The current TNM staging is not based the locations of positive lymph node and current system staging suggest that minimum 15 nodes must be evaluated for accurate staging.

The AJCC system is not specific for nodal location. Previous version of UICC (union internationale contrele cancer) TNM staging, N staging defined by location of lymph node metastasis according to the primary tumour. pN1- Positive nodes present within 3cm of primary tumour and pN2 defined as positive node present more than 3 cm from primary tumour.

THE AJCC TNM STAGING (EDITION 8)

DEFINITION OF PRIMARY TUMOR (T)

- Tx- primary tumour cannot be assessed
- T0- no evidence of primary tumour
- Tis- carcinoma insitu; intraepithelial tumour without infiltration to lamina propria, high grade dysplasia.
- T1-invades lamina propria or muscularis mucosae, submucosa
- T1a-invades lamina propria or muscularis mucosae
- T1b-invades submucosa
- T2- invades muscularis propria*
- T3-penetrates subserosal connective tissue without invasion of visceral peritoneum or adjacent structures**-***
- T4- invades serosa or adjacent structures**-***
- T4a-invades serosa (visceral peritoneum)
- T4b-invades adjacent structures/organ

*A tumour may penetrate the muscularis propria with extension into gastrocolic or gastrohepatic ligament or into greater or omentum, without penetrating visceral peritoneum covering these structures. In this case, the tumour classified as T3.

**ADJACENT STRUCTURES are spleen, transverse colon, liver, diaphragm, pancreas, abdominal wall, adrenal gland, kidneys, small intestine, and retroperitoneum.

***Intamural extension of duodenum or oesophagus is not considered invasion of adjacent structures, but is classified using depth of the greatest invasion in any of these sites.

DEFINITION OF REGIONAL LYMPH NODE (N)

- Nx- regional LNs cannot be assessed
- N0-no regional LN metastasis
- N1- 1 or 2 regional LNs
- N2- 3-6 regional LNS
- N3- more or equal to 7 regional LNs
- N3a- 7-15 regional LNs
- N3b- more or equal to 16 LNs

DEFINITION OF DISTANT METASTASIS (M)

- M0- No distant metastasis
- M1- distant metastasis

AJCC PROGNOSTIC STAGE GROUPS

CLINICAL TNM

STAGING	T	N	M
0	Tis	N0	M0
I	T1-T2	N0	M0
IIA	T1-T2	N1,N2,N3	M0
IIB	T3 OR T4a	N0	M0
III	T3 OR T4a	N1,N2 OR N3	M0
IV A	T4B	ANY N	M0
IV B	ANY T	ANY N	M1

AJCC TNM PROGNOSTIC STAGING in 8th edition stages IA, IB, IIIA, IIIB, and IIIC are removed. Stage IVA and IVB are formed.

JAPANESE CLASSIFICATION FOR GASTRIC CARCINOMA STAGING SYSTEM (JCGC)

The JCGC is established to describe the anatomical location of nodes removed during gastrectomy. 16 distinct anatomical locations of lymph nodes described with recommendation for nodal basin dissection dependents on the location of the primary. The presence of tumour deposit to each lymph node group

JAPANESE GASTRIC CANCER ASSOCIATION STAGING SYSTEM FOR GASTRIC CANCER

TUMOR STAGE

- T1-Tumor invasion of mucosa and/or muscularis mucosa (M) or submucosa (SM)
- T2-Tumor invasion of muscularis propria (MP) or subserosa (SS)
- T3-Tumor penetration of serosal (SE)
- T4-Tumor invasion of adjacent structures (SI)
- TX- Unknown

NODAL STAGE

- N0- No evidence of lymph node metastasis
- N1-Metastasis to group 1lymph nodes, but no metastasis to group 2 to 3 lymph nodes
- N2-Metastasis to group 2 lymph nodes, but no metastasis to group 3lymph nodes
- N3-Metastasis to group 3lymph nodes
- NX -Unknown

HEPATIC METASTASIS STAGE (H)

- H0-No liver metastasis
- H1-Liver metastasis
- HX-Unknown

PERITONEAL METASTASIS STAGE (P)

- P0-No peritoneal metastasis
- P1-Peritoneal metastasis
- PX-Unknown

PERITONEAL CYTOLOGY STAGE (CY)

- CY0-Benign/indeterminate cells on peritoneal cytology
- CY1-Cancer cells on peritoneal cytology
- CYX-Peritoneal cytology was not performed

OTHER DISTANT METASTASIS (M)

- M0-No other distant metastases (although peritoneal, liver, or cytological metastases may be present)
- M1-Distant metastases other than the peritoneal, liver or cytological metastases
- MX-Unknown

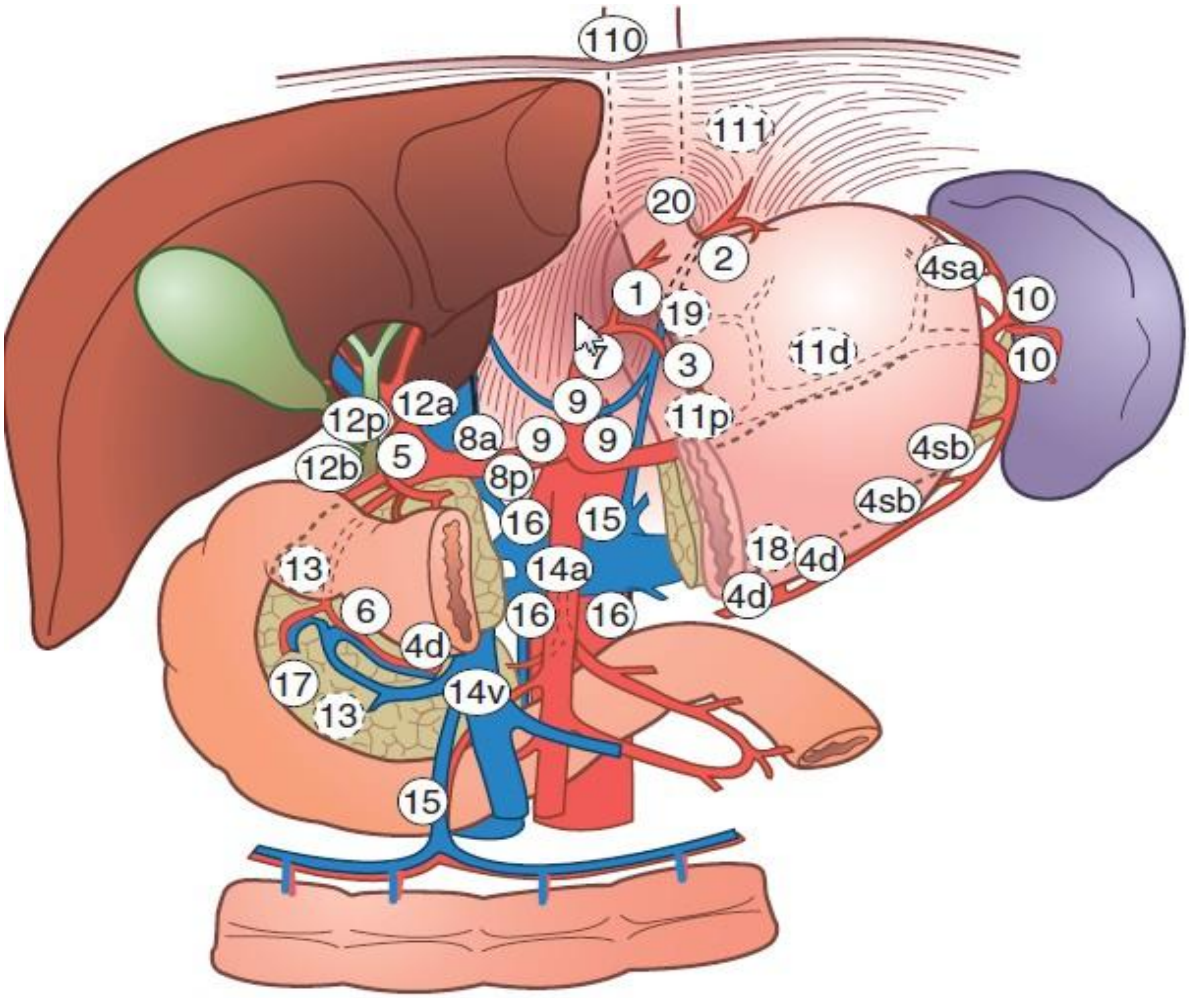

Fig-12: Lymph node station numbers as defined by the JCGC.

GROUPING OF REGIONAL LYMPH NODE BY LOCATION OF PRIMARY TUMOUR

		LOCATION OF PRIMARY TUMOUR IN STOMACH		
LYMPH NODE STATION	DESCRIPTION	UPPER THIRD	MIDDLE THIRD	LOWER THIRD
1	Right paracardial	1	1	2
2	Left paracardial	1	3	M
3	Lesser curvature	1	1	1
4sa	Short gastric	1	3	M
4sb	Left gastro epiploic	1	1	3
4d	Right gastroepiploic	2	1	1
5	suprapyloric	3	1	1
6	Infrapyloric	3	1	1
7	Left gastric artery	2	2	2
8a	Anterior common hepatic	2	2	2
8p	Posterior common hepatic	3	3	3
9	Celiac artery	2	2	2
10	Splenic hilum	2	3	M
11p	Proximal splenic	2	2	2
11d	Distal splenic	2	3	M
12a	Left hepatoduodenal	3	2	2
12b,p	Posterior hepatoduodenal	3	3	3
13	Retropancreatic	M	3	3
14v	Superior mesenteric vein	M	3	2
14a	Superior mesenteric artery	M	M	M
15	Middle colic	M	M	M
16al	Aortic hiatus	3	M	M
16a2, b1	Para-aortic, middle	M	3	3
16b2	Para-aortic,caudal	M	M	M

R status

In 1994, Hermanek described the term R status. R status used to describe tumour status after resection and it is helps in determine the adequacy of surgery. Extent of resection can influence survival.

R0- macroscopic and microscopically margin negative resection, it indicates no remaining tumour bed.

R1 – microscopically margin positive for tumour.

R2- gross residual disease

Long term survival achieved only by R0 resection.[6, 7, 25,30,31]

DUODENAL MALIGNANCY

Incidence is more in sixth decade, although the disease may develop in younger age group. Symptoms of duodenal malignancy are abdominal pain (15-60%), weight loss (30 to 60%), nausea and vomiting (25 to 30%), jaundice (20 to 30%), haemorrhage (10 to 38%). A palpable abdominal is found in less than 5% of cases.

PANCREATIC MALIGNANCIES

Pancreatic maliganancies especially periampullary carcinoma, head of pancreas are presents with painless, progressive jaundice, pruritus, clay coloured stools and high coloured urine and upper abdominal pain it may radiating to back due to infiltration of tumour into celiac and mesenteric plexus. Along with this nonbilious vomiting, early satiety, anorexia are suggestive of

gastric outlet obstruction. Pancreatic malignancy is most common cause of malignant gastric outlet obstruction in western countries. On physical examination abdominal mass may palpable in epigastrium, gallbladder palpable (30%) and visible gastric peristalsis may present. Gastric outlet obstruction occurs in pancreatic malignancies due to external compression or by direct invasion of pyloric antrum or second part of duodenum.

COMPLICATION OF GASTRIC OUTLET OBSTRUCTION

METABOLIC ABNORMALITY

In Gastric outlet obstruction the metabolic abnormalities is the most common complication and serious complication also. Long standing period of vomiting causes loss of HCL (hydrochloric acid) , sodium and increases the production of bicarbonate ions in plasma to compensate the loss of chloride ions, and it causes hypokalemic hypocholremic metabolic alkalosis. Increased pH of plasma shifts intracellular potassium to extracellular compartment; it increases the serum potassium level. Increased pH (alkalosis) causes increased execration of bicarbonate ion from kidney along sodium, urine become alkaline. If continues vomiting with renal loss of sodium increases renal excretion of potassium in order to prevent sodium excretion from kidney. Continues sodium loss causes hyponatraemia and profound dehydration it turn increase aldosterone level. Aldosterone increases the sodium reabsorbtion and it increase the loss of potassium from kidney and further aggravates the hypokalemia followed to prevent potassium loss, hydrogen ions excreted in urine. Now urine

becomes paradoxically acidic. Alkalosis leads to decreases circulating ionised calcium, and tetany can occur.

Correction of metabolic abnormality is started with correction of dehydration by using isotonic saline infusion. It corrects hypovolemia and increases extracellular fluid and it increases alkaline excretion. Potassium can be supplemented in severe hypokalemia by potassium chloride infusion at the rate of 20meq/day. It facilitates absorption of hydrogen ions from renal tubules.[7,25,31,32]

STASIS GASTRITIS

Dilated stomach with decreased peristalsis are increases the stasis of food in stomach it turns harbour more bacteria. It ferments the food and it produces the inflammation of gastric mucosa.

INVESTIGATION

BLOOD EXAMINATION

HEMOGLOBULIN

Anaemia is due to malena, hematemesis, and malnutrition. Blood loss and iron deficiency causes microcytic hypochromic anaemia. Due to deficiency of intrinsic factor in carcinoma stomach causes megalobalstic anaemia. Tumour releases some pro-inflammatory cytokines also suppress the bone marrow.

BLOOD GROUPING

Peptic ulcer more in persons with blood group O. Incidence of gastric carcinoma is higher in blood group A.

LIVER FUNCTION TEST

Patients with Abnormalities in liver function test may predict inoperability. Raised ALP and bilirubin levels indicate that obstructive jaundice may due to pancreatic malignancy, duodenal malignancies, gastric carcinoma, and cholangiocarcinoma.[33]

SERUM ELECTROLYTES

Gastric outlet obstruction may show hypochloremia, hypokalemia, hyponatremic metabolic alkalosis and it due to prolonged vomiting and dehydration.[32]

MEASUREMENT OF GASTRIC EMPTYING

Nasogastric intubation technique

Saline load test

In 1965, Goldstein and boyle introduced saline load test to measure the residual volume of stomach. Start with nasogastric tube intubation and gastric emptying followed that installation of 750 ml of isotonic saline done. After 30 min stomach content is aspirated. If aspiration more than 400ml it indicate gastric outlet obstruction. Disadvantage of this test is insensitive to measure the solid food digestion.[7]

THE FRACTIONAL TEST MEAL

This test first used to study the gastric secretion but it's also used to measure the gastric emptying. Starch containing meal given and aspiration done in every 15 minute till starch test become negative. It very difficult to determine the end point of test, because of starch test is very sensitive to tiny amount of starch containing food.

Gastric secretion tests

The Gastric secretion tests are performed to determine the basal secretion of stomach. For this test patient must be in overnight fasting and drug that alter the stomach secretory function should not take before 24 hours. During the test nasogastric tube is passed into stomach and stomach content is sucked out and after 60 min one gastric sample is collected. It used to estimate basal secretion.

PENTAGASTRIN TEST

The optimum dose of pentagastrin (6 microgram per body weight) is injected intramuscularly. Samples are collected every 15mintute for one hour after pentagastrin injection. Maximum acid output is defined by the maximum acid output in one hour of period after injection of pentagastrin. It helps in determine the effectiveness of treatment after vagotomy with drainage, low acid output noted in patients with vagotomy. Very low acid secretion noted in gastric carcinoma.[7,31,34]

Kays augmented histamine test

This test used to measures the total mass of oxyntic cells in the stomach. After keeping overnight fasting and fasting stomach content is aspirated. Mepyramine maleate is given to prevent the side effect of histamine except its stimulation of gastric acid. Histamine acid phosphate is given subcutaneously. Gastric content is aspirated for next one hour. The HCL is measured in mEq per hour. Results are in Gastric ulcer- 15; duodenal ulcer- 30 to 40; anastomotic ulcer – 30 to 35 mEq.[7,34]

RADIOLOGICAL INVESTIGATION

1. PLAIN XRAY ABDOMEN

Plain x-ray abdomen is used in detecting complicated peptic ulcer like perforation, foreign bodies in stomach, and in trichobezoar its show intra-gastric foreign body with outlined by air.

2. CHEST X-ray

It helps in detecting the complicated peptic ulcer like perforation, to rule out the secondaires lung, in primary gastric tuberculosis to see for the accompanying pulmonary tuberculosis.

3. BARIUM MEAL EXAMINATION

Barium meal study is done for chronic duodenal ulcer, gastric carcinoma, gastricjeunocolonic fistula. In chronic duodenal ulcer barium meal study shows absence of duodenal cap, dilated stomach, mottled stomach, and barium will not pass into duodenum. Carcinoma pylorus this study shows irregular filling defect, stomach lost its rugosity, delayed emptying, dilated stomach, margins of

the lesion projects outward from the ulcerative growth into the gastric lumen. Double contrast barium study is comparable to upper GI scopy in detecting gastric carcinoma and its sensitivity is 90 -95%. Double contrast barium study uses the barium and air as contrast medium. Stomach is inflated with barium and air, its forms thin layer on mucosa. Small and irregular lesions are identified by under tension normally stomach mucosal fold are disappeared, its absent in carcinoma of stomach.

Pancreatic carcinoma barium study shows dilated C loop of duodenum.[6, 7, 31, 35]

UPPER GI SCOPY

Upper GI scope is the most important diagnostic and therapeutic tool in gastric outlet obstruction. The procedure done in patient with alarm signs are recent onset weight loss, anemia, dysphagia, vomiting, and age more than 55 years with new onset dyspepsia. The procedure performed under laryngeal anaesthesia. Following an 8 hours fasting, the upper GI scope is introduced under direct vision into the oesophagus, stomach, and duodenum. Multiple biopsies are taken from suspicious mass or ulcer in stomach and to confirm the diagnosis histologically. More 8 biopsy specimen must be taken to achieve accurate diagnosis. Single biopsy has only 70% of diagnostic sensitivity. More 8 biopsies have 98% diagnostic sensitivity. This is the advantage over the double barium meal study. Narrow band imaging with magnified endoscopy is investigation of choice for early gastric carcinoma. NBI shows microvascular

architecture of the mucosa and microsurface pattern of lesion. Magnification endoscope is 50 to 150 time higher magnification than usual endoscope. [6, 7, 31, 34-37].

Therapeutic uses of endoscope are

1. Upper GI scope used in cicatrizing duodenal ulcer to dilate the stenosed part of stomach by using ballon dilation.
2. To remove the foreign body from stomach that causing gastric outlet obstruction.
3. Inoperable patients of gastric outlet obstruction the endoscopic palliative stenting can be done.
4. Early gastric cancer can be excisied by endoscopic mucosal resection or endoscopic submucosal dissection.
5. Polyps that causing gastric outlet obstruction can removed by endoscopic snare.

ABDOMINAL ULTRASOUND

The ultrasound is limited use in the gastric outlet obstruction due to gas in stomach deflect the sound waves. In malignant gastric outlet obstruction, USG may shows the pyloric thickening, ascites, liver metastasis, enlargement of regional, and para aortic nodes. In Hypertrophic pyloric stenosis the pyloric thickening more than 4mm or pyloric canal length more than 14mm are present and it can be measured by USG.

ENDOSCOPIC ULTRA SOUND

Endoscopic ultra sound is having ultrasound at the tip of insertion section. This is allows one get both endoscopic image and ultrasound image of stomach wall in better detail. EUS shows the gastric wall in five alternatively hyper and hypoechoic layers which are mucosa, muscularis mucosa, submucosa, muscularis propria, and serosa. The accuracy of T staging is around 90% and N staging of accuracy is about 80%.

Two types of EUS are linear and radial. Linear EUS is used for various therapeutic interventions. Linear EUS produce images parallel to axis of probe. It facilitates to see the pathological site and biopsy needle in real time.[34-38]

Uses of EUS

- For T and N staging of esophagus, stomach, pancreatic malignancy.
- To diagnose the bile duct stones
- To evaluate the cystic lesions of pancreas.
- EUS directed interventions are fine needle aspiration, coeliac plexus block, to guide the access to bile duct, to monitor glue injection of gastric varices.

CECT ABDOMEN AND PELVIS

Oral and IV contrast CT helps in preoperative staging the disease. It shows tumour location, lymph node enlargement and intraabdominal metastasis especially liver, peritoneal deposit, omental deposits. CECT abdomen shows more than 5mm metastatic deposit in abdominal cavity. Metastatic lesions less

than 5mm are called CT occult metastasis. CECT abdomen helps in staging gastric cancer by detecting depth of invasion (T) and lymph node enlargement (N) and it helps planning of procedure preoperatively.

POSITRON EMISSION TOMOGRAPHY

PET scan is a functional imaging technique that uses accumulation of tracer in metabolically active tumour tissue. PET scan is used to identify the distant metastasis by accumulation of 18 FDG in tumour cell and its helps in differentiating tumour from non-tumour cell. PET scan useful in locally advanced disease. PET with CT improves the accuracy of diagnosis, especially in locally advanced disease staging.[25, 31, 34]

STAGING LAPROSCOPY WITH PERITONEAL LAVAGE

Staging laparoscopy is helps determine the preoperative staging evaluation patients with locally advanced gastric carcinoma. Staging laparoscopy helps in preoperative staging by direct inspection of peritoneum and visceral surfaces for metastatic deposits, which are not identified by CECT. CECT abdomen cannot identify the low volume macroscopic deposits that are less the 5mm in size. Peritoneal lavage helps identify the micro metastasis. Patients with peritoneal lavage reported positive for metastatic deposits have poor prognosis. Staging laparoscopy can be performed either separate procedure or prior to planned laparotomy. Staging laproscopy have overall sensitivity of 89% and specificity of 100% in detecting CT occult M1 disease. It helps to avoid unnecessary procedures.[25]

TUMOUR MARKER

Tumour makers has very limited role in diagnosis of gastric cancer. Sometimes tumor makers also elevated in benign diseases. Tumor markers helps follow-up after primary treatment. Commonly used tumor markrs are carcinoembryonic antigen (CEA), cancer antigen (CA) 19-9, CA 50, CA 72-4, CA 125, human chorionic gonadotropin BETA. Combined tumor markers increase the sensitivity of diagnosis. Now days in gastric cancer HER 2U overexpression is reported around 13 % to 30% of patients. HER 2 targeting with trastuzumab is increases the prognosis of the patients with stage IV disease.[25]

TREATMENT

Preoperative preparation is most important part of management of gastric outlet obstruction otherwise these patients are carries a significant mortality during surgery. The presence of signs of dehydration indicates that fluid deficit of 4 litres and about 20gms of NaCl. Infusing sodium in the form of isotonic saline allows excretion of alkaline urine, its helps in correction of alkalosis. The hypokalemic, hypochloremic alkalosis is corrected with normal saline containing potassium chloride. More than 20meq of KCl per hour needs to be infused in severe hypokalaemia; it should be done under cardiac monitoring continuously because of arrhythmogenic nature of KCL. The general condition of patient must be improved before planning any surgical intervention. This must be done by helps of chest physiotherapy, asytmine supplements, multi-

vitamin supplement, and blood transfusion in case of anaemia. Outcome of treatment depends on improving condition of patients preoperatively. [32]

Treatment of benign gastric outlet obstruction

Chronic peptic ulcer

- Endoscopic intervention

Benjamin et al is the first reported the use of endoscopic balloon dilation of the pylorus as treatment of gastric outlet obstruction by using TTS (THROUGH THE SCOPE) endoscopic balloon dilation with good outcomes. Nowadays single or variable diameters balloons with hydrostatic inflation devices are available.

THROUGH THE SCOPE ENDOSCOPIC BALLOON DILATION;

Patients are kept overnight fasting before the day of procedure and stomach preparation also needed. Good preparation needed to visualization of pathological area. Under conscious sedation, endoscopy introduced into oesophagus and then into stomach. Followed by guide wire is pushed out of the TTS and it negotiated across the stricture and then balloon insufflate with either water or air. Pressure kept for 1 – 2 minitutes and then scope removed. Procedure repeated in every 1-2 weeks until 15- 18 mm dilation is achieved. For better results eradication of H. pylori is important.[39,40]

Causative ingestion induced stricture is difficult to dilate the stenosed part compared to chronic peptic ulcer.

Patients with gastric tuberculosis, crohns disease are dilation of stenosis with endoscopic balloon dilation have immediate relieve of symptoms but recurrence is more common. It needed to treatment the primary cause of stenosis for better results. Failed balloon dilation is indication for vagotomy and drainage procedures.

SURGICAL TREATMENT

Surgical treatment of chronic peptic ulcer with gastric outlet obstruction includes treatment of H.pyloric infection and vagotomy with drainage procedure or vagotomy with antrectomy. Truncal vagotomy and gastroenterostomy has very low mortality rate less than 1% and recurrence ulcer rate is 2-7%.[5,7,25,34]

Modified visick grading system used to assess the effectiveness of various procedures.

- Grade 1— no complaints, an asymptomatic patient
- Grade2—mild symptoms that are treated with diet modification
- Grade 3—moderate symptoms without substantial interference with modification of life style
- Grade 4—unsatisfactory results and includes all patients with recurrent ulcer.

For peptic ulcer were treated by variant of basic surgical procedures are parietal cell vagotomy, proximal gastric vagotomy, vagotomy and drainage. Among these procedures the lowest recurrences rate, but higher mortality and morbidity in vagotomy and drainage procedure. Low morbidity and mortality (1%) in

parietal cell vagotomy but recurrences rate is 5-15%. When we divide the nerve of latarjet it increases the complication like dumping syndrome (among 10% of patients), diarrhea. In patients with gastric outlet obstruction procedure of choice is truncal vagotomy and gastrojejunostomy.

TRUNCAL VAGOTOMY

Truncal vagotomy is indicated for patients with complicated peptic ulcer disease like bleeding or perforation or obstructing peptic ulcer disease. A drainage procedure is companied with truncal vagotomy, because when we denervate the vagus nerve, its motor supply to antropyloric region is lost. So it results in gastric stasis. Complications of truncal vagotomy includes

- Injuries to adjacent structures like distal esophagus, splenic artery and vein, liver.
- Early postoperative complications like delayed gastric emptying, dysphagia, anastomotic leak
- Late postoperative complications are diarrhoea, esophagitis due to reflux of gastric content, cholelithasis.
- Post – vagotomy diarrhoea
    - Its associated with Clinically significant dairrhoea in 5-10% of patients
    - Cause of post-vagotomy diarrhoea is unclear. Some proposed mechanism includes intestinal dysmotility, accelerated transient

time, bile acid secretion, rapid gastric emptying and bacterial overgrowth.

- Treatment; medical treatment like octerotide, cholestyramine, lopramide or codeine.
- Surgical treatment like decreasing transient time by reversing portion of bowel. 10 cm of reversed jejunal interposition placed in continuity 100cm distal to ligament of treitz.

HIGHLY SELECTIVE VAGOTOMY

Highly selective vagotomy can be performed by four phases and preserve the innervation of gastric antrum. In Highly selective vagotomy, vagus nerve are divided that supply the acid producing portion of the corpus and fundus of stomach.

- Expose the stomach and mobilisation of stomach
- Anterior leaf of the lesser omentum is dissected and
- Posterior leaf of the omentum is dissected
- Dividing the vagal nerve fibre starts from 5cm proximal to GE junction to 7cm proximal to pylorus.

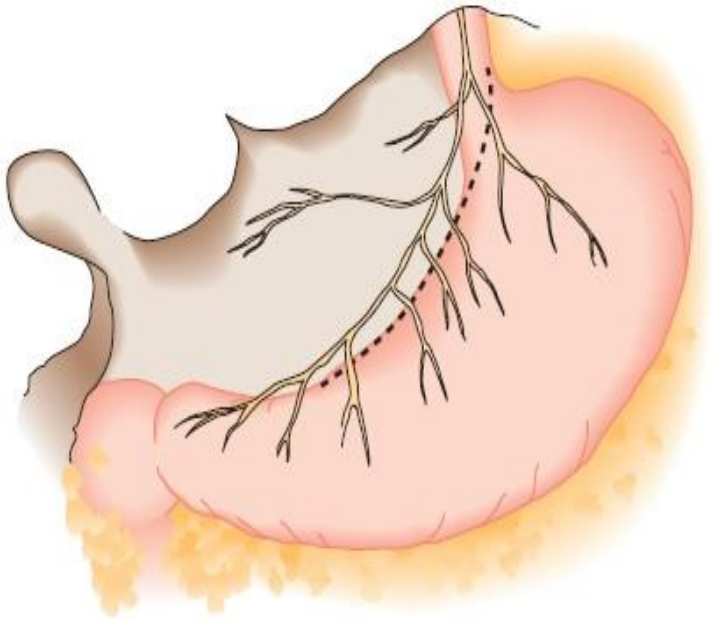

Fig-13: Highly selective vagotomy

DRAINAGE PROCEDURES

PYLOROMYOTOMY

In pyloromyotomy incision made in anterior surface of pyloric canal, its starts from 1 to 2cm proximal to 1cm distal to the pyloric ring. The seromuscular layer is opened without disrupting the mucosa by using fine tip hemostat or knife. Sometimes this area is covered with omental patch to prevent perforation.

PYLOROPLASTY

HEINEKE-MIKULICZ PROCEDURE

This procedure can be performed through the upper abdominal transverse or vertical incision, or laproscopically. Kocher maneuver is may not needed. The pylorus identified and a 3cm longitudinal thick fullness incision made it

starting from distal stomach to proximal duodenum. Incision is closed transversely with a 3-0 seromuscular silk suture without mucosal protrusion through the incision. Omental patch used cover the incision site.

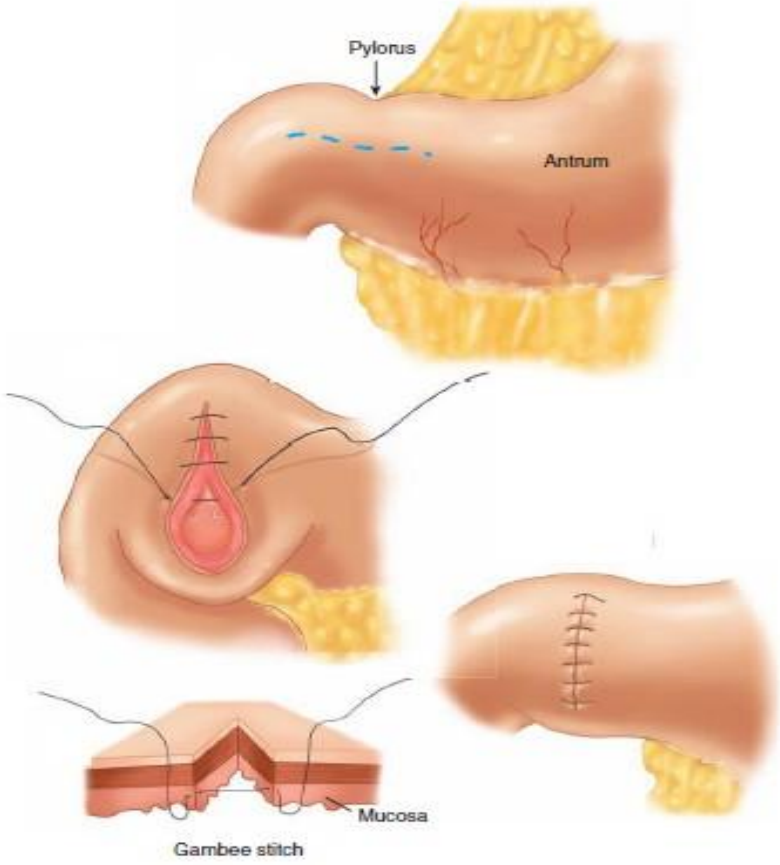

Fig-14: Heineke-mikulicz procedure

FINNEYS PYLOROPLASTY

It need more extensive dissection than Heineke mikulicz pyloroplasty and its need much larger incision than heineke mikulicz pyloroplasty. This Procedure is started with kochers maneuver is needed followed by 10 -12cm longitudinal gastrodunodenotomy is made. Pylorus must be in midpoint of incision and then closed as two layers in side to side gastroduodenostomy, with pylorus at apex. Nasogastric tube placed for decompression of stomach.

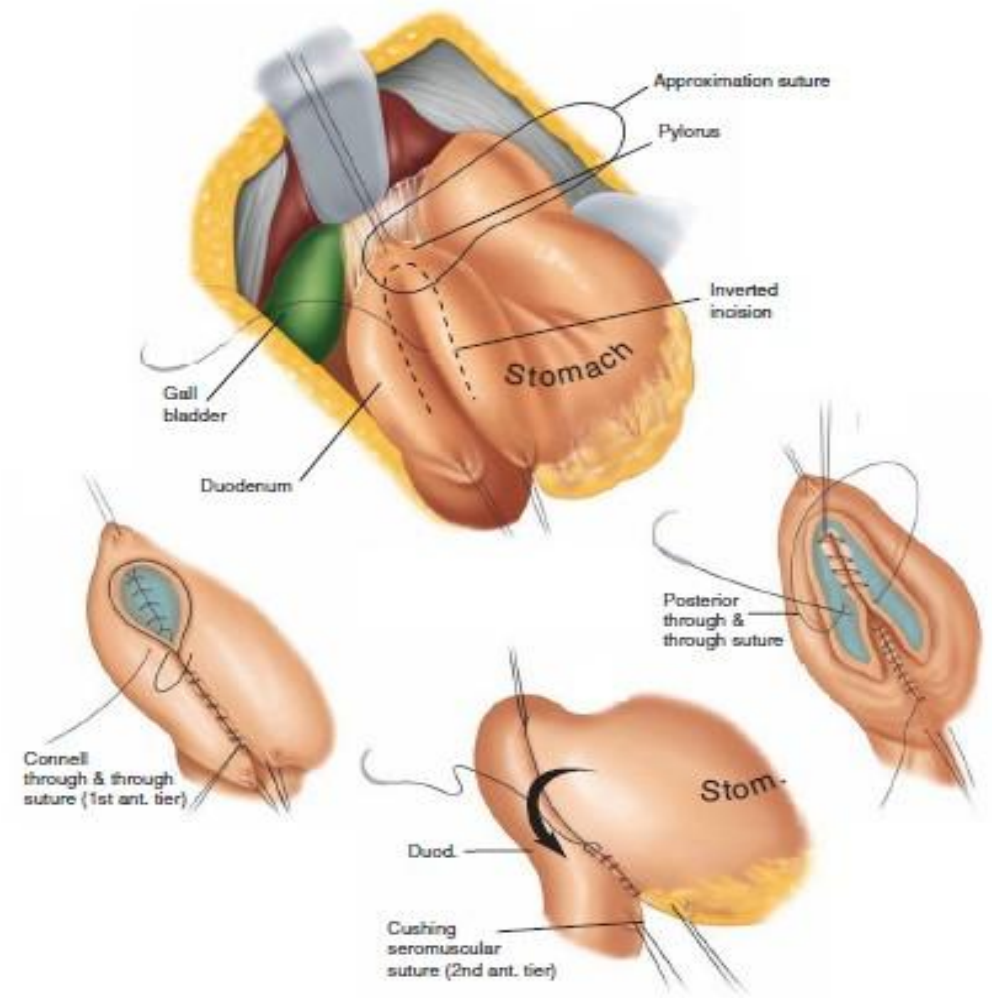

Fig-15: Finneys procedure

JABOULEYS PYLOROPLASTY

This procedure also started with kocher maneuver, 4 to 5cm incision made between the greater curvature side of the distal stomach and the anterior proximal duodenum. Incision is closed in side to side anastomosis in two layers by using silk and vicryl suture. Leak test performed.[5]

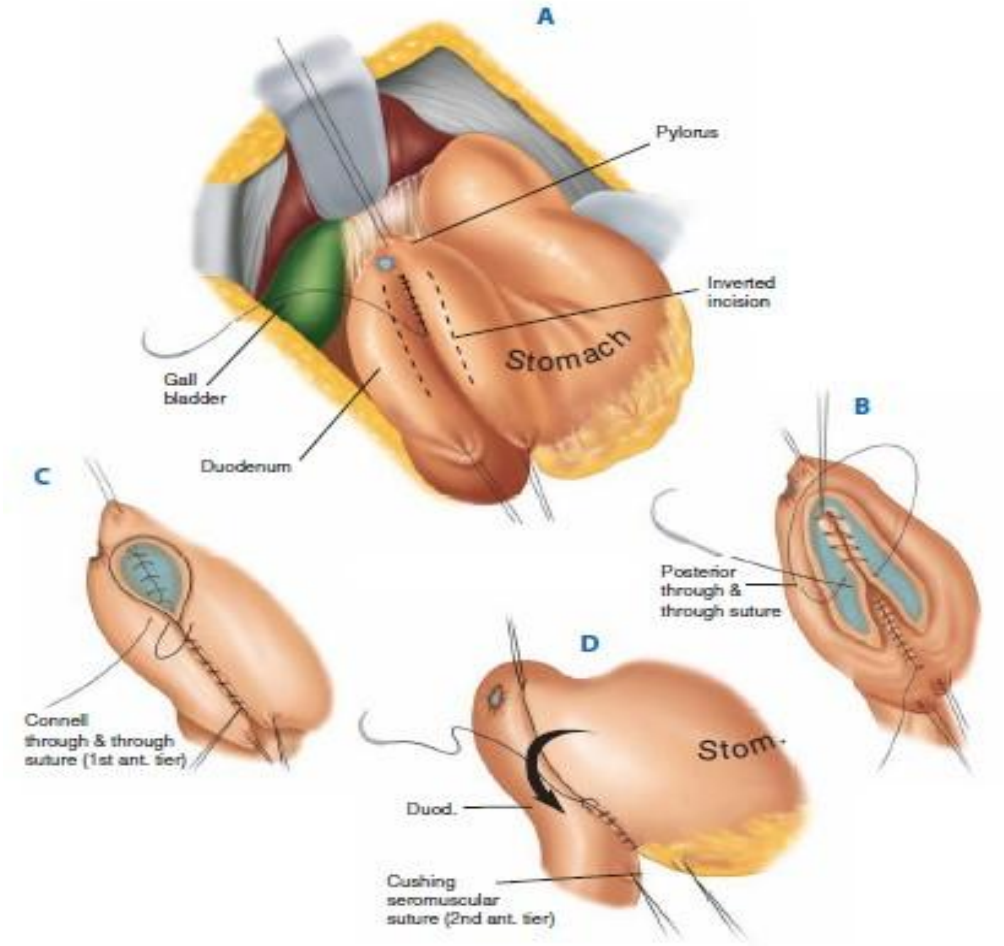

Fig-16: JABOULEYS PROCEDURE

GASTROENTEROSTOMY

In gastroenterostomy, a posterior retrocolic, isoperistalic, no loop and no tension, vertical gastrojejunostomy is made.[5]

GASTRIC OUTLET OBSTRUCTION DUE TO PRIMARY GASTRODUEDENAL TUBERCULOSIS

Primary gastroduodenal tuberculosis can be treated with endoscopic balloon dilation along with antitubercular therapy, but recurrence is more common. Gastrojejunostomy can be done in complete obstruction or failed

endoscopy therapy. Distal gastrectomy is done in patients with multiple tubercular ulcers.

CAUSATIVE INDUCTED STRICTURE

Causative induced stricture is difficult to treatment in endoscopic balloon dilation. Preferred procedure for complete obstruction is gastrojejunostomy or distal gastrectomy with gastrojejunostomy.

POSTOPERATIVE ADHESION

Postoperative adhesion causing gastric outlet obstruction may resolve spontaneously. Laproscopic or open adhesiolysis can be done if not resolve spontaneously. Repeated procedure increases the more adhesions. In suspected that patient may go for develop adhesion again in this patients gastrojejunostomy or gastrojejunostomy can be done.

PSEUDOCYST OF PANCREAS

Pseudocyst of pancreas may resolve spontaneously in only 10% of patients over 4- 6 weeks of time. Indication of treatment are gastric or duodenal or billiary compression or associated complication of bleeding, pancreaticopleural fistula. Endoscopic stenting shows improved outcome. Endscopic ultrasound guided aspiration increase visualisation of cyst, fluid collection, necrosis, and helps to avoid the vascular injury. Operative procedures like cystogastrostomy, Roux en y cystojejunostomy can be done in open or laproscopically. Obstruction due to chronic pancreatitis is treated by vagotomy with gastrojejunostomy.

ADULT ONSET IDIOPATHIC HYPERTROPHIC PYLORIC STENOSIS

AIHPS can be treated with endoscopic balloon dilation or pyloromyotomy with or without pyloroplasty. In some cases gastrectomy with billroth 1 gastroduodenostomy can be done. Laproscopic pyloroplasty is less invasive option. Endoscopic dilation has a high rate of failure and it's providing only temporary relief.

BEZOAR AND FOREIGN BODY

Endoscopic removal of bezoar can be with help of laser lithotripsy or mechanically. Laser lithotripsy is used to breakdown the bezoar and facilitates to easy removal of material. Enzymatic fragmentation is used to removal the trichobezoars. If endoscopic removal failed, gastrotomy may be needed for large bezoars.

Foreign body once cross the upper esophageal sphincter it can easily pass through alimentary tract without causing any harm. Indications for active treatment are

- Failure to progress
- Signs of perforation
- Sharp object unlikely to move on
- Multiple foreign bodies
- Gastrointestinal bleeding

For these patients retained foreign body can be removed by using upper GI scope. Gastrotomy or enterostomy need for removal large number of foreign body or complicated by perforation and intraoperative radiogram is taken to make sure no other foreign body materials in alimentary tract.

BOUVERETS SYNDROME

Patients with bouverets syndrome are elderly and usually associated with multiple comorbitities. For these patients endoscopic extraction can be done with help of laser lithotripsy, extracorporeal shockwave lithotripsy and intracorporeal eletrohydraulic lithotripsy. Endoscopic extraction is alternative to surgery for proximal gallstone obstruction. Main treatment modality for bouverets syndrome is surgery. Surgical options for extracting the stone are enterolithotomy and gastrotomy. Surgical extraction of the stone can be done along with or without cholecystectomy and fistula repair.

WILKIES SYNDROME

Wilkies syndrome sometime treated with correction of aggravating factor like lordosis, plaster cast for spinal injury. Main treatment option for wilkies syndrome is duodenojejunsotomy it will maintain the continuity of alimentary tract.

CROHNS DISEASE

Isolated gastroduodenal crohns disease is treated with infliximab or prednisone. Surgical treatment indicated whenever medical line of management fails or failed endoscopic balloon dilation or massive bleeding or gastric fistula.

Surgical procedure of choice is distal gastrectomy or bypass procedures like gastrojejunostomy.

POLYPS

Polyps are treated with endoscopic excision or snare. If polyps show dysphasia it can be treated with endoscopic mucosal resection.

MALIGNANT GASTRIC OUTLET OBSTRUCTION

CARCINOMA STOMACH

Surgery is the only curative treatment of choice for gastric cancer. Patients with clinically resectable locoregional disease are treated with gastrectomy with adequate lymphadectomy. The goal of curative resection is resection of all tumours with R0 margin and adequate lymphadectomy to acquire the accurate staging. 15 or more lymph node is resected to acquire the adequate staging and it markers of quality of care.[5,6,7,25,31,34]

EARLY GASTRIC CANCER

Early gastric cancer is defined as tumor confined to lamina propria (T1a) or muscularis mucosea (T1b). Early gastric cancer can be treated with endoscopic mucosal resection or endoscopic submucosal resection. Tumor must be removed in enbloc. Indications for endoscopic mucosal resection are

- Well differentiated adenocarcinoma, which is limited to mucosa.
- Less than 2cm and without signs of ulcer
- No nodal involvement

PROCEDURE OF EMR

- Saline injected in submucosal layer and lesion is elevated
- Mount of lesion is pulled up with forceps
- Snare placed at base of lesion
- Electrosurgical current applied through the snare to resect the mucosal lesion.

ESD (Endoscopic submucosal dissection)

ESD is indicated in patients meet the criteria for

- Intramucosal tumour with well differentiated lesion, and tumour size smaller than 30mm regardless of ulcer status.
- Tumour Limited to submucosal invasion (less than 500 micrometer) with smaller than 3cm and without ulcer.

Studies shows that ESD is higher rate of complete resection compared to the endoscopic mucosal resection. Early gastric cancer with size more than 2cm with ulceration or with submucosal invasion or histologically poorly differentiated carcinoma is treated with gastrectomy and lymphadectomy.

STAGE II AND III DISEASE

SURGERY

For stage II and III disease surgical resection of primary tumor with adequate lymphadectomy is needed to cure the disease. Aims of surgical resection are to resect the primary tumour with R0 margin and dissection of

regional lymph node and peritoneal surface in order to reduce the recurrence and staging and proper anastomosis techniques to restore the gastrointestinal continuity.[5,6,7,25,31,34]

Extent of resection for distal gastric cancer

Aim of gastrectomy to achieve gross and microscopically negative status of surgical margins. It can be achieved by resection of primary tumour with 5 cm grossly negative for tumor in proximally and distal margin of 2cm. Intraoperatively frozen section should be done to ensure the R0 resection in proximal and distal margins, when done in curative intention.

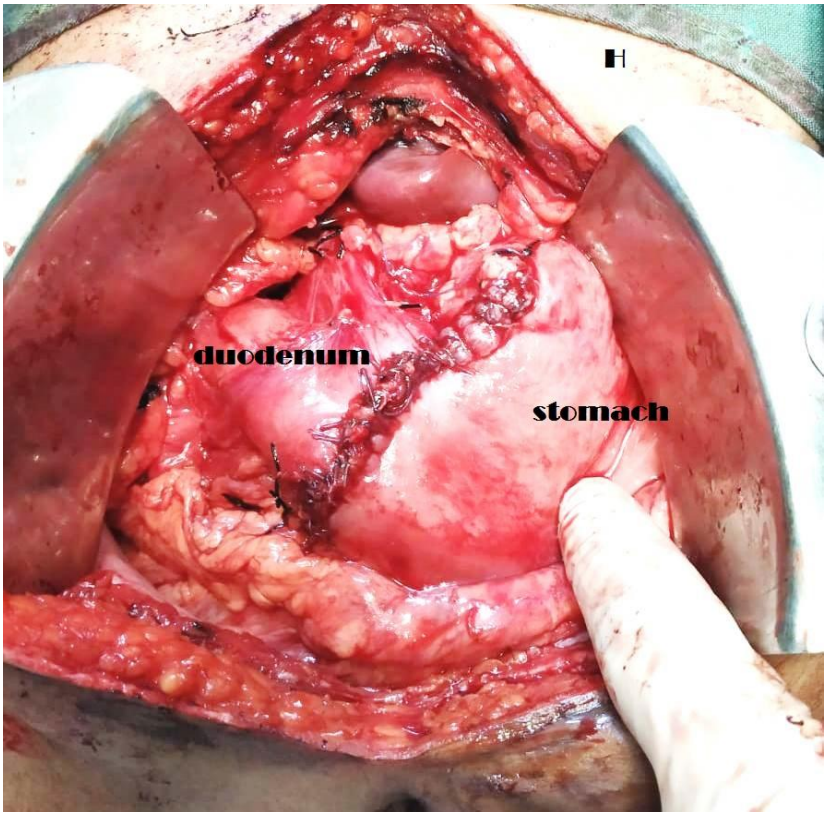

Fig -17; BILLROTH I GASTRODUODENAL ANASTOMOSIS

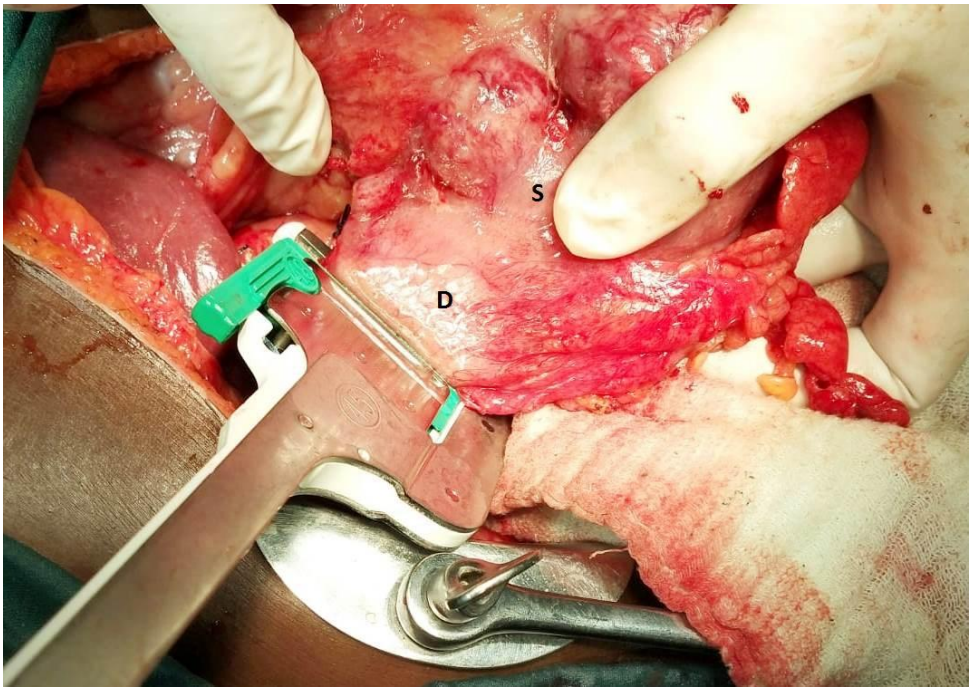

FIG-18: LINEAR STAPLER USED TO DIVIED THE DUODENUM.

EXTENT OF LYMPHADECTOMY

Number of pathologically positive lymph nodes shows prognostic significance. For accurate staging of gastric carcinoma needs removal and pathological analysis of at least 15 lymph nodes. But current AJCC staging system is emphasis that more than 16 lymph nodes needed for assign the pathological N stage.

D1 lymphadectomy indicate that removal that removal of perigastric lymph nodes

D2 lymphadectomy indicates remaoval of perigastric lymph node and celiac axis nodes with or without splenectomy.

D3 lympahdectomy indicates removal of D1 and D2 level nodes with removal of periaortic node

ADJUVANT CHEMOTHERAPY

ADJUVANT INTRAPERITONEAL CHEMOTHERAPY

Adjuvant intraperitoneal chemotherapy is used to prevent peritoneal recurrence in curative resection planned patients. Peritoneal recurrence have very poor prognosis even in patients underwent curative resection. Hyperthermic intraperitoneal chemotherapy (HIPEC), normothermic intra peritoneal chemotherapy (NIIC) is mode of delivery of chemotherapy. Use of Hyperthermic chemotherapy increases the microscopic tumour cytotoxicity. Common used regimen is 5FU, mitomycin C, and Cisplatin for intra peritoneal chemotherapy.[25]

ADJUVANT CHEMORADIATION

Inter group trial done in patients who underwent surgery alone and surgery followed by 5 FU based regimen in nonmetastatic adenocarcinoma. Regimen used in this trial is 5FU, leucovrin and 45Gy in 25 fractions. It shows significant prognostic improvement.[25,42]

PERIOPERATIVE AND NEOADJUVANT CHEMOTHERAPY

Perioperative chemotherapy has benefits of increase the R0 resection and micrometastasis treated in early. It's useful in patients with local advanced tumours. Currently pontentital regimen is FLOT it contains docetaxel, oxaliplatin, leucovorin and 5FU. It's administrated in four preoperative and four postoperative 2 week cycles.[25,41,42]

RADIATION THERAPY

Radiation therapy used as palliative treatment in advanced gastric carcinoma with bleeding or pain due to local infiltration. Radiotherapy also used in intraoperative to irradiate the stomach bed. Intra operative radiation therapy is delivered as a single large fraction at dose of 10- 35Gy to the stomach bed with protecting surrounding normal tissues.

TARGETED THERAPY

EPIDERMAL GROWTH FACTOR SUPER FAMILY; MONOCLONAL ANTIBODIES

Trastuzumab is used in patients with overexpression or amplification of HER2. Overexpression of HER2 occurs in 20 % of patients with gastric carcinoma and it more common in intestinal type of gastric cancer.

VASCULAR ENDOTHELIAL GROWTH FACTOR;

Bevacizumab is humanized monoclonal antibody. Its binds to vascular EGFA factor ligand.

Sunitinib is an oral tyrosine kinase inhibitor and also inhibits VEGFR1, VEGFR2, and VEGFR3, platelet derived growth factor receptors.

PROTEIN KINASE; Everolimus is an oral protein kinase inhibitor of rapamycin. It used in refractory metastatic gastric cancer.[25]

POLYMERASE INIBITORS; Olaparib is oral inhibitor of poly (ADP-ribose) polymerase

GASTRIC LYMPHOMA

Radical subtotal gastrectomy is done in primary gastric lymphoma involves the antrum. If it involves the proximal stomach the radical total gastrectomy is the treatment of choice. Anti H.pylori treatment also needed for early disease. Chemotherapy or radiotherapy or both is used as adjuvant therapy when lymph node positive for lymphoma.

GASTRIC OUTLET OBSTRUCTION DUE TO DUODENAL MALIGNANICES

Duodenal malignancies can be treated with pancreaticoduodenectomies when cure is possible. Segmental duodenectomy is possible whenever tumour is located in distal part of duodenum. Palliative procedure is performed for local extensive or Metastatic disease of duodenal malignancy. Palliative procedures include gastrojejunostomy and cholecystojejunostomy.

PANCREATIC MALIGNANCIES

Whenever curative resection is possible whipples pancreaticoduodenectomy can be done. Inoperable patients are treated with gastrojejunostomy to relieve gastric outlet obstruction, and cholecytojejunostomy to relieve the obstructive jaundice. inoperable cases have very poor prognosis so minimal invasive procedures like endoscopic duodenal stenting or ERCP and stenting can be done. Radiation therapy useful in these patients but it takes few weeks to relieve the gastric outlet obstruction.

MATERIALS AND METHOD

This study contacted in tirunelveli medical college and hospital, tirunelveli from January 2018 to June 2019. For this study 79 patients included who admitted with symptoms of gastric outlet obstruction

INCLUSION CRITERIA

- Age more than 12 yrs
- Patients presenting with symptoms of gastric outlet obstruction
- Patients willing for investigation and treatment

EXCLUSION CRITERIA

- Patients age less than 12 years
- Patients not willing for endoscopic examination
- Patients not willing for proposed surgery

Patients are admitted in general surgical wards in TVMCH. The comprehensive background analysis of these cases with regarding to presenting complaints, clinical presentations, routine and specific investigation to confirm the diagnosis, preoperative care and postoperative care and complications of surgical procedures are done.

Detailed history noted about presenting complaints, duration of complaints, symptoms of metabolic abnormalities, previous history regarding peptic ulcer, comorbidities, and personal history like smoking, alcohol, and bowel and bladder habits.

The comprehensive analysis of clinical examination done and its included the signs of dehydration, pallor, icterus, visible gastric peristalsis, palpable mass, succession splash, organomegaly like hepatomegaly or splenomegaly, shifting dullness or fluid thrill. Associated co-morbidities are treated before planned procedure with physician's opinion obtained wherever needed.

Complete blood count, liver function test, renal function test, serum electrolytes, coagulation profile, chest x-ray done to rule out metastasis, respiratory problems, cardiac status, blood grouping are done as part of routine workup for surgery. To confirm the diagnosis investigations like upper GI scope with biopsy from suspected lesion, USG abdomen, and CECT abdomen and chest are performed.

Patients with any one of the following criteria are included in study of gastric outlet obstruction.

- Non bilious, projectile, undigested food particle in the vomiting
- Palpable mass or hypertrophy of stomach
- Visible gastric peristalsis
- Saline load test; residual volume more than 400ml
- OGD scopy findings suggestive of GOO
- Intraoperative finding of GOO

PREOPERATIVE MANAGEMENT

It is crucial part of patient care and postoperative outcome. Preoperative care includes dehydration correction; normalize the metabolic alkalosis, correction of anaemia, H2 blockers, protein rich liquid diet, and antibiotics if needed. Nasogastric tube intubated; stomach wash given two times a day for minimum of three days it improve the stasis gastritis, decrease the mucosal edema, and improve the motor function of stomach. According to reports of special investigation and intraoperative findings, definitive procedure was undertaken.

For all patients who underwent for surgical procedures, general anaesthesia was given.

POSTOPERATIVE MANAGEMENT

After procedure patients are kept in strict observation. To decompress the stomach Ryles tube aspiration done, patients kept in nil per oral until bowel sounds appear. Intravenous fluid given for till bowel function recover, followed that liquid diet started. Few days' later frequent small solid diet is started. According to institution protocol the antibiotics are given. Continues monitoring of body temperature, pulse rate, respiratory rate, blood pressure and urine out are done.

All the details are registered in proforma and master chart is formed according to the details in proforma. Data analyzed and discussion carried out and end point of results made.

OBSERVATIONS AND RESULTS

AETIOLOGICAL DISTRIBUTION

Out of 79 cases studied, 71 are due to malignant causes out of which 69 are due to Carcinoma stomach, 2 had pancreatic malignancies and 7 cases due to cicatrizing duodenal ulcer and one cases of pseudocyst.

TABLE NO:1 DISTRIBUTION OF AETIOLOGICAL CAUSES

	FREQUENCY	PERCENT
DUODENAL ULCER	7	8.9
PSEUDOCYST OF PANCREAS	1	1.3
CA STOMACH	69	87.3
PERIAMPULLARY CARCINOMA	1	1.3
CA PANCREAS	1	1.3
TOTAL	79	100%

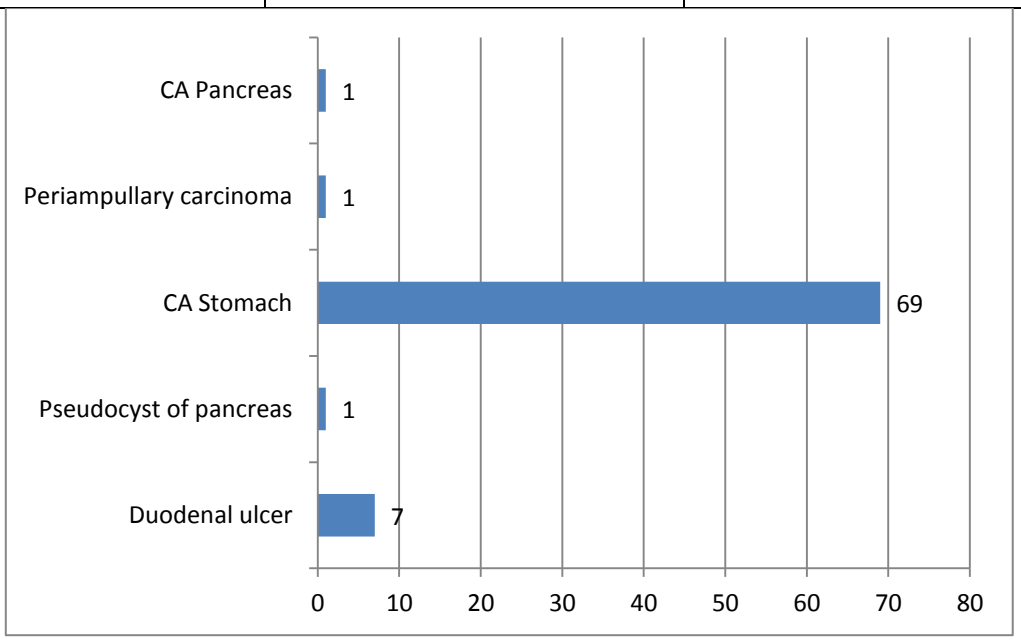

CHART NO 1: BAR CHART OF DISTRIBUTION OF AETIOLOGICAL CAUSES

AGE DISTRIBUTION

In our study most common age group affected by gastric outlet obstruction is 6th decade followed that 5th decade. Malignant gastric outlet obstruction is common in 5 to 6th decades. Benign gastric outlet obstruction is more common in younger age group.

		age_group				Total
		31-40	41-50	51-60	>61	
type	Benign	2	3	1	2	8
	Malignant	7	12	20	32	71
Total		9	15	21	34	79

TABLE NO 2 ; distribution of age in gastric outlet obstruction

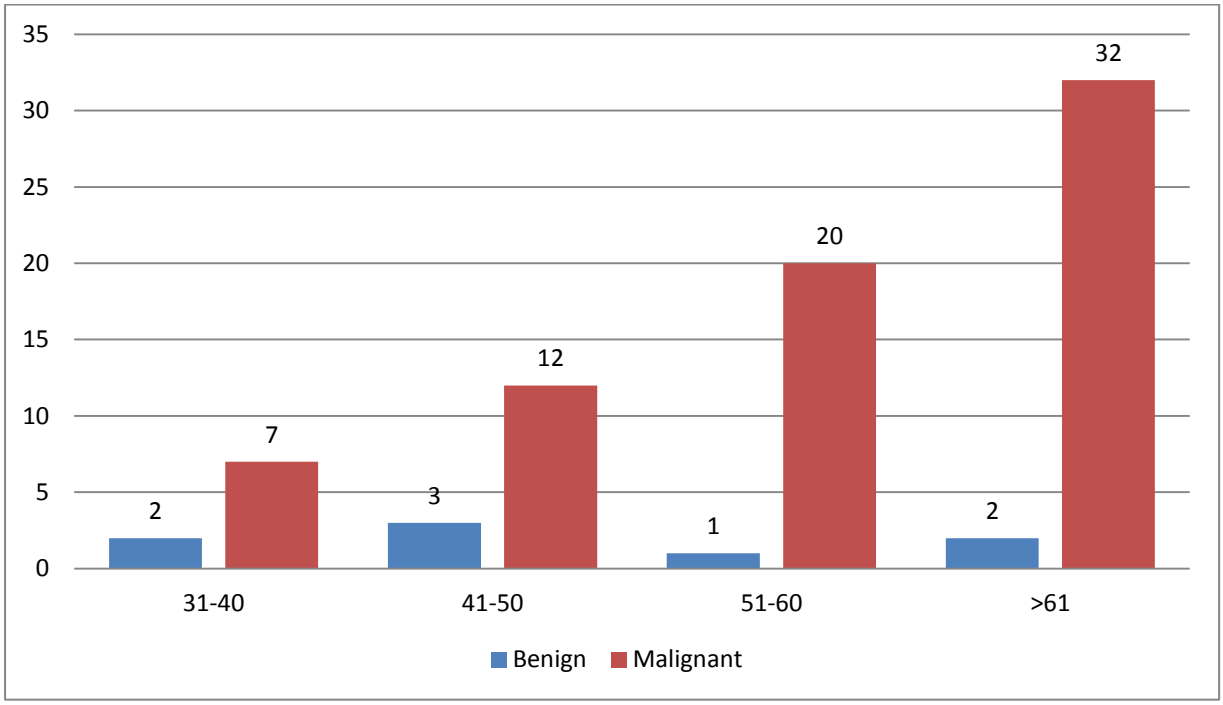

CHART NO ;2 DISTRIBUTION OF AGE IN GASTRIC OUTLET OBSTRUCTION

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PERSONAL HISTORY

SMOKING

In our study 49% of the patients are chronic smokers and 51% of the patients are non-smoker.

ALCOHOL

In this study 46% of patients are have history of alcohol intake.

DIET

Most of the patients are taking spicy diet with irregular dietary habits

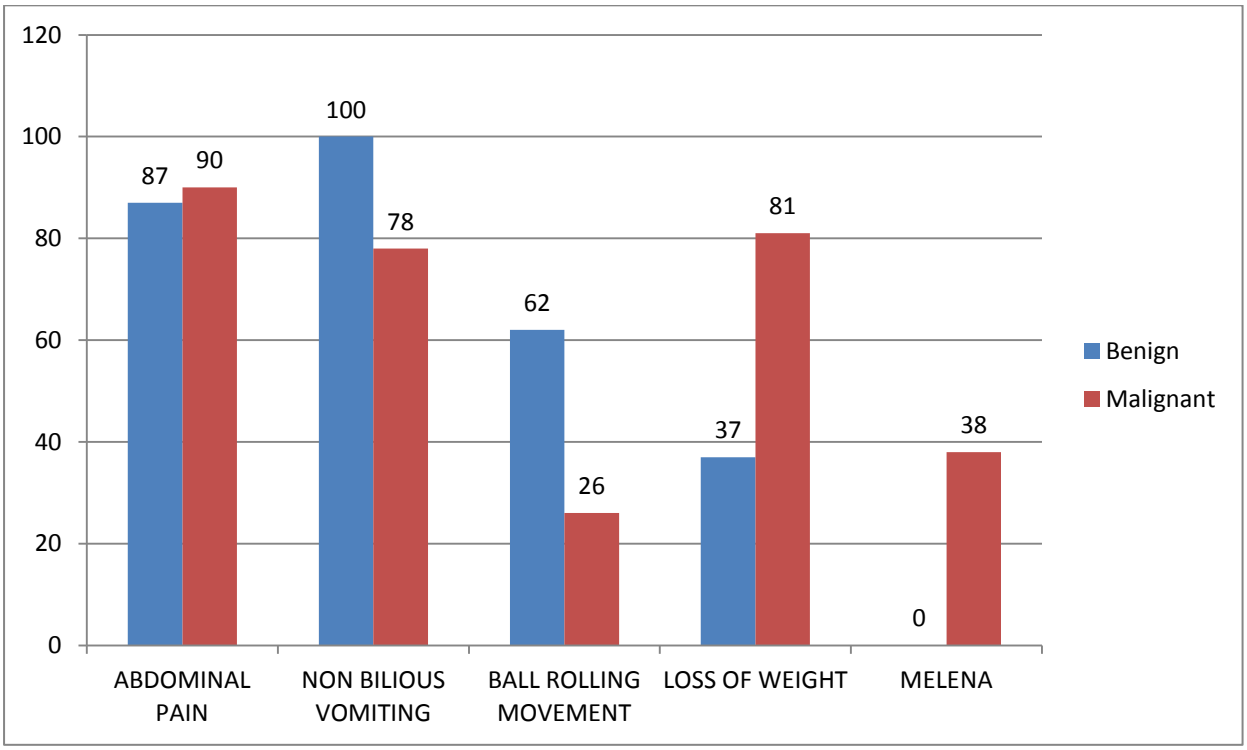
SYMPTOMS OF GASTRIC OUTLET OBSTRUCTION

In malignant GOO, abdominal pain (90%) is the most common symptom and weight loss (81%) is next common symptom. In Benign GOO, nonbilious vomiting (100%) is most common symptom and next common symptom is abdominal pain (87%).

TABLE NO:3 symptoms of the patients presenting as GOO

CAUSES	MALIGNANT GOO		BENIGN GOO	
	count	percentage	Count	percentage
Abdominal pain	64	90%	7	87%
Non bilious vomiting	56	78%	8	100%
Ball rolling movement	19	26%	5	62%
Weight loss	58	81%	3	37%
melena	27	38%	0	0

Chart no 3; symptoms of the patients presenting as GOO (percentage)



SIGNS OF GASTRIC OUTLET OBSTRUCTION

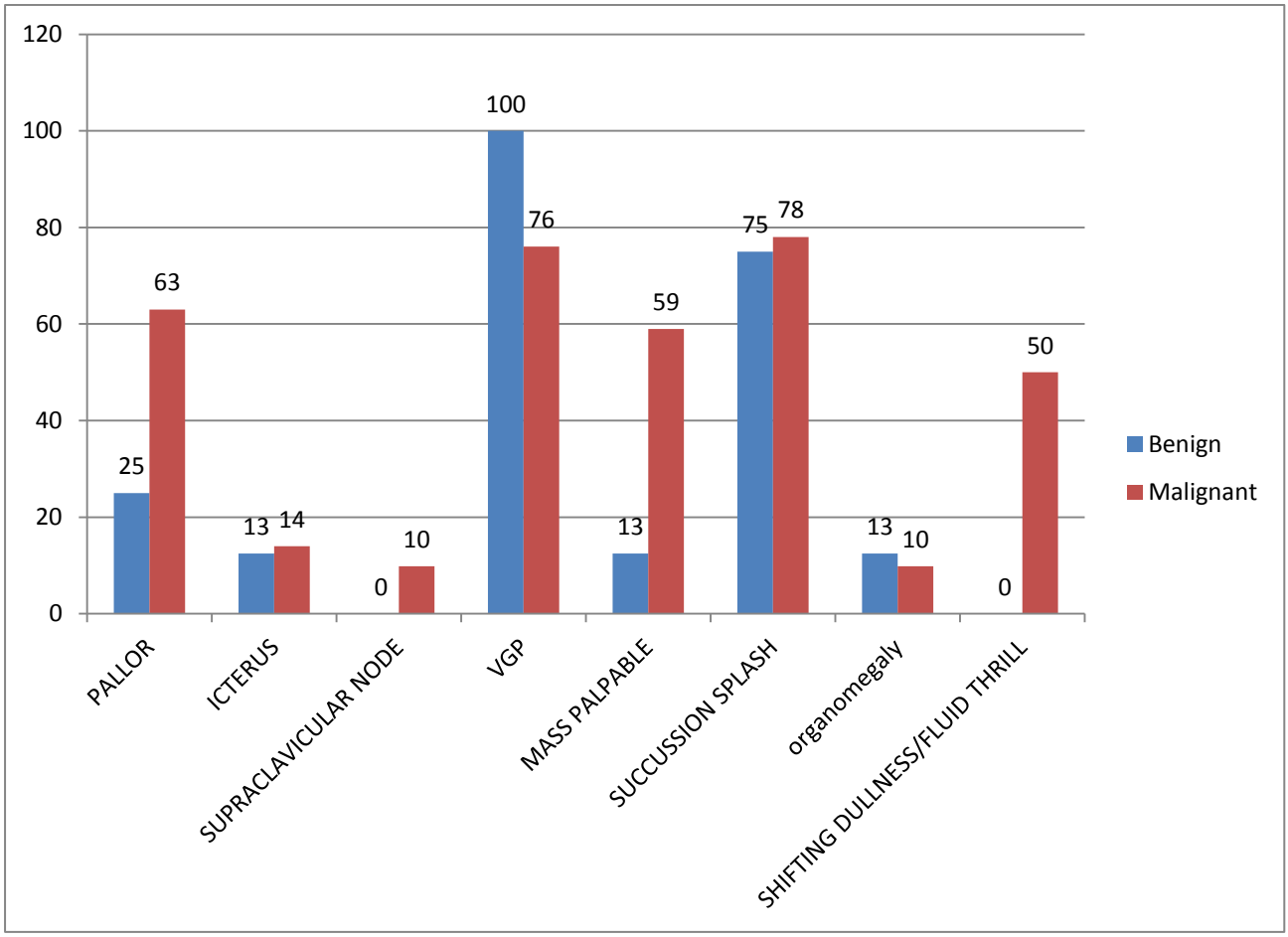
In Malignant gastric outlet obstruction, commonest clinical finding is succusion splash and it account for 78%, next common clinical signs is visible gastric peristalsis (76%).

In benign gastric outlet obstruction, common physical examination finding is visible gastric peristalsis (100%), it seen in all patients. Next common finding is succussion splash (75%).

TABLE NO: 4 SIGN IN THE PATIENT PRESENTING WITH GOO

	BENIGN GOO		**MALIGNANT GOO**	
	COUNT	**PERCENTAGE**	**COUNT**	**PRECENTAGE**
PALLOR	2	25%	45	63%
ICTERUS	1	12.5%	10	14%
SUPRACLAVICULAR NODE	0	0	7	9.8%
VGP	8	100%	54	76%
PALPABLE MASS	1	12.5%	42	59%
SUCCUSION SPLASH	6	75%	56	78%
ORGANOMEGALY	1	12.5%	7	9.8%
SHIFTING DULLNESS	0	0	36	50%

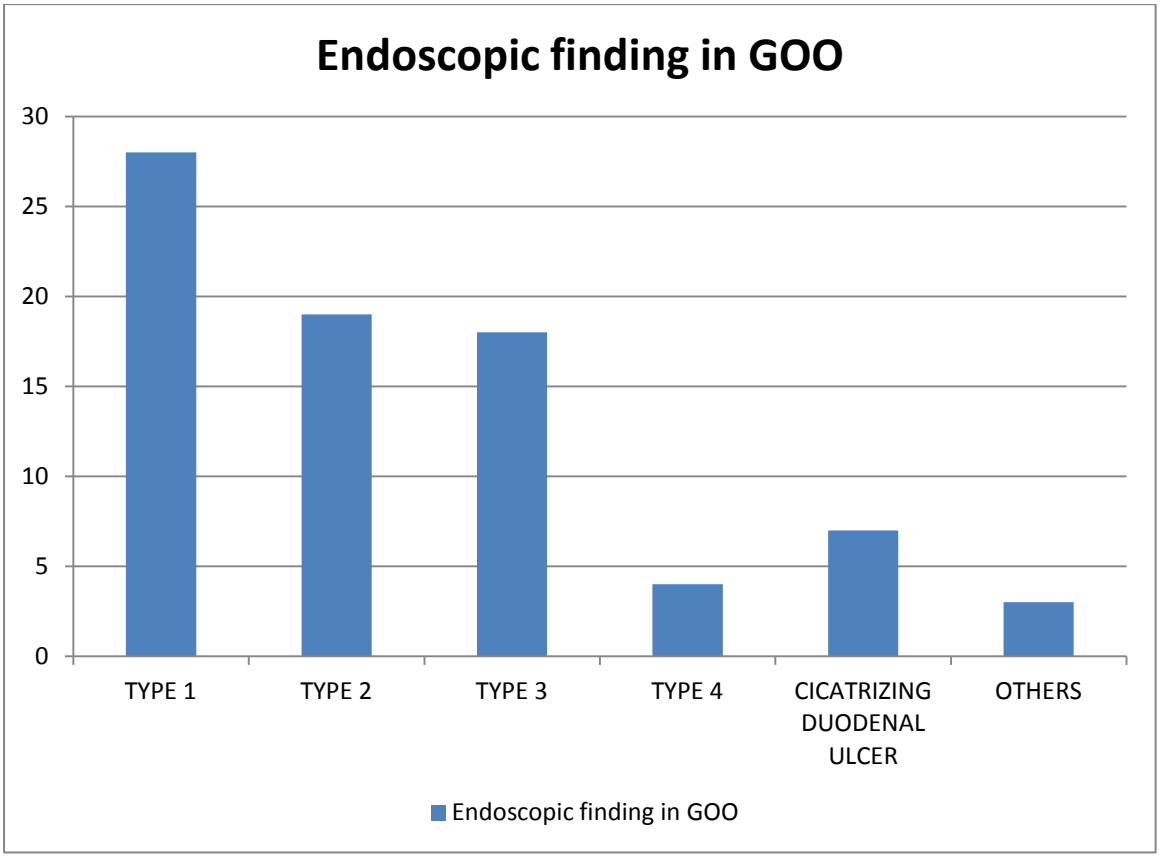
Bar chart 4 of sign of gastric outlet obstruction



ENDOSCOPIC FINDING IN GASTRIC OUTLET OBSTRUCTION

Upper GI endoscope is done for all case with signs and symptoms of gastric outlet obstruction. In gastric carcinoma, BORRMANNS classification used to describe the lesion. In gastric carcinoma, BORRMANNS classification type 1 lesion present in 28 cases (40%), type 2 present in 19 cases (28%), type 3 lesions present in 18 cases (26%) and remaining are type 4 lesions. In chronic duodenal ulcer, upper GI endoscopic finding is chronic cicatrizing duodenal ulcer.

Chart No.5 Endoscopic finding in GOO



CECT ABDOMEN

CECT abdomen is done for all patients with gastric outlet obstruction. It used in preoperative staging workup in gastric carcinoma. Perigastric node involved in most of the gastric carcinoma patients. Liver metastasis or ascites found in 21 cases.

HISTOLOGICAL STAGING OF GASTRIC CARCINOMA

In this study gastric carcinoma is graded according to degree of differentiation. Out of 69 cases 47 patients had poorly differentiated adenocarcinoma. Moderately differentiated carcinoma noted in 14 patients. 8 patients had well differentiated adenocarcinoma.

TYPES OF SURGICAL PROCEDURES PERFORMED IN THIS STUDY

Palliative anterior gastrojejunostomy with or without jejunojeunostomy is done for 32 patients. Palliative gastrojejunostomy with hepaticojejunostomy is done for a patient with obstructive jaundice and gastric carcinoma. Feeding jejunostomy performed for 9 patients who had diffuse infiltration to proximal stomach and infiltration to surrounding structures.

Palliative subtotal gastrectomy with Billroth II reconstruction is done in 21 cases of Carcinoma stomach with gastric outlet obstruction and melena but curative procedure cannot be done. It account for 29% of total cases. Triple bypass is performed in 2 patients who had gastric outlet obstruction due to pancreatic malignancy.

Total gastrectomy performed for two patients had tumour involvement in body of stomach and curable disease. Subtotal gastrectomy with D2 lymphadenectomy done for 15 cases (21%) in curative intention.

For cicatrizing duodenal ulcer the Truncal vagotomy and posterior gastrojejunostomy is performed. Cystogastrostomy is performed for a patient with pseudocyst of pancreas causing gastric outlet obstruction.

TABLE NO 5 SURGICAL PROCEDURES PERFORMED IN THIS STUDY

PROCEDURE	NUMBER OF PATIENT	PERCENTAGE
1.CHRONIC DUODENAL ULCER		
– TRUNCAL VAGOTOMY WITH GASTROJEJUNOSTOMY	**7**	100%
2.PSEUDOCYST OF PANCREAS		
– CYSTOGASTROSTOMY	**1**	100%
1.CARCINOMA STOMACH		
➢ FEEDING JEJUNOSTOMY	9	13%
➢ ANTERIOR GASTROJEJUNOSTOMY	5	7%
➢ ANTERIOR GASTROJEJUNOSTOMY WITH JEJUNOJEJUNOSTOMY	17	24%
➢ ANTERIOR GASTROJEJUNOSTOMY WITH HEPATICOJEJUNOSTOMY	1	1.4%
➢ PALLITIVE SUBTOTAL GASTRECTOMY WITH GASTROJEJUNOSTOMY	21	30%
➢ SUBTOTAL GASTRECTOMY WITH D2 LYMPHADECTOMY	14	20%
➢ TOTAL GASTRECTOMY	2	3%
2.PANCREATIC MALIGNANCIES		
➢ TRIPLE BYPASS	2	100%

POSTOPERATIVE COMPLICATION

Postoperatively all patients are kept in nasogastric tube and nil per oral till bowel sound heards or Ryle tube aspiration less than 100ml/day. Oral fluids started in between POD 4 to POD 6 for most of the patients. In this study most common complication encountered is surgical site infection in 6 (15%) patients and SSI is treated with regular dressing and higher antibiotics. Delayed gastric emptying is occurred in four patients. One patient had low output enterocutaneous fistula and patient is treated with conservatively. In this study mortality rate is approximately 6%.

TABLE NO 6 Distribution of Postoperative complication

	COMPLICATION	FREQUENCY
1	Surgical site infection	6
2	Postop pyrexia	3
3	pneumonia	3
4	Delayed gastric emptying	4
5	Postop diarrhoea	2
6	Feeding jejunostomy leak	2
7	Enterocutaneous fistula	1

DISCUSSION AND ANALYSIS

This discussion is based on analysis and observation derived from 79 patients, who admitted in general surgical wards, Tirunelveli medical college and hospital from January 2018 to June 2019. This study includes variables like etiological cause, symptoms and sign of GOO, investigations, management of gastric outlet. Of 79 patients,

Malignant gastric outlet obstruction secondary to gastric carcinoma is 69

Benign gastric outlet obstruction secondary to cicatrized duodenal ulcer is 7.

The most common cause of malignant gastric outlet obstruction is gastric carcinoma; next etiological cause is pancreatic malignancices. The most common cause of benign gastric outlet obstruction is cicatrized duodenal ulcer. It clearly shows that incidences of malignant gastric outlet obstruction is especially gastric carcinoma increased and incidences of cicatrized duodenal ulcer is decreasing trend due to last few decades evolution of peptic ulcer disease.

In this study malignant gastric outlet obstruction, patients were in sixth decade. Age group of 60-69 (43%) years is shown more incidence of malignancy. The average age of malignant gastric outlet obstruction is 56.6

year. In sushruta et al[43] series, average age is 62.5 years and age group is 60-69 years (38%). Incidence of malignant GOO is increases with older age.

In this study benign gastric outlet obstruction, patients were in fourth decades (40-49 years) and Average age of presentation is 50 years and it's comparable with Fisher et al.[44]

In this study 49% of patients are chronic smokers and 46% of patients are alcoholics. Most of patients are low socioeconomic status and they gave history of irregular diet intake and it's also comparable with the series of Donald D Kozoll et al.[45]

Most common symptom of gastric outlet obstruction is abdominal pain (90%) and vomiting 81% which is comparable to Yogiram and Chowdhary et al[46] series showed abdominal pain (87%), vomiting(81%) and it's also shown in Ranka Kishitiz et al[47], Kumar et al[48]. 77% patients had weight loss in this series and its comparable with Ranka Kishitiz et al and Jaka et al[49] and 59.5 % in Donald D Kozoll et al. Melena seen in 34% of patients in this study and it comparable with Ranka Kshitiz et al series showed 35% of patients had melena. Loss of weight is due to abdominal pain increases after food intake and long standing gastric outlet obstruction.

In malignant gastric outlet obstruction abdominal pain (90%) is leading complaint. Other symptoms included vomiting (78%), weight loss (81%), and melena in 38% of patients.

Non bilious vomiting (100%) and abdominal pain (87%) are most common symptom in benign gastric outlet obstruction due to cicatrized duodenal ulcer. Abdominal pain, vomiting are the presenting symptom of pseudocyst of pancreas.

In this series succussion splash (78%) and visible gastric peristalsis (78%) is most common sign and which is comparable with Jaka et al its shows succession splash in 78.3% of patients and Harold Ellis[50] observed that 64% of patients had succession splash. Visible gastric peristalsis present in 78% of patients and which is comparable with Yogiram and Chowdhary VGP present in 74% of patients.

Dehydration was present in 55% of patients in this series and same results observed in Jaka et al and Kumar et al.

Pallor was observed in 59% of patients and which is comparable with Kumar et al. In this series icterus seen in 14% of patients of malignant gastric outlet obstruction and it`s due to pancreatic malignancies, metastasis to periportal region or liver metastasis.

Pancreatic malignancy is present in 2 patients and they had jaundice, abdominal pain, vomiting, pruritus, epigastic mass, visible gastric peristalsis, shifting dullness.

In this series Blood grouping and cicatrized duodenal ulcer or gastric cancer has no correlation.In this series histological grading of gastric carcinoma showed poorly differentiated adenocarcinoma in 68% of patients with gastric carcinoma.

In the current study, truncal vagotomy with gastrojejunostomy done in 100% of patients with cicatrized duodenal ulcer. For a case of pseudocyst of pancreas, cystogastrosotmy was done.

In gastric carcinoma, 9 of patients (13%) underwent feeding jejunostomy and 5 patients (7%) underwent anterior gastrojejunostomy and anterior gastrojejunostomy with jejunojeunostomy was done in 17 patients (24%). 21 patients (30%) are had palliative subtotal gastrectomy with gastrojejunostomy and 14 patients (20%) underwent curative procedure subtotal gastrectomy with D2 lymphadectomy. Total gastrectomy with Roux en y anastomosis was performed in two patients. One patient with jaundice and GOO underwent anterior gastrojejunostomy with hepaticojejunostomy.

Two cases of pancreatic malignancy with GOO was had advanced inoperable disease and they are underwent triple bypass procedure.

All 79 patients are underwent laparotomy and postoperatively kept in nil per oral till bowel sound starts or ryles tube aspiration decreases grossly. Oral fluid started from 4^{th} POD to 6^{th} for most of the patients.

In this series 6 patients had surgical site infection and they are treated with frequent changing of dressing and antibiotics changed according pus culture report. 3 patients had Postop pyrexia and 3 patients had pneumonia is observed and treated with chest physiotherapy and antibiotics. One patients developed enterocutanous fistula with SSI and patient was treated with nil per oral, TPN supplement, and antibiotics.

In this series mortality rate was 6% of patients with gastric outlet obstruction.

CONCLUSION

This study used to determine the aetiology of gastric outlet obstruction in our geographic area. This study is based on smaller of patients, with limited follow up; arriving to final conclusions is difficult. However some conclusion are arrived from this series are as follows

1. In adults presenting with gastric outlet obstruction, must be treated as malignant disease until or otherwise proven. Because commonest cause of gastric outlet obstruction in our region is distal gastric carcinoma and its account for 87%.
2. Chronic cicatrized duodenal ulcer are decreasing trend, because better treatment available for H.pylori eradication and use of proton pump inhibitors and H2 blockers for peptic ulcer disease it prevent development chronic Sequalea of disease and change in dietary habits.
3. Diagnosis of gastric outlet obstruction can be made by clinical examination in most of cases.
4. Early recognition of symptoms and early diagnosis and early intervention by recent treatment modalities helps in achieve better prognosis.
5. All suspected cases of gastric outlet obstruction, should undergo upper GI endoscopy examination and biopsy to confirm the diagnosis.
6. Patient with advanced gastric carcinoma, newer minimally invasive modalities can be used to decrease the morbidities

7. Patients presenting with alarm signs of recent weight loss, dysphagia, evidence of GI bleeding, anaemia, and family history of gastric malignancy are should undergo upper endoscopy screening.

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A CLINICO PATHOLOGICAL STUDY OF GASTRIC OUTLET OBSTRUCTION AND ITS SURGICAL OUTCOME

PATIENT INFORMATION SHEET

Name: **Unit:**

Age: **Sex:** **Ip no:** **Address:**

DOA: **DOS:** **DOD/DOE:**

DIAGNOSIS :

Malignant/Benign

HISTORY:

Non bilious vomiting :

Abdominal pain :

Abdominal distension :

Loss of appetite :

Loss of weight :

Ball rolling movement :

Melena :

Hematemesis :

PAST H/O:

Peptic ulcer :

Corrosive poisoning:

FAMILY H/O:

PERSONAL H/O:

GENERAL EXAMINATION

- Anemic
- Icterus
- Dehydration
- Pedal edema

VITAL:

Pulse rate -

Blood pressure -

SYSTEMIC EXAMINATION:

Visible mass/Epigastric fullness:

Visible gastric peristalsis:

Abdominal mass:

Succussion splash :

Shifting dullness :

Heptomegaly :

Ausculto-percussion:

Left supraclavicular fossa: Other:

INVESTIGATION:

BLOOD GROUPING&TYPING:

INVESTIGATION	
HEMOGLOBULIN	
RBS	
UREA	
CREATININE	
TOTAL BILIRUBIN	
DIRECT	
INDIRECT	
ALP	
SR.ALBUMIN	
SERUM SODIUM	
SERUM POTASSIUM	

UGI SCOPY :

Findings-

Borrmanns classification-

Biopsy - taken or not taken

Biopsy report:

PROCEDURE DONE:

Intraop findings-

CECT ABDOMEN:

Nodal station-

POSTOPERATIVE COMPLICATION:

COMPLICATION	**POD**	**MANAGEMENT**

Follow up

நோயாளிகளுக்கு அறிவிப்பு மற்றும் ஒப்புதல் படிவம்
(மருத்துவ ஆய்வில் பங்கேற்பதற்கு)

ஆய்வு செய்யப்படும் தலைப்பு:
பங்கு பெறுவரின் பெயர்:
பங்கு பெறுவரின் வயது:

		பங்கு பெறுவர் இதனை ✓ குறிக்கவும்
1.	நான் மேலே குறிப்பிட்டுள்ள மருத்துவ ஆய்வின் விவரங்களை படித்து புரிந்து கொண்டேன். என்னுடைய சந்தேகங்களை கேட்கவும், அதற்கான தகுந்த விளக்கங்களை பெறவும் வாய்ப்பளிக்கப்பட்டுள்ளது என அறிந்து கொண்டேன்.	☐
2.	நான் இவ்வாய்வில் தன்னிச்சையாக தான் பங்கேற்கிறேன். எந்த காரணத்தினாலோ எந்த கட்டத்திலும், எந்த சட்ட சிக்கலுக்கும் உட்படாமல் நான் இவ்வாய்வில் இருந்து விலகி கொள்ளலாம் என்றும் அறிந்து கொண்டேன்.	☐
3.	இந்த ஆய்வு சம்பந்தமாகவோ, இதை சார்ந்து மேலும் ஆய்வு மேற்காள்ளும் போதும் இந்த ஆய்வில் பங்குபெறும் மருத்துவர் என்னுடைய மருத்துவ அறிக்கைகளை பார்ப்பதற்கு என் அனுமதி தேவையில்லை என அறிந்து கொள்கிறேன். நான் ஆய்வில் இருந்து விலகிக் கொண்டாலும் இது பொருந்தும் என அறிகிறேன்.	☐
4.	இந்த ஆய்வின் மூலம் கிடைக்கும் தகவலையோ, முடிவையோ பயன்படுத்திக் கொள்ள மறுக்க மாட்டேன்.	☐
5.	இந்த ஆய்வில் பங்கு கொள்ள ஒப்புக் கொள்கிறேன் எனக்கு கொடுக்கப்பட்ட அறிவுரைகளின் படி நடந்து கொள்வதுடன், ஆய்வை மேற்கொள்ளும் மருத்துவ அணிக்கு உண்மையுடன் இருப்பேன் என்று உறுதியளிக்கிறேன். என் உடல் நலம் பாதிக்கப்பட்டாலோ, அல்லது எதிர்பாராத, வழக்கத்திற்கு மாறான நோய்குறி தென்பட்டாலோ உடனே இதை மருத்துவ அணியிடம் தெரிவிப்பேன் என உறுதி அளிக்றேன்.	☐

பங்கேற்பவரின் கையொப்பம் / ..... இடம் .....
கட்டைவிரல் ரேகை
பங்கேற்பவரின் பெயர் மற்றும் விலாசம் .....
ஆய்வாளரின் கையொப்பம் /..... இடம் .....
ஆய்வாளரின் பெயர் .....
மையம் .....
கல்வியறிவு இல்லாதவற்கு (கைரேகை வைத்தவர்களுக்கு) இது அவசியம் தேவை
சாட்சியின் கையொப்பம் /..... இடம் .....
பெயர் மற்றும் விலாசம் ....

Sno	NAME	IP NO		Diagnosis	AGE	SEX	INPATIENTSTAY	ABDOMINAL PAIN	NONBILIOUSVOMITING	BALLROLLINGMOVEMENT	LOSSOFWEIGHT	MELENA	H/OPEPTICULCER	SMOKER	ALCOHOLIC	PALLOR	ICTERUS	SUPRACLVICULARNODE	dehydration	VGP	MASSPALPABLE	SUCCUSSIONSPLASH	organomegaly	AUSCULTOPERCUSSION	SHIFTINGDULLNESS/FLUIDTHRILL	HB	BLOODGROUPING	CT STAGING	ENDOSCOPICFINGING	HPE	PROCEDURE	COMPLICATION
1	THAMBIRATTI	109112	Malignant	CA Stomach	54	F	21	P	P	A	P	A	N	NS	NAL	P	A	NP	AH	P	NPA	P	NP	Mid	NP	11.2	O+VE	T3N1M0	TYPE 1	MDA	PSG+GJ+JJ	UE
2	BANUMATHI	109117	Malignant	CA Stomach	35	F	30	P	P	A	P	P	N	NS	NAL	P	A	NP	AH	P	PA	P	NP	GD	NP	8.9	B+VE	T4N1M0	TYPE 2	MDA	PAGJ	UE
3	PATHIRAKALI	111114	Malignant	CA Stomach	43	F	38	A	P	P	P	A	N	NS	NAL	NP	A	NP	MoDH	P	NPA	P	NP	Mid	P	10.6	A+VE	T4N1M1	TYPE 4	PDA	FJ	UE
4	PERYATHIRUVADI	118667	Malignant	CA Stomach	82	M	43	P	P	A	A	P	Y	S	AL	P	P	P	SDH	P	NPA	P	NP	Mid	NP	7.8	B+VE	T3N0M0	TYPE 1	PDA	PSG+GJ+JJ	DELAYED GASTRIC EMPTYING
5	SARASWATHY	126005	Malignant	CA Stomach	52	F	29	P	P	A	P	A	N	NS	NAL	P	A	NP	AH	P	NPA	P	NP	Mid	NP	6.5	A+VE	T4N1M0	TYPE 2	PDA	PSG+GJ+JJ	POST OP DIARRHOEA
6	ESWARAN	126008	Malignant	CA Stomach	47	M	28	P	P	P	P	A	N	S	AL	P	A	NP	AH	P	PA	P	NP	GD	NP	3.9	AB+VE	T2N1M1	TYPE 1	WDA	PSG+GJ+JJ	UE

7	CHELLAIAH	135551	Malignant	CA Stomach	69	M	22	P	A	A	P	A	Y	S	AL	NP	A	NP	SDH	P	NPA	P	NP	MiD	P	10	B+VE	T4N1M1	TYPE 1	PDA	PAGJ+JJ	UE
8	VELU	143342	Malignant	CA Stomach	60	M	20	P	A	P	P	P	Y	S	NAL	P	A	NP	AH	P	PA	P	NP	GD	NP	7.8	O+VE	T4N1M0	TYPE 1	MDA	PAGJ+JJ	UE
9	MADASAMY	203822	Malignant	CA Stomach	45	M	56	P	A	A	A	A	Y	S	AL	NP	A	NP	AH	NP	NPA	P	NP	ND	NP	14	A+VE	T3N0M0	TYPE 1	WDA	STG+D2	ENTEROCUTANEOUS FISTULA
10	MOHAMMEDHANIFA	206666	Malignant	CA Stomach	73	M	37	P	P	A	A	A	Y	S	NAL	NP	A	NP	AH	P	NPA	P	NP	ND	NP	10.2	B+VE	T3N1M0	TYPE 3	WDA	STG+D2	MYOCARDIAL INFARCTION
11	PERUMAL	239114	Malignant	Periampullary carcinoma	71	M	51	P	P	A	A	A	N	NS	NAL	P	P	NP	MoDH	P	PA	P	NP	MiD	NP	14.5	A+VE	PERIAMPUALLARY GROWTH	ULCEROPROFILERATIVE GROWTH	POORLY DIFFERENTIATED ADENOCARCINOMA	TRIPLE BYPASS	UE
12	THANGARAJ	275226	Malignant	CA Stomach	42	M	41	P	P	A	P	P	N	S	AL	NP	A	P	AH	P	PA	P	NP	MiD	P	14	AB+VE	T4N1M1	TYPE 2	PDA	FJ	CARDIOGENIC SHOCK
13	SARASWATHY	279662	Malignant	CA Stomach	66	F	10	P	P	A	P	P	N	NS	NAL	NP	A	NP	SDH	P	PA	P	NP	ND	NP	13.7	O+VE	T4N1M0	TYPE 4	PDA	FJ	POST OP PYREXIA
14	KANIAMMAL	362222	Malignant	CA Stomach	68	F	28	A	A	A	P	A	Y	NS	NAL	NP	A	NP	MoDH	NP	PA	NP	NP	ND	P	8.3	AB+VE	T4N2M0	TYPE 2	PDA	PSG+GJ+JJ	SSI

15	NATARAJAN	366627	Malignant	CA Stomach	68	M	22	P	P	P	P	A	N	S	AL	NP	A	NP	AH	P	NPA	P	NP	MiD	P	12.2	A+VE	T3N1M0	TYPE 1	PDA	STG+D2	UE
16	LINGAPANDI	381176	Malignant	CA Stomach	64	M	45	P	P	P	P	A	Y	S	AL	P	P	P	MoDH	P	PA	P	NP	MiD	P	7.5	B+VE	T4N1M1	TYPE 2	PDA	PAGJ+JJ	UE
17	KUMAR	384424	Malignant	CA Stomach	78	M	33	P	P	A	P	A	N	S	AL	P	A	NP	MoDH	P	PA	P	NP	MiD	NP	6.8	A+VE	T3N1M1	TYPE 1	WDA	PSG+GJ+JJ	UE
18	SUBRAMANIAN	384483	Malignant	CA Stomach	68	M	32	P	P	A	P	P	Y	S	AL	P	P	NP	MoDH	P	PA	P	NP	MiD	P	8.8	B+VE	T4N1M1	TYPE 1	PDA	PSG+GJ+JJ	PNEMONIA
19	RAJAKUMARI	434467	Malignant	CA Stomach	31	F	14	P	A	A	A	A	Y	NS	NAL	P	A	NP	AH	P	NPA	NP	NP	ND	NP	11	AB+VE	T3N1M0	TYPE 1	WDA	PSG+GJ+JJ	UE
20	KASIVISHVANATHAN	436117	Malignant	CA Stomach	62	M	23	P	A	A	P	A	N	S	AL	P	A	P	MoDH	NP	PA	P	NP	ND	P	12.6	B+VE	T4N1M1	TYPE 2	PDA	FJ	UE
21	ESSAKI	465447	Malignant	CA Stomach	59	M	29	P	P	A	P	A	Y	S	AL	NP	A	NP	SDH	P	NPA	P	NP	MiD	NP	13	B+VE	T3N1M0	TYPE 3	PDA	STG+D2	DELAYED GASTRIC EMPTYING
22	MURUGAN	473352	Malignant	CA Stomach	45	M	24	P	P	A	A	A	N	S	AL	NP	A	NP	AH	P	PA	P	NP	ND	P	15	A+VE	T3N1M0	TYPE 1	MDA	PSG+GJ+JJ	UE

23	MUTHAIAH	488899	Malignant	CA Stomach	38	M	39	P	P	A	P	A	N	NS	NAL	P	A	NP	AH	P	PA	P	NP	MiD	P	6.7	A+VE	T4N2M0	TYPE 2	PDA	FJ	UE
24	KALYANI	570021	Malignant	CA Stomach	50	F	43	P	P	A	P	P	N	NS	NAL	P	A	NP	AH	P	NPA	P	P	MiD	NP	7.1	B+VE	T3N1M0	TYPE 3	PDA	STG+D2	UE
25	SEENIPANDI	576999	Malignant	CA Stomach	60	M	29	P	A	A	A	A	Y	S	AL	P	A	NP	MoDH	NP	NPA	P	NP	ND	P	8.4	O+VE	T3N3M0	TYPE 1	MDA	FJ	FJ LEAK
26	MUPPIDATHY	578893	Malignant	CA Stomach	40	M	19	P	P	A	P	P	N	NS	NAL	P	A	NP	AH	P	NPA	NP	NP	ND	P	7.4	A+VE	T4N1M1	TYPE 2	WDA	FJ	UE
27	KRISHNAMOORTHY	580550	Malignant	CA Stomach	39	M	23	P	A	A	P	A	Y	S	AL	NP	A	NP	AH	NP	NPA	P	NP	ND	P	14.6	B+VE	T4N2M0	TYPE 4	PDA	FJ	UE
28	CHELLAPPA	591173	Benign	Chronic duodenal ulcer	61	M	42	P	P	P	P	A	Y	S	AL	NP	A	NP	MoDH	P	NPA	P	NP	GD	NP	10.9	B+VE	GROSSLY DISTENDED STOMACH	CICTRIZING DUODENAL ULCER	CHRONIC INFLAMMORTY CHANGES	AGJ+JJ	UE
29	CHELLAIAH	639900	Malignant	CA Pancreas	53	M	32	P	A	A	P	A	N	NS	NAL	P	P	NP	AH	NP	PA	NP	P	ND	P	9.9	B+VE	PANCREATIC MASS+STOMACH DISTENED	EXTERNAL COMPRESSION		TRIPLE BYPASS	UE
30	PALAMAMMAL	648446	Malignan	CA Stomach	60	F	49	A	P	A	P	P	Y	NS	NAL	P	A	NP	MoDH	P	NPA	NP	NP	MiD	NP	11.5	AB+VE	T3N0M0	TYPE 2	MDA	STG+D2	UE

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31	PARAMASIVAN	675044	Malignant	CA Stomach	60	M	24	P	A	A	P	P	N	S	AL	NP	A	NP	AH	NP	PA	NP	NP	MiD	NP	9.4	B+VE	T4NXMX	TYPE 3	PDA	PAGJ+JJ	UE
32	GANESHAN	685596	Benign	Chronic duodenal ulcer	45	M	46	A	P	P	A	A	Y	NS	NAL	NP	A	NP	AH	P	NPA	P	P	MiD	NP	9.6	A+VE	GROSSLY DISTENDED STOMACH	CICTRIZING DUODENAL ULCER	CHRONIC INFLAMMORTY CHANGES	TV+PGJ+JJ	POST OP DIARRHOEA
33	MADASAMY	714463	Malignant	CA Stomach	67	M	31	P	P	A	P	A	Y	S	NAL	NP	A	NP	SDH	NP	PA	P	P	MiD	NP	10.2	A+VE	T4NXM1	TYPE 2	PDA	PAGJ+JJ	UE
34	MARIMUTHU	722449	Malignant	CA Stomach	42	M	26	P	P	P	P	P	Y	S	AL	P	P	NP	SDH	P	PA	P	NP	GD	P	6.4	B+VE	T4N1MX	TYPE 1	PDA	PSG+GJ+JJ	UE
35	ARUMUGAM	729337	Malignant	CA Stomach	60	M	36	A	P	A	P	A	N	S	AL	P	A	NP	MoDH	P	NPA	P	NP	MiD	NP	12.3	O+VE	T3N1M0	TYPE 3	MDA	STG+D2	UE
36	ARUMUGAM	736227	Benign	Chronic duodenal ulcer	48	M	29	P	P	P	A	A	Y	S	AL	NP	A	NP	MoDH	P	NPA	P	NP	GD	NP	10.6	O+VE	NO E/O GASTRIC MALIGNANCY	CICTRIZING DUODENAL ULCER	CHRONIC INFLAMMORTY CHANGES	TV+PGJ+JJ	UE
37	ILAYAPERUMAL	754486	Malignant	CA Stomach	62	M	46	P	A	A	P	P	N	NS	NAL	P	A	NP	AH	NP	PA	NP	NP	ND	P	4.4	B+VE	T4N1M1	TYPE 1	PDA	PAGJ	SSI

38	SUBRAMANIAN	756944	Malignant	CA Stomach	63	M	22	P	P	P	P	A	N	NS	NAL	P	A	NP	MoDH	P	NPA	P	NP	GD	NP	14.2	A+VE	T3N0M0	TYPE 2	PDA	STG+D2	UE
39	VELPILLAI	793351	Malignant	CA Stomach	60	M	33	P	P	P	P	P	Y	A	AL	P	P	NP	SDH	P	PA	P	NP	GD	P	8.7	B+VE	T4N1M1	TYPE 3	MDA	PSG+GJ+JJ	POST OP PYREXIA
40	ARUMUGAKANI	809008	Malignant	CA Stomach	37	F	41	P	P	A	P	A	Y	NS	NAL	NP	A	NP	MoDH	P	PA	P	NP	MiD	P	12.4	B-VE	T3N2MX	TYPE 2	PDA	PAGJ+JJ	UE
41	ANANTHI	817006	Malignant	CA Stomach	51	F	30	P	A	A	P	P	N	NS	NAL	P	A	NP	SDH	P	NPA	P	P	GD	P	9.7	A+VE	T4N1M1	TYPE 1	PDA	PAGJ	UE
42	RAMASAMY	820339	Malignant	CA Stomach	68	M	43	P	P	A	P	A	N	S	AL	NP	A	NP	MoDH	NP	NPA	NP	NP	ND	P	8.4	O+VE	T3N3MX	TYPE 3	PDA	PSG+GJ+JJ	UE
43	SUMITHIRA	850339	Malignant	CA Stomach	62	F	35	A	A	A	P	P	Y	NS	NAL	P	A	NP	AH	NP	PA	NP	P	ND	P	7.5	AB+VE	T4N1MX	TYPE 1	PDA	PAGJ+JJ	UE
44	MURUGAN	861170	Malignant	CA Stomach	42	M	22	P	P	A	P	P	N	NS	NAL	P	A	NP	MoDH	NP	NPA	NP	NP	ND	P	10.1	B+VE	T4N1MX	TYPE 1	MDA	PAGJ+JJ	UE
45	CHELLAMMAL	870224	Malignant	CA Stomach	68	F	20	P	P	P	P	A	Y	NS	NAL	NP	A	NP	AH	P	NPA	P	NP	GD	P	14.2	O+VE	T4N1M1	TYPE 3	PDA	PSG+GJ+JJ	SEPTIC SHOCK

46	VELLASAMY	874449	Malignant	CA Stomach	59	M	34	P	P	A	P	P	N	S	AL	P	A	NP	AH	P	PA	P	NP	MiD	NP	7.1	B+VE	T4N1M1	TYPE 1	MDA	PAGJ+JJ	UE
47	MADASAMY	883022	Malignant	CA Stomach	70	M	32	P	P	P	P	A	Y	S	AL	P	A	NP	AH	P	NPA	P	P	GD	NP	11.3	B+VE	T3N1M0	TYPE 3	PDA	STG+D2	FJ LEAK
48	THANGAVEL	887001	Malignant	CA Stomach	50	M	34	P	P	A	P	A	N	NS	NAL	NP	A	NP	MoDH	P	PA	P	NP	MiD	NP	11.5	O-VE	T3N1M0	TYPE 3	PDA	STG+D2	DELAYED GASTRIC EMPTYING
49	MARIAPPAN	891003	Malignant	CA Stomach	50	M	43	P	A	A	P	A	N	S	AL	NP	A	NP	AH	P	PA	P	NP	MiD	NP	13.6	B+VE	T3N1M0	TYPE 2	MDA	STG+D2	UE
50	KANAGATHAI	905117	Malignant	CA Stomach	69	F	16	P	P	A	P	A	Y	NS	NAL	P	A	NP	MoDH	P	PA	NP	NP	ND	NP	8.7	A+VE	T4N1MX	TYPE 2	PDA	PAGJ+JJ	UE
51	SIVANANDAM	932885	Malignant	CA Stomach	68	M	31	P	P	A	P	A	Y	NS	NAL	NP	A	NP	AH	NP	NPA	NP	NP	ND	NP	10.8	B+VE	T4N1MX	TYPE 3	PDA	PSG+GJ+JJ	UE
52	KALIDOSS	2475	Benign	Chronic duodenal ulcer	39	M	45	P	P	P	P	A	Y	S	AL	P	P	NP	MoDH	P	NPA	NP	NP	GD	NP	10.7	O+VE	PANCREATIC MASS+STOMACH DISTENED	CICTRIZING DUODENAL ULCER	CHRONIC INFLAMMORTY CHANGES	AGJ+JJ	SSI

53	MARIAMMAL	26700	Malignant	CA Stomach	70	F	30	P	P	A	A	A	N	NS	NAL	P	A	NP	AH	P	NPA	P	NP	MiD	NP	9.3	AB+VE	T3N1M0	TYPE 3	PDA	STG+D2	UE
54	KADARKARAIANDI	65441	Malignant	CA Stomach	48	M	31	P	P	A	P	P	Y	NS	NAL	P	A	NP	MoDH	P	NPA	P	NP	GD	NP	5.3	B+VE	T4N3MX	TYPE 1	PDA	PSG+GJ+JJ	SSI
55	ALAGAMMAL	112246	Malignant	CA Stomach	56	F	28	P	P	A	P	A	Y	NS	NAL	NP	A	NP	AH	NP	PA	NP	NP	ND	NP	12.5	O+VE	T3NXMX	TYPE 3	WDA	PSG+GJ+JJ	UE
56	PONNAMMAL	115574	Malignant	CA Stomach	52	F	30	P	P	A	P	P	N	NS	NAL	P	A	NP	SDH	P	PA	P	NP	MiD	P	10.2	B+VE	T4N1MX	TYPE 1	WDA	PAGJ	UE
57	NATARAJAN	142228	Malignant	CA Stomach	57	M	30	P	P	A	P	P	Y	NS	NAL	NP	A	NP	MoDH	P	PA	P	NP	MiD	NP	10.5	B+VE	T3N0M1	TYPE 2	MDA	PSG+GJ+JJ	POST OP PYREXIA
58	MUNIYASAMY	167768	Benign	Chronic duodenal ulcer	37	M	34	P	P	A	A	A	Y	S	AL	NP	A	NP	AH	P	NPA	P	NP	GD	NP	13.5	B+VE	GROSSLY DISTENDED STOMACH	CICTRIZING DUODENAL ULCER	CHRONIC INFLAMMORTY CHANGES	TV+PGJ+JJ	UE
59	ABUBAKKAR	191105	Malignant	CA Stomach	55	M	36	P	P	A	P	A	Y	NS	NAL	P	A	NP	MoDH	P	NPA	P	NP	MiD	P	9.1	A+VE	T3NxM1	TYPE 2	PDA	PSG+GJ+JJ	UE
60	THENNARASU	194115	Benign	Chronic duodenal ulcer	55	M	25	P	P	A	A	A	Y	S	AL	NP	A	NP	AH	P	NPA	P	NP	GD	NP	12.5	O+VE	GROSSLY DISTENDED STOMACH	CICTRIZING DUODENAL ULCER	CHRONIC INFLAMMORTY CHANGES	TV+PGJ+JJ	UE

61	PARVATHI	197559	Malignant	CA Stomach	63	F	58	P	P	A	P	A	N	NS	NAL	P	A	NP	MoDH	NP	PA	P	NP	ND	NP	11.4	B+VE	T3N1M0	TYPE 1	PDA	TG+DISTAL PANCREATECTOMY&SPLENECTOMY+ROUXEJ+JJ+FJ	UE
62	RAKKAMMAL	206004	Malignant	CA Stomach	65	F	22	P	P	P	P	A	Y	NS	NAL	NP	A	NP	AH	P	PA	P	NP	MiD	NP	10.2	O+VE	T4N1MX	TYPE 3	PDA	PAGJ+JJ	UE
63	SUNDARALINGAM	218880	Malignant	CA Stomach	63	F	30	P	P	A	P	A	N	S	AL	P	A	NP	MoDH	P	NPA	P	NP	MiD	P	8.8	B+VE	T4N0M1	TYPE 2	MDA	PAGJ+JJ	UE
64	SAMUTHIRAM	221779	Malignant	CA Stomach	60	M	35	P	P	P	P	P	Y	S	AL	P	A	NP	AH	NP	PA	NP	NP	ND	P	6.2	A+VE	T4N1M1	TYPE 1	PDA	PAGJ	UE
65	KARPAGAM	223900	Malignant	CA Stomach	62	F	28	P	P	A	P	P	Y	NS	NAL	NP	A	P	SDH	P	PA	P	NP	MiD	P	4.5	B+VE	T4N1M1	TYPE 4	PDA	FJ	PNEMONIA
66	PITCHAMMAL	251116	Malignant	CA Stomach	64	F	26	A	P	P	A	A	Y	NS	NAL	P	P	P	MoDH	P	PA	P	NP	MiD	P	9.6	A+VE	T4N1MX	TYPE 3	PDA	PAGJ+HJ	UE
67	SANKARAMMAL	254118	Malignant	CA Stomach	55	F	28	P	A	A	P	A	Y	NS	NAL	NP	A	NP	MoDH	P	PA	P	NP	ND	P	8.2	B+VE	T3N1M1	TYPE 3	PDA	PAGJ+JJ	UE
69	KANAGAMMAL	258001	Malignant	CA Stomach	61	F	46	A	P	A	P	P	Y	S	AL	P	A	NP	AH	P	PA	P	NP	MiD	P	8.1	O-VE	T4N1MX	TYPE 1	PDA	PSG+GJ+JJ	SSI

70	RAMAKANI	280097	Malignant	CA Stomach	55	F	24	P	P	P	P	A	N	NS	NAL	P	A	NP	MoDH	P	PA	P	NP	GD	NP	9.2	B+VE	T3N1MX	TYPE 2	PDA	PAGJ+JJ	UE
71	MARIDURAI	289556	Benign	Pseudocyst of pancreas	42	M	37	P	P	P	P	A	N	S	AL	NP	A	NP	MoDH	P	PA	P	NP	GD	NP	12	A+VE	STOMACH STASIS	EXTERNAL COMPRESSION	CHRONIC INFLAMMORTY CHANGES	cystogastrostomy	UE
72	MUTHU	291198	Malignant	CA Stomach	53	M	34	P	P	P	P	P	N	S	AL	P	P	NP	SDH	P	PA	P	P	MiD	P	6.5	A+VE	T4N0M1	TYPE 3	PDA	PSG+GJ+JJ	DELAYED GASTRIC EMPTYING
73	SIVANANTHAPERUMAL	327422	Malignant	CA Stomach	70	M	31	P	P	A	A	A	N	NS	AL	P	A	NP	AH	P	NPA	P	NP	MiD	P	9.4	B+VE	T4N1MX	TYPE 1	MDA	PAGJ+JJ	UE
74	SANKARAPANDIYAN	343362	Malignant	CA Stomach	69	M	22	P	P	P	A	A	Y	NS	NAL	P	A	NP	SDH	P	NPA	P	NP	MiD	P	8.8	B+VE	T4N1MX	TYPE 3	PDA	PAGJ+JJ	UE
75	VELLASAMY	407229	Benign	Chronic duodenal ulcer	65	M	29	P	P	A	A	A	N	NS	NAL	P	A	NP	AH	P	NPA	NP	NP	GD	NP	12.6	B+VE	GROSSLY DISTENDED STOMACH	CICTRIZING DUODENAL ULCER	CHRONIC INFLAMMORTY CHANGES	TV+PGJ+JJ	UE
76	MURUGAN	380339	Malignant	CA Stomach	38	M	20	P	P	A	A	A	Y	S	AL	NP	A	NP	AH	NP	PA	NP	NP	ND	NP	14.6	B+VE	T3N0M0	TYPE 1	PDA	STG+D2	SSI
77	CHELLAPPA	487769	Malignant	CA Stomach	70	M	32	P	P	P	P	P	Y	S	AL	P	A	NP	MoDH	P	PA	P	NP	MiD	NP	8.1	B+VE	T3N1M0	TYPE 1	PDA	STG+D2	UE

78	PALANIAMMAL	465555	Malignant	CA Stomach	50	F	25	P	P	A	P	P	Y	NS	NAL	P	A	NP	SDH	P	PA	P	NP	MiD	NP	8.2	B+VE	T3N1M0	TYPE 1	PDA	TG+ROUXEJ+JJ+FJ	PNEMONIA
79	PAKKIRISAMY	379979	Malignant	CA Stomach	68	M	42	P	P	P	A	A	Y	NS	NAL	NP	P	P	MoDH	P	PA	P	NP	GD	P	11.6	A+VE	T4N1MX	TYPE 2	PDA	PAGJ+JJ	UE
80	KRISHNAMOORTHY	806678	Malignant	CA Stomach	78	M	29	P	P	P	P	P	N	NS	NAL	P	A	NP	MoDH	P	PA	P	NP	GD	P	6.7	AB+VE	T3N1M1	TYPE 1	PDA	PSG+GJ+JJ	CCF