A CLINICAL STUDY ON GASTRIC OUTLET OBSTRUCTION

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ABSTRACT

INTRODUCTION
Gastric outlet obstruction (GOO) is obstruction in the first part of duodenum secondary to cicatrised duodenal ulcer or proximally where the diagnosis of carcinoma is most probable. GOO can be a diagnostic and treatment dilemma. Once a mechanical obstruction is confirmed, differentiate between benign and malignant processes. Cicatrized duodenal ulcer was the most common cause of GOO. But with increased awareness, change in the dietary habits and availability of H2 receptor blockers and Proton Pump Inhibitors and H pylori kits all have resulted in decreased incidence of patients requiring surgery and also the complications like pyloric stenosis have reduced. At the same time the incidence of antral carcinoma of stomach producing GOO has comparatively increased, which may be due to increased early diagnosis of the condition with the help of flexible fibro optic endoscope.

AIMS & OBJECTIVES
To study and identify the cause of cases of GOO with respect to benign peptic ulcer and malignancy of stomach.

MATERIALS AND METHODS
An observational study comparing of 30 cases of GOO. An elaborate study of the cases with regard to history, clinical features, routine and special investigation, pre operative treatment, operative findings, post operative management and complications in the post operative period is done. Apart from routine surgical profile special investigations like serum electrolytes, barium meal study, Upper GI Endoscopy and ultrasound abdomen and pelvis will be carried. For peptic ulcer disease truncal vagotomy with posterior gastrojejunostomy was done and for carcinoma partial gastrectomy with Billroth II reconstruction or anterior GJ or palliative resection with anterior GJ were done.

INCLUSION CRITERIA
1. Peptic ulcer disease 2. Carcinoma pyloric antrum, 3. Benign neoplasm of stomach.

EXCLUSION CRITERIA
1. Carcinoma stomach with liver metastasis, ascites, peritoneal implantation, 2. Gastro duodenal tuberculosis.

ETHICAL ISSUES
Informed written consent will be taken from patients before including them in the study.

SUMMARY OF RESULTS
1. The most common cause of gastric outlet obstruction is carcinoma stomach with antral growth in 53.3% producing GOO in the study. 2. Number of cases with cicatrized duodenal ulcer causing GOO were 43.3%. 3. One case of stenosis following corrosive acid ingestion was also present as the reason for GOO in the study 3.3%. 4. Males are more commonly affected than female and the male female ratio is 7:1 in malignancy. 5. This study was undertaken in an adult rural population. 6. The most common presenting complaints were vomiting (100%), abdominal pain (90%) and loss of appetite (90%). In malignant cases loss of weight (93%) was also a common complaint. 7. Visible gastric peristalsis and succession splash were less prominent in malignant cases when compared to stenosing duodenal ulcer cases. 8. 31% of the malignant cases presented with mass in the upper abdomen. 9. Only 5 cases of malignancy could be able to undergo definite surgical procedure. All others underwent palliative procedures.10.The surgical procedure undertook in the cicatrized duodenal ulcer patients were truncal vagotomy and posterior GJ and there were no recurrence of symptoms in any of the casers which turned up for follow up. 11. The mortality rate was 18.8% in malignant cases.

CONCLUSION
1. The most common cause of gastric outlet obstruction in adults are carcinoma stomach with antral growth producing GOO in 53.3% and cicatrized duodenal ulcer causing GOO were 43.3%. 2. In the vast majority of cases the diagnosis can be established clinically. 3. The saline load test was found to be effective bedside investigations to assess the degree of GOO. 4. Upper GI endoscopy should be mandatory in all suspected cases of GOO. 5. Number of cases with cicatrized duodenal ulcer as the chief etiological factors for GOO is diminishing and the number of cases of antral carcinoma of stomach as the cause of GOO is increasing. 6.Effective treatment in carcinoma stomach depends on early diagnosis.

KEYWORDS
Gastric Outlet Obstruction; Cicatrized Duodenal Ulcer; antral carcinoma.

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INTRODUCTION: Gastric outlet obstruction (GOO) is the more accurate term for the commonly used term 'pyloric stenosis' as the site of obstruction is rarely the pylorus itself. The obstruction is usually in the first part of duodenum secondary to cicatrised duodenal ulcer or proximally where the diagnosis of carcinoma is most probable.[1]

GOO is the clinical and pathophysiological consequence of any disease process that produces a mechanical impediment to gastric emptying. GOO can be a diagnostic and treatment dilemma. As part of initial work up, exclude the possibility of functional non mechanical causes of obstruction such as Gastroperesis

Once a mechanical obstruction is confirmed, differentiate between benign and malignant processes because definitive treatment is based on recognition of the specific underlying cause.

Cicatrized duodenal ulcer was the most common cause of GOO. It is more common in south India when compared to rest of the country which may probably be due to bulky and irritative diet consumed by people in the south.[2] surgery for chronic duodenal ulcer and its complications was the most common surgical procedure done in this part of country decades ago.[2]

But with increased awareness of the disease, change in the dietary habits and availability of H2 receptor blockers and Proton Pump Inhibitors and recent findings of association of H pylori with the causation of peptic ulcer diseases and its effective eradication with H pylori kits all have resulted in decreased incidence of patients requiring surgery and also the complications like pyloric stenosis have reduced[3]. At the same time th incidence of antral carcinoma of stomach producing GOO has comparatively increased, which may be due to increased early diagnosis of the condition with the help of flexible fibro optic endoscope.[4,5]

So this study has been taken up to review the changes in incidence and presentation, evaluation methods and management of GOO.

AIMS & OBJECTIVES:

1. To study cases of GOO with respect to benign peptic ulcer and malignancy of stomach.
2. To identify the cause of GOO in the adult rural patients coming to S.V.R.R.G.G.Hospital, Tirupathi.

MATERIALS AND METHODS: This is a clinical observational study comparing of 30 cases of GOO. The Patients of this study have been selected from surgical wards of S.V.R.R.G.G.Hospital, Tirupati during the period from November 2012- November 2014.

An elaborate study of the cases with regard to history, clinical features, routine and special investigation, pre operative treatment, operative findings, post operative management and complications in the post operative period is done

In history details about presenting complaints and duration, history of acid peptic disease, features of metabolic disturbances, occupation, personal history including diet, bowel and bladder habit, smoking and alcoholism will be noted.

Physical examination will be carried out in detail noting status of hydration and nutritional status, presence of Visible Gastric Peristalsis, succussion splash, hepatomegaly and ascites.

Apart from routine surgical profile special investigations like serum electrolytes, barium meal study, Upper GI Endoscopy and ultrasound abdomen and pelvis will be carried. Any one of the following criteria was used to diagnose a case to be having GOO.

- Projectile vomiting of undigested food consumed previous day.
- Visible gastric peristalsis.
- Gastric succusion splash 3 to 4 hrs after last meal.
- Palpable hypertrophied stomach.
- Delayed emptying of stomach on barium meal study.
- Difficulty in negotiating tube on upper gi endoscopy.
- A gastric residue of more than 500ml in an adult.
- Demonstration of narrowed gastric outlet during surgery.

In managing GOO, measures employed are;

- Improve local condition of stomach
- Correct dehydration, fluid and electrolyte imbalance
- Correct anemia,hypoprotienemia and vitamin deficiency
- According to investigations reports and operative findings, definitive surgery was undertaken.

Surgeries were performed under general, combined spinal and epidural.

For peptic ulcer disease truncal vagotomy with posterior gastrojejunostomy was done and for carcinoma partial gastrectomy with Billroth II reconstruction or anterior GJ or palliative resection with anterior GJ were done.

Post operatively ryles tube aspiration and iv fluids were given till stomach recovers normal tone and bowel sounds appear. Oral feedings with fluids was then commenced followed by solids. Early ambulation was encouraged in elderly.

Inclusion Criteria:

- Peptic ulcer disease.
- Carcinoma pyloric antrum.
- Benign neoplasm of stomach.

Exclusion Criteria:

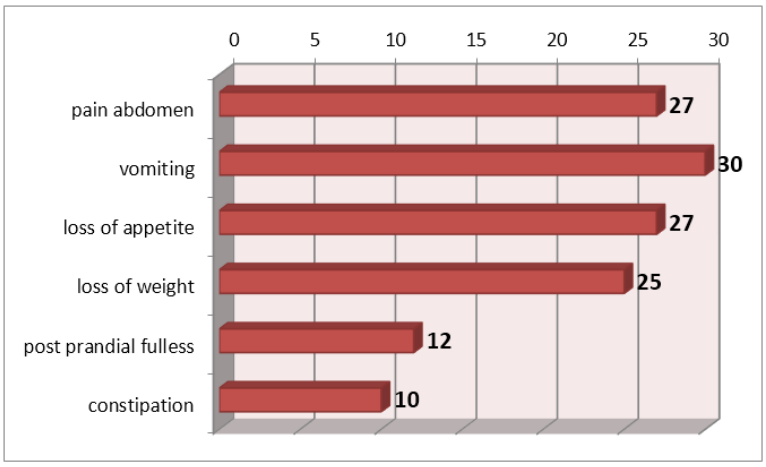
- Carcinoma stomach with liver metastasis, ascites, peritoneal implantation.
- Gastro duodenal tuberculosis.

Ethical Issues: Informed written consent will be taken from patients before including them in the study.

RESULTS: Of the 30 cases, 16 cases had malignant growth in the antrum (53.33%) and 13 cases had cicatrized duodenal ulcer (43.33%) and 1 case had stenosis following corrosive ingestion (3.33%) as the cause of GOO.

Symtoms	No. of cases	%	Malignancy cases	%	No. of cicatrised duodenal ulcer	%
Pain abdomen	27	90	16	100	10	76
Vomiting	30	100	16	100	13	100
Loss of appetite	27	90	15	93.7	11	84
Loss of weight	25	83.3	15	93.7	9	69.2
Post prandial fulless	12	40	6	37.5	6	46.2
Constipation	10	33.3	6	37.5	3	23.1

TABLE NO 1: DISTRIBUTION OF SYMPTOMS



GRAPH NO 1

Pain abdomen was present in 27 (90%) cases. It was mainly present in upper abdomen and in 2 cases of malignancy; there was radiation to the back suggestive of involvement of pancreas. In 18 cases, the duration of pain was less than 1 year, of which 13 were malignant cases. Of these 18 cases 7 cases gave history suggestion of acid peptic disease and 4 of them were malignant case suggesting malignancy developing in gastric ulcer. 1 case did not give history of pain abdomen gave history suggestive of acid peptic disease. In 16 cases food was the aggravating factor while vomiting was the relieving factor in all 15 cases.

Vomiting was present in all 30 (100%) cases. It was spontaneous and projectile in 29 cases while in 2 cases it was induced. 1 case where vomiting was induced was malignant case. Frequency of vomiting was variable and was present occasionally in majority of the cases. The vomitus contained mainly undigested food contents of meals taken earlier and was foul smelling in 2 cases. The vomitus was bile stained in one of the cases. 1 patient with cicatrized duodenal ulcer gave past history of one episode of hematemesis.

Loss of appetite was present in 27 cases (90%). 1 patient with malignancy did not have loss of appetite. Loss of weight was present in 25 cases. 15 out of the 16 malignant cases gave history of loss of weight.

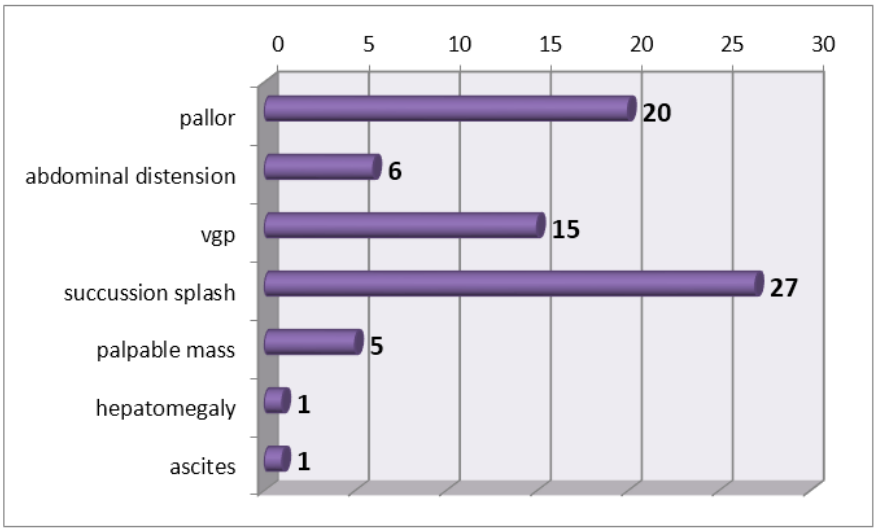
12 cases gave history of post prandial fullness, of which 2 were malignant cases. 3 cases gave a past history of melena and only one case had malignancy. In 1 case melena was associated with an episode of hemetemisis.

Totally 18 cases were smokers and 16 cases consumed alcohol regularly. In the malignant cases 12 patients were smokers while 9 patients consumed alcohol regularly.

Signs	No. of Cases	%	Malignancy cases	%	No. of cicatrised duodenal ulcer	%
Pallor	20	66.7	12	75	7	53.9
Abdominal distension	6	20	4	25	2	15.4
VGP	15	50	7	43.7	8	61.5
Succussion splash	27	90	13	81.3	13	100
Palpable mass	5	16.7	5	31.3		

Hepatomegaly	1	3.3	1	6.3		
Ascites	1	3.3	1	6.3		

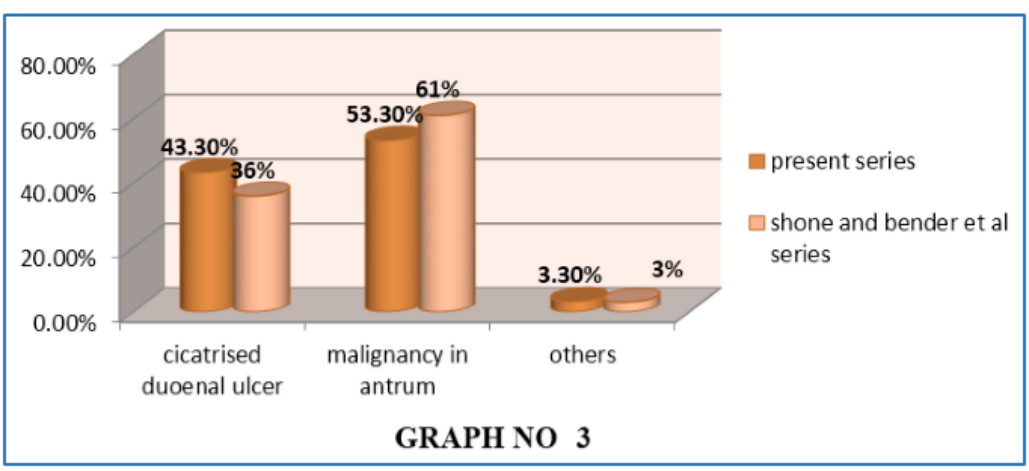
Table No. 2: DISTRIBUTION OF SIGNS



GRAPH NO 2

Cause of obstruction	Present series	Shone and Bender et al series[31]
Cicatrised duodenal ulcer	43.30%	36%
Malignancy in antrum	53.30%	61%
Others	3.30%	3%

TABLE NO 3: DISTRIBUTION OF CAUSES OF GOO

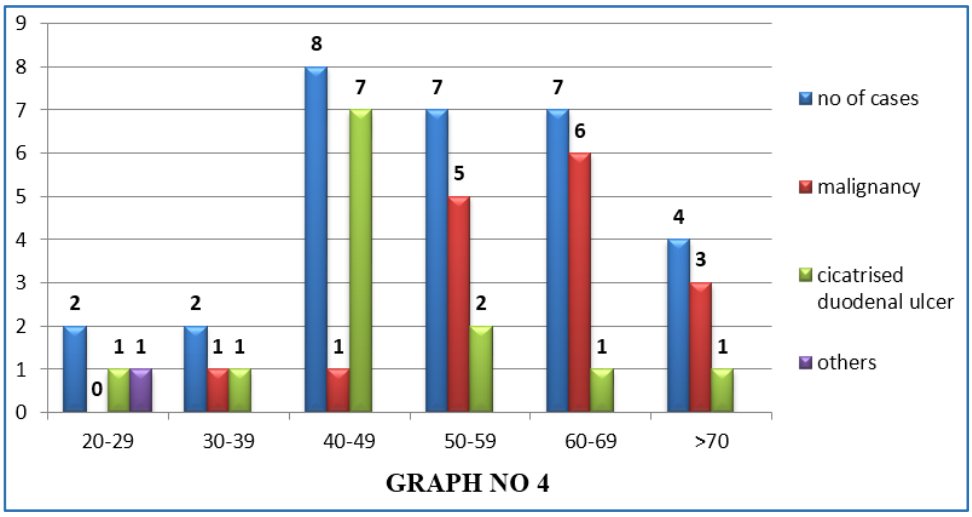


GRAPH NO 3

These observations reveal that the incidence of GOO secondary to chronic duodenal ulcer has come down while that of malignancy has relatively gone up.

Age	No. of cases	Malignancy	%	Cicatrised duodenal ulcer	%	Others	%
20-29	2	0	0	1	7.7	1	7.7
30-39	2	1	6.3	1	7.7		
40-49	8	1	6.3	7	53.8		
50-59	7	5	31.3	2	15.4		
60-69	7	6	37.5	1	7.7		
>70	4	3	18.8	1	7.7		

TABLE NO 4: AGE AND SEX INCIDENCE

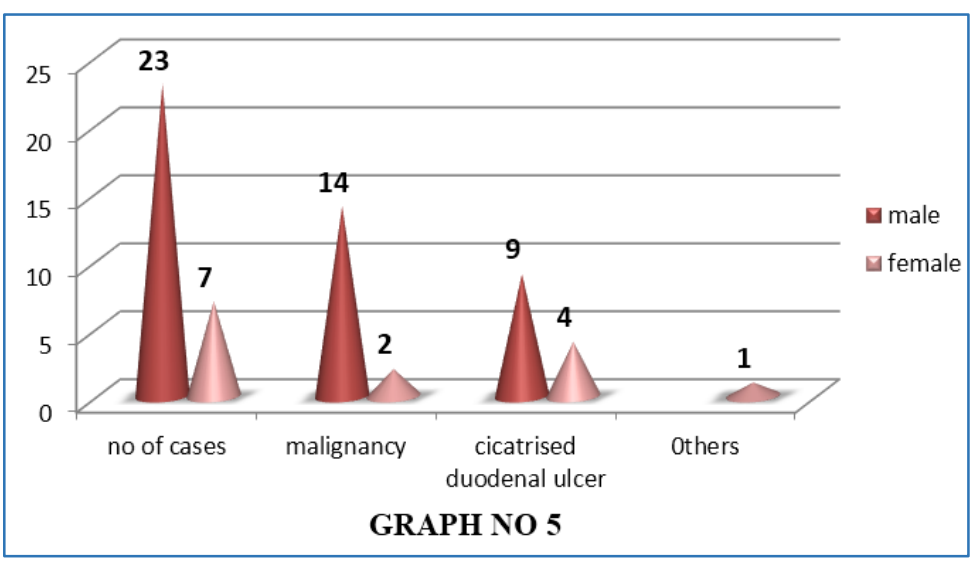


GRAPH NO 4

The study showed that maximum incidence of malignant cases was seen in 50 -70 age group. Ther maximum incidence of cicatrized duodenal ulcer cases was seen in the 21-49 age groups.

Sex	No. of cases	Malignancy	Cicatrised duodenal ulcer	0thers
Male	23	14	9	
Female	7	2	4	1

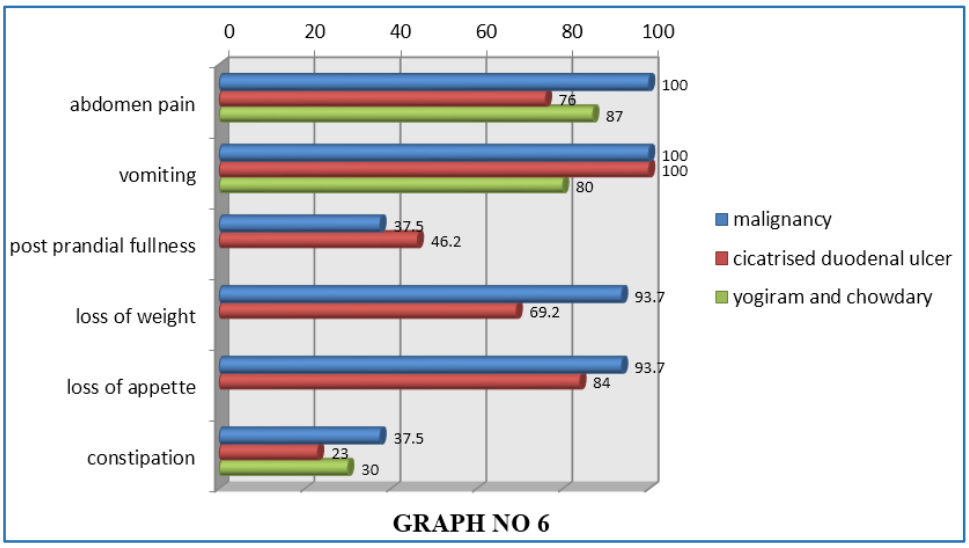
TABLE NO 5: DISTRIBUTION OF SEX



GRAPH NO 5

Incidence of symptoms	Malignancy	Cicatrised duodenal ulcer	Yogiram & chowdary[2]
Abdomen pain	100	76	87
Vomiting	100	100	80
Post prandial fullness	37.5	46.2	
Loss of weight	93.7	69.2	
Loss of appette	93.7	84	

TABLE NO 6 : INCIDENCE OF SYMPTOMS IN THE CASES



GRAPH NO 6

Saline load test was positive for all cases minimum residual saline being 250ml and maximum being 380ml.

INVESTIGATIONS: Apart from routine investigations upper GI endoscopy was done in all. In the 16 cases of malignancy, 14 cases showed antral growth as the cause for GOO. In the other 2 cases prepyloric growth was seen and in one case lesser curvature growth was noted.

In 13 cases of cicatrized duodenal ulcer cases endoscopy showed duodenal ulcers as the cause of GOO. 10 cases showed duodenal ulcers in d1 few cases showed ulcers at D1 and D2 junction. Stomach showed varying degree of dilatation.

In case of stenosis due to corrosive acid ingestion producing GOO pyloric narrowing with stenosis was noted.

Barium meal was done in very few selected cases. In malignant cases irregular filling defect was noted. In cicatrized duodenal ulcer deformed duodenal cap and dilated stomach was found.

Gastric lavage was given with normal saline for 4-5 days prior to surgery. Blood transfusions were given. Dehydration, Hypoproteinaemia was corrected. In 16 cases of malignant cases 5 cases where mass was not fixed underwent partial gastrectomy and GJ. 6 cases with fixed masses underwent anterior GJ. In the 3 advanced cases one with liver secondaries and ascites underwent palliative resection and anterior GJ. In one case only feeding jejunostomy was done.

In 13 cases of cicatrized duodenal ulcer all underwent truncal vagotomy and posterior GJ was done

In case of stenosis with acid ingestion laparotomy and posterior GJ and jejuno jejunostomy was done

In the post operative period all patients were managed with IV fluids, antibiotics, Ryles tube aspiration and analgesics. Suture removal was done on 7^{th} – 10^{th} post op day.

1 patient with cicatrized duodenal ulcer developed persistent vomiting following oral feeds. On investigations, his potassium levels were low while endoscopy revealed a patent and adequate stoma. He recovered with conservative management with iv fluids, iv potassium chloride and ryles tube aspiration.

3 patients of malignancy died in the post op period. In which two patients were advanced cases they died due to severe electrolyte imbalance. One case died of chest pain on 6^{th} POD.

THEORY PART:

AETIOLOGY: Gastric outlet obstruction is caused by various pathological conditions caused by varied etiologies[6]

A. Congenital:

- Congenital hypertrophic pyloric stenosis.
- Duodenal atresia/stenosis.
- Malrotation/incomplete rotation,
- Duplication of stomach and/or duodenum.

B. Acquired:

I. Inflammatory:

- Chronic cicatrizing duodenal ulcer.
- Gastroduodenal tuberculosis.
- Strictures due to corrosives, curling's ulcer or post-operative scarring.
- Pancreatic pseudocyst.
- Gastric syphilis.
- Crohn's disease.
- Adhesions due to chronic cholecystitis or post-cholecystectomy.
- Post-radiation gastritis.
- Allergens.
- Cholecystitis.
- Acute pancreatitis.

II. Neoplastic:

- Carcinoma stomach (pylorus).
- Benign tumors of stomach and duodenum.

1. Epithelial polyp.
2. Mesenchymal neoplasm.
3. Miscellaneous tumours.
   - Malignant mesencymal tumours.

1. Carcinoid tumours.
2. Sarcoma.
3. Melanoma.

III. Miscellaneous:

- Hypertrophic pyloric stenosis in adults.
- Wilkie's syndrome (duodenal ileus).

Foreign bodies and bezoars:

- Intramural hematoma
- Gall stone obstruction of duodenum
- Crohn's disease
- Aberrant vessels
- Duodenal diverticula
- Paraduodenal hernia
- Radiation induced, especially second part of duodenum
- Chemotherapy induced–continuous hepatic artery infusion of 5 FU

AETIOPATHOGENESIS:

A. Gastric Outlet Obstruction due to Peptic Ulcer Cicatrisation: Stenosed peptic ulcers are the commonest cause of gastric outlet obstruction. Obstruction may be due to stenosed duodenal ulcer, pyloric or low lesser curve ulcers.

Diet and Nutrition: The south Indian diet plays an important role in the genesis of peptic ulceration. The ploopy south Indian diet consists mainly of rice and soups, full of irritant condiments, chilly and spices and is deficient in certain essential nutrients.[6]

Genetic Factors: Family studies have shown that peptic ulcers occur 2-2.5 times more frequently among first degree relatives of patients unlike in relatives of controls. Subjects with blood group 'O' are about 37% more likely to develop duodenal ulcers than people with other groups. Other contributing factors are race, personal habits, rapid gastric emptying etc.

PATHOLOGY: Gastric outlet obstruction occurs during course of duodenal ulcer disease because recurrent episodes of ulceration and inflammation around the ulcer, followed by healing and deposition of scar tissue. Over a period of 10 -15 years, with repeated episodes of duodenal ulceration, healing and fibrosis, the pyloric channel or the duodenal bulb becomes relatively scarred and stenotic causing obstruction. An active ulcer crater is frequently, but not always present. The obstruction is usually incomplete and some food can be demonstrated to leave the stomach slowly. In few patients, obstruction mat be complete. In some patients a gastric ulcer may develop due to persistent stasis. Four histological zones are described surrounding chronic peptic ulcer – Superficial layer of fibrin and exudates with successive underlying zones of fibrinoid necrosis, granulation tissue and fibrosis.[7]

B. Gastric Outlet Obstruction due to Carcinoma Stomach[8]: It is a leading cause of cancer deaths second only to lung cancer. Etiological factors can be divided into;

1. Acquired Factors:

A. Nutritional:

- High salt intake.
- High nitrate consumption.
- Low dietary Vit A and C.
- Smoked and salt cured food.
- Lack of refrigeration.
- Poor drinking water (well water).

B. Occupational:

- Rubber workers.
- Coal workers.

C. Cigarette smoking.
D. H. pyloric infection.
E. Epstein Bar virus.
F. Radiation Exposure.

2. Genetic Factors:

- Type A blood.
- Pernicious anemia.
- Family history.
- Hereditary nonpolyposis colon cancer.
- Li-Fraumeni syndrome.

3. Precursor Lesions:

- Adenomatous gastric polyps.
- Chronic atrophic gastritis.
- Dysplasia.
- Intestinal metaplasia.
- Menetrier's disease.

The most celebrated example of genetic predisposition toward stomach cancer is illustrated in Bonaparte family. Napoleon, his father and his grandfather all died of gastric carcinoma.

I. GENERAL CLINICAL FEATURES[9,10,11]:

a. Pain: Pain or discomfort in upper abdomen especially after taking food is present in most of the times. It is dull aching and relieved by vomiting.

b. Vomiting: It is a constant feature and in its absence the diagnosis of gastric outlet obstruction is to be reconsidered. Typically the vomiting is projectile and effortless, large in amount and may contain food particles ingested 2-3 days previously. It may be foul smelling because of fermented food particles because increased infection which is due to hypoacidity which can be a result of chronic gastritis.

c. Loss of Appetite: Seen usually in late cases with advanced stenosis, profound in cases of malignancies.

d. Loss of Weight: Improper digestion, vomiting and loss of appetite lead to loss of weight.

e. Constipation: Because of repeated vomiting and dehydration, recent onset of constipation is a common complaint.

f. Movement of Some Lump in the Abdomen: Patients sometimes attribute gastric peristalsis to some lump moving in the abdomen which occurs usually after taking food.

ON EXAMINATION:

a. **Wasting:** Due to malnutrition and vomiting.
b. **Anemia:** Due to improper nourishment (deficiency of vitamins and minerals) and due to hematemesis and malaena.
c. **Dehydration:** Dehydration commences if the fluid losses are over 6% of body weight and here it is because of vomiting there is loss of water, K+, Na+ and Cl-.
d. **Visible peristalsis[12,13]:** It is a ball-rolling movement in upper abdomen because of gastric peristalsis from left to right and its course varies depending upon the shape of the stomach. Clinically it is elicited after making the patient drink as much water he can and can be made more prominent on putting alcohol or ether over epigastric region.
e. **Dilated stomach:** In most cases of gastric outlet obstruction stomach will be dilated below the level of umbilicus. In advanced cases it can be in the pelvis. Following methods can be adopted to make out the dilatation of stomach.
    I. **Percussion Method:** The area of stomach is percussed centrifugally and greater curvature is marked at the point of change of note.
    II. **Auscultoscraping:** Bell of stethoscope is placed just below and left of xiphisternum. Abdomen is scraped radially and centrifugally with a blunt object and the points of change of note mark the greater curvature.
f. **Succussion splash[12,13]:** Performed after three hours after food, by hearing for the gurgle produced by shaking the patient after keeping the ear close to anterior abdominal wall.

II. SPECIFIC CLINICAL FEATURES[14]:

A. Cicatrised Peptic Ulcer:

a. **Vomiting:** The patient usually will have non-bilious painless vomiting once in the evening. In advanced cases vomiting may occur at any time.
b. **Pain:** Periodicity is lost. The patient can usually eat breakfast and a small lunch and nothing after that.
c. **Features of Nutritional Disturbance:** Vomiting and dehydration may cause anorexia, coated tongue, thirst, unpleasant taste in the mouth and weakness A history of previous GI bleed can be obtained in about 25% of cases and perforation or obstruction in the past in 30% of cases.[15]

On examination, the patient may have typical "ulcer facies" and mentally confused and have tetany because of electrolyte imbalance. Skin is dry, wrinkled and turgor is lost. Rarely, a peptic ulcer forms an inflamed mass with head of pancreas, the adjacent omentum or the hepatic flexure of colon.

B. Carcinoma stomach[8]: Carcinoma stomach causing gastric outlet obstruction will have short history of about few months and may not have any history suggestive of peptic ulcer. The vomitus is offensive and contains coffee ground coloured altered blood. Patient may give history of malaena and jaundice. In late cases there may be abdominal distension with ascitis. Deep palpation of relaxed abdomen may reveal an epigastric mass, which is hard, irregular, mildly tender and may move with respiration. Liver, when palpable with hard nodules indicates secondary involvement. One may get hard, subcutaneous metastatic nodules in umbilical region (Sister Mary Joseph's nodules). Left supraclavicular (Virchow's) or left axillary (Irish) lymph nodes may be palpable or migratory thrombophlebitis (Trousseau's sign) may be present. One may get shifting dullness and fluid thrill in late cases. Digital rectal examination may reveal transcoelomic deposition of metastatic cells into rectovesical pouch (Bulmer's shelf). Vaginal examination may reveal Krukenburg's tumor.

COMPLICATIONS OF GASTRIC OUTLET OBSTRUCTION:

1. **Metabolic effects[16,17]:** As a result of the loss of gastric juice in vomitus or even its sequestration\ within the dilated stomach profound metabolic disturbances may occur. Gastric juice contains an average of 100 meq/L of Cl-, 45 meq/L of Na+ and 10 meq/L of K+. Initially vomiting causes loss of hydrochloric acid in excess of sodium and potassium.

The hydrogen ions are derived from carbonic acid with residual bicarbonate passing into ECF. So, there will be fall in plasma Cl- and rise in HCO3. In early stages, this alkalotic tendency is compensated for by renal excretion of excess of NaHCO3 which maintains the pH levels within normal limits. It is of interest that there is little evidence of respiratory compensation of excretion of this excess of carbon dioxide in exposed air. Urine is alkaline because of diminished chloride content and increased bicarbonate and is concentrated because of reduced volume because of dehydration. In longstanding cases, because of loss of large amounts of gastric juice that is rich in hydrogen chloride and potassium ions, there is hypokalemic, hypochlorohydric alkalosis. Sodium deficit increases as loss is both in vomitus and in urine. In urine it is excreted with bicarbonate which must be accompanied by a cation. This causes decrease in ECF which sometimes can cause shock. This sodium deficit stimulates aldosterone secretion which conserves sodium at the cost of K+ and H+.Pre-renal azotemia occurs as a result of decreased GFR and urea clearance. H+ excretion in urine in place of Na+ in an alkalotic condition is called as "paradoxical aciduria". As potassium is lost from the cells, it is replaced by sodium and hydrogen ions which produce an intra-cellular acidosis and aggravates still further the extracellular alkalosis.

Further, "gastric tetany" can result from a shfit of weakly alkaline ionized calcium phosphate to its unionized form in an attempt to reduce alkalosis. So, there will be apparent fall in plasma calcium ion levels although total calcium levels are normal. Because of dehydration, the

concentration of electrolytes tends increase and K+ and Cl- levels tends to decrease in some instances, the degree of metabolic alkalosis is better reflected by level of standard bicarbonate level than electrolyte analysis. Isotonic saline replacement is primarily needed for the correction of dyselectrolytemia. It restores the volume of ECF and plasma, and hence renal blood flow. It helps the kidney to excrete an alkaline urine. Potassium can be supplemented in severe cases and dilute HCl in very severe cases. K+ supplementation of > 20meq/hour requires strict cardiac beat monitoring.

Clinical Presentation: Along with features of dehydration, there will be drowsiness, muscular hypotonia, loss of reflexes, urinary incontinence. Cheyne-strokes respiration, fall in BP with bounding pulse, sometimes tetany and abdominal distension due to paralytic ileus may be present.

2. **Gastritis:** Due to stasis and fermentation of gastric contents.
3. **Gastric Ulcer:** Due to stasis and gastritis, lesser curve ulcers are seen. There may be augmented release of gastrin due to stasis.
4. **Gastric Atony:** Initially emptying of the stomach may be normal or near normal and is associated with presence of giant peristaltic waves in barium meal examination. These excessive peristalses result in hypertrophy of stomach wall. After this stomach enters a phase of decompensation where it is grossly dilated and atonic which may be compared to cardiac muscle decompensation which occurs in stenotic valvular heart disease.

Decompensation may be dependent on intracellular shift of K+ and Mg2+. Effortless or profuse vomiting in late stages of obstruction may be well caused by this failure in gastric peristalsis.

Investigations:

1. **Blood examinations:**
   a. **Hb%:** Anemia is present in majority of the patients and is due to under nutrition, hematemesis and malena. It is microcytic, hypochromic due to loss of blood and iron deficiency but in carcinoma stomach where it is megaloblastic where it is due to intrinsic factor deficiency.[9]
   b. **Complete Hemogram:** ESR is raised in all chronic conditions. Hematocrit may be elevated in cases of dehydration. Peripheral smear shows type of anemia.
   c. **Blood group and Rh typing:** Carcinoma stomach is common in blood group A and peptic ulcers in blood group O.[10]
   d. **Liver function tests:** To assess liver function in related cases.
   e. **Serum electrolytes:** It is often overlooked. It may show hypochloremic, hypokalemic, hyponatremic metabolic alkalosis.[17]
2. **Urine:** Initially urine is alkaline which later becomes acidic with large amounts of Na+ and H+. Urine is concentrated with high specific gravity.[17] Albumin, sugar and microscopy are done to rule out associated conditions. Bile salts and bile pigments are done in cases of carcinomas and duodenal tuberculosis.
3. **Stool:** Done to look for occult blood. It may be black in cases of carcinoma stomach.
4. **Chest X-ray:** To know lung secondaries, aspiration pneumonia and for pre-operative lung function assessment.
5. **ECG in all leads:** May show features of hypokalemia and hypocalcemia in late stages.
6. **Saline load test[6]:** It remains the most widely used bedside for estimation of gastric motility since 1965, when introduced by Boyle and Goldstein. Through Ryle's tube 750 ml of saline (at room temperature) is infused over 3-5 minutes and the tube is clamped. Stomach is aspirated off its contents after half an hour. Recovery of more than 400 ml of residual volume suggests gastric outlet obstruction. Stomach should be emptied by nasogastric suction before infusing saline and patient can be put into right lateral position hasten gastric emptying.
   300-400 ml - Possible gastric outlet obstruction.
   200-300 ml - Indeterminate gastric outlet obstruction.
   < 200 ml – Normal.

   Repeat saline load test after giving stomach wash for four days. If residual volume decreases, it may be due to previous pylorospasm or not an organic cause of gastric outlet obstruction.
7. **Barium meal study[18]:** Only flat malignancies in the body of the stomach may go unnoticed. Irregular persistent filling defect with short history suggests malignancy. Three Ds are suggestive of cicatrized duodenal ulcer deformed duodenal cap delayed gastric emptying dilated stomach.

   In decompensation phase the peristaltic waves fade away after 1 to 3 hours with good emptying initially. So, considerable amount (> 50%) of barium remains in the stomach even after 4-6 hours. Pylorospasm may be distinguished by giving IV propantheline bromide after gastric emptying attains normalcy. Double contrast study is considered by many to be superior to endoscopy. Ulcerative growths show an irregular crater with rolled edges of the growth forming a half shadow around the crater (Carman's sign).
8. **Upper GI endoscopy[8,11]:** The likelihood of a positive yield on biopsy is greater than 95% when 6 to 10 tissue samples are obtained in suspected cases of gastric carcinoma. Vital dyes like 0.1% indigocalmin can be sprayed to increase the yield. Cicatrised peptic ulcer may show large voluendoscope cannot be passed.

9. **USG:** Endoscopic Ultrasonography (EUS) has been used to stage the depth of invasion and regional lymphnode extent in potentially curable cancer. It is more accurate than CT scan for staging T and N status.[9]
10. **CT Scan:** Overall accuracy of EUS for assessing all regional lymph nodes is less satisfactory than CT. With newer triphasic spiral CT scanning methods, greater emphasis has been placed on identifying low volume disease and predictinge fluid with irregular and narrowed pylorus through which Tstage. It has an accuracy of 82% for staging T-status in advanced cancer and 15% early gastric cancer and can detect carcinoma pancreas and lymph nodes
11. **Tumor markers:** CEA, CA 19-9, CA 50, CA 72-4, are the important tumor markers to evaluate recurrence and prognosis. Most important is CA 72-4which disappears with surgery and reappears with regional or distant recurrence.
12. **PET Scan:** Whole body FDG-PET scan is being applied increasingly in GI malignancies. It has 100% specificity and 60% sensitivity and 94% accuracy in identifying gastric cancer.
13. **Laparoscopy[19]:** Since CT may identify mets to distant sites (liver, adrenals and ovaries), thus avoiding an operation, CT, EUS and laparoscopy are all considered complementary tests. Laparoscopic ultrasonography, though more invasive than endoscopic ultrasonography, is superior in identifying unsuspected mets in liver and lymph nodes.
14. **Endoscopic Biopsy:** A minimum of three samples should be taken. H. pylori can be stained by Gram technique with same results as that with H and E stain by experienced workers. Special stains are useful for less experienced are modified Giemsa or staining with cresyl fast violet (similar to the technique used to demonstrate sex chromatin Warthin and Starry is a popular technique but requires expertise in staining and is time consuming. A modification of Warthin-Starry (Genta) stain can also be used. In cases of malignancy typing, grading, mitotic activity, mucosal invasion, growth pattern, cellularity, nuclear pleomorphism, necrosis, etc. can be made out.[20]
15. **Immunohistochemistry and Molecular Biology:** These are basically the investigations of research and good tertiary care centers and measure TS, P53, vascular endothelial growth factor, glutathione S-transferase which predict the response and resistance to chemotherapy.
16. **Fasting Serum Gastrin [FSG]:** If Zollinger Ellison's syndrome is suspected FSG is done after 3-10 days of continuous gastric suction. Initially through wide bore NGT (Ewald's) followed by salem sump tube as chronic distension of any cause can give rise to hypergastrinemia and acid hypersecretion.
17. **Urease tests:** Least expensive and excellent screening test for H. pylori with specificity and sensitivity greater than 90%. Agar gel slide tests take 24 hours while Pylori Tek membrane tests take less than two hours.[21]
18. **ELISA:** IgG antibody response to H. pyloric is quantitated and is as sensitive andn specific as biopsy based tests.
19. **Urea breath tests:** A quite accurate non-invasive method of H. pylori detection.

TREATMENT: Careful preparation is important as the surgery in these cases otherwise carries a significant mortality. The presence of dehydration signs suggests an approximate fluid deficit of 4 litres and about 20 gms of NaCl.[22] Provision of sodium allows excretion of alkaline urine, so that alkalosis becomes correctable. The hypokalemic, hypochloremic acidosis is best corrected by administering normal saline containing potassium chloride. When hypokalemia is severe and more than 20 meq of KCl per our needs to be infused. Cardiac rhythm should be monitored continuously. Rarely central venous monitoring becomes necessary, but if it is, the administration of large quantities of KCl centrally must be avoided. In rare cases with severe alkalosis, IV ninfusion of isotonic solution of dilute HCl may be required. The patient should not be given solids and milky drinks should be encouraged. The general condition should be improved by treatment of dental caries, chest physiotherapy, and vitamin supplementation. Nutritional support may be provided by a central line or internally via laparoscopically placed jejunostomy feeding tube. Success is indicated by clinical improvement in the state of hydration, increased urine output, fall in blood urea and hematocrit and normal electrolyte concentration. Repeat saline load test after decompression will gauge the progress and residual volume of less than 200 ml indicates resolution which occurs in about 50% of cases of duodenal ulcers leading to partial gastric outlet obstruction. Definitive diagnosis requires endoscopy which is usually delayed for 3-5 days after decompression. This resolution is due to spasm, edema, inflammation, pyloric dysmotility, etc. rather than malignancy or dense scarring. Full dose intravenous antisecretion therapy followed by H2RA syrup when patient starts taking orally and empties liquids. Emptying of solids is required for better absorption of PPIs. Many surgeons believe that nasogastric suction of 7-10 days is useful to regain gastric tone. Culture of gastric lavage should be done as obstructed stomach is nearly always colonized by bacteria. Gastric lavage to be given the previous night of surgery and is followed by instillation of non-absorbable broad spectrum antibiotics (neomycin and erythromycin base). Intravenous broad spectrum antibiotics should be given in preoperative period.

GASTRIC OUTLET OBSTRUCTION DUE TO CHRONIC PEPTIC ULCER:

A. **Endoscopic Intervention:** About 85% of gastric outlet obstruction cases are amenable to dilatation and 80% of these had immediate relief from the symptoms.[21] Only 40% of these sustained

improvement after three months. The normal pyloric canal is 15-20 mm in diameter and dilates to 25 mm without difficulty. If a standard endoscope (11-12 mm) could not be passed, pyloric stenosis probable. A pyloric ring of less than 6 mm is generally associated with symptoms of gastric outlet obstruction.

a. **Through The Scope (TTS) balloon dilatation:** A well lubricated balloon is passed through the biopsy channel. Balloon is inflated to maximum pressure in stenosed area with water or diluted contrast medium using a pressure gauge. Pressure is maintained for one minute and repeated for 3-4 times. Duodenal bulb and beyond is inspected.
b. **Over the Wire Balloon Dilatation:** A guide wire is advanced far enough in the strictured area. Dilatation is done under fluoroscopic guidance using dilute radiographic contrast medium. Pyloric ring and duodenal bulb are examined endoscopically. A 15 mm balloon diameter is good for the first attempt whereas 18-20 mm may be the best final diameter. If there is any perforation following dilatation, a postprocedure gastrograffin study usually detects perforation. For those with greatly dilated stomach nasogastric suction is continued for 24-48 hours.

B. Surgical Treatment: It involves the relief of obstruction and antiulcer treatment. The choice of operation is between vagotomy and antrectomy and vagotomy and drainage.[23] If the disease in duodenal bulb is not severe and duodenal stump can be closed, vagotomy with antrectomy with Billroth II anastomosis is the procedure of choice. If duodenum appears unsafe for transection, vagotomy and drainage procedure is safer, although ulcer recurrence rates are higher than after transection. Truncal vagotomy and gastroenterostomy conserves the gastric reservoir and can be done with a lower risk. It has 0.5 to 0.9% mortality rate and 6 to 7% ulcer recurrence rate. In this, there is delayed emptying of solids and early emptying of liquids. 75% to 85% of patients were in Visick grade I. Visick score has four grades to assess the quality of life after gastric surgeries.

I - Only fullness after extra meals.
II - Mild, occasional and easily controlled symptoms.
III - Mild and uncontrolled symptoms.
IV - Not improved.

A newer approach is to perform a highly selective vagotomy (HSV) or proximal selective vagotomy with either a pyloroplasty or gastrojejunostomy. This preserves antral mill and reduces the incidence of dumping, gastric stasis and bile reflux gastritis. Early dumping was reported in only 8% of cases by Donahue et al. HSV and pyloric dilatation was performed by McMohan et al. for the first time. Produced superior nutritional status and iron was better absorbed as duodenum was nnot bypassed. It has 0.1% mortality rate, 17% recurrence rate and 15% of patients present with GERD. Problems associated are dysphagia due to dissection of last 7 cm of esophagus and damage to nerve of Latarjet. Simplest procedure to perform through minimally invasive technique is truncal vagotomy and gastrojejunostomy. Gastrojejunostomy can be accomplished through endoscopic stapling device.

Truncal Vagotomy (TV)[25]: The main indication is gastric outlet obstruction with a long-standing history of ulcer symptoms or bleeding or perforation. It should be performed in conjunction with some drainage procedure. Operative complications include injury to spleen, distal esophagus, diaphragm, liver, pancreas, splenic flexure and vessels (intraoperative) delayed emptying, dysphagia (early post-operative), diarrhoea, reflux esophagitis and cholelithiasis (late post-operative). Post-vagotomy diarrhoea is disabling and etiology is unclear. Factors implicated are gastric alkalization, alteration in receptive relaxation and alteration of bile emptying into duodenum. HSV, used in conjunction with finger or endoscopic dilatation involves exposure of stomach and its mobilisation, dissection of both the leaves of lesser omentum and dissection of vagal fibres from nerves of laterjet from 3 cm proximal to GE junction upto 7 cm proximal to pylorus preserving the fibres to antrum.

Drainage Procedures: There are four types of drainage procedures.

1. **Pyloric dilatation:** This can be accomplished endoscopically with a balloon 15 mm in length inflated to 45 psi for 10 minutes or through 3-4 cm gastrotomy by finger. This is advocated in the belief that drainage is required in early post-operative period not permanently.
2. **Pyloromyotomy:** This can be done easily with laparoscopic Truncal vagotomy or SV.
3. **Pyloroplasty:** The pyloric ring division can be done by two methods.
   a. **Heineke Mikulicz Pyloroplasty:** Kocherisation is not always required. Two stay sutures 1 cm apart are placed over pyloric ring on anterior aspect and lumen opened for 3.5 cm on gastric side and 2.5 cm on duodenal side. Giving traction to stay sutures, diamond shaped opening closed transversely.
   b. **Finney's pyloroplasty:** After Kocherisation, an inverted U-shaped incision made through all coats of distal stomach and proximal duodenum and gastroduodenostomy performed.
4. **Gastroenterostomy:** The most dependent part of stomach is anastomosed to jejunum through isoperistaltic, retrocolic, short loop, oblique, posterior, gastro-jejunostomy.

Post-operative Management: Delayed gastric emptying is the major post-operative problem. In very few patients it lasts for more than 5-10 days, rarely for months where obstruction was a long standing one and hence the need

for feeding jejunostomy. After 10-14 days, a gastrograffin swallow is obtained to rule out mechanical obstruction. After three weeks OGD scopy can be done. Prokinetics are not advised. PPIs can be administered via jejunostomy to reduce gastric secretions and gastrostomy tube losses.

GASTRIC OUTLET OBSTRUCTION DUE TO CARCINOMA STOMACH:

Surgical Treatment[8]: The only potentially curative treatment for localised gastric cancer is complete gastric resection. In general surgical resection of gastric cancer involves a wide enough resection to achieve negative margins as well as en bloc resection of lymph nodes and any adherent organs. A gross margin of 6 cm is usually needed to ensure adequate margins by final histological analysis. The signs of inoperability are:

Preoperative signs:

1. Malignant ascites.
2. Jaundice.
3. Gross cachexia.
4. Secondaries in rectovesical or rectouterine pouch (Blumer's shelf).
5. Sister Mary Joseph's nodules.
6. Krukenburg tumour.
7. Secondaries in left supraclavicular lymphnodes.

Per-operative signs:

1. > H2
2. > P2
3. ≥ N4
4. Omental deposits

Growth is resectable even if it has involved pancreas, mesocolon or transverse colon but unresectable if portal vein, aorta, base of mesentery, hepatic or celiac artery is involved. Cure is possible in cases with H0, P0, negative cut margins and D number more than N number. The specific type of resection will depend upon on the location, stage, and pattern of spread of the particular tumor.

Proximal Tumors: Proximal tumors and tumors of gastro esophageal junction comprise 35% to 0% of all gastric cancers and require different considerations for resection and reconstruction.

Patients with type 2 or 3 tumors can be resected by either a total gastrectomy or a proximal subtotal gastrectomy. Total gastrectomy may offer an advantage in that patients are unlikely to have reflux esophagitis after a total gastrectomy and Roux-en- Y reconstructon.

Midbody Tumors: Mid-stomach tumors comprise 15% to 30% of all gastric cancers because adequate margins often leaves very little residual stomach, a total gastrectomy is usually required.

Distal Tumors: For tumors of the distal stomach, which comprise about 35% of gastric cancers, a distal sub-total gastrectomy is performed. In general 5-6cms gross margin is recommended to ensure a microscopically negative resection margin.

Lymphadenectomy: The extent of lymph node dissection is designated by 'D'.

- **D1:** only perigastric lymph nodes.
- **D2:** nodes along the hepatic,left gastric celiac and spleenic arteries as well as those in the splenic hilum in addition to perigastric nodes more than 3 cms from the primary tumor.
- **D3:** nodes along the porta hepatic retro-pancreatic and peri aortic regions.

Reconstruction after gastrectomy[24]: Common options for reconstruction after subtotal gastrectomy include duodenostomy [Billroth-I], an antecolic or retro colic gastro jejunostomy [Billroth-II], or an antecolic Roux-en-Y gastro jejunostomy. After a total gastrectomy, options for reconstruction include a standard Roux-en-Y oesophago jejunostomy, construction of a pouch or jejunal interposition

RARE CAUSES OF GASTRIC OUTLET OBSTRUTION IN ADULTS:

A. Gastroduodenal Tuberculosis[25]: Usually the ulcer occurs at the junction of first and second part of the duodenum. Isolated duodenal tuberculosis, without involvement of other parts of GIT does occur, though infrequently. It occurs in two forms. 1. True form: Primarly affecting pylorus and resulting in fibrosis. 2. False form: Obstruction is caused by extrinsic pressure by involved lymph nodes. Mesenteric, peripancreatic and paraoartic lymphnodes may show caseation. Usually this is associated with pulmonary or intestinal tuberculosis whose clinical features may be elicited. Antitubercular therapy is only that is required in partial obstruction. Gastrojejunostomy is advised for complete obstruction while distal partial gastrectomy if multiple tubercular ulcers are present.

B. Corrosive Strictures: In most of the cases of corrosive poisoning, esophagus escapes from the effect of corrosives and gastric outlet obstruction develops in about 1-6 weeks after ingestion of corrosives. Hydrochloric acid, nitric acid, sulphuric acid, carbolic acid, ferrous sulphate, copper sulphate, formaldehyde, tincture iodine, etc. are the corrosives causing obstruction. Surgery is treatment of choice. Gastrojejunostomy is preferred. Partial gastrectomy can also be done.

C. Post-operative Adhesions: The common surgery causing post-operative adhesion leading to gastric outlet obstruction, is cholecystectomy because of the formation of band between pyloroduodenal junction and liver surface resulting in kinking. The obstruction may be partial or complete. There will be history of surgery and one may find operative scar over the abdomen. Patient may give history of acute abdominal pain suggesting obstruction.

Adhesiolysis is enough except when an organic stenosis present where gastrojejunostomy or partial gastrectomy is done.

D. Crohn's Disease: A rare cause of gastric outlet obstruction usually associated with ileal disease.

General features of gastric outlet obstruction may be accompanied with intermittent pyrexia, diarrhoea and steatorrhoea. Resection of the segment if it is short or else bypass is a satisfactory procedure.

E. Pseudopancreatic Cyst[26]: Most of the times it is found in lesser sac behind the stomach. When large enough, causes obstruction to gastric outlet by mechanical pressure. Patient may be an alcoholic. On examination, a mass may be felt in epigastric region, may move with respiration. It is round, smooth, usually tense, and resonant as it is covered by stomach. Surgical treatment is required in patients who continue to have symptoms even after 3-4 weeks of medical line of management to reduce inflammation and edema of duodenal wall. Obstruction may be relieved by release of inflammatory adhesions around the duodenum or drainage of pseudocyst. Obstruction due to chronic pancreatitis is treated by vagotomy or gastro-jejunostomy or resection. Gastrojejunostomy is enough to relieve duodenal obstruction. Vagotomy is added if a patient has a past history of acid peptic disease.

F. Post-radiation Gastritis: Radiation tolerance of stomach is high and gastritis occurs occasionally. Usually 4000 rads are delivered over a period of 3-4 weeks. If it exceeds, then radiation gastritis occurs and may lead to gastric ulceration, fistula or antral stenosis.

G. Other malignancies of stomach:

1. Benign tumors[27]: These account for less than 2% of all gastric neoplasms.

I. Epithelial polyps:

i. Adenomatous polyps: These are common in antrum.

ii. Hyperplastic polyps: These are distributed throughout the stomach and constitute for 75% of all gastric epithelial polyps. Atrophic gastritis is usually associated with hyperplastic polyps. Epithelial polyps may present with epigastric pain, hematemosis or malena. Gastric leiomyoma presents with hematemesis or malena. Bleeding from tumour may be massive or intermittent.

II. Benign smooth muscle tumors: These are common in distal stomach and obstruction may be produced by prolapse of the tumour into duodenum. Though fibromas, schwannomas, neurofibromas, lipomas, etc. occur leiomyomas are the commonest.

III. Gastric carcinoids: These account for less than 2% of all carcinoid tumours as compared with 70% in appendix and 20% in ileum and caecum.

2. Malignant tumors:

I. Malignant Smooth Muscle Tumours: These comprise of 1-2% of all malignant lesions of stomach. The common conditions are leiomyoblastoma and leiomyosarcoma. Other mesenchymal tumours are neurofibrosarcoma, Kaposi's sarcoma, fibrosarcoma, hemangiopericytoma and rhabdomyosarcoma. Distal subtotal radical gastrectomy is performed for distal lesions and adjuvant CT in metastatic disease with cyclophosphamide, dacarbazine, vincristin and adriamycin.

II. Gastric lymphomas: Primary gastric lymphoma is the commonest site of extranodal NHL and constitutes 2% of all gastric malignancies. They are common in posterior wall and along lesser curvature. Abdominal pain occurs in more than 80% of patients. It may be associated with early satiety, nausea, night sweats, fever, haemorrhage. On examination, a mass may be felt in left upper abdomen, spleenomegaly may be present. Distal subtotal radical gastrectomy is performed if the lesion is present in body or antrum. Spleen is removed at all operations and needle biopsy specimen taken from liver. Following histologic proof, radiation therapy is used as primary treatment in some centers. Chemotherapy is used when lymph nodes are positive or if there is evidence of systemic disease.

H. Duodenal Malignancies: Carcinoma of first part of duodenum is rare. The incidence is maximum at periampullary region. Both ulcerative and papilliferous growths can cause duodenal obstruction. Other tumours of duodenum are carciniods, melanomas and sarcomas, which are still rarer. Though they are rare may present with obstruction as opposed to periampullary carcinoma which slough off before obstruction is developed. On examination there may be jaundice and palpable mass in right upper quadrant. Pancreaticoduodenectomies are performed when cure is possible. Palliative procedures include duodenoduodenostomy, duodenojejunostomy, gastrojejunostomy and cholecystojejunostomy.

I. Carcinoma head of the pancreas: Accounts for about 5% of cancer related deaths. Two-thirds of the cases present with painless, progressive jaundice. Usually associated with pruritis, pale coloured stools and dark coloured urine. Epigastric pain may radiate to back due to involvement of coeliac and mesenteric plexus. In 30% of cases gall bladder may be palpable and liver in 50% of cases. In cases of splenic vein thrombosis, spleen may be enlarged. Exocrine insufficiency due to duct obstruction may result in malabsorption and steatorrhoea. Glucose intolerance is present in the majority of patients with pancreatic cancer, because of altered β-cell function and impaired tissue insulin sensitivity. Symptoms of gastric outlet obstruction are due to external compression or by direct invasion of pyloric antrum or second part of duodenum. Whipple's pancreaticoduodenectomy is done if the growth is resectable. Otherwise cholecystojejonostomy to relieve biliary obstruction and gastrojejunostomy to relieve gastric outlet obstruction.

J. Miscellaneous Causes of Gastric Outlet Obstruction:

I. Adult pyloric stenosis[28]: A rare condition whose relationship to childhood condition is unclear, although some patients have a long history of problems with gastric emptying. Normal pyloric canal measures 0.5 to 1 cm in length and thickness of its musculature ranging from 3 to 9 mm with an average of 5.5 mm. The longitudinal muscles are not affected by the disease. There may be moderate thickening due to effects of obstruction. Finney's or Heineke-Mikulicz pyloroplasty is advised. Ramstedt's pyloromyotomy has the disadvantage of producing a diverticulum. When diagnosis is in doubt, limited partial gastrectomy with a Billroth I type anastomosis is done.

II. Duodenal ileus: Also called as Wilkie's syndrome, gastromesenteric ileus, arteriomesenteric ileus, superior mesenteric artery syndrome. Normal aortomesenteric angle is 50°-60° and distance between them is 10 mm to 20 mm. In duodenal ileus these averages are 18° and 2-5 mm. Thus increased lordosis, a short mesentery, narrow aorto-mesenteric root, short length and high attachment of ligament of Treitz, marked loss of weightmay decrease aorto-mesenteric angle. Acute presentation is less common and may be precipitated by application of plaster cast or bedrest in supine position. Chronic duodenal ileus presents with epigastric pain, fullness after meals and foul eructations. Symptoms may get relieved on knee-chest position or left lateral position. Patient may be thin and asthenic. Epigastric pulsations may be present. Conservative treatment is successful in most cases associated with orthopedic conditions. In chronic SMA syndrome, if the patient fails to respond to conservative treatment (turning to prone or knee-elbow position after meals, prokinetic drugs and weight gain) duodenojejunostomy is the treatment of choice. Gastrojejunostomy plays no role because of high incidence of stomal ulceration.

K. Bezoars: Phytobezoars may occur in patients after gastric surgery (because of reduced peristatisis, hypochlorhydria, inadequate chewing, high fibre intake, loss of gastric pump mechanism. Trichobezoars (hair balls) are unusual and are virtually exclusively found in female psychiatric patients, often young. Epigastric mass is characteristic finding in bezoars. Bezoars can be broken of endoscopically by laser lithotripsy or mechanically and allowed to pass through. ESWL and enzymatic (cellulase) fragmentation are also used. Large trichobezoars need gastrotomy.

L. Foreign Bodies (FBs): Usually long foreign bodies get stuck at gastric outlet as they cannot get through fixed curvature. These could have been swallowed purposefully or accidentally. Large foreign bodies, which fail to negotiate through gastric outlet may present features of obstruction due to ball-valve mechanism, while long foreign bodies obstruct the outlet mechanically and there may be ulceration and pylorospasm. May present with hematemesis and malena. Indications for active treatment are;

1. Failure to progress.
2. Signs of penetration or actual perforation.
3. Objects unlikely to move-on.
4. Large number of F Bs.
5. Evident GI haemorrhage.

Many of the retained F Bs can be removed through OGDscope after they are protected by a sheath. In surgical intervention, gastrotomy or puncture in cases of slender and sharp objects which are closed after a preoperative radiogram, when retrieval of all the F Bs is dubious.

M. Bouveret's syndrome: This is the impaction of gall stones at duodenal bulb. It is more common in elderly women. Vomiting is bilious in nature and vomitus may contain gall stones along with other features of cholelithiasis. Endoscopic fragmentation or retrieval is advised. If surgical intervention is planned, duodenolithotomy, division of fistula and cholecystectomy is the treatment of choice.

SUMMARY:

1. The most common cause of gastric outlet obstruction is carcinoma stomach with antral growth in 53.3% producing GOO in the study
2. Number of cases with cicatrized duodenal ulcer causing GOO were 43.3%
3. One case of stenosis following corrosive acid ingestion was also present as the reason for GOO in th study 3.3%
4. Males are more commonly affected than female and the male female ratio is 7:1 in malignancy
5. This study was undertaken in an adult rural population
6. The most common presenting complaints were vomiting (100%), abdominal pain (90%) and loss of appetite (90%). In malignant cases loss of weight (93%) was also a common complaint.
7. Visible gastric peristalsis and succession splash were less prominent in malignant cases when compared to stenosing duodenal ulcer cases.
8. 31% of the malignant cases presented with mass in the upper abdomen
9. Only 5 cases of malignancy could be able to undergo definite surgical procedure. All others underwent palliative procedures.
10. The surgical procedure undertook in the cicatrized duodenal ulcer patients were truncal vagotomy and posterior GJ and there were no recurrence of symptoms in any of the casers which turned up for follow up.
11. The mortality rate was 18.8% in malignant cases

The incidence of GOO secondary to cicatrized duodenal ulcer has been reduced probably due to increased awareness of the disease, change in the dietary habits, and availability of H2 receptor blockers and Proton Pump Inhibitors eradication of H pylori with kits.

12. The availability of flexible fiber optic endoscope has helped the surgeon to know the cause of obstruction pre operatively and biopsy taken from the suspected lesion can confirm the diagnosis also.

CONCLUSION:

1. The most common cause of gastric outlet obstruction in adults are carcinoma stomach with antral growth producing GOO in 53.3% and cicatrized duodenal ulcer causing GOO were 43.3%.
2. In the vast majority of cases the diagnosis can be established clinically.
3. The saline load test was found to be an effective bedside investigations to assess the degree of GOO.
4. Upper GI endoscopy should be mandatory in all suspected cases of GOO.
5. Number of cases with cicatrized duodenal ulcer as the chief etiological factors for GOO is diminishing and the number of cases of antral carcinoma of stomach as the cause of GOO is increasing.
6. Effective treatment in carcinoma stomach depends on early diagnosis.

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