"A PATHOLOGICAL STUDY ON GASTRIC OUTLET OBSTRUCTION"

Dissertation submitted in partial fulfillment of requirements for

M.S. DEGREE IN GENERAL SURGERY

Of

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CERTIFICATE

This is to certify that this dissertation titled "A PATHOLOGICAL STUDY OF GASTRIC OUTLET OBSTRUCTION" is a bonefide work of Dr. P. Selladurai, and has been prepared by him under my guidance, in fulfillment of regulations of The Tamilnadu Dr.M. G. R. Medical University,for the award of M.S. degree in General Surgery in the year 2012.

Prof. Dr. S. Soundararajan M.S.,

Place: Tirunelveli

Date:

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DECLARATION

I solemnly declare that this dissertation entitled "A PATHOLOGICAL STUDY ON GASTRIC OUTLET OBSTRUCTION." was done by me at Tirunelveli Medical College and Government Hospital, during the academic year 2010-2011 under the guidance and supervision of Prof.R.MAHESWARI M.S., This dissertation is submitted to the Tamil Nadu Dr.M.G.R. Medical University towards the partial fulfillment of requirements for the award of M.S. Degree in General Surgery.

Place: Tirunelveli

Signature of the candidate

Date:

TABLE OF CONTENTS

AIM

1. Age and sex incidence of gastric outlet obstruction.
2. Pathological study on causes of gastric outlet obstruction.
3. Correlate the results of upper gastrointestinal endoscopy, biopsy and peroperative findings in gastric outlet obstruction.

A PATHOLOGY STUDY ON GASTRIC OUTLET OBSTRUCTION

INTRODUCTION

Gastric outlet obstruction is due to obstruction in first part of duodenum at the site of chronic scarring from ulceration or antrum where a benign gastric ulcer (type II and type III) or carcinoma is a problem.

Stenotic complications of peptic ulcer disease are hour glass deformity and tea pot deformity (gastric ulcer).

Stenotic complications arise from repeated cycles of ulceration and healing resulting in dense fibrosis with narrowing and deformity.

Common causes of gastric outlet obstruction are

1. Chronic duodenal ulceration / fibrosis,

2. Antral gastric carcinoma,

3. Carcinoma of the head of pancreas.

Rare causes of gastric outlet obstruction

1. Lymphomas,

2. Crohn’s disease,

3. Duodenal haematoma,

4. Adult pyloric hypertrophy,

5. Annular pancreas,

6. Mucosal diaphragm,

7. Megaduodenum,

8. Arteriomesenteric compression (Wilke’s disease).

Clinical features of gastric outlet obstruction

a. Abdomen pain,
b. Upper abdominal discomfort,
c. Vomiting (effortless, projectile, absence of bile, presence of partially digested food eaten hours or days previously),
d. Constipation.

SIGNS

1. Malnourished,
2. Dehydrated,
3. Pallor,
4. Succussion splash,
5. Visible peristalsis,
6. Dilated stomach.

Metabolic features: Hypochloraemic, hyponatremic, hypokalemic, metabolic alkalosis. In late stage metabolic alkalosis with paradoxical acid urine occurs.

The two common causes of gastric outlet obstruction are

1. Gastric cancer,

2. Pyloric stenosis secondary to peptic ulceration.

ANATOMY

STOMACH

Stomach is the widest part of the alimentary tract and lies between the oesophagus and the duodenum. The mean capacity of the stomach increases from approximately 30 ml at birth, to 1000 ml at puberty, and approximately 1500 ml in adults.

PARTS OF THE STOMACH

The stomach is divided into fundus, body, pyloric antrum and pylorus.

VASCULAR SUPPLY AND LYMPHATIC DRAINAGE

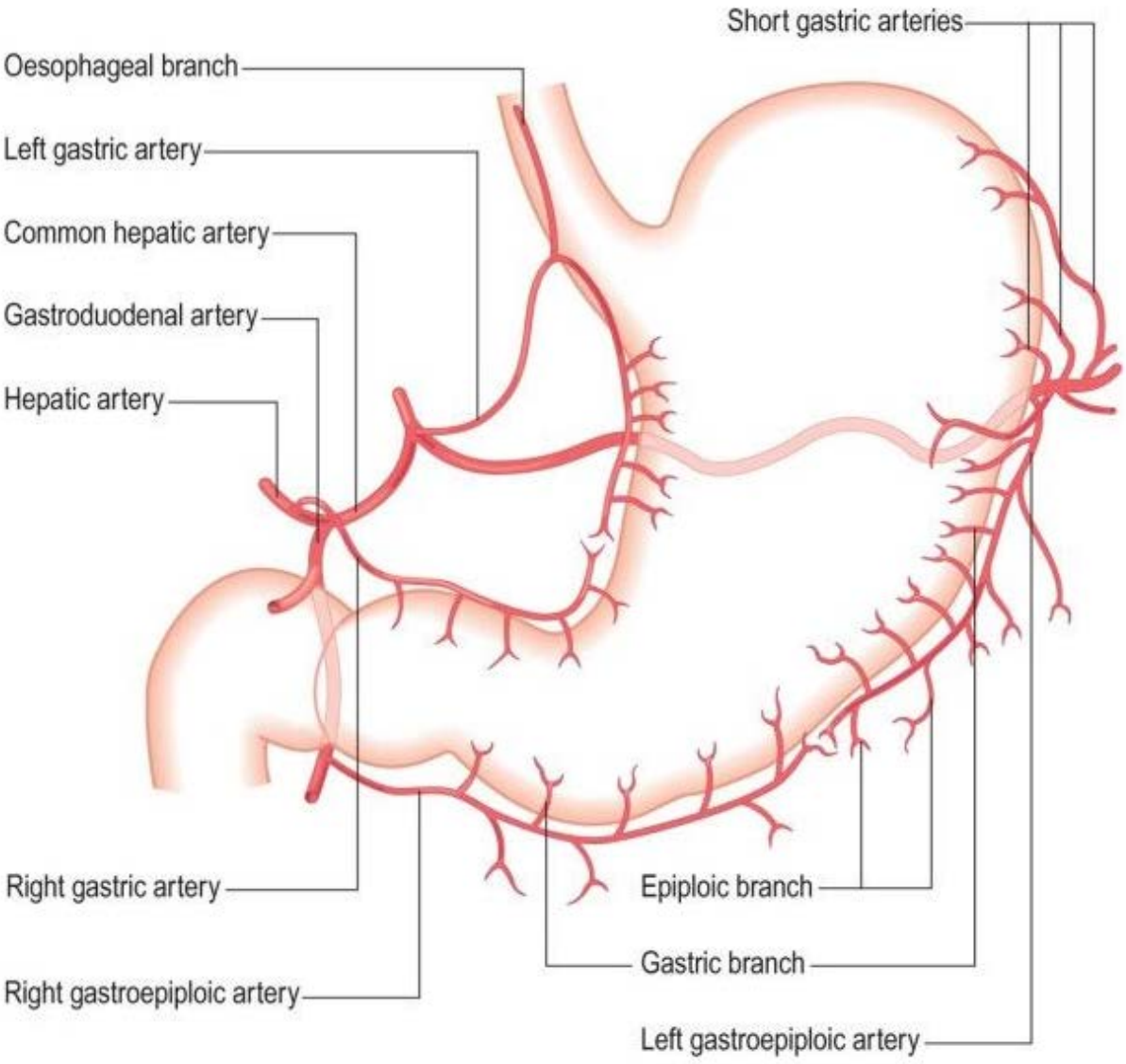
Arteries: The arterial supply to the stomach is from

1. The coeliac axis

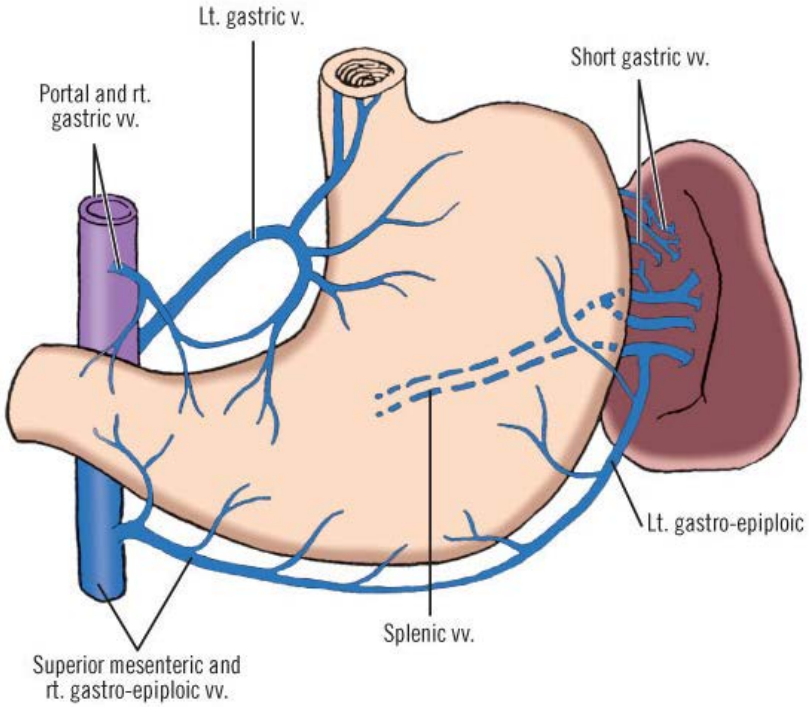
2. The left gastric artery arises directly from the coeliac axis.

3. The splenic artery gives origin to the short gastric arteries and the left gastroepiploic artery.

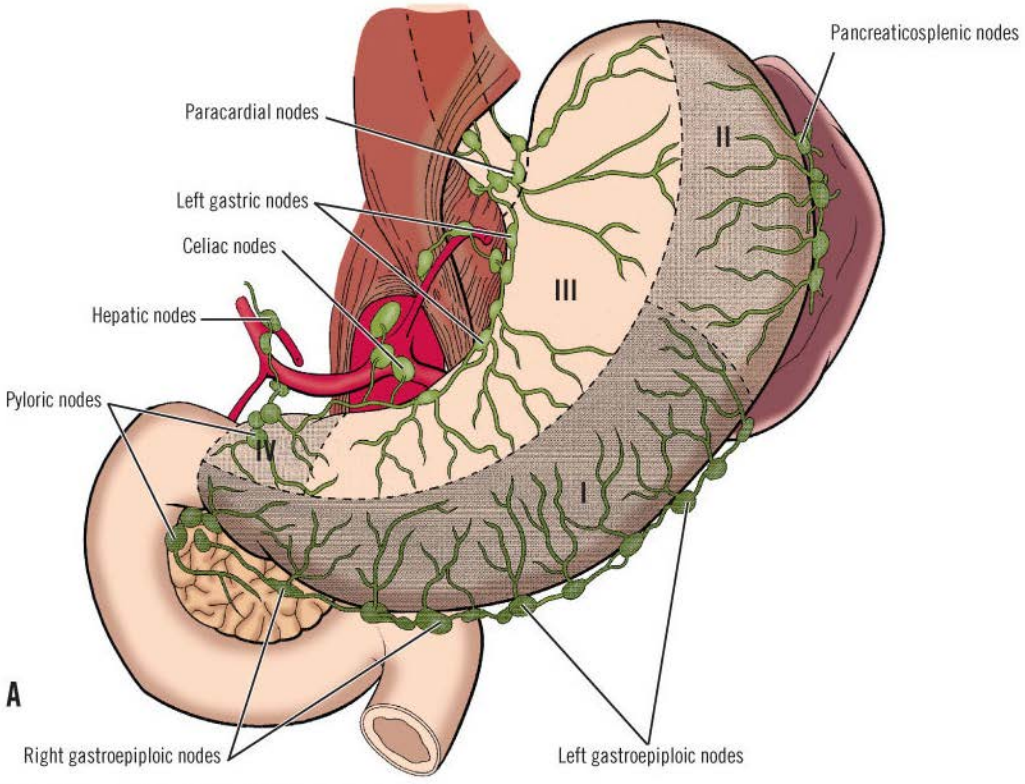
4. The hepatic artery gives origin to the right gastric artery and to the gastroduodenal artery, which in turn gives origin to the right gastroepiploic artery.



Arterial supply of stomach



Venous drainage of stomach



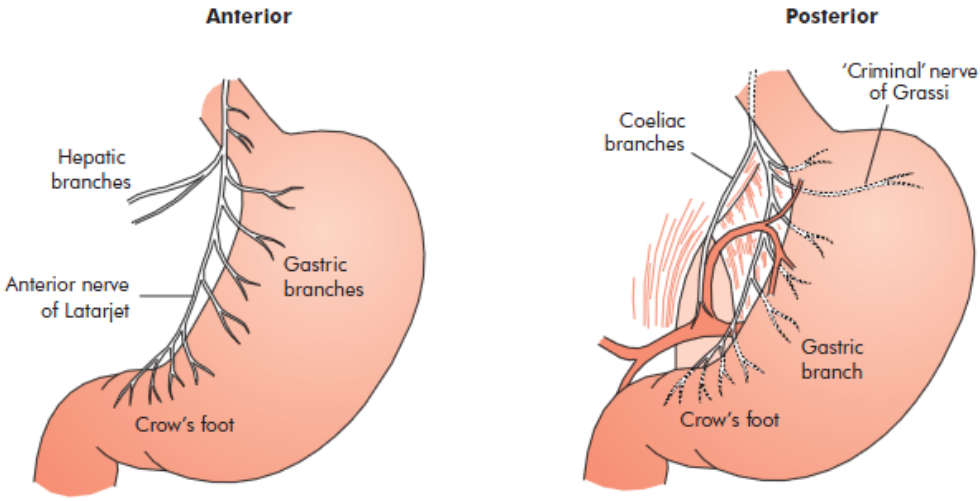
Lymphatics of stomach

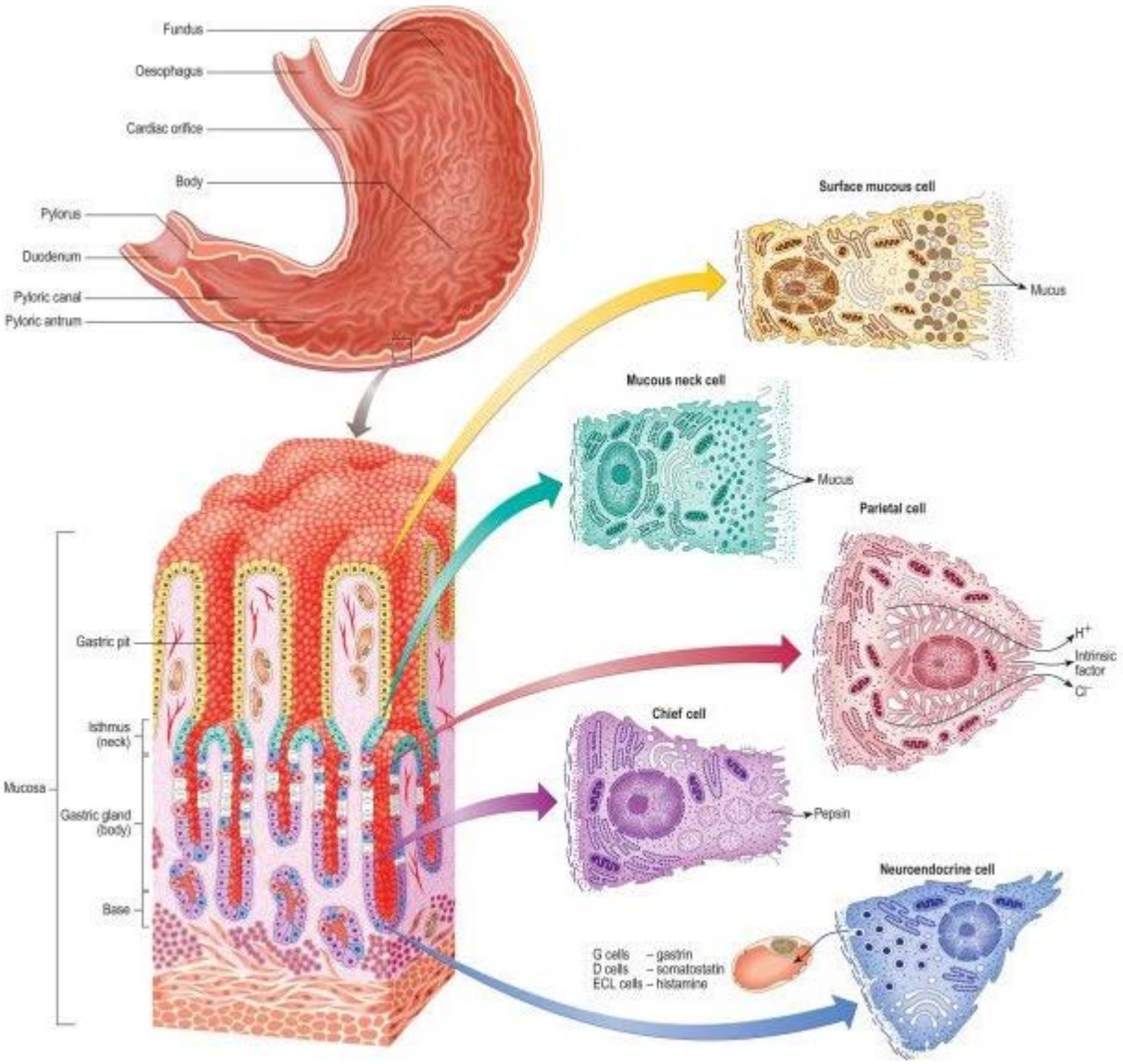
INNERVATION

The stomach is innervated by sympathetic and parasympathetic fibres.

Sympathetic innervations: The sympathetic supply to the stomach originates from the fifth to 12th thoracic spinal segments, and is mainly distributed to the stomach via the greater and lesser splanchnic nerves via the coeliac plexus. Additional innervation comes from fibres of the hepatic plexus. The gastric sympathetic nerves are vasoconstrictor to the gastric vasculature and inhibitory to gastric musculature. The sympathetic supply to the pylorus is motor, and brings about pyloric constriction. The sympathetic supply also conducts afferent impulses that mediate sensations, including pain.
Parasympathetic innervations: The parasympathetic supply to the stomach is from the anterior and posterior vagus nerves . The anterior vagus formed mainly from fibres from the left vagus originating from the oesophageal plexuses. The nerve divides near the oesophageal end of the lesser curvature into gastric and pyloric/hepatic branches. The upper anterior gastric branches radiate on the anterior surface of the upper body and fundus.

Anatomy of the anterior and posterior vagus nerves in relation to the stomach.



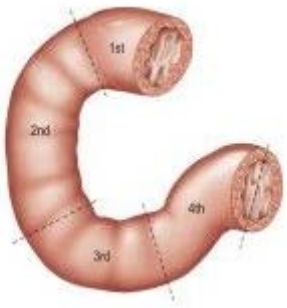


Secretion of acid

DUODENUM

The adult duodenum is 20–25 cm long and is the shortest, widest part of the small intestine.It is only partially covered by peritoneum, the proximal 2.5 cm is intraperitoneal, and the remainder is retroperitoneal. The head and uncinate process of the pancreas lie within the concavity of the duodenum .

The duodenum lies entirely above the level of the umbilicus and is described as having four parts.



FIRST (SUPERIOR) PART: The first, and most mobile, part of the duodenum is about 5 cm long. It starts as a continuation of the duodenal end of the pylorus and ends at the superior duodenal flexure. The first 2 or 3 cm have a bland internal mucosal appearance and referred as the duodenal ‘cap'.

SECOND (DESCENDING) PART :

The second part of the duodenum is 8–10 cm long. The common bile duct and pancreatic duct enter the medial wall obliquely and usually unite to form the common hepatopancreatic ampulla. The narrow distal end opens on the summit of the major duodenal papilla (ampulla of Vater), which is situated on the posteromedial wall of the second part, 8–10 cm distal to the pylorus.

THIRD (HORIZONTAL) PART:The third part of the duodenum is approximately 10 cm long.

FOURTH (ASCENDING) PART: The fourth part of the duodenum is 2.5 cm long.

VASCULAR SUPPLY AND LYMPHATIC DRAINAGE

Arteries:

Duodenum are supplied by the superior and inferior pancreaticoduodenal arteries. The first and second parts also receive contributions from the right gastric, supraduodenal, right gastroepiploic, hepatic and gastroduodenal arteries. Branches of the superior pancreaticoduodenal artery supply the pyloric canal.

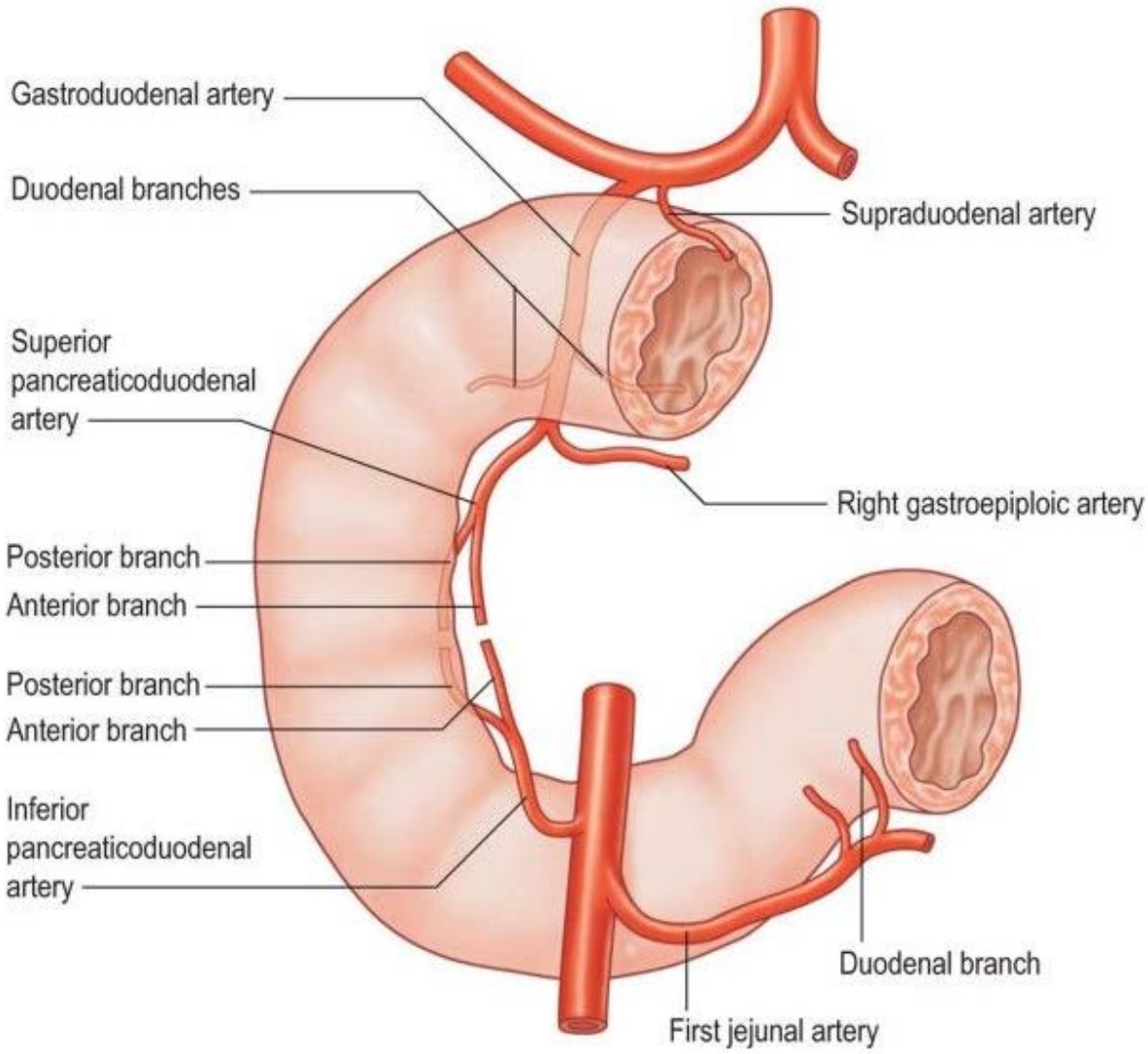
Veins

The duodenal veins drain ultimately into the portal vein.

Lymphatics

Duodenal lymphatics run to anterior and posterior pancreatic nodes that lie in the anterior and posterior grooves between the head of the pancreas and the duodenum: and drain into

the supra-pyloric, infrapyloric, hepatoduodenal, common hepatic and superior mesenteric nodes.



INNERVATION

The duodenum is innervated by both parasympathetic and sympathetic neurones.Preganglionic sympathetic axons originate from neurones in the intermediolateral columns of the grey matter in the fifth to the 12th thoracic spinal segments. They travel via the greater and lesser splanchnic nerves to the coeliac plexus where they synapse on neurones in the coeliac ganglion.postganglionic axons are distributed to the duodenal wall via

periarterial plexuses on the branches of the coeliac axis and superior mesenteric artery. The sympathetic nerves are vasoconstrictor to the duodenal vasculature and inhibitory to duodenal musculature.

The preganglionic parasympathetic supply is carried by vagal axons that are distributed via the coeliac plexus and which synapse on neurones in the duodenal wall. The parasympathetic supply is secretomotor to the duodenal mucosa and motor to the duodenal musculature. The sympathetic nerves are vasoconstrictor to the duodenal vasculature and inhibitory to duodenal musculature.

PHYSIOLOGY

The stomach stores food and facilitates digestion through a variety of secretory and motor functions.

Acid Secretion

PARIETAL CELL

The parietal cell is stimulated to secrete acid when one or more of three membrane receptor types is stimulated by

Acetylcholine (from vagal nerve fibers),

Gastrin (from D cells), or

Histamine (from ECL cells).

The enzyme H+ /K+ -ATPase is the proton pump and is the final common pathway for gastric acid secretion.

PHYSIOLOGIC ACID SECRETION

Food ingestion is the physiologic stimulus for acid secretion . The acid secretory response that occurs after a meal is described in three phases: cephalic, gastric, and intestinal.

CEPHALIC PHASE:

The cephalic or vagal phase begins with the thought, sight, smell, and/or taste of food. These stimuli activate several cortical and hypothalamic sites . The cephalic phase accounts for no more than 30% of total acid secretion in response to a meal.

GASTRIC PHASE:

When food reaches the stomach, the gastric phase of acid secretion begins and accounts for about 60% of the total acid secretion in response to a meal. Amino acids and small peptides directly stimulate antral G cells to secrete Gastrin. Proximal gastric distention stimulates acid secretion via a vagovagal reflex arc.

INTESTINAL PHASE:

This phase starts when gastric emptying of ingested food begins, and continues as long as nutrients remain in the proximal small intestine. It accounts for about 10% of meal induced acid secretion. Interprandial basal

acid secretion is 2 to 5 mEq hydrochloric acid per hour, about 10% of maximal acid output (MAO), and it is greater at night.

Pepsinogen secretion

The stimulus for pepsinogen secretion from chief cells is food ingestion; acetylcholine is the most important mediator.

Pepsinogen I is produced by chief cells.

Pepsinogen II is produced by SECs.

Intrinsic Factor

Activated parietal cells secrete intrinsic factor. Intrinsic factor binds to luminal vitaminB12, and the complex is absorbed in the terminal ileum via mucosal receptors.

Gastric Mucosal Barrier (mucosal defences) :

Mucous barrier

Bicarbonate secretion

Epithelial barrier

Hydrophobic phospholipids

Tight junctions

Restitution

Microcirculation (reactive hyperemia)

Afferent sensory neurons

Mediators:

Prostaglandins

Nitric oxide

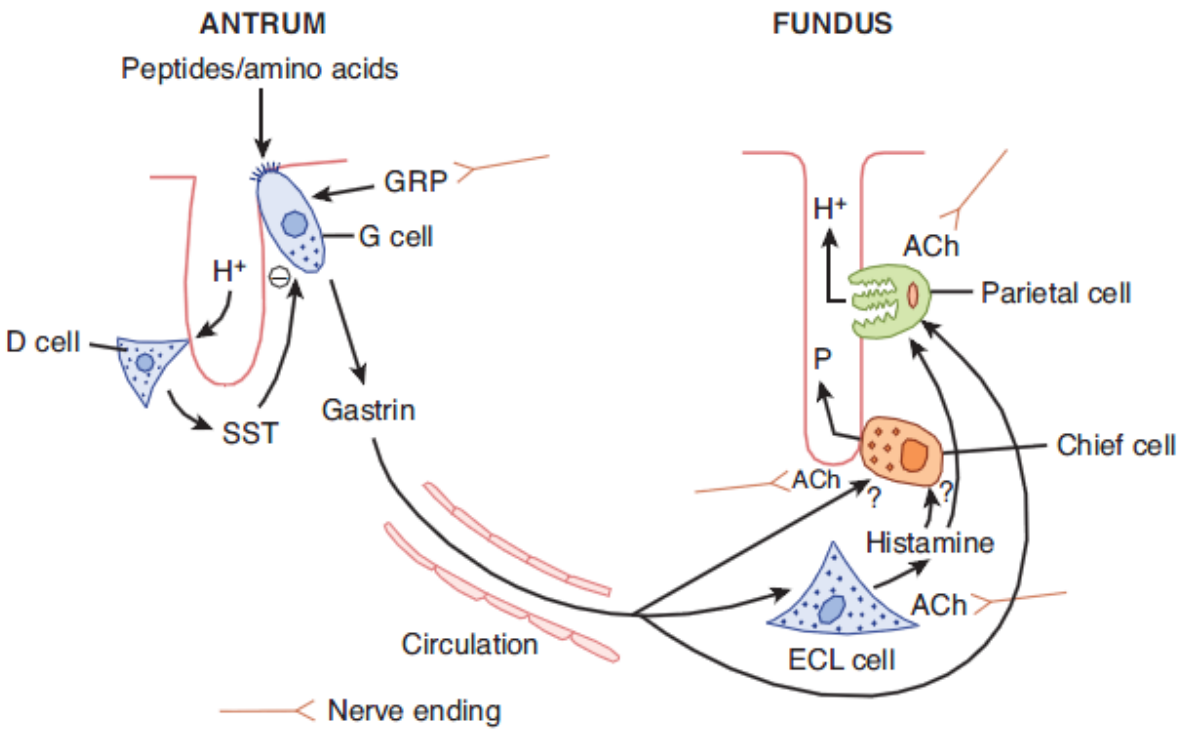
Epidermal growth factor

Calcitonin gene-related peptide

Hepatocyte growth factor

Histamine

Gastrin-releasing peptide



Regulation of gastric acid secretion

INTRINSIC GASTRIC INNERVATION

The intrinsic innervation consists of ganglia and nerves that constitute the enteric nervous system.

Excitatory neurotransmitters:

1. Acetylcholine, 2. Tachykinins,

3. Substance P, and

4. Neurokinin A.

Inhibitory neurotransmitters: 1. nitric oxide (NO) and

2. Vasoactive intestinal peptide (VIP).

Serotonin has been shown to modulate both contraction and relaxation. Specialized cells in the muscularis propria are interstitial cells of Cajal , amplify both cholinergic excitatory and nitrergic inhibitory input to the smooth muscle of the stomach and intestine.

SEGMENTAL GASTRIC MOTILITY

The proximal stomach regulates basal intra gastric tone, and the distal stomach mixes and grinds the food. The pylorus facilitates retropulsion of the solid food bolus back into the body of the stomach for additional breakdown. The pylorus intermittently allow emptying of liquids and small solid particles into the duodenum.

When food is ingested, intragastric pressure falls as the proximal stomach relaxes. This proximal relaxation is mediated by two important vagovagal reflexes: receptive relaxation and gastric accommodation.

Receptive relaxation is the reduction in proximal gastric tone associated with the act of swallowing.

Gastric accommodation is the proximal gastric relaxation associated with distention of the stomach.

Slow waves of myoelectric depolarization sweep down the distal stomach at a rate of about three per minute.

These waves originate from the proximal gastric pacemaker, high on the

greater curvature. During fasting, distal gastric motor activity is controlled by the migrating motor complex (MMC), the "gastrointestinal housekeeper". The MMC lasts approximately 100 minutes (longer at night, shorter during daytime) and is divided into four phases.

Phase I is a period of relative motor inactivity.

Phase II consists of some irregular, high amplitude, non propulsive contractions.

Phase III, a period of intense, regular, propulsive contractions.

Phase IV is a transition period.

GASTRIC EMPTYING (LIQUID EMPTYING)

The gastric emptying of water or isotonic saline follows first-order kinetics, with a half emptying time around 12 minutes. This emptying pattern of liquids is modified considerably as the caloric density, osmolarity, and nutrient composition of the liquid changes. Up to an osmolarity of about 1 M, liquid emptying occurs at a rate of about 200 kcal per hour.

SOLID EMPTYING

Normally, the half-time of solid gastric emptying is <2 hours. Solids have an initial lag phase during which little emptying of solids occurs followed by a linear emptying phase follows, during which the smaller particles are metered out to the duodenum.

PATHOPHYSIOLOGY

DUODENAL ULCER

Decreased duodenal bicarbonate secretion: 70%

Increased nocturnal acid secretion: 70%

Increased duodenal acid load: 65%

Increased daytime acid secretion: 50%

Increased pentagastrin stimulated maximal acid output: 40%

Increased sensitivity to gastrin: 35%

Increased basal gastrin: 35%

Increased gastric emptying: 30%

Decreased pH inhibition of gastrin release: 25%

Increased postprandial gastrin release: 25%

Modified Johnson Classification of Gastric Ulcers

Type	Location	Secretion
I	Lesser curvature	Low
II	Body of the stomach and duodenum	High
III	Prepyloric (within 2-3 cm of the pylorus)	High
IV	High on the lesser curve, near gastroesophageal junction	Low
V	Anywhere, induced by medication	Low

Treatment of uncomplicated peptic ulcers

The aim of treatment is

(i) to relieve symptoms;

(ii) to heal the ulcer as quickly as possible;

(iii) to prevent recurrence;

(iv) to prevent complications.

The classic triple therapy of 'bismuth', metronidazole, and amoxycillin (amoxicillin) for 2 weeks eliminates H. pylori in 90 per cent of patients.
A proton-pump inhibitor, with metronidazole 400 mg and amoxycillin 500 mg three times a day, results in eradication rates of 85 to 95 per cent in 1 week.
If patients are allergic to amoxycillin, clarithromycin may be substituted; this antibiotic combined with ranitidine–bismuth citrate heals 93 per cent of patients.
For the 5 to 15 per cent that do not respond to the proton-pump inhibitor, bismuth should be added and the course extended for 2 weeks.

QUDRAPLE THERAPY

SURGERY

The goals of surgical procedures are to

1. Permit ulcer healing,

2. Prevent or treat ulcer complications,

3. Address the underlying ulcer etiology,

4. Minimize postoperative digestive consequences.

SURGERY FOR INTRACTABLE DUODENAL ULCER

Vagotomy:

1. Truncal Vagotomy: involves resecting a 2- to 3-cm section of the anterior and posterior nerve trunks between the gastroesophageal junction and diaphragm.
2. Selective Vagotomy: involves a bilateral vagotomy distal to the branching of the hepatic and celiac vagal branches.
3. Highly selective Vagotomy: involves dividing the vagal branches to the fundus and body of the stomach while preserving the motor branches to the antrum and pylorus.

Surgical techniques

Intractable gastric ulcer :

Type I:

Partial gastrectomy and Billroth I or Ulcer excision and Highly selective vagotomy.

Type II

Truncal vagotomy and antrectomy or Highly selective vagotomy.

Type III

Truncal vagotomy and antrectomy

Type IV

If ulcer within 2 cm of GE junction, Kelly-Madlener or Csendes procedure,

For more distal lesions, Pauchet's procedure

TYPE V

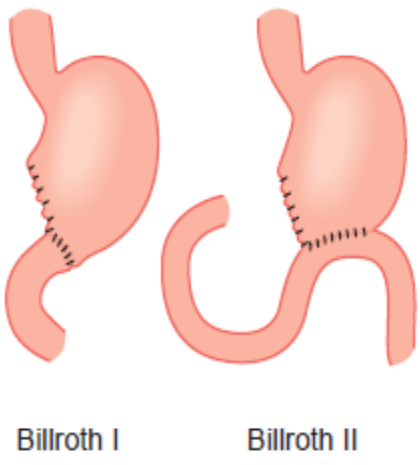
If medication cannot be stopped, excision and truncal vagotomy.

BILLROTH I PARTIAL GASTRECTOMY

This is the standard operation for gastric ulcer. The distal stomach and pylorus, including the ulcer, is excised and an end-to-end gastro duodenal anastomosis is made to restore continuity. A new lesser curve is made to narrow the stoma of the gastric remnant to the size of the duodenum.

PYLORIC PRESERVING SURGERY

The pyloric function is retained and therefore prevent the rapid gastric emptying and dumping that may occur when the pylorus is excised. About 1.5 cm proximal to the pylorus is preserved with the pyloric branches of the vagus nerve.

Billroth II (Polya)  partial gastrectomy

This can be used for both duodenal and gastric ulcers, and involves resecting two-thirds of the stomach, closing the duodenum distal to the pylorus (usually beyond the ulcer), and an end-to-side gastrojejunal anastomosis.

VAGOTOMY AND ANTERECTOMY

Resection of the antrum (gastrin-secreting area) is used in addition to a truncal vagotomy. The re-anastomosis may be gastro duodenal or gastro jejunal, the gastro duodenal being preferable if the duodenum is not too scared.

Truncal vagotomy and pyloroplasty or gastro jejunostomy

Truncal vagotomy is carried out as high as possible on the abdominal oesophagus above the area where the vagi start to branch. The peritoneum over the anterior surface of the oesophagus is divided and the phreno-oesophageal ligament identified and elevated, exposing the anterior vagal trunk. The posterior trunk lies on the right posterior aspect of the oesophagus, often well away from the oesophageal wall, and the encircling finger around the oesophagus may pass medial to it and therefore it may be missed. A 1-cm piece is taken out of each of the trunks and the surgeon must carefully search the oesophageal wall for small branches. A 4 cm-long segment of the oesophagus should be denuded of its attachments.

Pyloroplasty: These are of several different types, for example Heineke–Mikulicz, Finney (gastroduodenostomy), and ulcer-excluding. They all bypass or destroy the pyloric mechanism and so affect gastric emptying and duodenogastric reflex.

Anterior seromyotomy and posterior truncal vagotomy

The operation is based on the principle that only the anterior nerve to the antrum need be preserved to ensure antral function. In about 3 per cent of patients, the only supply to the antrum is from the posterior nerves, such that they would get delayed gastric emptying.

LAPRASCOPY PROCEDURES:

1. Truncal vagotomy,
2. Gastroenterostomy,
3. Seromyotomy .

DRAINAGE PROCEDURES

Pyloroplasty:

1.Heineke Mikulicz: A longitudinal incision through pylorus and reconstructing duodenotomy in transverse fashion.

2. Finney: Indicated primarily for patients with a J-shaped stomach or extensive scarring and narrowing of a significant portion of the duodenal bulb.A long incision from the stomach, through the pylorus, and well into

the duodenal bulb, with closure of the inferior duodenum to the inferior stomach and the superior duodenum to the superior stomach.

3. Jaboulay Gastroduodenostomy: The procedure involves anastomosis of the distal part of the stomach to the first and second portions of the duodenum, and bypassing the pylorus.

Gastrojejunostomy: Indicated as a drainage procedure when there is duodenal obstruction and the duodenal bulb is so scarred.

CARCINOMA OF STOMACH

Epidemiology and aetiology

Carcinoma of the stomach occurs predominantly between the fifth and seventh decades of life and in people in the lower socioeconomic groups. The variations in geographical incidence are not irrevocable, as environmental factors play an important part.

Site of gastric carcinoma

The tumour is moving proximally, with a rising incidence of adenocarcinoma of the cardia. Tumours in the fundus are more aggressive, with a greater tendency to submucosal invasion regardless of the histological type. Proximally placed tumours have a worse prognosis than those sited distally because proximally placed tumours appear to be at a more advanced stage at presentation.

Aetiological factors

Genetic

E-cadherin, a cell-adhesion molecule, was poorly expressed in those patients who developed early, poorly differentiated, diffuse gastric cancer.

Aird, in 1953, described an association between blood-group A and gastric carcinoma. The relative risk over patients with blood group O is 1.2 times. This difference has been related to the nature of muco polysaccharide secretion in the stomachs of group-A individuals, and greater susceptibility to ingested carcinogens.

ENVIRONMENTAND DIET

Low-quality diets poor in milk, animal protein, and vitamins but rich in starch, heavily salted pickles, smoked fish and meats. These smoked foods contain polycyclic hydrocarbons (1,4-benzpyrene), which are the probable carcinogens.

In man ingested nitrates and nitrites present may be converted to nitrosamines (N-nitroso compounds) by the action gastrointestinal bacteria. Presence of atrophic gastritis and the associated achlorhydric stomach might predispose to the production of N-nitroso carcinogens.

PREMALIGANT CONDITION

Mucosal atrophy and intestinal metaplasia .The first lesion is atrophic

gastritis, followed by progressive intestinalization of the mucosa to intestinal metaplasia, then dysplasia, and finally carcinoma.

HELICOBACTER PYLORI

The particular subtype of H. pylori infection is becoming important. Infected individuals who had CagA antibodies were 5.8-fold more likely to develop gastric cancer, whereas those without CagA antibodies had an odds ratio of 2.2. The most important strain has been characterized further as being CagAvacAS1a-positive.

Primary low grade lymphoma of the mucosal associated lymphoid tissue (**MALT**) of the stomach. This tumour is strongly associated with H. pylori infection.

Strickland has divided chronic atrophic gastritis into two subgroups:

Type A, which is associated with pernicious anaemia, predominantly affects the fundus and body, and is autoimmune in origin.

Type B affects the antrum and is related to environmental factors.

GASTRIC POLYPS

Adenomatous polyps are truly premalignant. They are often larger (80 per cent greater than 2 cm), and are tubulovillous or villous on microscopic examination. PREVIOUS SURGERY

The risk of developing gastric cancer following gastrectomy is reportedly between 3 and 10 per cent.

MENETRIER'S DISEASE

Gastric carcinoma has been described as a complication of Menetrier disease. In Menetrier disease there is giant hyperplasia of the gastric mucosal folds.

Pathology

The Borrmann classification: (By its macroscopic appearance).

Type I - Polypoid or fungating cancers,

Type II -Fungating and ulcerated and surrounded by elevated borders,

Type III -Ulcerated lesions infiltrating the gastric wall,

Type IV- Cancers infiltrate diffusely, and

Type V -That are unable to be classified.

The Lauren classification:Intestinal and Diffuse.

The intestinal variant arises from the gastric mucosa and is glandular in origin.

Diffuse-type pathology appears to arise from the lamina propria.

The World Health Organization Classification:

Depending on the degree of intestinal metaplasia.

Adenocarcinoma (intestinal and diffuse),

Signet cell,

Mucinous,

Tubular, and Papillary.

Risk Factors and Protective Factors in the Pathogenesis of Gastric Cancer

Intestinal variant

Arises from precancerous areas (gastric atrophy, metaplasia)

5:1 Male-to-female ratio

Older population

Dominant histology in areas where stomach cancer is epidemic

Declining in incidence

Diffuse Variant

Women >> men

Younger patients

Higher association in familial occurrence (genetic cause)

Major histologic type in endemic areas

Worse overall prognosis

Acquired Factors

1.High-salt diet

2.High-nitrate diet

3.Smoked/cured food

4.Low vitamin A and C

5.Well water

6.Cigarette smoking

7.Helicobacter pylori

8.Epstein-Barr virus

9.Radiation exposure

10.Previous gastric surgery

11.Coal workers

12.Rubber workers

Genetic Factors

1.Type A blood

2.Pernicious anemia

3.Family history

4.Hereditary nonpolyposis colorectal cancer

5.Li-Fraumeni syndrome

Precursors

1.Adenoma

2.Atrophic gastritis

3.Dysplasia

4.Intestinal metaplasia

5.Ménétrier’s disease

Protective Factors

1.Raw vegetables

2.Citrus fruits

3.Antioxidants—vitamins A and C

4.Selenium, zinc, iron,

5.Green tea.

Precise description of the morphology of advanced gastric cancer by the Japanese has defined three types according to strict morphovolumetric types. The ratio of the amount of muscle invasion to mucosal involvement gives

(i) a funnel type (mucosal greater than muscle involvement; ratio less than 0.75),

(ii) a column type (equal involvement; ratio 0.75 to 1.25), and

(iii) a mountain type (muscle greater than mucosal involvement; ratio greater than 1.25).

MULTIPLE SYNCHRONOUS GASTRIC ULCER

Multiple synchronous gastric cancer was described by Moertel, who defined strict criteria for diagnosis. Each lesion must be proved histo pathologically malignant, all lesions must be separated by normal gastric wall, and the possibility that the lesions represent a local extension or metastasis must be ruled out beyond reasonable doubt.

GASTRIC LYMPHOMA AND MALTOMA

Between 2 to 8 per cent of gastric malignancies are lymphomas .The initial stage preceding lymphoma is the excessive development of MALT ('maltoma'). MALT an immunological defence system to control the local infection. It is clear that the pre neoplastic cells are proliferating in response to an antigenic stimulus that is T-cell dependent. The proliferation is composed of reactive T cells, plasma cells, and B cells, and mimics the appearance of lymphoid follicles in lymph nodes. Transformation from this low-grade tumour to high-grade lymphoma occurs when the mass becomes dominated by large blast cells.

In gastric cancer, oncogenes coding for

1.Tyrosine kinase receptors (c-met, c-erbB2),

2.Epidermal growth factor,

3.Intracellular signal transduction (ras), and

4.Tumour suppressor genes (p53, APC)

have been identified as of potential significance of gastric cancer.

PATTERNS AND SREAD OF METASTASIS

The poorly differentiated mucinous and signet-ring tumours are more invasive. Early gastric cancer with tumour confined to the mucosa and submucosa; those with poorly differentiated carcinomas are more prone to metastasis.

PENETRATION OF GASTRIC MUCOSA

If the muscularis propria and then the serosa are breached the tumour is likely to spread by transcoelomic implantation of shed cells.These cells characteristically become implanted in the ovaries, producing Krukenberg tumours (bilateral ovarian tumours from a signet-ring carcinoma), or in the pelvis, producing a shelf-like mass palpable rectally (Blummer's shelf).Gastric tumours may spread by direct extension and invasion of adjacent structures including liver, pancreas, spleen, peritoneum, mesentery, and omentum.

Lymphatic drainage and lymph-node involvement

Four zones of lymph drainage corresponding to the blood supply are identified.

Zone 1 is located in the gastrocolic omentum along the right gastroepiploic vessels,

draining to the pylorus and then to coeliac and aortic nodes.

Zone 2 is in the gastrocolic and gastrosplenic omentum around the left gastroepiploic vessels, draining to the pancreaticosplenic lymph nodes and to the aortic nodes.

Zone 3 surrounds the left gastric artery and drains into perioesophageal lymph nodes.

Zone 4 drains along the hepatic artery and drains into para-aortic lymph nodes.

The Japanese have adjusted and stratified the classification of lymph drainage.

GroupI (N1) are perigastric lymph nodes;
Group II (N2) are nodes along and at the roots of the major vessels;
Group III (N3) are lymph nodes at the root of the superior mesenteric artery, in the hepaticoduodenal ligament, and behind the pancreas; and
Group IV (N4) are distant lymph nodes.

1. Right cardial
2. Left cardial
3. Lesser curvature
4. Greater curvature
5. Supra pyloric
6. Infra pyloric
7. Left gastric
8. Common hepatic
9. Celiac
10. Splenic hilus
11. Splenic artery

12. Hepatoduodenal ligament

13. Retropancreatic

14. Mesentric root

15. Trancsverse mesocolon

16. Para aortic

AJCC STAGING OF GASTRIC CARCINOMA

Tumor

T1 Tumor invades the lamina propria or submucosa

T2 Tumor invades the muscularis propria (a) or submucosa (b)

T3 Tumor invades through the serosa without invading adjacent structures

T4 Tumor directly invades adjacent structures

Lymph Nodes

N0 0 lymph nodes

N1 1 to 6 positive lymph nodes

N2 7 to 15 positive lymph nodes

N3 >15 positive lymph nodes

Distant Metastases

M0 No distant metastases

M1 Distant metastases

TNM Grouping

Stage IA T1, N0, M0

Stage IB T1, N1, M0

T2a, N0, M0

T2b, N0, M0

Stage II T1, N2, M0

T2a, N1, M0

T2b, N1, M0

T3, N0, M0

Stage IIIA T2a, N2, M0

T2b, N2, M0

T3, N1, M0

T4, N0, M0

Stage IIIB T3, N2, M0

Stage IV Any T4 + any N1, Any N3 or M1

CLINICAL FEATURES

1.Abdomen pain,

2.Nausea,

3.Vomiting,

4.Loss of weight,

5.Anorexia,

6.Early satiety.

SIGNS

1.Palpable abdominal mass,

2.Ascites,

3.Blumer’s shelf nodules,

4.Krukenberg tumors,

5.Periumbilical lymphadenopathy (Sister Mary Joseph’s Nodule),

6.Palpable supra clavicular lymphadenopathy(Virchow’s node).

7.Radiography,

8.Barium contrast studies of the upper gastrointestinal tract,

9.The double-contrast, air–barium study ,

10.Computed tomography (CT),

11.Ultrasonography, and

12.Magnetic resonance imaging ,

13.Imunolocalization with monoclonal targeted isotopes,

14.Endoscopic lymphography,

15.Endoluminal ultrasonography, and

16.Dynamic CT,

17.Endoscopic ultrasonography,

18.Positron emission tomography (PET) with fluorodeoxyglucose (FDG),

19.Endoscopy and biopsy.

The development of fibreoptic, flexible, forward-viewing endoscopes with a controllable tip has been a major advance. It allows direct visualization of the stomach and accurate biopsy of any lesion identified. If up to 10 biopsies were taken from each lesion, the diagnostic accuracy was 100 per cent. The working recommendation is that four to six biopsies be taken from each lesion, and from the inner border of the edge of any ulcer.

Improvements in endoscopic techniques include

1.Dye-spraying,

2.Fluorescence endoscopy,

3. Magnified endoscopy,

4.Electronic endoscopy,

5.Endoscopic ultrasonography.

6.Magnified endoscopy,

7.Electronic endoscopy, and

8.Endoscopic ultrasonography.

Endoscopic Instrumentation and Patient Preparation: Flexible endoscopes now use fiber optics only for transmission of light, and the image is transmitted via a CCD (charge-coupled device) computer chip at the tip of the endoscope. Flexible endoscopes with smaller outer diameters and larger biopsy channels have resulted in better patient tolerance and comfort and the performance of complex interventions. Double-channel endoscopes allow "two handed techniques" such as mucosal resection and tissue approximation in the absence of more effective endoscopic suturing devices. Preparation for diagnostic and therapeutic endoscopy of the stomach requires merely 6 to 8 hours of fasting before the procedure. Patients with gastric outlet obstruction or profound gastroparesis require a longer period of fasting, and tube decompression before the procedure may be prudent.

Delivery of conscious sedation requires adequate monitoring with pulse oximetry, blood pressure recordings, and regular documentation of respiration. As the endoscope advances into the stomach, it assumes a "greater curve position," with the posterior wall at 3 o'clock, the greater curvature at 6 o'clock, the anterior wall at 9 o'clock, and the lesser curvature in the 12- o'clock position. When the scope is initially advanced into the stomach, rugal folds are identified in the fundus and body and are typically absent at the junction of the distal body and antrum. Evaluation of the incisura angularis is important to rule out type I gastric ulcers. A side-

viewing endoscope is necessary to obtain a full endoscopic view of this portion of the duodenum.

Gastric Pathology

Gastric ulcers may be identified in the prepyloric, body, and fundic portions of the stomach and angularis. Gastric ulcers found at the time of endoscopy require aggressive biopsy of all margins at the junction of the edges of the base and surrounding gastric mucosa Suspicion of malignancy may be supported by the presence of heaped edges, deeper ulcerated bases, or diffuse infiltrative processes. Follow-up endoscopy within 8 to 12 weeks is necessary for ulcers that are benign by initial biopsy but have an atypical appearance, are larger than 2 cm, appear suspicious pathologically, or are leading to persistent symptoms. Absence of healing at the time of second endoscopy may be an indication for surgical excision

Cytology

Cytological study of gastric aspirate for exfoliated malignant cells has been used in patients with advanced disease, but has a variable accuracy of between 40 to 90 per cent.

Methods of collecting the cytological specimen include

1.Gastric washings,

2.Washings with the addition of a mucolytic agent, and

3.The passage of a balloon.

Cytological analysis of brushings directly collected from suspicious lesions can alone be accurate in 81 per cent, but when combined with biopsy has an improved accuracy of 91 per cent. Immunocytochemical stains such as fetal sulfaglycoprotein may be useful for cytological preparations. Serum tumor markers, including carcinoembryonic antigen (CEA), CA 19-9, CA-125, CA 72-4, and human chorionic gonadotropin (HCG), can be elevated in patients with gastric cancer, although the individual sensitivities are generally low—in the 40% to 50% range.

TREATMENT AND OUTCOME

ONCOLOGICAL APPROACH

FACTORS AFFECTING PROGNOSIS

There are undoubtedly predetermined factors that govern survival,

1.The site of the tumour,

2.The extent of nodal and,

3.Serosal involvement,

4.The manner of execution of the resection.

Poor prognostic factors in relation to the presentation of the tumour are

1.Serosal invasion,

2.Presence of lymph-node metastasis,

3.Presence of free carcinoma cells in the peritoneum,

4.Lauren's classification (intestinal type better than diffuse type),

5.Cardial tumour,

6.Histological evidence of the invasion of lymph vessels,

7.Tumour stage,

8.Tumour depth and size, and

9.Patient's age.

The poor prognostic variables that relate to surgery are

1.Positive resection margins,

2.Inadequate extent of lymphadenectomy, and

3.Associated splenectomy.

The basic oncological approach for resection of mucosal cancers is wide excision of the primary tumour with en bloc removal of the draining network of lymph node.

ENDOSCOPIC LOCAL EXCISION

The Japanese in particular have pioneered endoscopic local excision of early gastric cancer. The technique involves the use of an endoscopic snare, with pathological assessment of the adequacy of excision. If the lesion is not amenable to removal by snare, the injection of saline beneath it may allow its removal. Laser ablation is an alternative, but fails to allow histological analysis; it can be used to treat tumours that penetrate deep into the submucosa.

EXTENT OF GASTRIC RESECTION

Adequate gastrectomy implies surgical margins in the stomach free of tumour; thus gastrectomy may be partial or subtotal if the tumour is distal, or total if it is more proximal. The definition of adequate resection margins is an 5- to 6-cm clearance proximally and 2cms distally in the unstretched stomach. Failure to resect the stomach widely with microscopically clear margins is highly detrimental to survival. If the resection margin is not confirmed free of microscopic disease, the prognosis of a stage-II tumour falls to that of a stage IV. In general, multiple cancers should be treated by total gastrectomy.

Lesions in the body and fundus of the stomach present different problems. Total gastrectomy is often advocated in these circumstances because the small amount of stomach that remains has little reservoir capacity. Both procedures require an oesophagoenteric anastomosis. Total gastrectomy with a Roux-en-Y reconstruction is also superior to proximal gastrectomy since these patients will be less prone to alkaline and biliary reflux. Tumours of the body and fundus are often far advanced at presentation because they reach considerable size before producing symptoms. They have a less favourable prognosis than tumours in the antrum.

The preferred method of reconstruction after total gastrectomy is as a Roux-en-Y with a 60-cm Roux to prevent bile reflux. The creation of a pouch to act as a reservoir to prevent early satiety has been advocated. The Hunt–Lawrence pouch is created from a long Roux loop with an enteroenteric anastomosis at 80 cm. It is simply folded on itself and a 10 cm, side-to-side anastomosis created below the oesophageal anastomosis. A partial gastrectomy is best reconstructed as an antecolic, Polya type of gastrectomy.

Lymph nodes removed in D1 versus D2 versus D3 lymphadenectomy during resection of gastric cancer.

Reconstruction After Gastrectomy

After distal subtotal gastrectomy,

Billroth I gastroduodenostomy,

Billroth II gastrojejunostomy (either antecolic or retrocolic), and

Roux-en-Y gastrojejunostomy (also either antecolic or retrocolic).

After total gastrectomy, options for reconstruction include a standard

Roux-en-Y esophagojejunostomy,

Construction of a pouch, and

Jejunal interposition

Anastomosis can be done in a stapled fashion (circular or linear) or hand sewn in one or two layers.

When constructing an esophagojejunostomy, one may use a jejunal pouch to provide for a neostomach reservoir.

Adjuvant Therapy

1.Adjuvant Chemotherapy

Combination chemotherapy with 5-fluorouracil (5-FU), epirubicin, mitomycin, and methyl-CCNU have been associated with better response rates.

2.Adjuvant Chemoradiotherapy

5-FU/leucovorin (LV), followed by 45 Gy radiotherapy, followed by additional

5-FU/LV.

3.Adjuvant Intraperitoneal Therapy

Continuous hyperthermic peritoneal perfusion (CHPP) has been used in patients with gastric cancer in both the adjuvant and palliative setting. Drugs used are 5 flurouracil, mitomycin-c, cisplatin.

Neoadjuvant Therapy

1. Neoadjuvant Radiotherapy

The radiotherapy group had a higher overall resection. Tumor down-sizing and nodal down-staging were also noted, and there was no increase in

operative mortality with the use of preoperative radiotherapy.patient with node-positive disease and T4 lesions had a significant survival advantage with the radiotherapy regimen, and again there was no increase in perioperative mortality or morbidity.

2. Neoadjuvant Chemotherapy

Neoadjuvant drugs are etoposide, cisplatin, and 5-FU. A higher proportion of patients in the chemotherapy group underwent curative resection and were noted to have significantly smaller tumors at surgery, as well as significantly lower T and N stages.

3. Neoadjuvant Chemoradiotherapy

5-Fluorouracil, folinic acid, and cisplatin, followed by 5 Fluorouracil potentiated radiotherapy (45 Gy). Surgical resection after this preoperative regimen was safe, with a complete pathologic response in 30% and a partial pathologic response in 24%.

Management of Advanced Disease

Surgical palliation may include resection alone or in combination with endoscopic, percutaneous, or radiotherapeutic interventions or chemotherapy and radiotherapy.

Surgery for Palliation

Exploratory laparotomy alone, GI bypass, or palliative resection, resection was associated with longer survival in patients with both local and distant spread of

disease.

Endoscopic Palliation

In patients who are not good candidates for palliative resection but have symptoms of obstruction, endoscopic palliative techniques may be useful, including placement of metal expandable stents and laser recanalization, dilatation or stenting.

After relief of obstruction, many of these patients can later receive palliative chemotherapy.

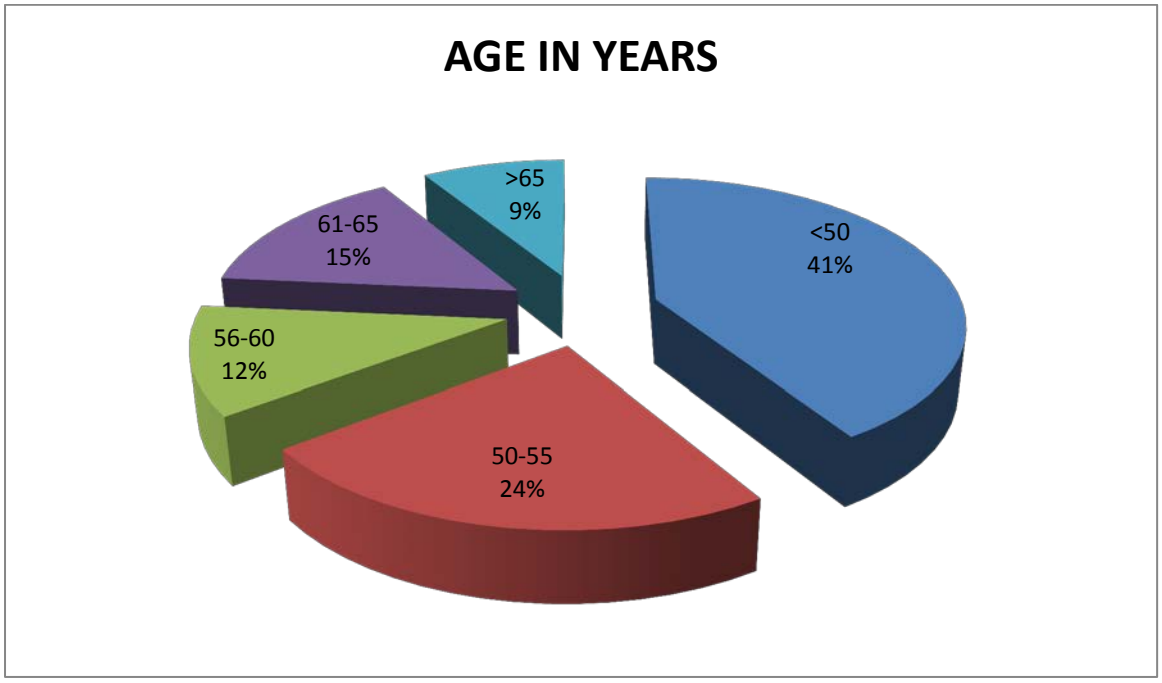
Palliative Chemotherapy

Multiagent chemotherapy cisplatin, paclitaxel, and irinotecan given to all patients with advanced disease who have reasonable performance status.

Palliative Radiotherapy

It is effective in controlling symptoms such as bleeding and pain, most patients have diffuse metastatic disease.

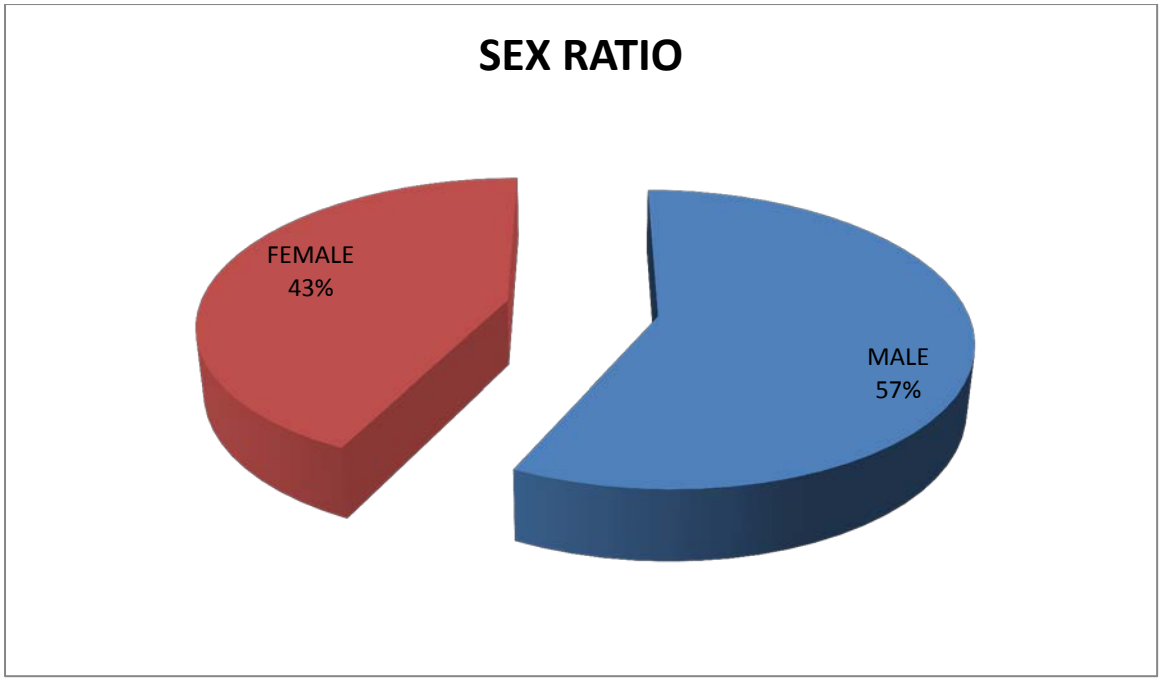
OBSERVATIONS AND RESULTS



AGE IN YEARS	NUMBER OF PATIENTS	PERCENTAGE
<50	15	41
50-55	8	24
56-60	4	12
61-65	5	15
>65	3	9

Gastric outlet obstruction is common in age less than 50 years is 14 (40%). In 10 patients it is due to malignancy and in 4 patients it is due to benign lesion. In age between 50 to 55 years 8 pateints (24%) develop gastric outlet

obstruction. In 7 patients the cause is malignant lesion and in one patient the cause is cicatrized duodenal ulcer in 1st part of duodenum. In age between 56 to 60 years 4 patients had gastric outlet obstruction and in all 4 patients cause was malignant lesion. In age between 61 to 65 years 5 patients had gastric outlet obstruction. In 3 patients the cause is malignant lesion and in 2 patients it was due to benign lesion. In age above 65 years 3 patients develop gastric outlet obstruction and the cause is malignant lesion. In this study young age to develop gastric outlet obstruction is 30 years due to cicatrized duodenal ulcer and oldest age is 75 years due to gastric carcinoma. The age incidence is 30 to 75 years with mean of 52.5 years. The young age to develop gastric carcinoma is 35 years.



SEX RATIO

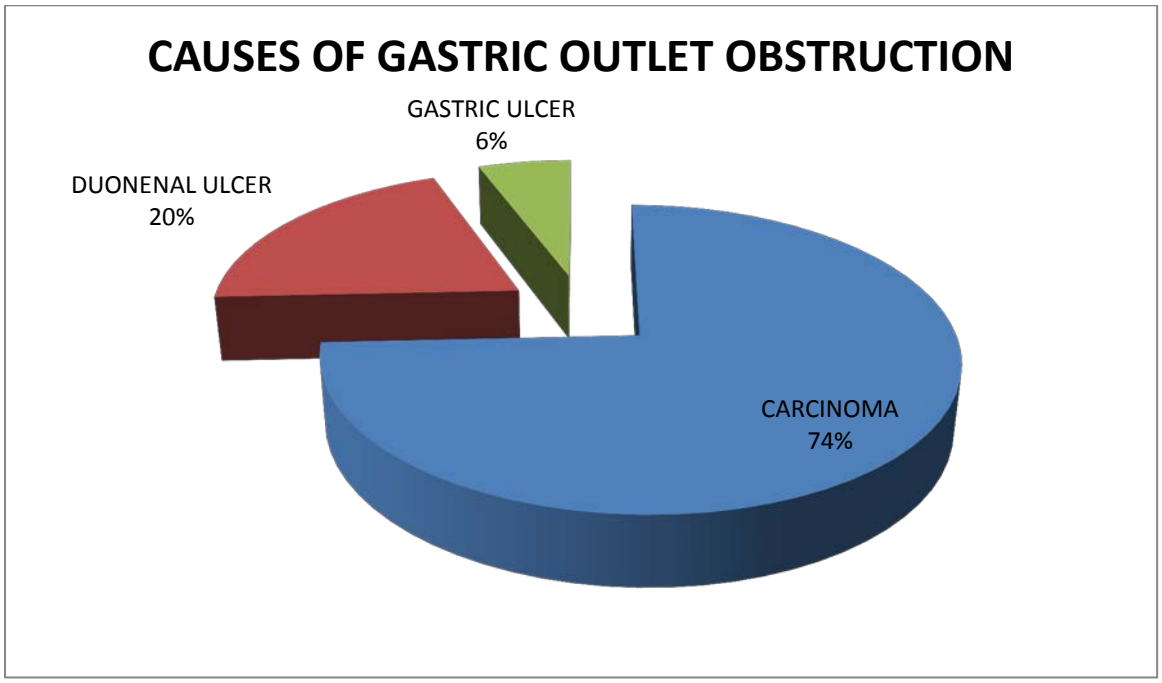
SEX	NUMBER OF PATIENTS	PERCENTAGE
MALE	20	57
FEMALE	14	43

Gastric outlet obstruction is common in male patients. In this study 20 male patients (57%) develop gastric outlet obstruction. In 14 male patients the cause is malignant lesion and in 6 male patients it is due to benign lesion. 14 female patients (43%) develop gastric outlet obstruction. In 12 female patients the cause is malignant lesion and in 2 female patients it is due to benign lesion. In this study malignancy is most common cause of gastric outlet obstruction in both sexes. In this study in age less than 60 years gastric

outlet obstruction is more common in female patients (14 female patients and 12 male patients).

In this study 3 male patients (100%) above 65 years develop gastric outlet obstruction. In age between 50 to 55years the cause of gastric outlet obstruction is equal in both sexes (4 patients in each sex).

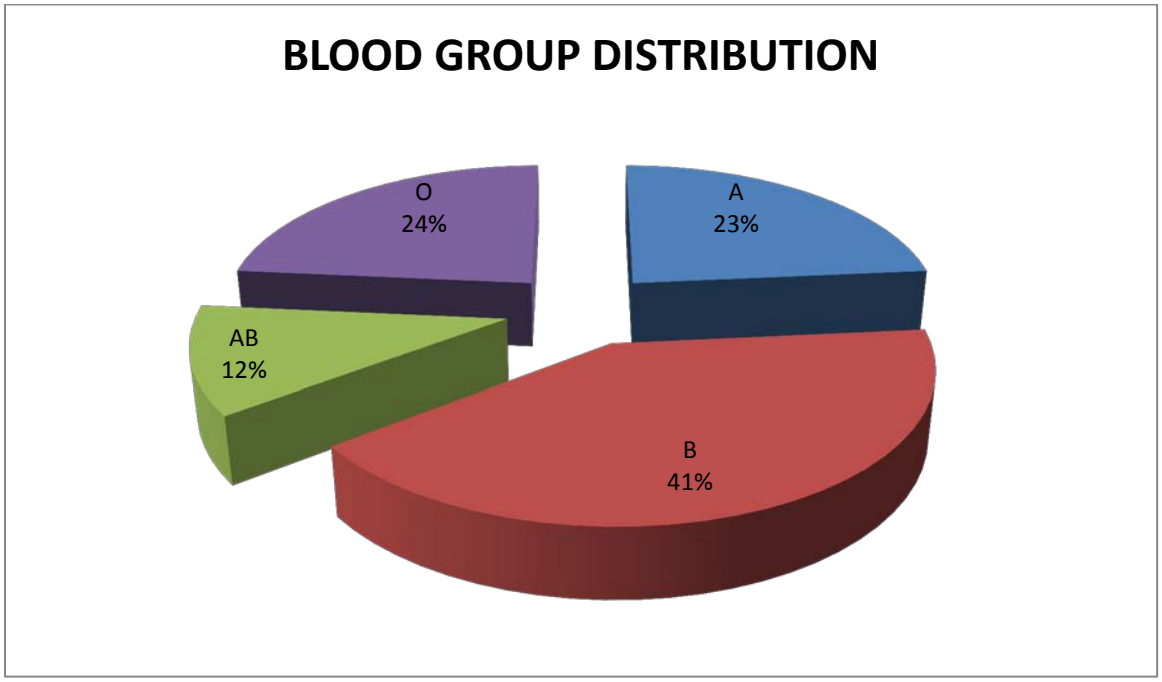
ETIOLOGY



ETIOLOGY OF GASTRIC OUTLET OBSTRUCTION

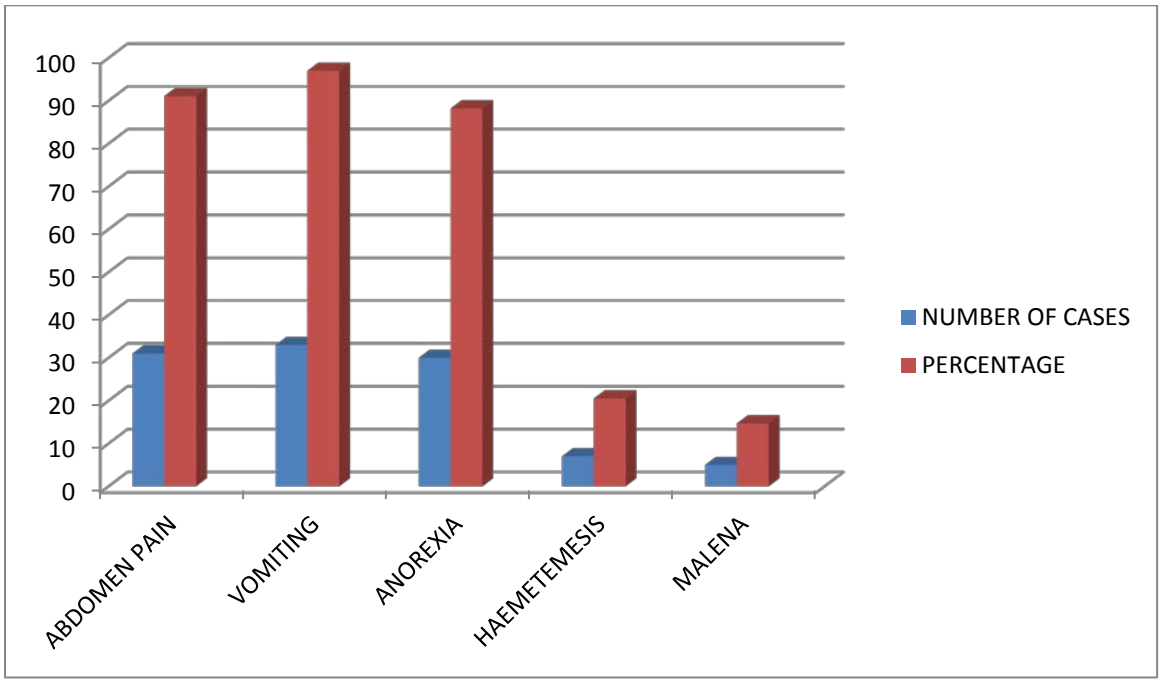
CAUSES OF GASTRIC OUTLET OBSTRUCTION	NUMBER OF PATIENTS	PERCENTAGE
CARCINOMA	26	74
DUODENAL ULCER	7	20
GASTRIC ULCER	1	6

In this study gastric carcinoma in pyloric region is the common cause of gastric outlet obstruction in 26 patients (74%). In 8 patients the cause is benign lesion. In 7 patients the cause is cicatrized duodenal ulcer in 1st part of duodenum. In 1 patient it is due to chronic gastric ulcer in pyloric region.



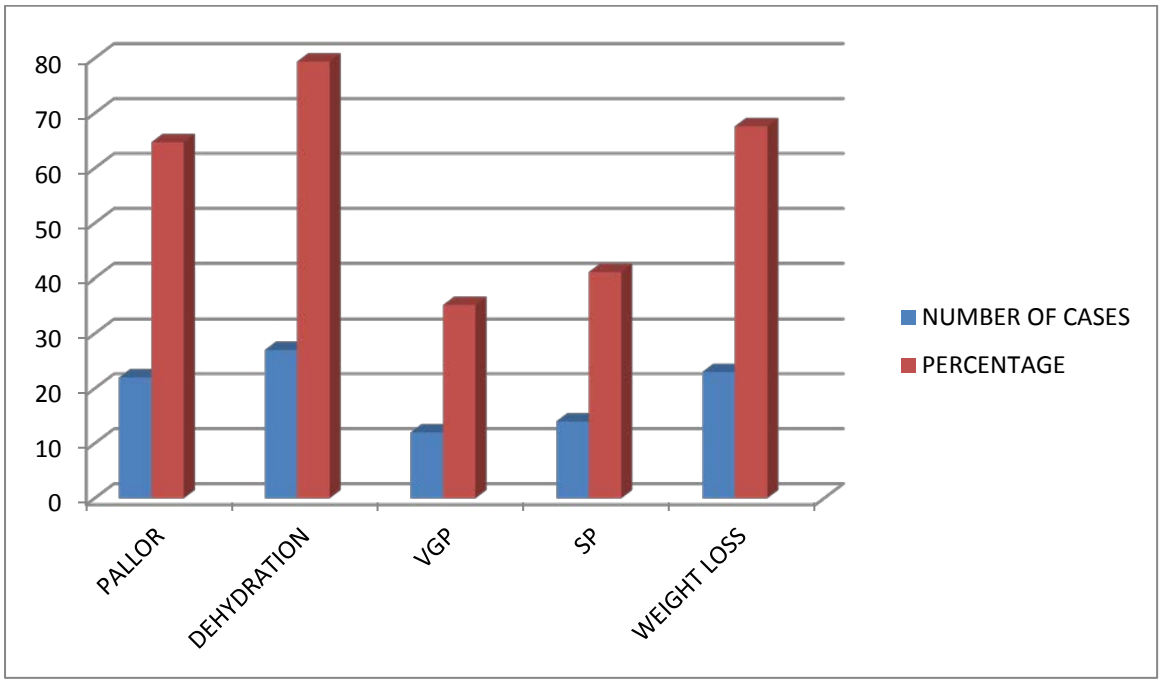
BLOOD GROUP	NUMBER OF PATIENTS	PERCENTAGE
A	9	23
B	13	41
AB	4	12
O	8	24

In this study majority of patients (14) were having blood group B (41%) and next common in blood group is A (23%). In benign lesion 4 patients had B blood group, 2 patients had O blood group, 1 patient had AB and A blood group each. In malignant lesion 10 patients had B blood group, 7 patients had A blood group, 6 patients had O blood group, 3 patients had AB blood group.



SYMPTOMS	NUMBER OF PATIENTS	PERCENTAGE
ABDOMEN PAIN	31	91.1
VOMITING	33	97
ANOREXIA	30	88.2
HAEMETEMESIS	7	20.5
MALENA	5	14.7

In this study the common symptom in gastric outlet obstruction was vomiting in 33 patients (97%). Vomiting is projectile, effortless, non bilious and vomitus contains undigested food particles. In 7 patients vomiting was blood stained and 5 patients had history of passing black tarry stools. The next complaint was abdomen pain (91.1%) followed by anorexia (88.2). Pain was dull aching in nature. In case of carcinoma stomach pain was aggravated immediately after food intake and relived by vomiting. In duodenal ulcer patients abdomen pain appears 2 hours after food intake and relieved by taking food or medicines. In 30 patients they had a history of reduced food intake. Patients developed early satiety and pain after taking food.

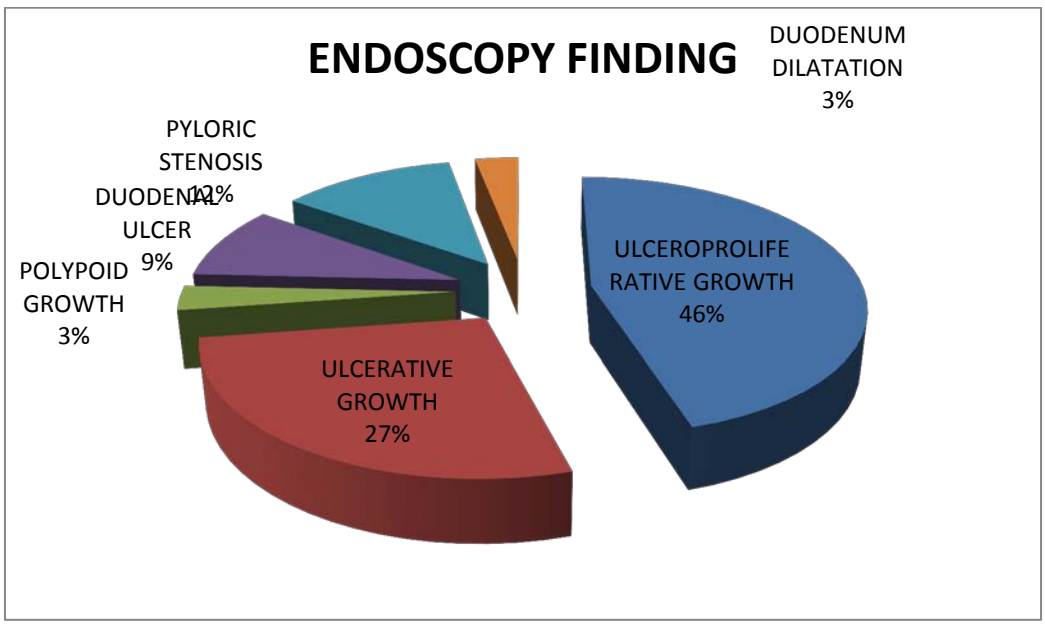


Note: VGP: Visible gastric peristalsis

SP: Succussion splash

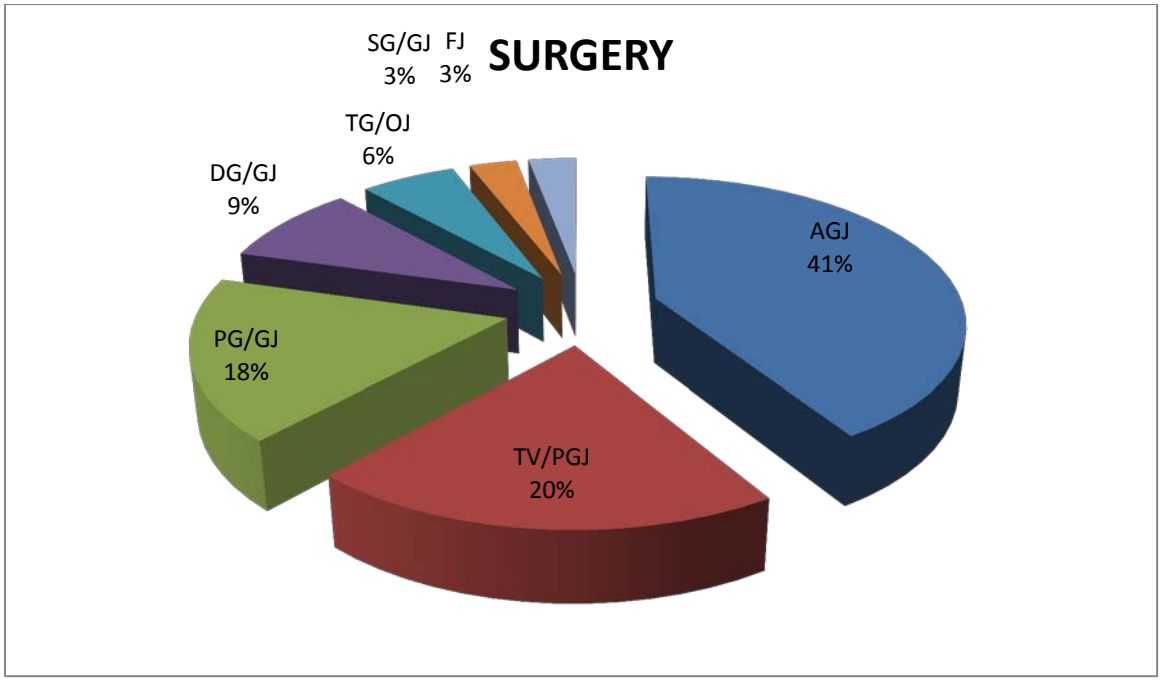
SIGNS	NUMBER OF PATIENTS	PERCENTAGE
PALLOR	22	64.7
DEHYDRATION	27	79.4
VISIBLE PERISTALSIS	12	35.2
SUCCUSSION SPLASH	14	41.1
WEIGHT LOSS	23	67.6

The common sign in gastric outlet obstruction was dehydrated and malnourished patients (79.4%). Next common sign is pallor (64.7%). Visible gastric peristalsis is seen 12 (35.2%) patients. In 14 (41.1%) patients succession splash was present. Patients with gastric carcinoma were more malnourished and dehydrated than patients with benign lesion. Patients with gastric carcinoma lost weight in short duration than benign lesion.



ENDOSCOPY	NUMBER OF CASES	PERCENTAGE
ULCEROPROLIFERATIVE	15	46
ULCERATIVE	9	27
POLYPOID	2	3
DUODENAL ULCER	3	9
PYLORIC STENOSIS	4	12
DUODENUM DILATATION	1	3

All the patients were subjected to endoscopy and biopsy. Ulceroproliferative growth was present in 15 patients. 1n 9 patients it was of ulcerative growth. In 4 patients endoscopy could not be passed beyond pyloric end. 2 patients had polypoid growth in pyloric region. In one patient 1st and 2nd part of duodenum was dilated. In all patients biopsy was taken and sent for histopathology study.



SURGERY	NUMBER OF CASES	PERCENTAGE
AGJ	14	41
TV/PGJ	7	20
PG/GJ	6	18
DG/GJ	3	9
TG/OJ	2	6
SG/GJ	1	3
FJ	1	3

AGJ- ANTERIOR GASTRO JEJUNOSTOMY

DG/GJ- DISTAL GASTRECTOMY, GASTRO JEJUNOSTOMY

TV/PGJ- TRUNCAL VAGATOMY, POSTERIOR GASTROJEJUNOSTOMY

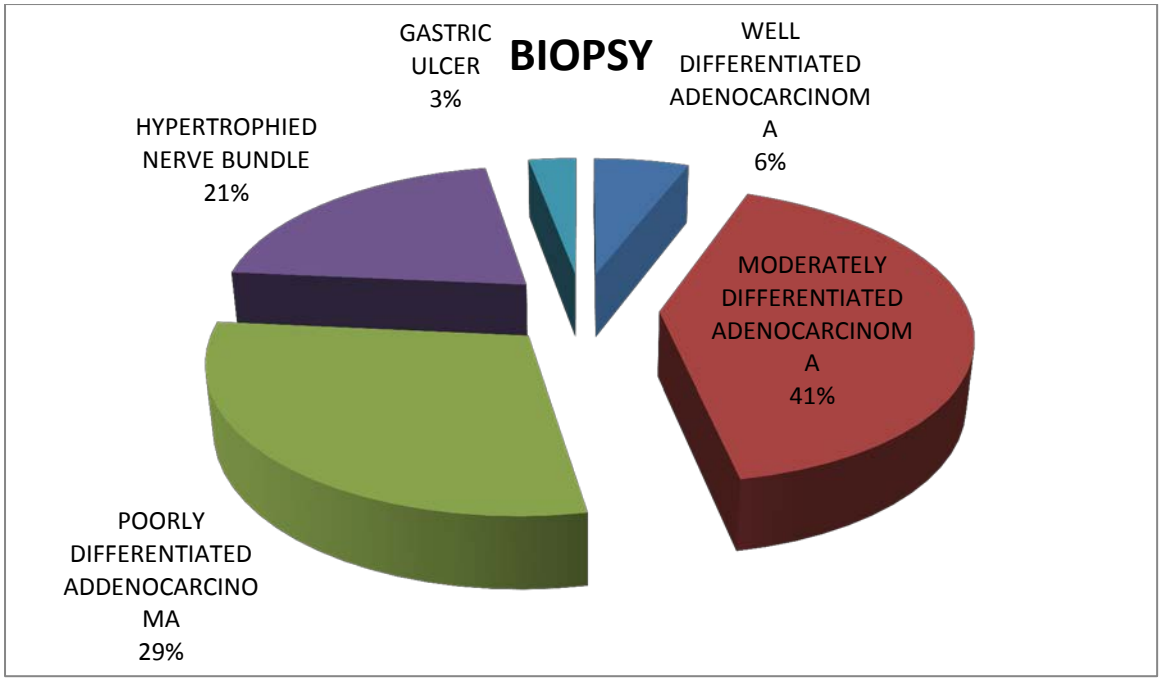
TG/OJ-TOTAL GASTRECTOMY, OESOPHAGOJEJUNOSTOMY

PG/GJ- PARTIAL GASTRECTOMY, GASTRO JEJUNOSTOMY

SG/GJ- SUB –TOTAL GASTRECTOMY, GASTRO JEJUNOSTOMY

FJ- FEEDING JEJUNOSTOMY

Truncal vagotomy with posterior gastro jejunostomy was done for patients with duodenal ulcer and gastric outlet obstruction. Among carcinoma stomach patients 6 of them underwent partial gastrectomy with gastrojejunostomy. In 2 patients carcinoma stomach total gastrectomy with oesophagus jejunum anastomosis was done. In 3 patients of gastric carcinoma distal gastrectomy with gastrojejunsotomy was done. In one patient with gastric carcinoma subtotal gastrectomy was done. In 14 patients advanced gastric carcinoma palliative anterior gastrojejunsotomy was done. Palliative procedure was done because the tumor was inoperable during surgery. In 1 patient with advanced gastric carcinoma feeding jejunostomy was done. Per operative finding was identified and noted. Specimen was sent for histopathology study.



BIOPY REPORT	NUMBER OF PATIENT	PERCENTAGE
WDA	2	6
MDA	14	41
PDA	10	29
HYPERTROPHIED NERVE BUNDLE	7	21
GASTRIC ULCER	1	3

WDA- WELL DIFFERENTIATED ADENOCARCINOMA

MDA- MODERATELY DIFFERENTIATED ADENOCARCINOMA

PDA - POORLY DIFFERENTIATED ADENOCARCINOMA

In this study the biopsy reports of the specimen were

14 patients had moderately differentiated adenocarcinoma,

10 patients had poorly differentiated adenocarcinoma,

2 patients had well differentiated adenocarcinoma,

7 patients had hypertrophied nerve bundle of vagus nerve,

1 patient had chronic gastric ulcer.

PEROPERATIVE FINDING

In benign lesion 4 patients had dilated stomach with cicatrized ulcer in 1st part of

duodenum. 3 patients had dilated stomach with cicatrized ulcer in pyloric antrum.

One patient had ulcer in pyloric antrum with stenosis in pylorus region.

In malignant lesion 14 patients had growth in pyloric region with dilated stomach. In 4 patients there was ulceroproliferative growth in pyloric region. 2 patients had ulcerative mass in pyloric region. 2 patients had mass in pyloric antrum with tumor fixed to posterior stomach bed structures and the tumor was in operable. 2 patients had metastases to liver, sigmoid colon, omentum, peritoneal deposits and the tumor was inoperable. One patient had polypoid growth in pyloric region. In one patient there was ascites with serosal involvement and the tumor was inoperable.

MATERIALS AND METHODS

This was a pathological study on gastric outlet obstruction comprising of 34 cases of gastric outlet obstruction. The patients have been selected from Tirunelveli Medical College Hospital in the Surgery department from December 2010 to December 2011.

The cases were selected with following inclusion and exclusion criteria.

Inclusion Criteria

1. Peptic ulcer disease with gastric outlet obstruction.
2. Carcinoma pyloric region with gastric outlet obstruction.

Exclusion Criteria

1. Infantile hypertrophic pyloric stenosis
2. Congenital lesion
3. Gastro intestinal tuberculosis

An elaborate study of all the patients with regard to history, clinical features, routine investigations, endoscopy and biopsy report, pre operative management, per-operative findings, post operative management and complications during post operative period is managed. Patient general condition, nutrition status, hydration and co-morbid conditions were managed before surgery.

Complete haemogram, blood urea, serum creatinine, serum electrolytes, electro cardiogram, chest x-ray, blood grouping and Rh typing, bleeding time, clotting time, blood sugar (fasting and post prandial), was done. Ultra sonogram of abdomen and pelvis, upper gastro intestinal endoscopy and biopsy from the lesion, were taken. Biopsy sent for histopathological study.

Criteria for diagnosing the patient clinically by

1. Abdomen pain,
2. Vomiting (projectile, non bilious, undigested food particles),

3. Anorexia,

4. Malnutrition,

5. Visible gastric peristalsis,

6. Succussion splash,

7. Palpable mass,

8. Loss of weight.

Preoperative management

Patient general condition, anaemia, hydration, electrolyte imbalance, comorbid conditions were managed. Informed consent obtained and patient was prepared for surgery.

Post operatively patients were monitored under intensive care. Half hourly pulse, temperature, respiratory rate chart , 4^{th} hourly blood pressure chart, input and output chart, ryles tube aspiration, drain collection, soakage of dressing were maintained.

Higher antibiotics, analgesics, H2 blockers, proton pump inhibitors were given. Patient was discharged after complete recovery from illness. Then patients were regularly followed and managed.

DISCUSSION AND ANALYSIS

The observations and results of this study were compared with previous studies and the results were analysed.

This study includes 34 cases of gastric outlet obstruction in adults. Young age of presentation is 30 years and old age is 75 years. Average age of presentation is 52.5 years.

Male patients accounted for 57% of the cases and female patients accounted for 43%. In less than 50 years old, 6 were male patients and 8 were female patients. In 10 patients the cause was malignant and 4 patients had benign lesion. This study shows that the malignant lesion occurs in young age. In age between 50 to 55 years 4 patients were male patients and 4 patients were female. In 7 patients the cause is malignant lesion and in 1 patient the cause is benign lesion. In age 56 to 60 years old patients there were 3 cases and the cause is malignant lesion. In age between 61to 65 years there were 6 patients and in 4 patients the cause is malignant lesion. In 2 patients the cause is benign lesion. Malignancy is the cause in 3 patients above 65 years old.

In 14 patients the blood group is “B” and in 8 patients the blood group is “A”. In 8 patients the blood group is “O” and in 4 patients the blood group is “AB”.

The causes of gastric outlet obstruction in 34 patients were as follows

In 26 patients the cause was carcinoma stomach,

In 7 patients the cause was cicatrized duodenal ulcer,

In 1 patient the cause was chronic ulcer in pyloric antrum.

In carcinoma stomach 14 patients had moderately differentiated adenocarcinoma,

10 patients had poorly differentiated adenocarcinoma, 2 patients had well differentiated adenocarcinoma.

In 33 patients vomiting was the main complaints and it was projectile, effortless, non bilious and contains undigested food particles. 31 patients developed abdomen pain which was dull aching and continuous in nature. Patient developed pain immediately after taking food and was relieved by vomiting. 30 patients had reduced food intake and had early satiety due to which patients were dehydrated and malnourished. 7 patients had episodes of blood vomiting and 5 patients had malena.

Clinically 27 patients were dehydrated due to increased frequency of vomiting. Hence patients were malnourished and emaciated. 23 patients had weight loss and the weight loss is rapid in malignant lesion.22 patients were pallor and anaemic on investigations. In 14 patients there were succussion splash and dilated stomach clinically. In 12 patients there were visible gastric peristalsis from left hypochondrium to right hypochondrium. Some patients had palpable mass in epigatric region.

All patients were subjected to upper gastrointestinal endoscopy and biopsy was taken from the lesion and sent for histopathological study. In 15 patients there was ulceroproliferative growth in pyloric region, in 9 patients there was ulcerative growth in pyloric region, in 4 patients there was pyloric stenosis and the scopy could not be passed beyond the pyloric region, in 3 patients there was duodenal ulcer in 1st part of duodenum, in 2 patients there was

polypoid growth in pyloric region, in one patient the 1st and 2nd part of duodenum was dilated. Many patients had dilated stomach with food debris. The present study results and findings were compared and analysis was done.

Comparison of etiological factors in various studies

Etiology	Present study		Johns Hopkins		Ishra university		JSLS study		Mishra study	
	No:	%	No	%	No	%	No	%	No	%
Malignant	26	74	20	61	27	51.9	16	57	56	76
Benign	8	26	13	39	25	48.1	12	43	18	24
Total	34	100	33	100	52	100	28	100	74	100

From various studies the commonest cause of gastric outlet obstruction was found to be malignant lesion. The present study values are close to values observed in mishra study. The cause of rise in malignancy was due to the use of H2 blockers, proton pump inhibitor, eradication of helicobactor pylori infection.

In present study upper gastrointestinal endoscopy was done in 34 cases (100%), and biopsy sent for histopathological study. 26 patients (74%) had carcinoma antrum pyloric region, 7 patients (20%) had cicatrized duodenal ulcer, 1 patients (6%) had gastric ulcer in pyloric region.

In this study 7 patients with cicatrized duodenal ulcer underwent truncal vagotomy with posterior gastrojejunostomy. One patient with gastric ulcer in pyloric region underwent distal gastrectomy with gastro jejunostomy.

Among 26 patients with gastric carcinoma in pyloric region, 14 of them with advanced gastric carcinoma underwent anterior gastro jejunostomy as a palliative procedure. In all these patients the tumor was in operable and curative surgery was not possible. In 6 patients partial gastrectomy with gastro jejunostomy was done. In 2 patients total gastrectomy with oesophagus jejunostomy and feeding jejunostomy was done. In 3 patients distal gastrectomy with gastro jejunostomy was done. In one patient subtotal gastrectomy with gastro jejunostomy was done. One patient with advanced gastric carcinoma underwent feeding jejunostomy as a palliative procedure.

CONCLUSION

The present study is a pathological study on gastric outlet obstruction. The observations from the data and results obtained in the present study were

Male patients are more commonly affected by gastric outlet obstruction.

Carcinoma in pyloric antral region was the most common cause of gastric outlet obstruction.

Vomiting and dehydration are the common symptoms and signs of gastric outlet obstruction.

Upper gastro intestinal endoscopy and biopsy are the Gold standard

investigation for gastric outlet obstruction. It has been used for both diagnostic purpose and taking biopsy from the lesion.

Patient general condition, correction of anemia, fluid and electrolyte imbalance and preparation of stomach must be done preoperatively.

Patients with gastric outlet obstruction due to cicatrized duodenal ulcer require truncal vagotomy with posterior gastrojejunostomy.

In patients with carcinoma pyloric antral region require curative surgery in early stage or a palliative surgery depending on the stage of the disease.

All patients above 40 years with symptoms of dyspepsia should undergo upper gastro intestinal endoscopy and biopsy examination.

PROFORMA

Name: Age:

Sex: I.P. No.:

Occupation:

DOA: DOS:

DOD:

Income: Address:

Chief Complaints

A. Pain abdomen -- B. Vomiting-

C. Mass per abdomen -- D. Others -

History of Presenting Illness

A. Abdominal pain

Duration: Site: Nature: Relation to food:

Radiation: Periodicity: Aggravating factors:

Relieving factors:

B. Vomiting

Duration: Frequency: Quantity:

Relation to food:

Induced/Spontaneous: Projectile/Regurgitant:

Digested/Undigested: Foul smelling:

Bile/Blood:

C. Mass per abdomen

Duration: Site: Progress:

Ball rolling movement:

D. Post-prandial fullness

E. Malena / Haematemesis

F. Loss of appetite

G. Loss of weight

H. Associated symptoms

Fever

Occupation

Jaundice

Abdominal distension

Others

Past History: History of peptic ulcer disease

Family History: Married/Unmarried

History of peptic ulcer disease/deaths due to carcinoma stomach/ tuberculosis.

Personal History:

A. Diet: Vegetarian/Mixed Spicy: Yes/No

Irregular food habits: Yes/No

B. Smoking: Type: Quantity: Duration:

C. Alcohol intake: Type: Quantity: Frequeny: Duration:

D. Tobacco chewing:

E. Micturition: Menstrual history:

General Physical Examination

Built: Pulse: Blood pressure:

Nourishment: Pallor: Cachexia: Icterus:

Signs of dehydration: Lymphadenopathy:

Systemic Examination

Respiratory system: Cardio vascular system:

Abdominal Examination

Inspection: Distension: Visible gastric peristalsis:

Palpation: Site of tenderness:

Mass: Tenderness: Site: Size:

Shape: Surface: Consistency:

Movement with respiration: Mobility:

Organomegaly: Succession splash:

Shifting dullness: Auscultopercussion:

Per rectal digital examination: Per vaginal examination:

Investigations

A. Routine Investigations

Blood: Haemoglobin % Bleeding time: Clotting time:

Blood glucose: mg% Blood urea: mg% Serum creatinine: mg%

Blood group: Serum electrolytes:

Urine: Albumin: Sugar: Microscopy:

Stool: Ova/Cyst Occult blood:

Chest X-ray/Screening

ECG in all leads:

Upper Gastrointestinal Endoscopy/ Biopsy

Ultra sonogram of Abdomen

Pre-operative Management:

Surgery:

Date of surgery: Anaesthesia: Incision:

Findings:

Procedure:

Histopathological report:

Final Diagnosis

Post-operative management:

Complications:

Post-operative treatment: Chemotherary/Radiotherapy

Outcome: Date of Discharge: Condition:

Advice:

Follow-up:

ANNEXURE II

CONSENT FORM

For Operation / Anesthesia

I_____Hosp. No._____ in my full senses hereby give

my complete consent for _____ or any other procedure deemed fit

which is a/ and diagnostic procedure / biopsy / transfusion / operation to be

performed on me / my ward_____ age _____under any

anesthesia deemed fit. The nature and risks involved in the procedure have been

explained to me to my satisfaction. For academic and scientific purpose, the

operation / procedure may be televised or photographed.

Date: Signature / Thumb impression

Name: of patient / Guardian

Designation:

Guardian

Relationship:

Full Address

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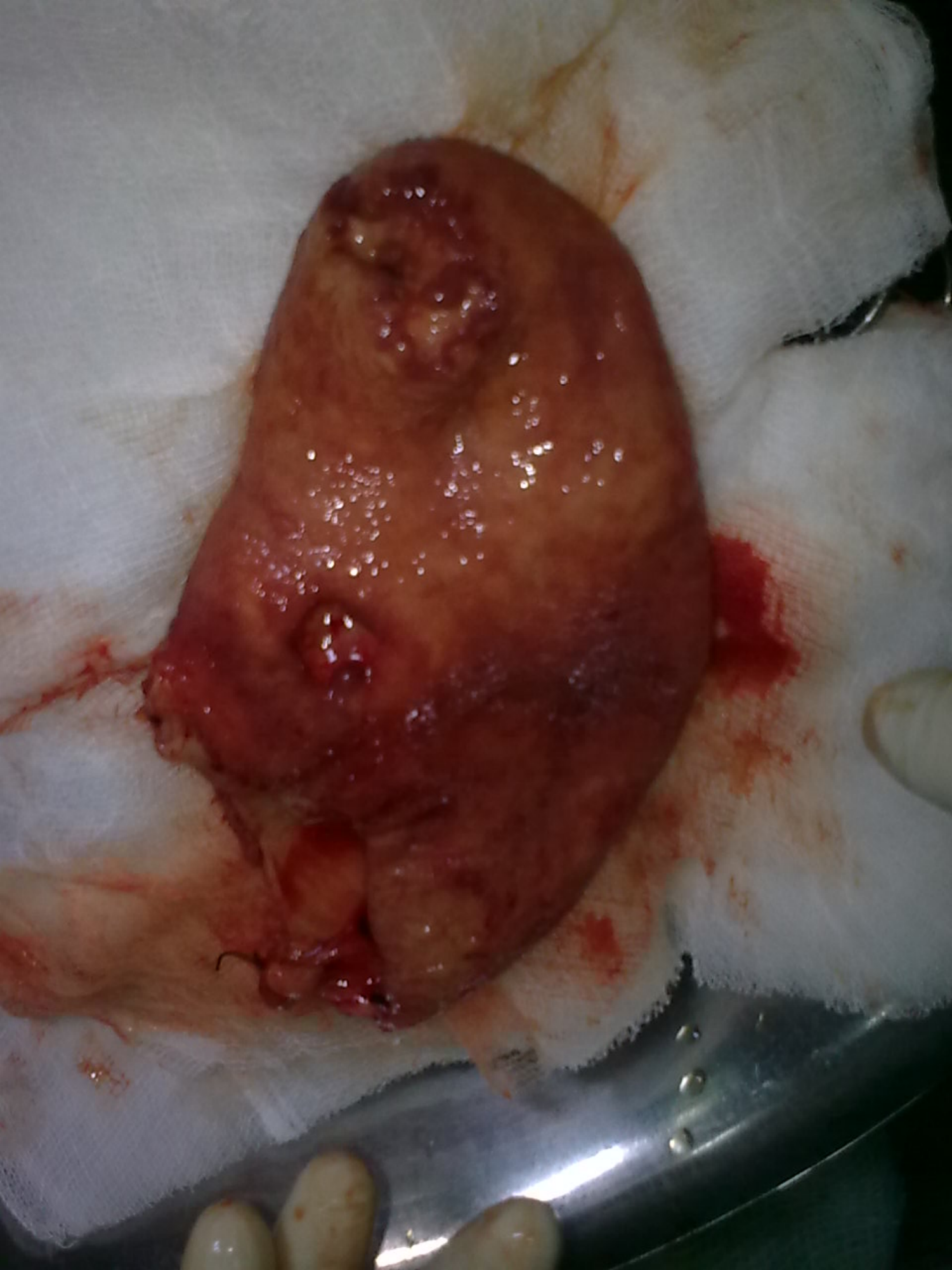

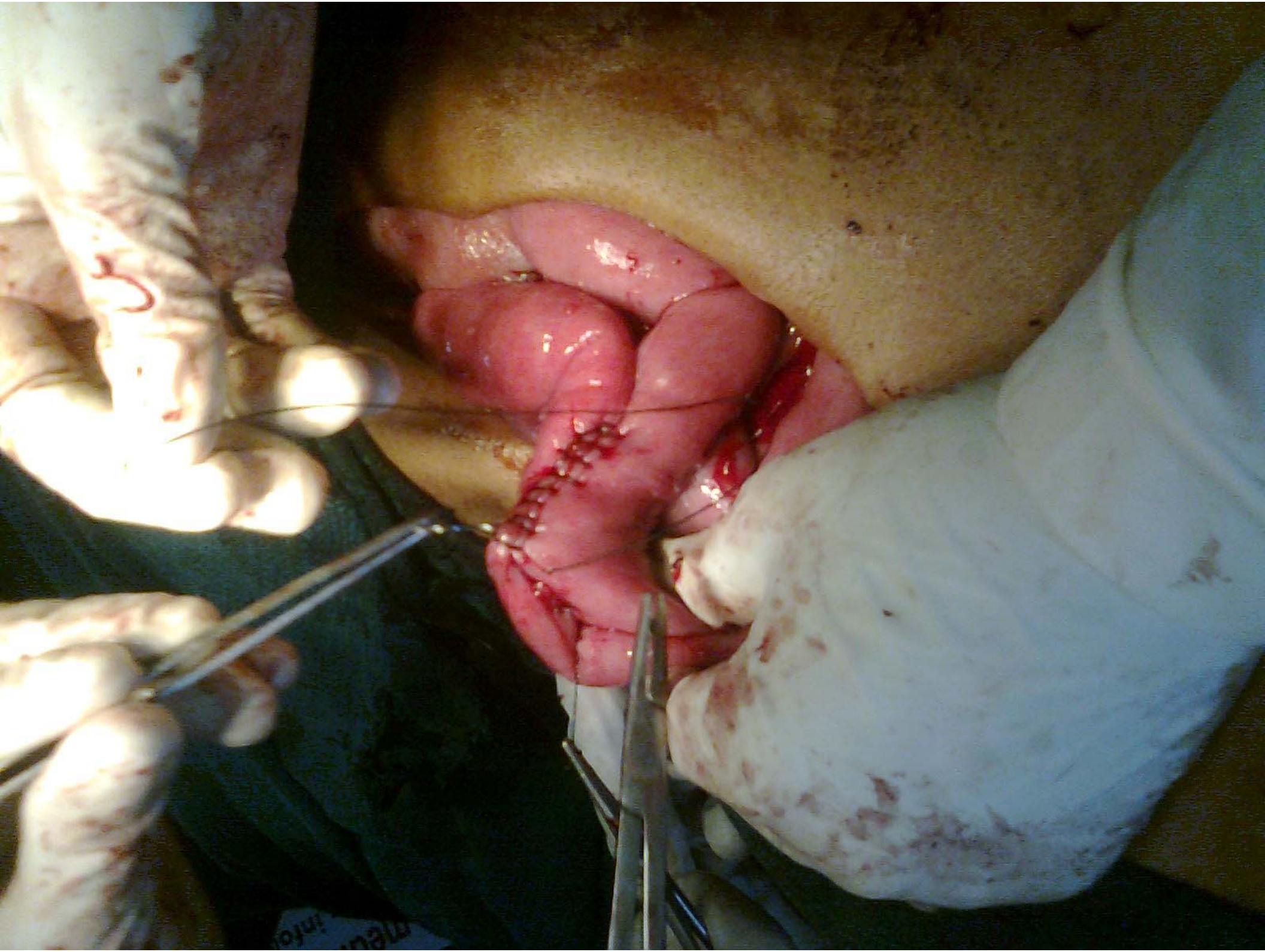

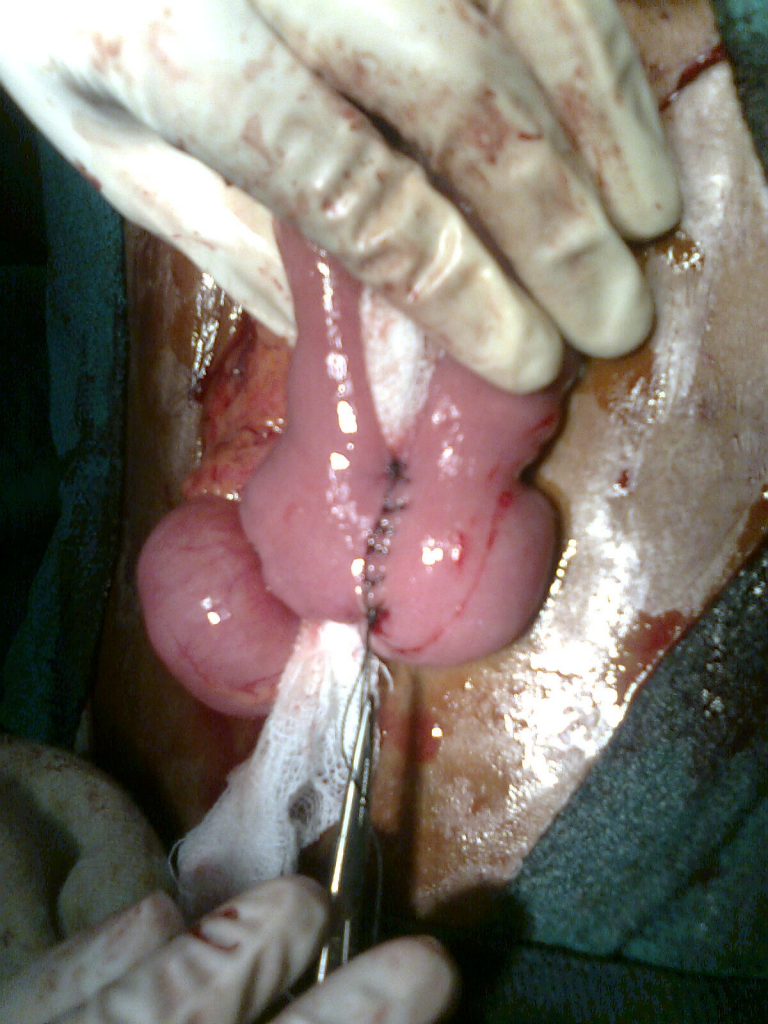

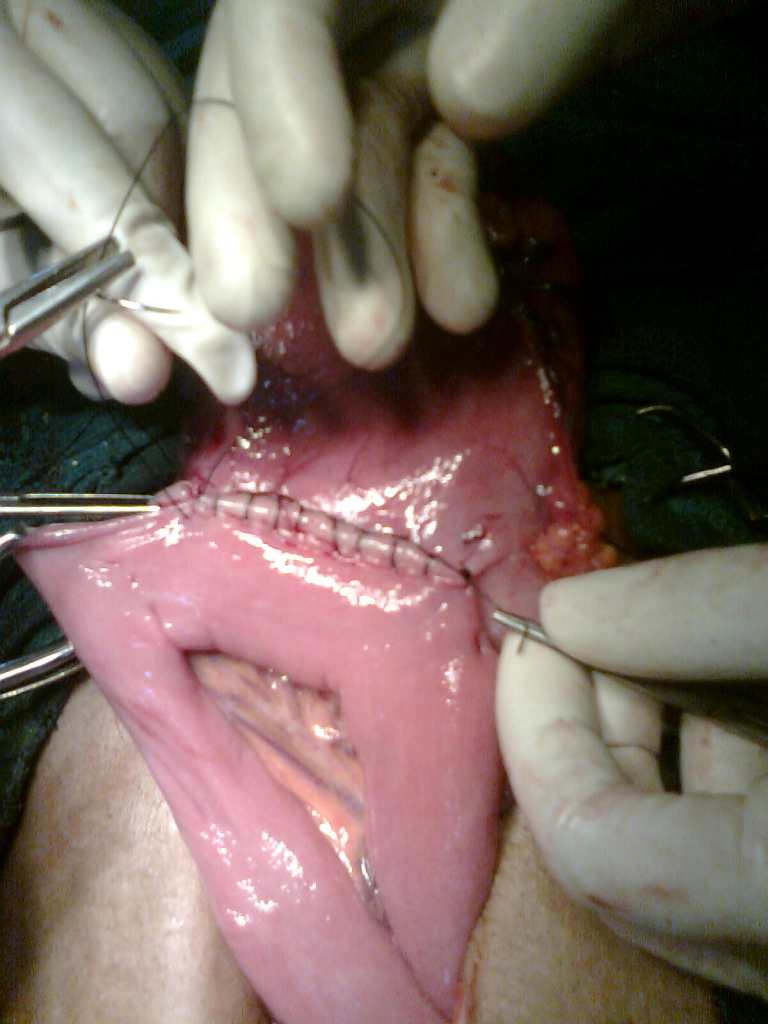

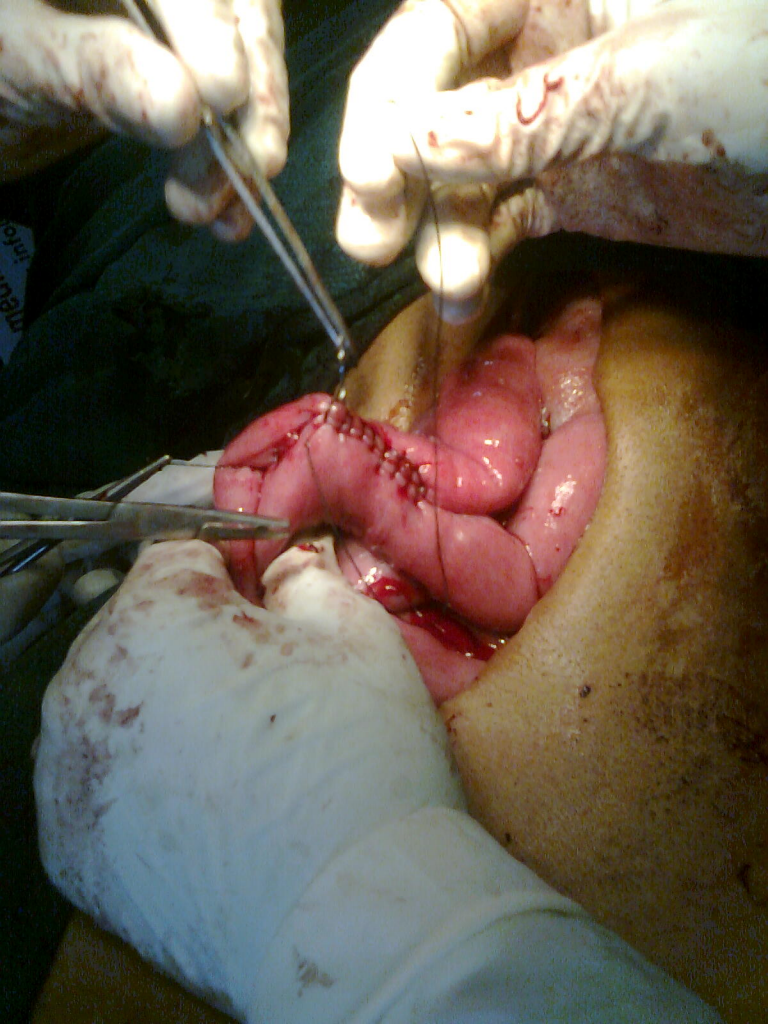

S.NO	NAME	AGE/YRS	SEX	I.P.NO	Bl:GROUP
1	ARUNACHALAM	62	M	39878	B+ve
2	GOMATHY	40	F	57027	B+ve
3	THAVASIRAJAN	60	M	55130	B+ve
4	PANDIAN	65	M	58068	O+ve
5	GOMATHI	55	F	53908	B+ve
6	ARUMUGAM	55	M	55826	AB+ve
7	CHELLAMMAL	55	F	50038	A+ve
8	SUDALAIMUTHU	75	M	42664	A+ve
9	RAJAMANI	65	M	10288	A-ve
10	AADINACHIYAR	55	F	16649	AB+ve
11	KUMAR	47	M	15487	B+ve
12	SUNDAR	48	M	39244	B+ve
13	GOMATHI	60	F	9653	O+ve
14	CHOKKALINGAM	65	M	27941	A+ve
15	RATHINASAMY	35	M	26665	A+ve
16	MUNIYANDI	51	M	26764	A+ve
17	VANAMAMALAI	50	M	49828	B+ve
18	VIMALA LINCY THANGAM	61	F	33249	A+ve
19	LAXMI	55	F	33888	B+ve
20	MUTHULAKSHMI	47	F	31290	A+ve
21	PATTURAJ	39	M	37201	B+ve
22	SHANMUGAIAH	68	M	32495	B-ve
23	MUTHAMMAL	60	F	39234	AB+ve
24	ANGAMMAL	41	F	38554	O+ve
25	MAHESWARI	41	F	41510	B+ve
26	CHELLAMMAL	30	F	7708	AB+ve
27	ANNABABU	50	F	50670	A+ve
28	BOOTHAPAANDI	75	M	16010	B+ve
29	PALANI	48	M	2033	AB+ve
30	RAMALAKSHMI	49	F	7805	O+ve
31	KURUVIAMMAL	48	F	33931	O+ve
32	AMMAPONNU	45	F	53172	O+ve
33	ARUNACHALADEVAR	66	M	8346	O+ve
34	MADASAMY	47	M	43566	O+ve

NAME	UPPER GASTRO INTESTINAL ENDOSCOPY
ARUNACHALAM	1st AND 2nd PART OF DUODENUM DILATED
GOMATHY	ULCEROPROLIFERATIVE MASS PRESENT IN PYLORIC REGION
THAVASIRAJAN	ULCEROPROLIFERATIVE GROWTH PRESENT IN PYLORIC REGION
PANDIAN	ULCEROPROLIFERATIVE MASS PRESENT IN PYLORIC REGION
GOMATHI	ULCERATIVE GROWTH IN PYLORUS REGION
ARUMUGAM	ULCEROPROLIFERATIVE GROWTH PRESENT IN PYLORIC REGION
CHELLAMMAL	ULCERATIVE GROWTH IN PYLORUS REGION
SUDALAIMUTHU	ULCEROPROLIFERATIVE GROWTH PRESENT IN PYLORIC REGION
RAJAMANI	ULCER IN PYLORUS REGION WITH STENOSIS IN PYLORIC REGION
AADINACHIYAR	POLYPOID GROWTH IN PYLORUS REGION
KUMAR	ULCEROPROLIFERATIVE GROWTH PRESENT IN PYLORIC REGION
SUNDAR	ULCEROPROLIFERATIVE GROWTH PRESENT IN PYLORIC REGION ALONG LESSER CURVATURE
GOMATHI	ULCEROPROLIFERATIVE GROWTH PRESENT IN PYLORIC REGION ALONG LESSER CURVATURE
CHOKKALINGAM	DILATED STOMACH WITH ULCEROPROLIFERATIVE GROWTH PRESENT IN PYLORIC REGION
RATHINASAMY	ULCEROPROLIFERATIVE GROWTH PRESENT IN PYLORIC REGION
MUNIYANDI	DILATED STOMACH WITH ULCEROPROLIFERATIVE GROWTH PRESENT IN PYLORIC REGION
VANAMAMALAI	PYLORIC STENOSIS WITH DILATATION, SCOPY COULD BE PASSED BEYOND PYLORUS
VIMALA LINCY THANGAM	ULCEROPROLIFERATIVE GROWTH PRESENT IN PYLORIC REGION ALONG LESSER CURVATURE
LAXMI	GROWTH PRESENT IN PYLORIC REGION ALONG LESSER CURVATURE
MUTHULAKSHMI	PYLORUS LINITIS PLASTICA
PATTURAJ	GASTRIC EROSION , ULCER IN PYLORUS, DUODENAL ULCER WITH GASTRIC OBSTRUCTION
SHANMUGAIAH	ULCER IN 1st PART OF DUODENUM
MUTHAMMAL	ULCEROPROLIFERATIVE GROWTH PRESENT IN PYLORIC REGION ALONG GREATER CURVATURE
ANGAMMAL	ULCEROPROLIFERATIVE GROWTH PRESENT IN PYLORIC REGION ALONG GREATER CURVATURE
MAHESWARI	ULCEROPROLIFERATIVE GROWTH PRESENT IN PYLORIC REGION ALONG GREATER CURVATURE
CHELLAMMAL	DUODENAL ULCER WITH LUMEN NARROWING IN 1st PART, SCOPY COULD NOT BE PASSED BEYO
ANNABABU	ULCEROPROLIFERATIVE GROWTH PRESENT IN PYLORIC REGION
BOOTHAPAANDI	ULCEROPROLIFERATIVE GROWTH PRESENT IN PYLORIC REGION ALONG GREATER CURVATURE
PALANI	SUPERFICIAL ULCERATIVE LESION IN PYLORUS REGION
RAMALAKSHMI	ULCEROPROLIFERATIVE GROWTH PRESENT IN PYLORIC REGION
KURUVIAMMAL	ULCEROPROLIFERATIVE GROWTH PRESENT IN PYLORIC REGION
AMMAPONNU	DUODENAL ULCER WITH OUTLET OBSTRUCTION
ARUNACHALADEVAR	ULCERATIVE GROWTH IN PYLORUS REGION
MADASAMY	STOMACH GROSSLY DILATED , PYLORIC OPENING STENOSED, SCOPY COULD NOT BE PASSED BEY

NAME	HISTO PATHOLOGICAL REPORT REPORT	SURGERY
ARUNACHALAM	DUODENAL ULCER WITH GASTRIC OUTLET OBSTRUCTION	TV/PGJ
GOMATHY	POORLY DIFFERENTIATED ADENO CARCINOMA	PG/GJ
THAVASIRAJAN	WELL DIFFERENTIATED ADENO CARCINOMA	AGJ
PANDIAN	POORLY DIFFERENTIATED ADENO CARCINOMA	TG/OJ
GOMATHI	POORLY DIFFERENTIATED ADENO CARCINOMA	AGJ
ARUMUGAM	MODERATELY DIFFERENTIATED ADENOCARCINOMA	AGJ
CHELLAMMAL	MODERATELY DIFFERENTIATED ADENOCARCINOMA	AGJ
SUDALAIMUTHU	MODERATELY DIFFERENTIATED ADENOCARCINOMA	PG/GJ
RAJAMANI	CHRONIC GASTRIC ULCER WITH OUTLET OBSTRUCTION	DG/GJ
AADINACHIYAR	MODERATELY DIFFERENTIATED ADENOCARCINOMA	AGJ
KUMAR	MODERATELY DIFFERENTIATED ADENOCARCINOMA	DG/GJ
SUNDAR	POORLY DIFFERENTIATED ADENO CARCINOMA	AGJ
GOMATHI	MODERATELY DIFFERENTIATED ADENOCARCINOMA	AGJ
CHOKKALINGAM	POORLY DIFFERENTIATED ADENO CARCINOMA	AGJ
RATHINASAMY	POORLY DIFFERENTIATED ADENO CARCINOMA	PG/GJ
MUNIYANDI	POORLY DIFFERENTIATED ADENO CARCINOMA	SG/GJ
VANAMAMALAI	PYLORIC STENOSIS WITH GASTRIC DILATATION	TV/PGJ
VIMALA LINCY THANGAM	MUCINOUS ADENOCARCINOMA	TG/OJ/FJ
LAXMI	MODERATELY DIFFERENTIATED ADENOCARCINOMA	AGJ
MUTHULAKSHMI	MODERATELY DIFFERENTIATED ADENOCARCINOMA	AGJ
PATTURAJ	DUODENAL ULCER WITH GASTRIC OUTLET OBSTRUCTION	TV/PGJ
SHANMUGAIAH	CHRONIC DUODENAL ULCER WITH OUTLET OBSTRUCTION	TV/PGJ
MUTHAMMAL	MODERATELY DIFFERENTIATED ADENOCARCINOMA	PG/GJ
ANGAMMAL	POORLY DIFFERENTIATED ADENO CARCINOMA	PG/GJ
MAHESWARI	POORLY DIFFERENTIATED ADENO CARCINOMA	PG/GJ
CHELLAMMAL	DUODENAL ULCER WITH GASTRIC OUTLET OBSTRUCTION	TV/PGJ
ANNABABU	WELL DIFFERENTIATED ADENO CARCINOMA	TG/OJ
BOOTHAPAANDI	MODERATELY DIFFERENTIATED ADENOCARCINOMA	PG/GJ
PALANI	MODERATELY DIFFERENTIATED ADENOCARCINOMA	AGJ
RAMALAKSHMI	MODERATELY DIFFERENTIATED ADENOCARCINOMA	AGJ
KURUVIAMMAL	MODERATELY DIFFERENTIATED ADENOCARCINOMA	AGJ
AMMAPONNU	CHRONIC DUODENAL ULCER WITH OUTLET OBSTRUCTION	TV/PGJ
ARUNACHALADEVAR	MODERATELY DIFFERENTIATED ADENOCARCINOMA	CT
MADASAMY	DUODENAL ULCER WITH GASTRIC OUTLET OBSTRUCTION	TV/PGJ

NAME	PER OPERATIVE FINDING
ARUNACHALAM	DILATED STOMACH, CICATRIZED ULCER IN 1st PART OF DUODENUM
SUBBAMMAL	STOMACH GROSSLY DILATED, MASS OF 3 X 3 CMS IN PYLORIC REGION
THAVASIRAJAN	ULCEROPROLIFERATIVE MASS IN PYLORIC REGION
PANDIAN	MASS IN ANTRUM FIXED TO POSTERIOR STOMACH BED STRUCTURES
GOMATHI	ULCERATIVE MASS IN PYLORIC REGION
NARAYANAN	ASCITES, GROWTH IN PYLORIC PART, SEROSAL INVOLVEMENT
CHELLAMMAL	STOMACH GROSSLY DILATED, MASS IN PYLORIC REGION
SUDALAIMUTHU	STOMACH DILATED, MASS OF 12 X 7 CMS IN PYLORIC REGION
RAJAMANI	ULCER 1 X 1 CM IN PYLORIC ANTRUM WITH STENOSIS IN PYLORUS
AADINACHIYAR	POLYPOID MASS OF IN PYLORIC REGION
KUMAR	MASS OF 5 X 3 CMS IN PYLORIC REGION
SUNDAR	MASS OF IN PYLORIC REGION
GOMATHI	ULCERATIVE MASS OF IN PYLORIC REGION
CHOKKALINGAM	ULCEROPROLIFERATIVE GROWTH OF IN PYLORIC REGION
RATHINASAMY	ULCERPROLIFERATIVE GROWTH OF 10 X 4 CMS IN PYLORIC REGION
MUNIYANDI	GROWTH OF 5 X 3 CMS IN PYLORIC REGION
VANAMAMALAI	DILATED STOMACH, CICATRIZED ULCER IN PYLORUS
VIMALA LINCY THANGAM	ANTERIOR WALL INFILTRATION, LIVER METASTSIS, ASCITES
LAXMI	MASS IN PYLORUS
HARICHANDRAN	MASS IN ANTRUM FIXED TO POSTERIOR STOMACH BED STRUCTURES
PATTURAJ	DILATED STOMACH CICATRIZED DUODENAL ULCER IN 1st PART OF DUODENUM
MUTHU	STOMACH DILATED, CICATRIZED ULCER IN 1st PART OF DUODENUM
MUTHAMMAL	GROWTH IN PREPYLORIC REGION OF 7 X 6 CMS
ANGAMMAL	MASS OF 6 X 2 CMS IN PYLORIC REGION
MAHESWARI	ULCEROPROLIFERATIVE GROWTH OF 8 X 5 CMS IN PYLORIC REGION
CHELLAMMAL	DILATED STOMACH, CICATRIZATION IN PYLORUS WITH PYLORIC OBSTRUCTION
ANNABABU	MASS OF 9 X 5 CMS IN PYLORIC REGION
BOOTHAPAANDI	MASS OF 6 X 4 CMS IN PYLORIC REGION
PALANI	GROWTH IN STOMACH WITH METASTASES IN LIVER, SIGMOID COLON
RAMALAKSHMI	GROWTH IN PYLORIC REGION
KURUVIAMMAL	GROWTH IN PYLORIC REGION
CHELLAIAH	DILATED STOMACH WITH CICATRIZED ULCER IN PYLORIC ANTRUM
ARUNACHALADEVAR	GROWTH IN PYLORIC REGION
MADASAMY	CICATRIZED DUODENAL ULCER IN 1st PART OF DUODENUM