STUDY ON CARCINOMA STOMACH WITH SPECIAL EMPHASIS ON THE SIGNIFICANCE OF PERITONEAL WASH CYTOLOGY DURING LAPROSCOPY AND LAPAROTOMY IN UNSEEN METASTASIS

A Dissertation submitted to
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In partial fulfillment of the requirements
for the degree of

MASTER OF SURGERY
IN
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CHENNAI – 600 001.

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CERTIFICATE

This is to certify that the dissertation entitled **"STUDY ON CARCINOMA STOMACH WITH SPECIAL EMPHASIS ON THE SIGNIFICANCE OF PERITONEAL WASH CYTOLOGY DURING LAPROSCOPY AND LAPAROTOMY IN UNSEEN METASTASIS"** is a bonafide research work done by **Dr. G. RANGARAJAN**, post graduate (2014-2017) in the department of general surgery, Govt. Stanely Medical College and Hospital, Chennai under my direct guidance and supervision, in partial fulfillment of the regulations of the TAMILNADU DR. MGR MEDICAL UNIVERSITY Chennai for the award of M.S. degree (General Surgery) Branch-I examination to be held in APRIL 2017.

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DECLARATION BY THE CANDIDATE

I hereby declare that this dissertation entitled in **"STUDY ON CARCINOMA STOMACH WITH SPECIAL EMPHASIS ON THE SIGNIFICANCE OF PERITONEAL WASH CYTOLOGY DURING LAPROSCOPY AND LAPAROTOMY IN UNSEEN METASTASIS"** is a bonafide and genuine research work carried out by me under the guidance and supervision of my unit chief **Dr. J. LALITHKUMAR, M.S.,** and my Head of Department **Dr. D. NAGARAJAN, M.S.**

This dissertation is submitted to The Tamilnadu Dr. M.G.R. Medical University, Chennai in partial fulfillment of the university regulations for the award of M.S., Degree (General Surgery) Examination to be held in April 2017.

Date:
Place: Chennai G. RANGARAJAN

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It gives me great pleasure in preparing this dissertation and I take this opportunity to thank everyone who made this possible.

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LIST OF ABBREVIATIONS

UGI – Upper Gastro intestinal

PWC – Peritoneal Wash Cytology

SL – Staging Laparoscopy

CECT – Contrast enhanced Computerized Tomography

CA - Carcinoma

D-Lap – Diagnostic Laparoscopy

LOA & LOW – Loss of Appetite & Loss of Weight

GJ – Gastro Jetenostomy

EGC – Early Gastric cancer

CT – Computerized Tomography

PET – Positron Emission Tomography

PR – Pulse Rate

BP – Blood Pressure

TLC – Total leucocyte count

DC – Differential count

LFT – Liver Function Test

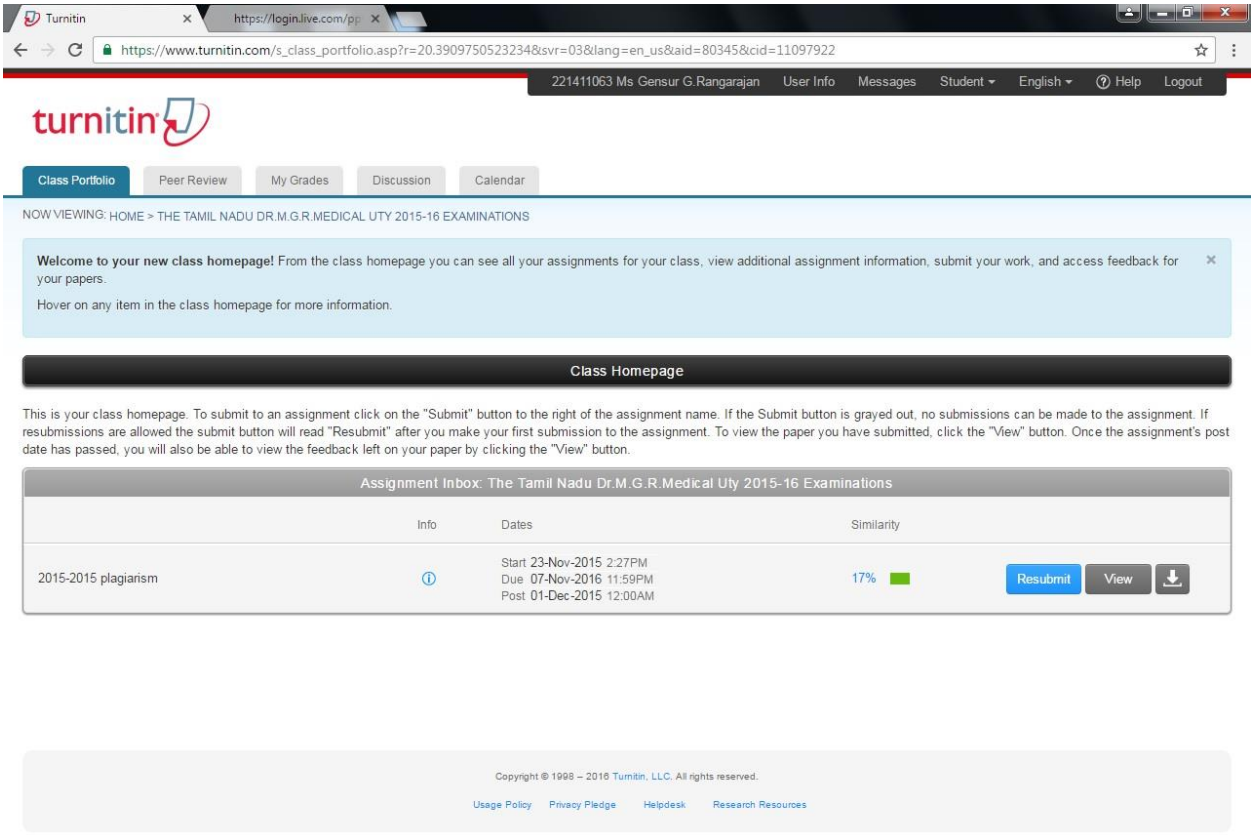
DBR – Direct Bilirubin

ALP – Alkaline Phosphatase

RFT – Renal Function Test

ECG – Electro Cardiogram

PLAGIARISM_CERTIFICATE



ETHICAL CLEARANCE CERTIFICATE

INSTITUTIONAL ETHICAL COMMITTEE,
STANLEY MEDICAL COLLEGE, CHENNAI-1

Title of the Work	:	Study on Carcinoma stomach with special Emphasis On the Signficance of peritoneal wash cytology During Laparoscopy/Laparotomy in unseen metatasis.
Principal Investigator	:	Dr. G. Rangarajan
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The request for an approval from the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 14.06.2016 at the Council Hall, Stanley Medical College, Chennai-1 at 2PM

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4. You should abide to the rules and regulation of the institution(s).
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6. You should submit the summary of the work to the ethical committee on completion of the work.

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ABSTRACT

Background:

Carcinoma stomach is one of the leading causes of cancer related deaths. The potentially curative treatment for gastric adenocarcinoma is a complete margin negative (R_0) resection. Hence, surgery forms the mainstay of treatment of carcinoma stomach. The commonest pattern of gastric cancer relapse is peritoneal metastasis which is the foremost cause of death even after curative resection is done. Studies have shown that despite radiological findings ruled out metastasis, staging laparoscopy contributes substantially in upstaging the disease.

Also positive peritoneal wash cytology during laparoscopy or laparotomy & proceed indicates peritoneal micromets and patients with positive peritoneal wash cytology has been regarded as stage IV disease and should be treated with palliative intent.

Objectives:

1. To study the Age & Sex incidence of CA Stomach
2. To study the presenting complaints and mode of presentation of CA stomach
3. To categorize the anatomical site of tumor
4. To classify the patients into curative or palliative group
5. To study the significance of peritoneal lavage cytology during laparoscopy/ laparotomy procedure

Methods:

The study was conducted on 22 patients diagnosed with adenocarcinoma of the stomach who were admitted in Government Stanley Medical College between June 2015 to September 2016. Patients admitted with gastric malignancy who satisfied the inclusion and exclusion criteria were included in the study. Upper GI scopy and CECT Abdomen were done as a routine to confirm the disease and to rule out metastasis respectively. Patients without obvious metastasis in CECT Abdomen were subjected to either staging laparoscopy or laparotomy and proceed.

During diagnostic laparoscopy, patients with obvious peritoneal and liver mets were noted and analyzed. These patients were excluded from taking peritoneal wash cytology.

Remaining patients with no evidence of mets (during CECT and Laparoscopy) were subjected to peritoneal wash cytology and their results were noted and analysed.

Results:

Out of 22 patients studied, 4 were women (18%) and remaining 18 were men (82%). Highest number of patients, 11 cases (50%) found to be in the age group of 40 to 60 years, 5 cases (22%) found to be in the age group of <40 years and 6 cases (28%) were in the age group of >60 years.

Out of 11 patients with Metastatic CA, 9 patients were greater 50 years. Out of 10 patients with Locally Advanced CA, 7 patients were less than 50 years.

Most common presenting complaints are Abdomen Pain (63%), Vomiting (63%) and LOA & LOW (77%).

Out of 22 patients studied, 9 cases are smoker and alcoholic and remaining 13 cases were non smoker and non alcoholic.

During general examination of patients included in my study, 10 cases were found to be anaemic. Out of 10 patients who are Anaemic, 7 patients were Metastatic and 3 patients were Locally Advanced.

Out of 22 patients, 9 cases were presented with Gastric outlet obstruction, 7 cases had Abdominal pain, 5 cases had epigastric mass for evaluation and only 1 case with UGI Bleed.

The most common blood group in my study were O +ve and A +ve (7 cases each).

During Upper GI Scopy, the most common site of growth were located in Antro pylorus of stomach (16 cases) percentage being 72%.

In CECT (Abdomen and pelvis) of study patients, 8 patients were metastatic. The remaining 14 patients subjected to D - lap/Laparotomy, 2 cases were found to be metastatic (peritoneal mets) and 12 cases were subjected to peritoneal wash cytology out of which 1 case (8.33%) was found to be positive for peritoneal cytology.

Conclusion:

Peritoneal Wash cytology during Laparoscopy/Laparotomy though many studies have shown to **upstage** the disease process from locally Advanced to Metastatic.

Moreover, Positive cytology rate 4.4 – 11% reported in literature which reflects the heterogeneity of patient cohorts with variable disease severity, Experience of Pathology, Duration of sample retrieval to Sample Analysis & Differences in diagnostic criteria.

In my study, there were only **1 positive cytology patients** out of 12 patients (D-Lap negative) examined. Positive cytology rate being 8.33%

Hence institutional based study should be done to establish the significance of Peritoneal Wash cytology in CA Stomach.

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Introduction

INTRODUCTION

The fourth most common cancer is Gastric Cancer and it is the second leading cause of cancer death worldwide. High occurrence of local and distant recurrence even in patients with resectable gastric cancer suggests the notorious behavior of gastric cancer. These indications suggest the systemic spread of cancer very early in the disease process. Rate of gastric cancer in India is less compared to worldwide incidence.

In India, gastric cancer seems to be increasing in North east and Southern parts of the country.

Incidence of gastric cancer is four times higher in South India than North India and the incidence in Chennai is 13.1/1,00,000 population.

There are only two options available for the Management of Gastric cancer i.e., Curative or Palliative.

Complete operative resection remains the only potentially curative modality for gastric carcinoma.

Gastric cancer is more prone for metastasis apart from CECT Abdomen and chest. Laparoscopic staging must form a part of workup to assess Peritoneal and Liver mets which helps to avoid unnecessary laparotomy for advanced cases.

Laparoscopic staging may substantially reduce the need for exploratory laparotomy. Also, peritoneal washings can be performed during staging laparoscopy to detect intraperitoneal free cancer cells thus patients with radiologically resectable gastric cancer whereas with positive peritoneal wash cytology is associated with early disease recurrence and poor survival despite Ro resections.

In this study, apart from Study of Age, Sex, Incidence, Mode of presentation and Site of tumor, we subject the patients for peritoneal wash cytology taken either during laparoscopy or laparotomy & proceed. Hence patients with locally advanced stage are subjected to peritoneal wash cytology during surgery and their results are analyzed.

Objectives

OBJECTIVES

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Review of Literature

REVIEW OF LITERATURE

ANATOMY

Anatomically stomach is divided into:

1. Fundus – Part of the stomach lying above a horizontal plane from cardiac notch to greater curvature

2. Body of the stomach – Part lying between the Fundus and Pyloric part of stomach being demarcated from the pyloric part of the stomach by a plane drawn from incisura angularis to the greater curvature.

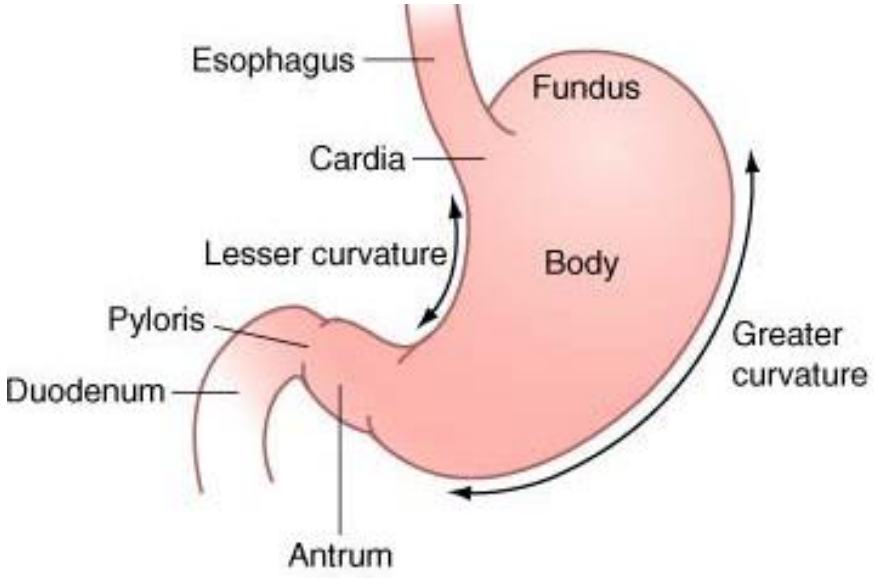
3. Pyloric portion – further subdivided into

    a. pyloric antrum – extends from incisura angularis to another plane drawn from the right end of the bulging of greater curvature.

    b. Pyloric canal – narrowed part of distal stomach extending from end of pyloric antrum to pyloric orifice.

4. Lesser curvature – concave border of the stomach and is continuous with right free border of esophagus.

5. Greater curvature of the stomach – this is the convex border of the stomach and starts at left border of esophagus where it joins the stomach.



Interior of Stomach:

1. The stomach shows the same four layers as any other part of G.I.T.
2. Mucous membrane
3. Sub mucous layer.
4. Muscularis propria
5. Serous layer.

Mucosa:

1. Thrown into folds called 'Rugae'.

2. The epithelial surface is divided into small areas called mamillated areas – studded with numerous depressions called gastric pits.[gastric glands open in these pits]

3. The mucous membrane has (i) surface epithelium (ii) lamina propria (iii) glands of stomach (iv) muscularis mucosa.

4. Surface epithelium – Is similar from cardiac to pyloric region – tall columnar cells – starts abruptly at the junction between oesophagus & stomach.

5. Lamina propria – made up of delicate connective tissue and contains glands of the stomach and also infiltrated with lymphocytes through out.

6. Muscularis mucosa – made up of smooth muscle fibres arranged in inner circular and outer longitudinal layers.

Sub Mucosa:

Loose connective tissue with collagenous, reticular & elastic fibres along with lymph vessels & blood vessels.

Muscularis propria:

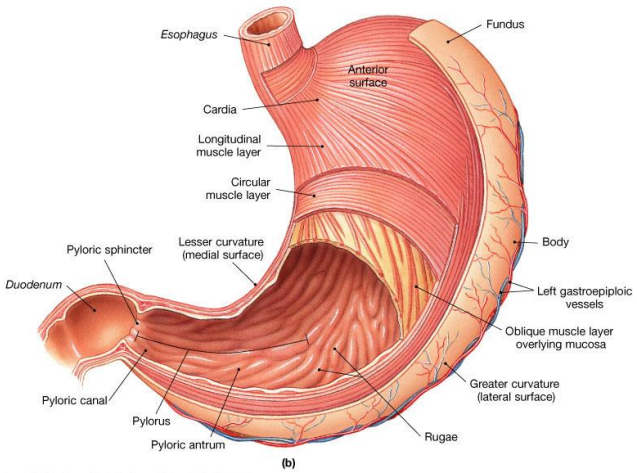
1. Outer longitudinal layer – continuous with the outer longitudinal layer of oesophagus.

2. Middle circular layer – continuous with the inner layer of oesophagus.

3. Intl. oblique layer – not a complete layer. In some places it forms loops of muscle fibres extending from cardiac orifice around the fundus & body.

Serous layer:

Thin layer of loose connecting tissue underlying a layer of simple squamous mesothelium.

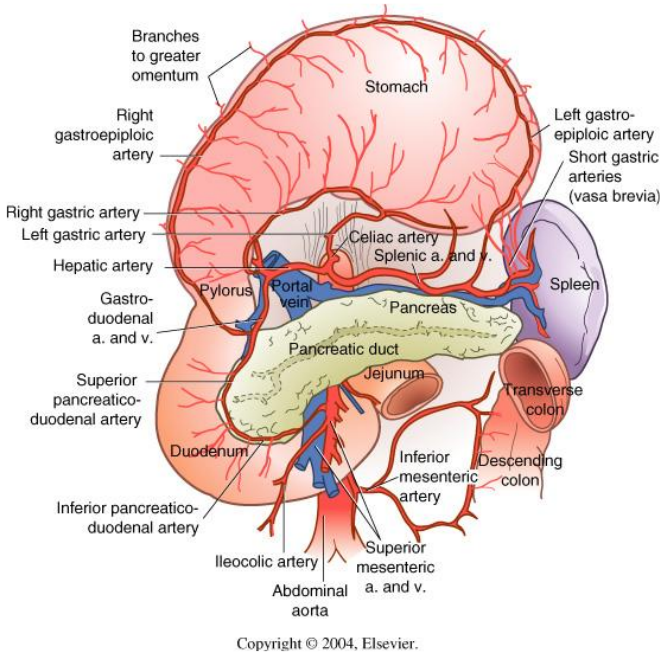


Glands of Stomach:

1. Cardiac portion – contains mucus secreting glands only.
2. Fundic portion – lies between the pyloric gland area and the cardia – mucosa contain parietal cells [acid secreting] & chief cells **[**pepsin secreting]
3. Pyloric portion – contains mucus secreting cells & gastrin secreting cells.

Blood Supply:

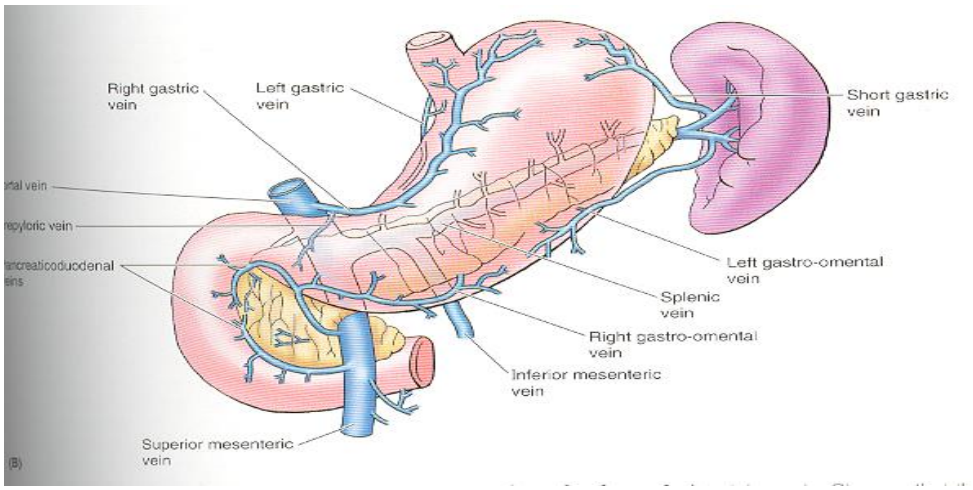
1. Rich arterial supply of stomach arises from the celiac trunk and its branches
2. Blood is mostly supplied by anastomoses formed along the lesser curvature by the right and left gastric arteries, and along the greater curvature by the right and left gastro-omental (gastroepiploic) arteries.
3. Short and posterior gastric arteries supplies blood to fundus and upper body.
4. The veins of the stomach parallel the arteries in position and course
5. Lt. gastric artery: Arises from the coeliac axis and divides into an ascending [oesophageal] & descending branch.
6. The descending branch lying between the layers of lesser omentum is closely opposed to lesser curvature & sends branches to the stomach.
7. Rt. Gastric artery: Arises from common hepatic artery & divides into many branches along the lesser curvature.
8. Anastamoses with the left gastric artery.
9. Rt. Gastro epiploic artery: Arises from the gastro duodenal artery – anastamoses with Lt. gastro epiploic & forms an arcade supplying the greater curvature.
10. Lt. gastro epiploic artery: Arises from splenic artery.
11. Short gastric [vasa brevia]: 5 – 7 small branches arising from splenic artery to supply fundus.



Venous Drainage:

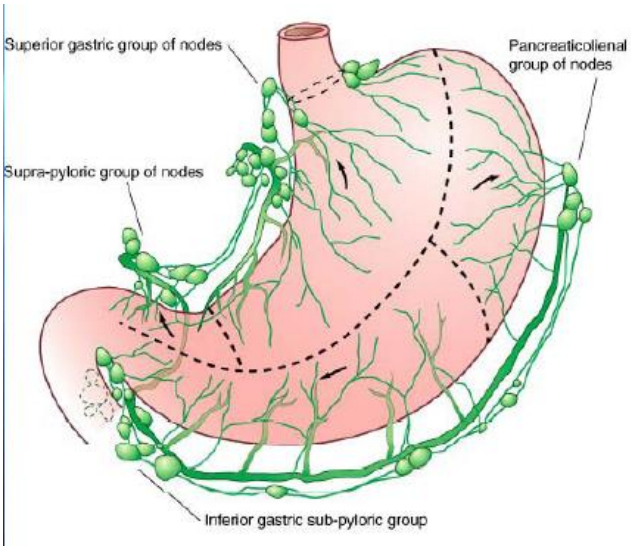
1. Venous Drainage Veins: Accompany the arteries, drain into the portal, supr. Mesentric & splenic vein.

2. Lt. gastric [coronary] vein: receives branches from oesophagus



Lymphatic Drainage of stomach:

TABLE – Lymphatic Drainage of Stomach	
Zone I (Inferior gastric)	Nodes around right gastroepiploic and gastroduodenal arteries to nodes around hepatic artery to celiac nodes
Zone II (splenic)	Nodes around left gastroepiploic and short gastric arteries to pancreaticosplenic nodes to splenic artery nodes to celiac nodes
Zone III (superior gastric)	Nodes around the left gastric artery to celiac nodes
Zone IV (hepatic)	Nodes around the right gastric artery to celiac nodes



Eight groups of lymph nodes of stomach from a surgicoanatomic standpoint are:

1. Paracardial nodes
2. Left gastric nodes at the left gastric artery
3. Celiac nodes at the celiac artery
4. Suprapyloric nodes
5. Infrapyloric nodes
6. Right gastroepiploic nodes at the pathway of the right gastroepiploic artery.

Nerve supply of stomach:

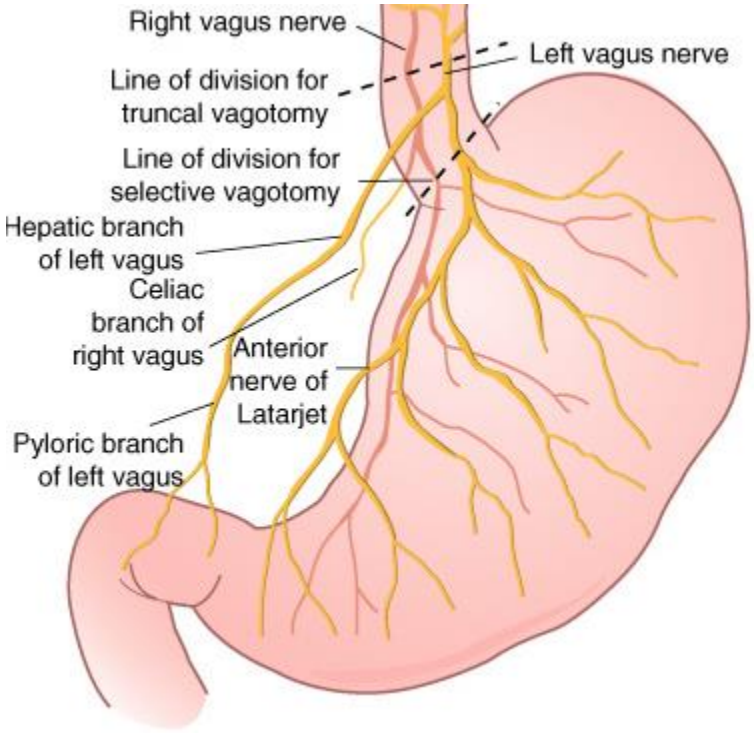
Sympathetic – Derived from segments T6 to T10 of the spinal cord via the greater splanchnic nerves & the coeliac and hepatic plexuses.

Vasomotor – Motor to the pyloric sphincter but inhibitory to the rest of gastric musculature.

Parasympathetic – Derived from the vagi, through the oesophageal plexus & gastric nerves. Motor – increases motility of stomach. Secreto motor – For secretion of gastric juice.

Antr. Gastric nerve – mainly the Lt. vagal fibres – pylorus & antr.surface.

Postr. Gastric nerve – mainly Rt. Vagal fibres – coeliac branches & postr. Surface.



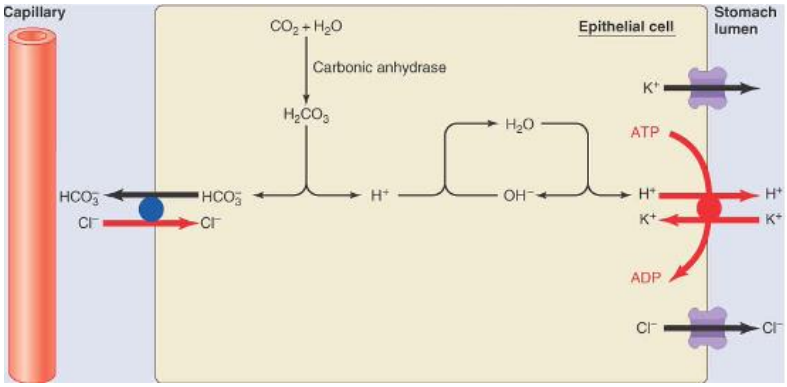
PHYSIOLOGY

Functions of stomach:

1. Bulk storage of undigested food
2. Mechanical breakdown of food
3. Disruption of chemical bonds via acids enzymes (pepsin)
4. Production of intrinsic factor
5. Very little absorption of nutrients
    a. Some drugs, however, are absorbed
6. Enteroendocrine cells

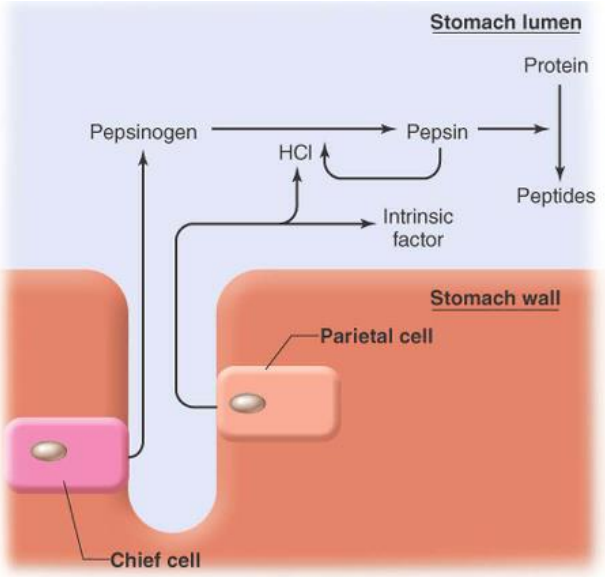
Gastric Acid Secretion:

Acid production by the parietal cells in the stomach depends on the generation of carbonic acid; subsequent movement of hydrogen ions into the gastric lumen results from primary active transport.



1. One inhibitory and three stimulatory signals that alter acid secretion by parietal cells in the stomach.
2. Gastrinand Ach work by increasing [Ca++] and activate Protein Kinases
3. Histamine works via a H2receptor and by a cAMP mechanism
4. All 3 work synergistically.

The acidity in the gastric lumen converts the protease precursor pepsinogento pepsin; subsequent conversions occur quickly as a result of pepsin's protease activity.



Functions of the Gastrointestinal Hormones

Site of Production	Method of Stimulation	Secretory Effects	Motility Effects
Gastrin			
Stomach and duodenum	Distention; partially digested proteins, autonomic stimulation, ingestion of alcohol or caffeine	Increases gastric secretion	Increases gastric emptying by increasing stomach motility and relaxing the pyloric sphincter
Secretin			
Duodenum	Acidity of chyme	Inhibits gastric secretion; stimulates pancreatic secretions high in bicarbonate ions; increases the rate of bile and increases intestinal secretion; mucus secretion	Decreases gastric motility
Cholecystokinin			
Intestine	Fatty acids and other lipids	Slightly inhibits gastric secretion; stimulates pancreatic secretions high in digestive enzymes; and causes contraction of the gallbladder and relaxation of the hepatopancreatic ampullar sphincter	Decreases gastric motility
Gastric Inhibitory Polypeptide			
Duodenum and proximal jejunum	Fatty acids and other lipids	Inhibits gastric secretions	Decreases gastric motility

EPIDEMIOLOGY OF ADENOCARCINOMA STOMACH

The fourth most common cancer is Gastric Cancer and it is the second leading cause of cancer death worldwide. In India, gastric cancer seems to be increasing in North east and Southern parts of the country. Incidence of gastric cancer is four times higher in South India than North India and the incidence in Chennai is 13.1/1,00,000 population. Over the past few decades incidence of gastric cancer is decreasing in trend. This decrease is more in the intestinal form of gastric cancer rather than diffuse variety.

Gastric cancer is the disease of elderly.

In younger patients, tumors are

Diffuse variety,

Tends to be large and aggressive,

Poorly differentiated.

Incidence of gastric cancer is shifting from distal to proximal stomach.

ETIOLOGY OF GASTRIC CANCER

Gastric cancer is more common in patients with pernicious anemia, blood group A, family history of gastric cancer.

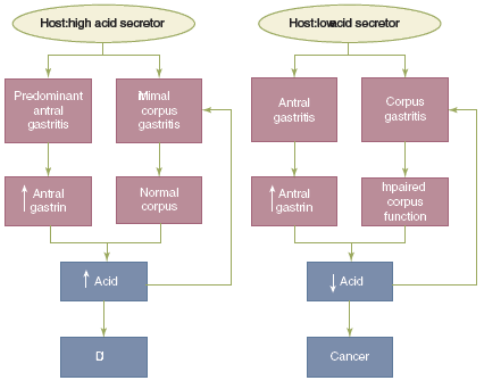
Factors increasing or decreasing the risk of gastric cancer

- Increase risk
  - Family history
  - Diet (high in nitrates, salt, fat)
  - Familial polyposis
  - Gastric adenomas
  - Hereditary nonpolyposis colorectal cancer
  - *Helicobacter pylori* infection
    - Atrophic gastritis, intestinal metaplasia, dysplasia
  - Previous gastrectomy or gastrojejunostomy (>10 y ago)
  - Tobacco use
  - Ménétrier's disease
- Decrease risk
  - Aspirin
  - Diet (high fresh fruit and vegetable intake)
  - Vitamin C

Diet and drugs – Starchy diet high in pickled, salted or smoked food carries high risk of gastric cancer. Dietary nitrates are also a possible cause of gastric cancer. Diets high in fresh fruits and vegetables, rich in vitamin C & D decreases the risk of gastric cancer. Tobacco use probably increases the risk of stomach cancer.

Helicobacter pylori – the risk of gastric cancer in patients with chronic H.pylori infection is increased threefold. Patient with history of gastric ulcer are more likely to develop gastric cancer than patients with duodenal ulcer. This is because these patients develop corpus predominant gastritis resulting in hypochlorhydria and predisposing to gastric ulcer and gastric cancer.

Patients infected with virulent cag A, vacA strains have increased chance of getting gastric cancer. Infection decreases acid – pepsin secretion.



Epstein - Barr virus – 10% of adenocarcinoma harbor EBV virus. EBV transcripts are present in cancer cells but not in metaplastic cells of precursor epithelium.

Genetic abnormalities

a. **Hereditary diffuse gastric cancer**

a.i. Inherited form of gastric cancer resulting from a gene mutation for the cell adhesion molecule E-cadherin,

a.ii. 80% lifetime chance of developing gastric cancer

a.iii. Prophylactic total gastrectomy should be considered

b. **Familial Adenomatous Polyposis**

b.i. 85% of the patients have fundic gland polyps out of which 40% have some type of dysplasia and over 50% have sometic apc mutation.

c. Li – Fraumeni syndrome – Auto fomal dominant disorder caused by mutation in tumor suppressor p53 gene. These patients are at risk of gastric cancer.

d. Hereditary non polyposis colorectal cancer or Lync syndrome – Associated with microsatellite instability. Increased risk of gastric and overian cancer.

Genetic abnormalities in gastric cancer

ABNORMALITIES	GENE	APPROXIMATE FREQUENCY %
Deletion/suppression	*p53*	60–70
	FHIT	60
	APC	50
	DCC	50
	E-cadherin	<5
Amplification/ overexpression	*COX-2*	70
	HGF/SF	60
	VEGF	50
	c-met	45
	AIB-1	40
	β-*catenin*	25
	k-sam	20
	ras	10–15
	c-erb B-2	5–7
Microsatellite instability		25–40
DNA aneuploidy		60–75

Premalignant conditions of Stomach

Polyps:

Benign gastric polyps are classified as neoplastic (adenoma and fundic gland polyps) or non-neoplastic (hyperplastic polyp, inflammatory polyp, hamartomatous polyp.

Fundic gland polyps seen in patients on long term proton pump inhibitor can cause dysplasia in patients with FAP. Hyperplastic polyps > 2cm may harbor dysplasia or carcinoma insitu.

Gastric adenomas are premalignant. Patients with FAP have a high prevalence of gastric adenomatous polyp. They have 10 times more chance of developing adeno carcinoma of the stomach than the general population.

Gastric polyps more than 1 cm need to be removed to confirm diagnosis and to prevent further risk of malignancy.

Atrophic Gastritis:

It is the commonest precursor for gastric cancer especially intestinal subtype.

In most patients H.pylori is involved in pathogenesis of atrophic gastritis.

Correa described 3 patterns of chronic atrophic gastritis.

a) **Auto immune** (involves the acid secreting proximal stomach)

b) **Hypersecretory** (involving in distal stomach)

c) **Environmental** (involving multiple random areas at the junction of the oxyntic and antral mucosa).

Intestinal Metaplasia:

Gastric carcinoma often occurs insites of intestinal metaplasia.

Complete type of intestinal metaplasia, glands are completely lined with goblet cells and intestinal absorptive calls.

Eradication of H.pylori infection leads to regression of intestinal metaplasia and improvement in atropic gastritis.

Benign gastric ulcer:

All gastric ulcers are cancer until proven otherwise with adequate biopsy and follow up.

Carcinomas are occasionally found when biopsied benign ulcers are resected for non healing.

Gastric Remnant cancer:

Stomach cancer can develop in gastric remnant following distal gastrectomy for PUD.

Tumors develop >10 years after the initial surgery and arise in the area of chronic gastritis, metaplasia and dysplasia.

Site is more common near stoma.

This condition is reported following billroth II gastroenterostomy.

Menetriers disease:

Giant hypertrophy of gastric mucosal folds carries 5 to 10% risk of adenocarcinoma.

CLASSIFICATION SYSTEMS IN ADENO CARCINOMA

1. JAPANESE CLASSIFICATION FOR EARLY GASTRIC CANCER (EGC):

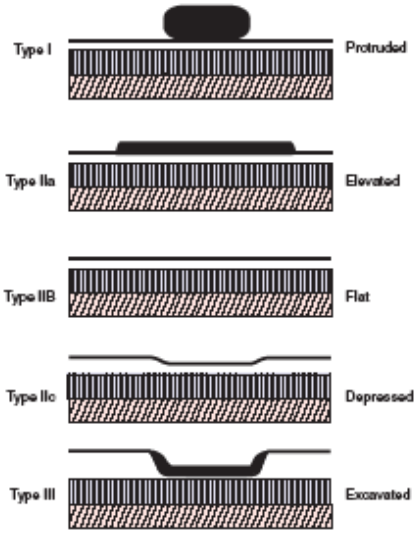
    a. EGC is gastric cancer continued to mucosa and submucosa irrespective of lymph node status.

    b. 10% of the patients with EGC have lymph node metastatis.

    c. 70% of EGC are well differentiated while 30% are poorly differentiated.

Macroscopic types of superficial gastric cancer	
Type 0-I (protruding)*	Polypoid tumors
Type 0-II (superficial)	Tumors with or without minimal elevation or depression relative to the surrounding mucosa.
Type 0-IIa	Slightly elevated tumors. (superficial elevated)
Type 0-IIb	Tumors without elevation or depression. (superficial flat)
Type 0-IIc	Slightly depressed tumors. (superficial depressed)
Type 0-III (excavated)	Tumors with deep depression

*Tumors with less than 3-mm elevation are usually classified as –IIa, with more elevated tumors being classified as 0-I.



2. CLASSIFICATION BASED ON GROSS MORPHOLOGY:

    a. Polypoidal – Here bulk of tumor mass is intraluminal, not ulcerated.

    b. Fungating – Here tumors are elevated intraluminally but also ulcerated.

    c. Ulcerative – This type of CA Stomach arises most commonly in pyloric antrum region towards lesser curvature side.

    d. Colloid – Appear on massive tumor with gelatinous appearance.

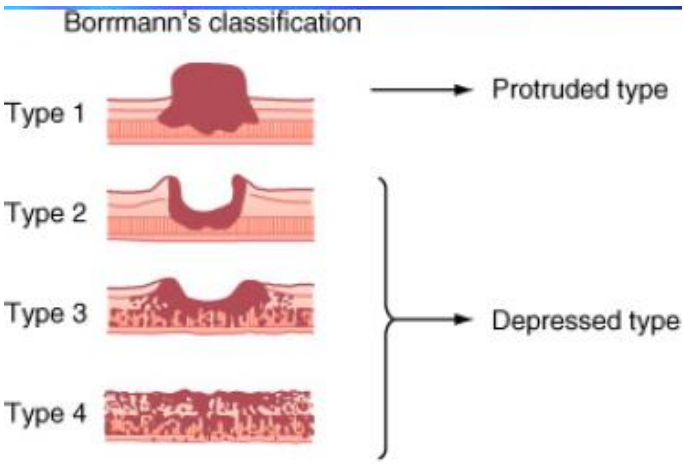
    e. Scirrhous – Rare variety, poor prognosis and involve entire stomach (submucosa, submucosal muscle coat without protruding into the lumon of the stomach)

3. BORMANN'S CLASSIFICATION FOR ADVANCED GASTRIC CANCER:

Advanced gastric cancer extends beyond submucous coat and involves muscularis propria. Classification is as follows:

Type I – Protruded

Type II, III, IV – Depressed type



4. LAURENS CLASSIFICATION OF GASTRIC CANCER BASED ON HISTOLOGY:

Type I (53%):

Intestinal type,

Arises insite of intestinal metaplasia

Forms polypoid tumor or ulcer.

Associated with Chronic Atrophic Gastritis, severe intestinal metaplasia and dysplasia

Tends to be less aggressive.

Type II (33%):

i. Diffuse type,

ii. Infiltrates deeply in the stomach wall without producing obvious mass lesion.

iii. Associated in younger patients with proximal tumor

iv. Poorly differentiated.

Type III (14%) – Otherwise not specified.

Lauren Classification System	
INTESTINAL	**DIFFUSE**
Environmental	Familial
Gastric atrophy, intestinal metaplasia	Blood type A
Men > women	Women > men
Increasing incidence with age	Younger age group
Gland formation	Poorly differentiated, signet ring cells
Hematogenous spread	Transmural,lymphatic spread
Microsatellite instability	Decreased E-cadherin
APC gene mutations	
p53, p16 inactivation	*p53, p16* inactivation

5. WHO HISTOLOGICAL TYPING OF GASTRIC CANCER:

World Health Organization histologic typing of gastric cancer
Adenocarcinoma
Papillary adenocarcinoma
Tubular adenocarcinoma
Mucinous adenocarcinoma
Signet-ring cell carcinoma
Adenosquamous carcinoma
Squamous cell carcinoma
Small cell carcinoma
Undifferentiated carcinoma
Others

RELATIVE SITE DISTRIBUTION OF GASTRIC CANCER:

a. Pyloric region – 47%
b. Body of Stomach – 23%
c. Cardia – 21%
d. Fundus – 2%
e. Limits Plastica – 7%

Recently there may be proximal migration of tumor site. So currently 40% distal, 30% middle and 30% proximal.

PROGNOSTIC FACTORS IN CA STOMACH:

1. Tumor – Depth of invasion
2. Metastasis – Lymph node or distant
3. Histological grading and type – well, moderately or poorly.

SPREAD OF CANCER STOMACH:

Carcinoma stomach spread in the different ways:

1. Direct spread – Spread from mucosa to serosa. Involves adjacent part of stomach to adjacent structures like colon, pancreas and liver, esophagus, mesocolon and rarely duodenum.
2. Lymphatic spread – Occurs by permeation and embolization
    a. First tier of lymph nodes – perigastric nodes lying within 3cm of primary growth (lymph node station 1 to 6)
    b. Next tier of lymph nodes – nodes around main and intermediate arterial trunk (lymph node station 7 to 11)
    c. Regional lymph node - lymph node station 12 to 18
    d. Lymphatic spread may occur
        d.i. Left Virchow's gland in neck via thoracic duct

d.ii. Along the lymphatics in falciform ligament leading to formation of subcutaneous nodule.

3. Blood borne spread – Liver, lungs and brain.
4. Transperitoneal spread – Tumor cells may exfoliate and drop in peritoneal cavity giving rise to Krukenberg's tumor.
5. Transluminal spread may occur
6. Transplantation – At the time of surgery cancer cells may dislodge and implant at the sites of abdominal incisions.

CLINICAL MANIFESTATION:

Most common symptoms are weight loss and decreased food intake and most common mode of presentation is silent.

EARLY GASTRIC CANCER:

1. Specific symptoms
2. Early vague epigastric discomfort
3. Indigestion
4. Constant pain, non-radiating and not relieved by food intake.

ADVANCED GASTRIC CANCER:

1. Vomiting due to obstruction
2. Dysphagia
3. UGI Bleed

SPECIFIC SYMPTOMS:

1. Proximal tumor – Dysphagia
2. Distal tumors – GOO
3. Diffuse variety – Early satiety
4. GI Bleed is rare

PHYSICAL SIGNS:

1. Weight loss and anemia
2. Palpable mass
3. Gastric outlet obstruction
4. Ascites
5. Jaundice

SIGNS OF INOPERABILITY:

1. Left supraclavicular node (Virchow's node)
2. Ascites
3. Fixed mass/posterior fixation
4. Liver metastases (Hepatomegaly, Jaundice)
5. Blumer's shelf deposit (peritoneal metastasis felt as firm shelf on rectal examination)
6. Irish node
7. Sister Mary Joseph nodule (Periumblical nodule)
8. Krukenberg's tumor (drop metastasis to ovary)

PATHOLOGIC STAGING:

The most widely used staging system is AJCC TNM Staging system. This is based on

Depth of tumor invasion (T)

Number of involved nodes (N)

Presence or absence of metastasis (M)

Minimum of 15 nodes must be evaluated for accurate staging.

TNM Classification of Carcinoma of the Stomach

PRIMARY TUMOR (T)†			
TX	Primary tumor cannot be assessed		
T0	No evidence of primary tumor		
Tis	Carcinoma in situ; intraepithelial tumor without invasion of the lamina propria		
T1	Tumor invades lamina propria, muscularis mucosae, or submucosa		
T1a	Tumor invades lamina propria or muscularis mucosae		
T1b	Tumor invades submucosa		
T2	Tumor invades muscularis propria*		
T3	Tumor penetrates subserosal connective tissue without invasion of visceral peritoneum or adjacent structures‡‡		
T4	Tumor invades serosa (visceral peritoneum) or adjacent structures‡‡		
T4a	Tumor invades serosa (visceral peritoneum)		
T4b	Tumor invades adjacent structures		
REGIONAL LYMPH NODES (N)§			
NX	Regional lymph node(s) cannot be assessed		
N0	No regional lymph node metastasis§		
N1	Metastasis in 1-2 regional lymph nodes		
N2	Metastasis in 3-6 regional lymph nodes		
N3	Metastasis in 7 or more regional lymph nodes		
N3a	Metastasis in 7-15 regional lymph nodes		
N3b	Metastasis in 16 or more regional lymph nodes		
DISTANT METASTASIS (M)			
M0	No distant metastasis		
M1	Distant metastasis		
ANATOMIC STAGE	**Prognostic Group**		
0	Tis	N0	M0
IA	T1	N0	M0
IB	T2 T1	N0 N1	M0 M0
IIA	T3 T2 T1	N0 N1 N2	M0 M0 M0
IIB	T4a T3 T2 T1	N0 N1 N2 N3	M0 M0 M0 M0
IIIA	T4a T3 T2	N1 N2 N3	M0 M0 M0
IIIB	T4b T4b T4a T3	N0 N1 N2 N3	M0 M0 M0 M0
IIIC	T4b T4b T4a	N2 N3 N3	M0 M0 M0
IV	Any T	Any N	M1

From Edge S, Byrd D, Compton C, et al (eds): AJCC cancer staging manual, ed 7, New York, 2010, Springer.

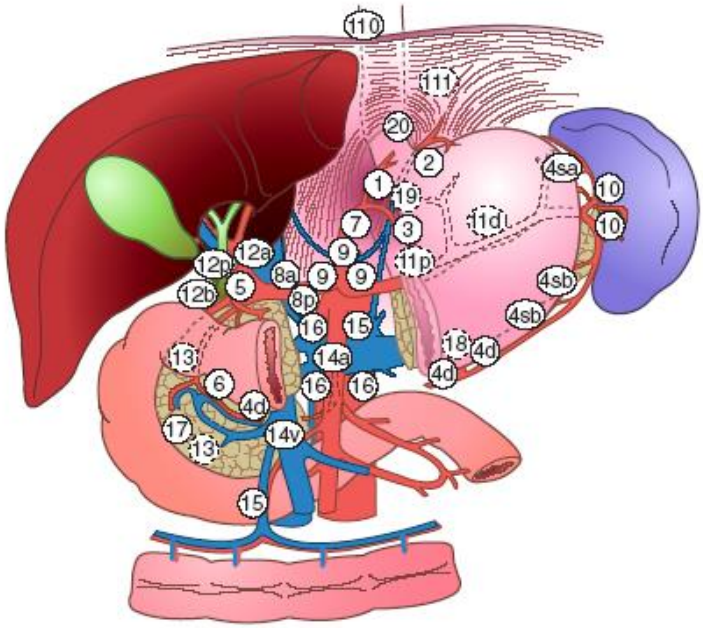
*A tumor may penetrate the muscularis propria with extension into the gastrocolic or gastrohepatic ligaments, or into the greater or lesser omentum, without perforation of the visceral peritoneum covering these structures. In this case, the tumor is classified T3. If there is perforation of the visceral peritoneum covering the gastric ligaments or the omentum, the tumor should be classified T4.

†The adjacent structures of the stomach include the spleen, transverse colon, liver, diaphragm, pancreas, abdominal wall, adrenal gland, kidney, small intestine, and retroperitoneum.

‡Intramural extension to the duodenum or esophagus is classified by the depth of the greatest invasion in any of these sites, including the stomach.

§A designation of pN0 should be used if all examined lymph nodes are negative, regardless of the total number removed and examined.

Following is the Japanese Gastric Cancer Association definition of Lymph node station number.



R status described by Hermanek describes tumor status post resection and is important for determining the completeness of surgery.

1. R0 – microscopically margin-negative resection, in which no gross or microscopic tumor remains in the tumor bed.
2. R1 - removal of macroscopic disease, but microscopic margins are positive for tumor.
3. R2 - gross residual disease.

STAGING WORKUP:

1. To ascertain prognosis to counsel the patient and family.
2. To diagonise the extent of disease and determine course of treatment.

ORDER OF INVESTIGATIONS IN STAGING WORKUP:

1. Upper GI endoscopy
2. Endoscopic Ultrasound
3. USG/CT Abdomen and Pelvis
4. X-ray chest
5. Blood investigations
6. Barium meal
7. Laparoscopy and wash cytology
8. PET Scan

Upper GI endoscopy:

1. Flexible endoscopy is the goal standard tool for the analysis of gastric cancer.
2. It allows
    a. Visualization of tumor
    b. Provides tissue for pathological diagnosis
    c. Serve as treatment for patient with obstruction or bleeding
3. Pre-requisites for doing endoscopy:
    a. 8 hours fasting
    b. Facilities for resuscitation – needed
    c. Pulsoximeter – must
    d. Sedation - small incremental doses of Diazepam/Midazolam

e. Spasmolytics (Buscopan) allows visualisation of antrum and negotiation into duodenum

4. The following point should be noted in endoscopic findings:

    a. Size

    b. Location

    c. Morphology

    d. Extent

    e. Biopsy

    f. Other parts of Stomach

    g. Endoscopic USG

    h. Chromo endoscopy

5. Endoscopic society classification of EGC:

    a. Type 1 – Protruding

    b. Type 2 – Superficial

        b.i. 2 i – elevated

        b.ii. 2 ii – flat

        b.iii. 2 iii – depressed

    c. Type 3 – excavated

6. BENIGN VS MALIGNANT:

    1. If benign ulceration is diagnosed, endoscopy and biopsies are to be repeated after four to eight weeks of medical treatment to confirm ulcer healing and benign nature of the lesion.

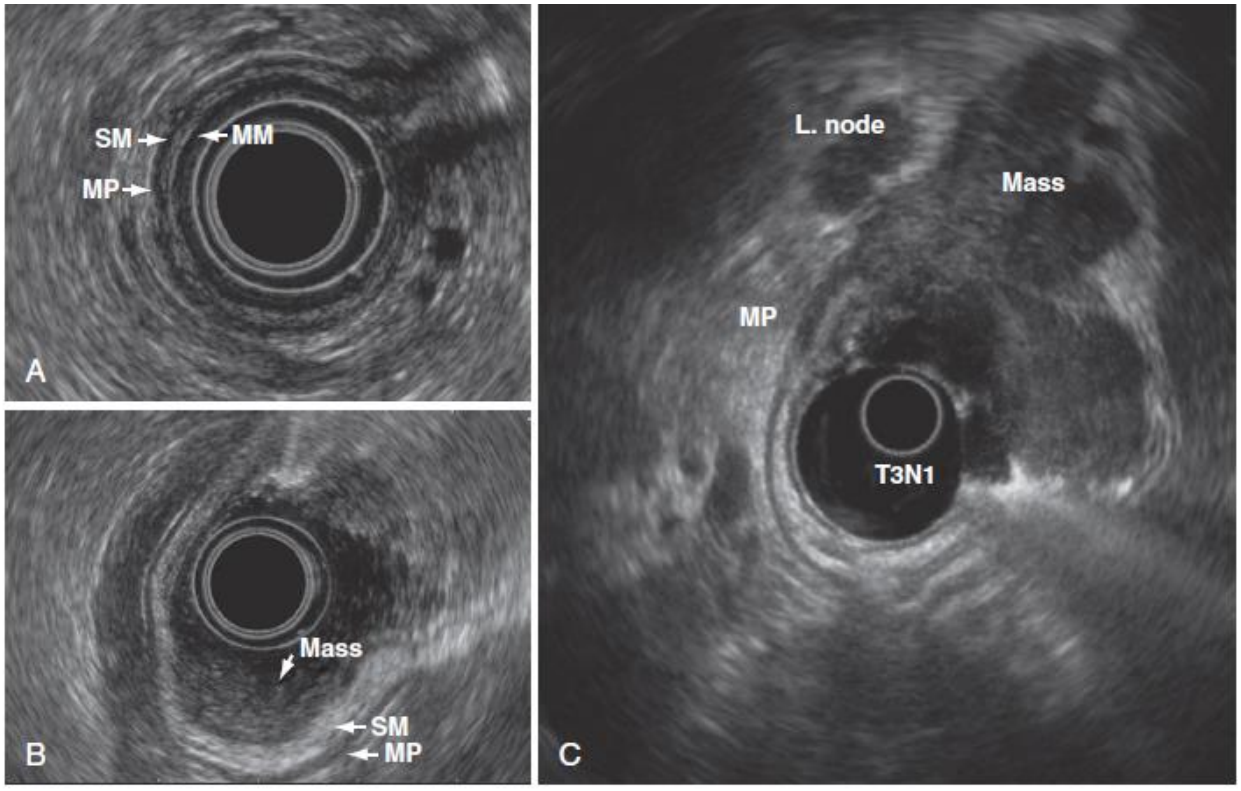
2. Benign and malignant gastric ulcers differentiation at endoscopy can be difficult, hence several biopsies need to be taken (preferably six) from all parts of the ulcer. 100% diagnostic accuracy can be attained if 10 samples are taken.

BENIGN ULCER Vs MALIGNANT ULCER:

BENIGN ULCER	MALIGNANT ULCER
1. PROJECTS BEYOND GASTRIC LUMEN. 2. SMOOTH CONTOUR. 3. MAY BE DEEP. 4. SYMMETRICAL EDEMA MOUND. 5. UNDERMINING OF ADJACENT TISSUES. 6. SMOOTH CONVERGING FOLDS. 7. HEALS COMPLETELY.	1. DOES NOT PROJECT. 2. IRREGULAR CONTOUR. 3. USUALLY SHALLOW. 4. ADJ. TISSUES USUALLY IRREGULAR, NODULAR OR RIGID. 5. NO UNDERMINING. 6. NODULAR OR IRREGULAR FOLDS. 7. RARELY HEALS COMPLETELY.

Endoscopic Ultrasound (EUS):

1. EUS is performed using flexible endoscope with 7.5 to 12 MHz ultrasound transducer.
2. The stomach is filled with water to distend the stomach and stomach wall is visualized as five alternating hypoechoic and hyperechoic layers.
3. The mucosa and submucosa represent the first three layers (T1). The fourth layer is the subserosa, invasion represents T2 tumor. The serosa is the fifth layer and tumor penetration is the T3 tumor.
4. Local lesion,regional lymph nodes and left hepatic lobe can be assessed.
5. Bulk of right hepatic lobe and peritoneum is outside the range of the probe which is approx 10cm



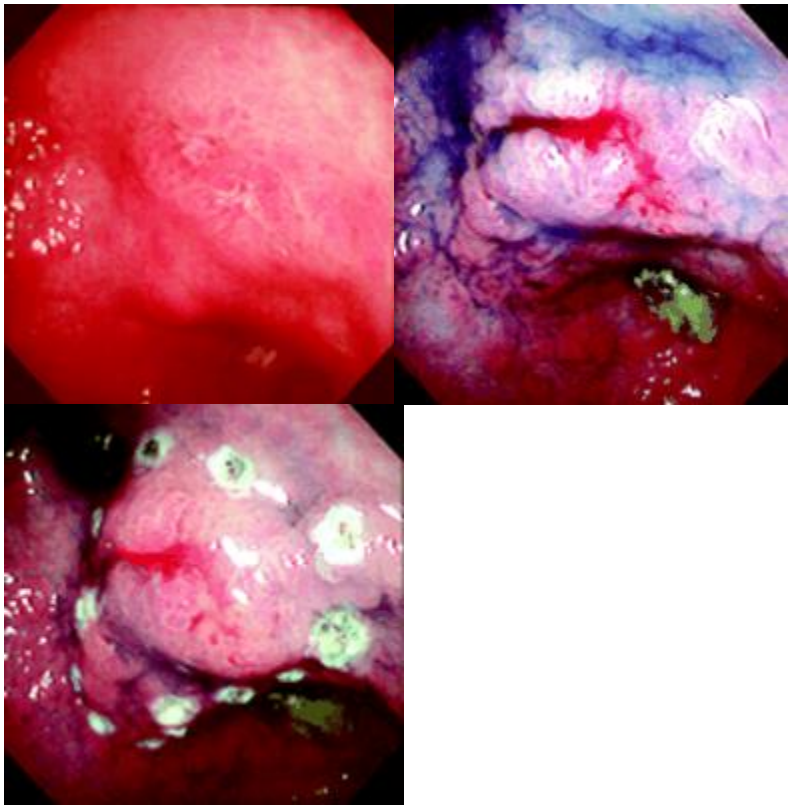
Endoscopic ultrasound views of normal stomach **(A)**, T1 N0 gastric cancer **(B)**, and T3 N1 gastric cancer **(C)**.

Gastric CA is seen hypoechoic disruption of the layers and depth of penetration can be determined in relation to this:

1. T1 tumours – disruption of first 3 layers
2. T2 tumours - 4 layers disrupted
3. T3 tumours – penetrates through the 5th layer
4. T4 lesion – invades adjacent structures
5. N stage – malignant nodes are larger in size with hypoechoic or mixed echogenicity with round and sharp borders
6. Limited use in determining M stage

Chromo endoscopy:

1. To target early lesions
2. To define their margins
3. Due to indigo carmine dye it is blue color



Barium Meal:

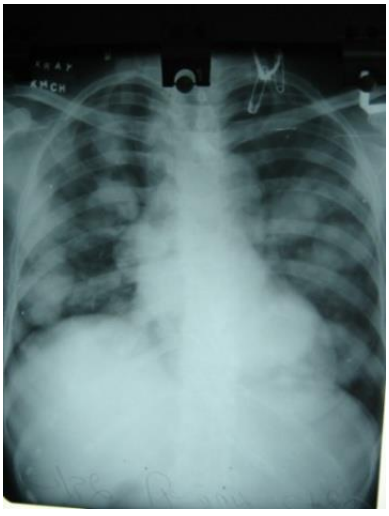
1. Endoscopy and Barium studies are interdependent.
2. In case, first investigation in a patient with sinister symptoms is negative then the other test is indicated
3. Degree of obstruction
4. Diagnosing linitis plastica that can be missed at gastroscopy

BENIGN Vs MALIGNANT ulcer:

1. Benign ulcer
    a. Extends beyond the luminal margin
    b. radiating gastric folds
2. Malignant ulcer
    a. Doesnot extend beyond luminal margin
        a.i. Folds - Parallel/ interrupted/ fused/ nodular
    b. a/w mass
    c. Ulcer with irregular filling defect

CHEST X-RAY:

Multiple metastases would stop further investigations unless there is a need for trials.



Computerized Tomography:

1. CT is the primary method for detection of intra-abdominal metastatic disease (detection rate of approximately 85%).
2. The ability to image peritoneal metastases is only 50%.
3. Used in locoregional staging but the accuracy of T and N stages as determined by CT is less accurate than EUS.
4. Detects the following:
    i. Spread beyond the mucosa
    ii. Presence of local tumour invasion
    iii. Regional lymph node metastasis
    iv. Metastasis
    v. Egc has no detectable findings on ct
5. CT finding in Advanced Gastric CA:

    Infiltrative type - focal or diffuse wall thickening, infiltrated wall shows marked contrast enhancement

    Mucinous adeno ca – low density masses with stippled or punctate calcification

    Polypoid tumours – soft tissue masses that protrude into gastric lumen

    Ulcerative type – tumour with central cavitation
6. CT staging of gastric CA:

    STAGE I - Intraluminal mass only

    STAGE II - Gastric wall thickening > 1 cm

    STAGE III – Gastric wall thickeneing with direct invasion of adjacent structures and or local adenopathy

STAGE IV - Distant metastasis present

7. Helical CT abdomen:
    a. Staging and assessment of operability
    b. Extent of mural involvement
    c. Extra gastric extension
    d. Involvement of retrogastric organs
    e. Detection of lymphnodes
8. Limitations of CT:
    a. In evaluating early gastric cancers
    b. In detecting small (< 5 mm) mets in liver and peritoneal surface
    c. Accuracy for LN – 25 – 36%

PET:

1. PET is not a primary staging modality for gastric cancer.
2. Principle of PET Scanning is based on tumor cells preferentially accumulate positron emitting 18 – F fluorodeoxy glucose.
3. Used for evaluating distant metastasis in gastric cancer and also in loco regional staging.
4. Sensitivity - 60%
5. Specificity - 100%
6. Possible role:

a. Staging

b. Assess response to treatment(NEO-ADJUVANT CHEMO)

Staging Laparoscopy and Peritoneal Wash cytology:

For patients with locally advanced gastric cancer and those who received neo adjuvant chemotheraphy, Staging Laparoscopy is a safe and effective tool,. The most frequent metastasis and recurrence in patients with gastric cancer is peritoneal carcinomatosis.

Disseminated lesions originate from free cancer cells shred from cancer invaded mucosa. Several Japanese Institutions performed Peritoneal Wash cytology to detect these cells. Robustic value of positive cytology was also confirmed recently in the West.

Why Staging Laparoscopy should be done?

Conventional Imaging techniques often understage the extent of intra abdominal spread of advanced gastric cancer which results in high rate of unnecessary exploratory laparotomy. Clinical staging may be improved by laparoscopy since this may identify the abdominal tumor deposits on peritoneal surfaces which are not detectable by contrast CT. Also peritoneal wash cytology can be taken during laparoscopy, which found positive can be considered as Gastric Malignancy.

Advantages of Staging laparoscopy:

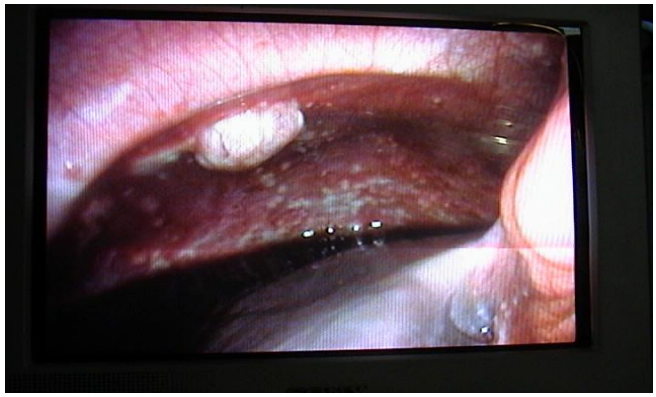
1. Staging patients for preoperative treatments.
2. Rate of detecting occult metastatic disease 13 – 37%
3. Disease missed by CT are mostly peritoneal metastases
4. Eliminate the need for Laparotomy
5. Laparoscopic USG
    a. Adjacent organ invasion

b. Liver metastases

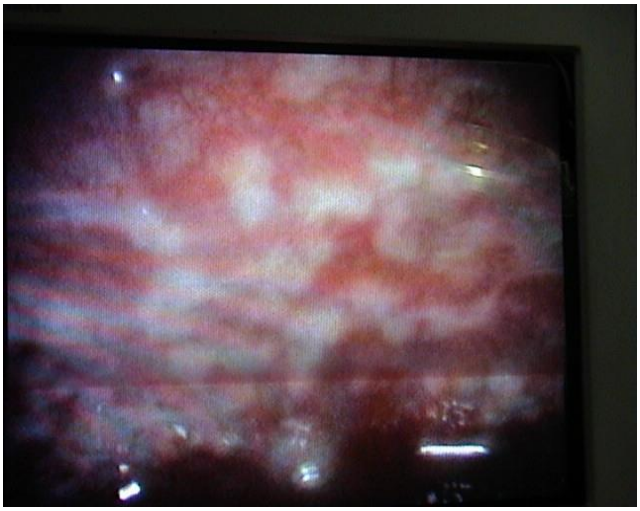
c. Guided biopsy of doubtful lesion in liver to r/o benign pathology

6. Peritoneal wash cytology can be taken during laparoscopy which if found positive can be treated with palliative intent.

Laparoscopy showing liver mets:



Laparoscopy showing peritoneal mets:



Peritoneal Wash cytology:

Positive peritoneal cytology (PC) is associated with poor prognosis. In locally advanced gastric cancer, patient should undergo staging laparoscopy and PC to select those requiring different treatment.

Peritoneum is the most frequent site of recurrence following R0 resection, possibly due to intra peritoneal presence of shed from the serosal surface of primary tumor. Majority of patients with intra peritoneal free cancer cells (IPFCC) do not escape postoperative peritoneal recurrence. Clinical studies have shown peritoneal cytology findings as an independent prognostic factor in gastric cancer. Hence peritoneal cytology should be included as a Staging process in gastric cancer as followed by Japanese research society for gastric cancer.

Peritoneal Washing Procedure:

Peritoneal wash fluid should be collected either during staging laparoscopy or laparotomy and proceed.

Immediately on opening the abdomen, 200 ml of warm saline was instilled into the serosa overlying the tumor. The fluid was then aspirated from the abdominal cavities including the pouch of Douglas. After gentle stirring, atleast 50 ml of the fluid was recovered subsequently. Fluid was then centrifuged for 15 mins at 1500 rpm. Supernatant fluid should be discarded. Sediment was made on two glass slides and stained by Papanicolau's method.

All cytological examinations were performed by experienced cytopathologists.

Cytological findings were classified as

Positive,

Negative or

Hemorrhagic

Although IFCC are detected in a considerable number of gastric patients, the probability of peritoneal recurrence far exceeds the IFCC detected. The use of real time RT – PCR (real time quantitative reverse transcriptase – polymerase chain reaction) has been reported to increase the sensibility of IFCC detection.

Methodology

METHODOLOGY

Patients admitted in various surgical units of Stanley Medical College from June 2015 to September 2016 constitute the materials of this study. Clearance was obtained from hospital ethical committee.

All patients admitted with diagnosis of carcinoma stomach by endoscopic biopsy and their staging and management were included in this study. A total of 22 patients were analysed.

A detailed history including dietary factors, life style habits and mode of presentation were elicited in all patients and thorough clinical examinations were done in them.

All patients were subjected to basic blood, urine and biochemical evaluation including liver function test and USG abdomen, CECT abdomen/pelvis, diagnostic laparoscopy. Peritoneal wash cytology was taken for patients with negative macroscopic metastasis.

Patients were categorized into various stages (by CECT, biopsy, DPL) and treatment plan was made according to stages.

Patients were operated and operative findings were noted, recorded and analyzed. Epidemiological factors relevant to age, sex distribution were noted.

Specimen was sent for HPE, histological type and grading of tumour was analysed.

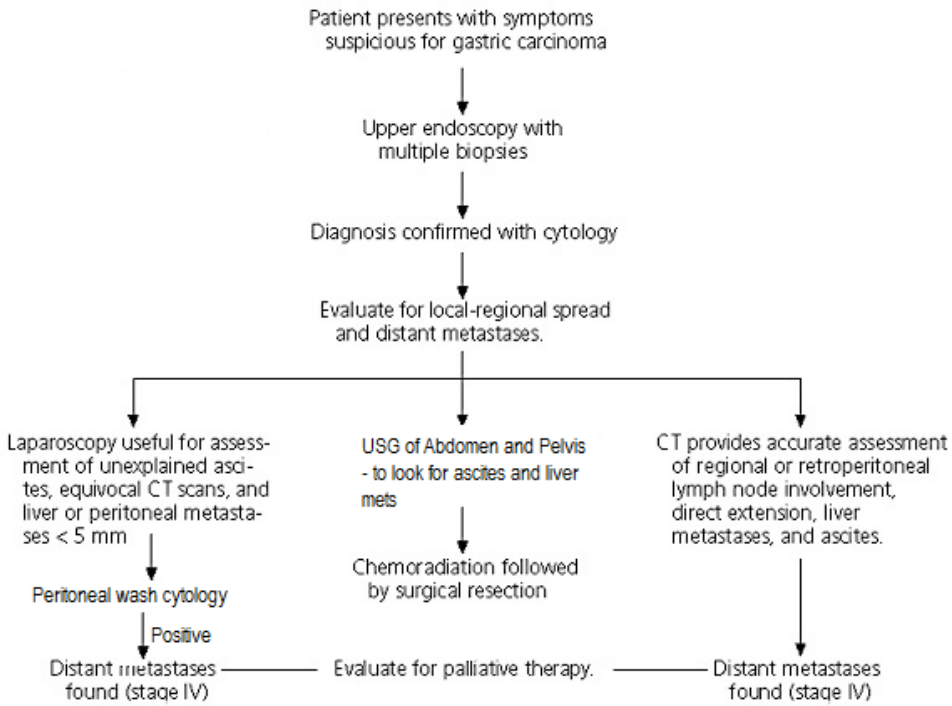
INCLUSION CRITERIA:

1. Patients with histologically proven CA stomach after upper GI endoscopy.
2. Patients with negative macroscopic metastasis will be subjected to peritoneal wash cytology.

EXCLUSION CRITERIA (for peritoneal wash cytology):

1. Patients with obvious metastatic disease will be excluded for peritoneal lavage cytology.

DIAGNOSTIC WORK UP:



PERITONEAL WASHING:

1. Peritoneal wash cytology was done either during laparoscopy/laparotomy and proceed.
2. On opening the abdomen, before manipulating the tumor, 200 ml of warm normal saline were introduced and manually stirred in the Douglas cavity, para-colic gutters and in the right and left subphrenic cavity.
3. After gentle stirring, atleast 50 ml of the fluid was recovered subsequently from several regions of the abdominal cavity.
4. The fluid was then centrifuged for 15 min at 1500 rpm.
5. The sediment was smeared onto one or more glass slides and stained using the Papanicolau's method.
6. All cytological examinations were performed by experienced cytopathologists.
7. Cytological findings were classified as positive, negative or suspicious.
8. The following cell characteristics were used to determine the presence of malignant cells:
    1. Presence of aggregate
    2. size
    3. shape
    4. type of cytoplasm
    5. cytoplasmic vacuoli, mainly nuclear abnormalities
    6. nuclear chromatin
    7. nuclearcytoplasmic ratio
    8. mitotic figures
    9. nucleolar prominence

    Staging Laparoscopy is done to look for evidence of peritoneal, liver metastasis (M+) or ascites. The T factor is assessed by looking for serosal involvement or involvement of adjacent organs; and the N factor is assessed by involvement of adjacent lymph nodes.

    If either D-Lap shows evidence of metastasis or peritoneal cytology is found to be positive, patient is treated with Palliative intent.

Results

RESULTS

Table 9: Age Distribution

Age	No of patients
<=40	5
41 to 50	4
51 to 60	7
>60	6

Graph 1: Age Distribution

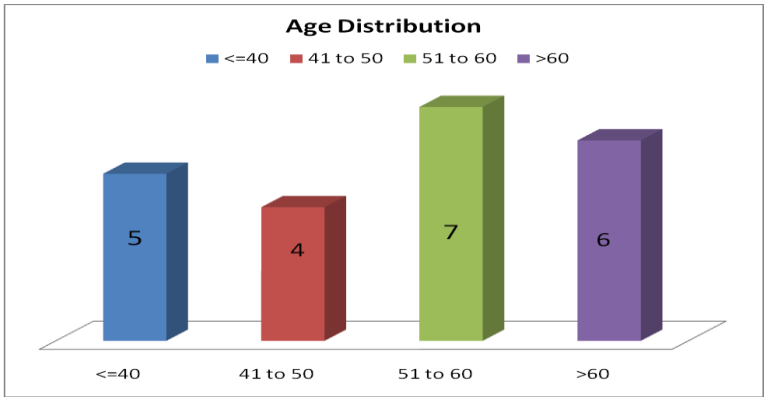


Table 2: Sex Distribution

Sex	No of Patients
Male	18
Female	4

Graph 1: Sex Distribution

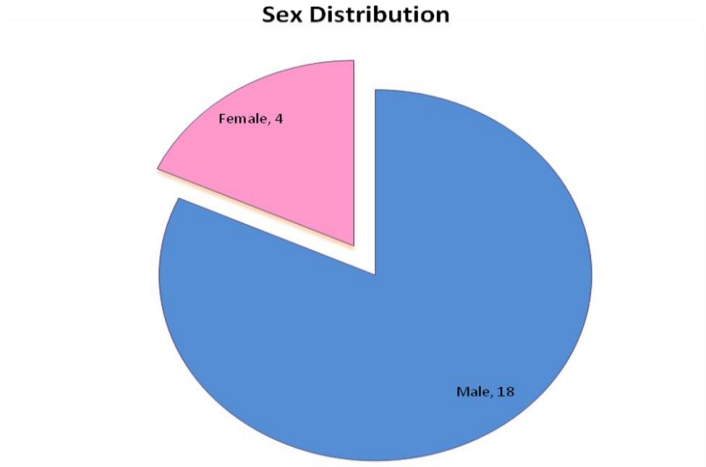
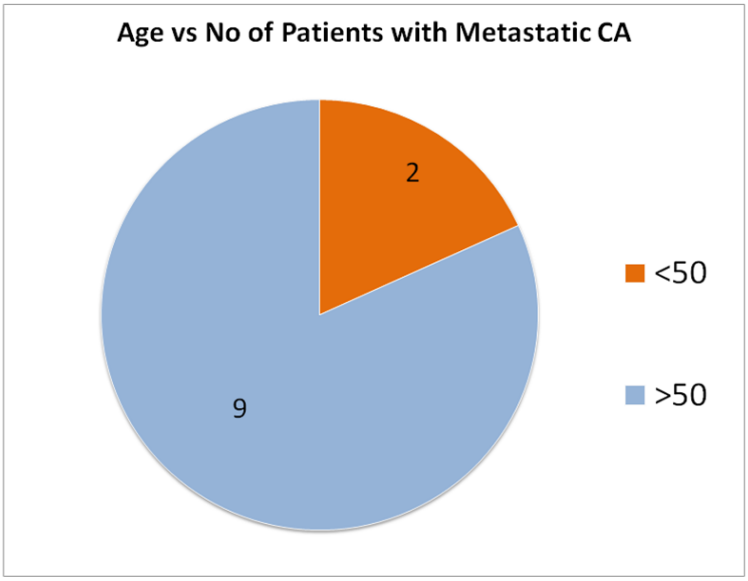


Table 10: Age vs No of Patients with Metastatic CA

Age	No of Patients with Metastatic CA
<50	2
>50	9

Graph 2: Age vs No of Patients with Metastatic CA

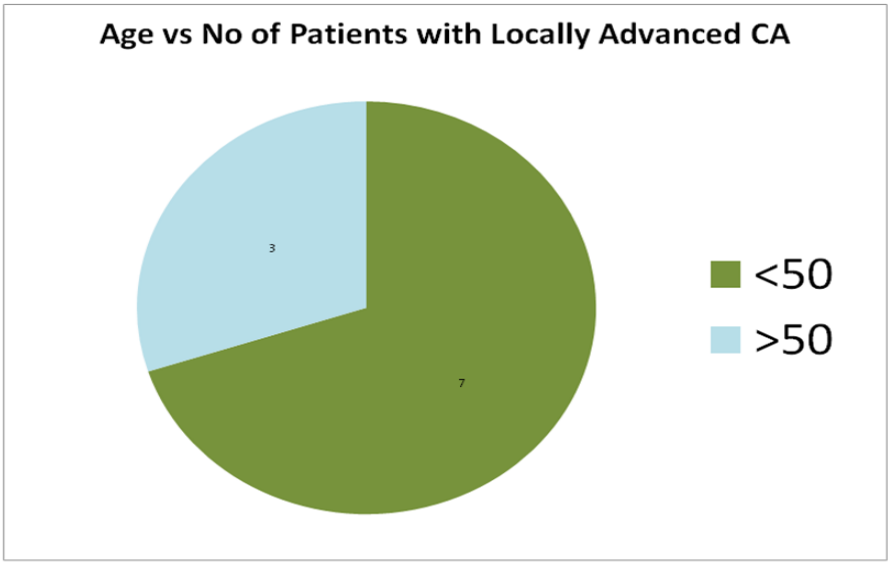


Out of 11 patients with Metastatic CA, 9 patients were greater 50 years.

Table 11: Age vs No of Patients with Locally Advanced CA

Age	No of Patients with Locally Advanced CA
<50	7
>50	3

Graph 3: Age vs No of Patients with Locally Advanced CA

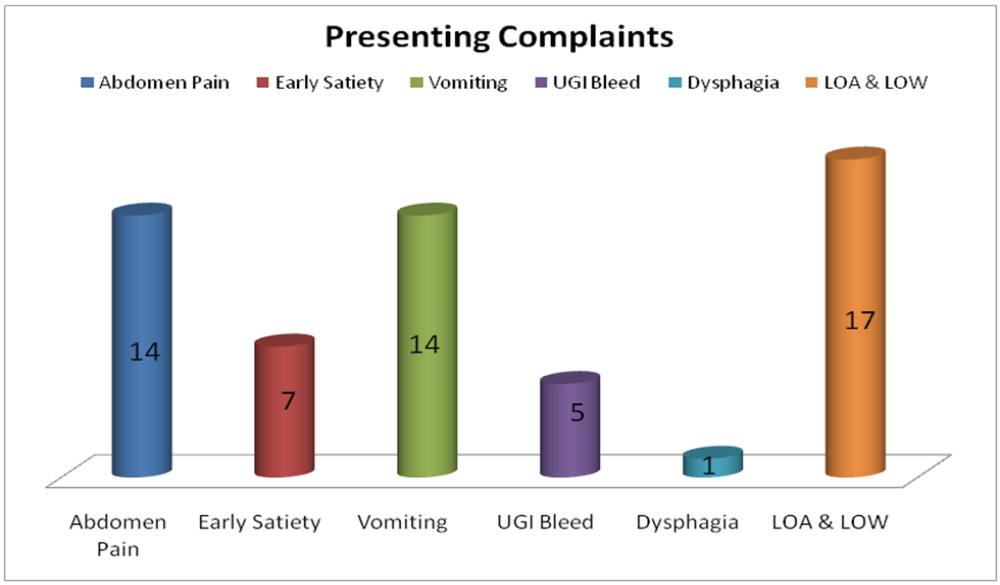


Out of 10 patients with Locally Advanced CA, 7 patients were less than 50 years.

Table 12: Presenting Complaints

Complaints	No of patients
Abdomen Pain	14
Early Satiety	7
Vomiting	14
UGI Bleed	5
Dysphagia	1
LOA & LOW	17

Graph 4: Presenting Complaints



Most common presenting complaints are Abdomen Pain, Vomiting and LOA & LOW

Table 13: Dietary Habits

Diet Habits	No of Patients
Vegetarian Diet	1
Mixed Diet	21

Graph 5: Dietary Habits

Table 13: Personal Habits

Personal Habits	No of Patients
Smoking and Alcoholic	9
Non smoker and Non alcoholic	13

Graph 5: Personal Habits

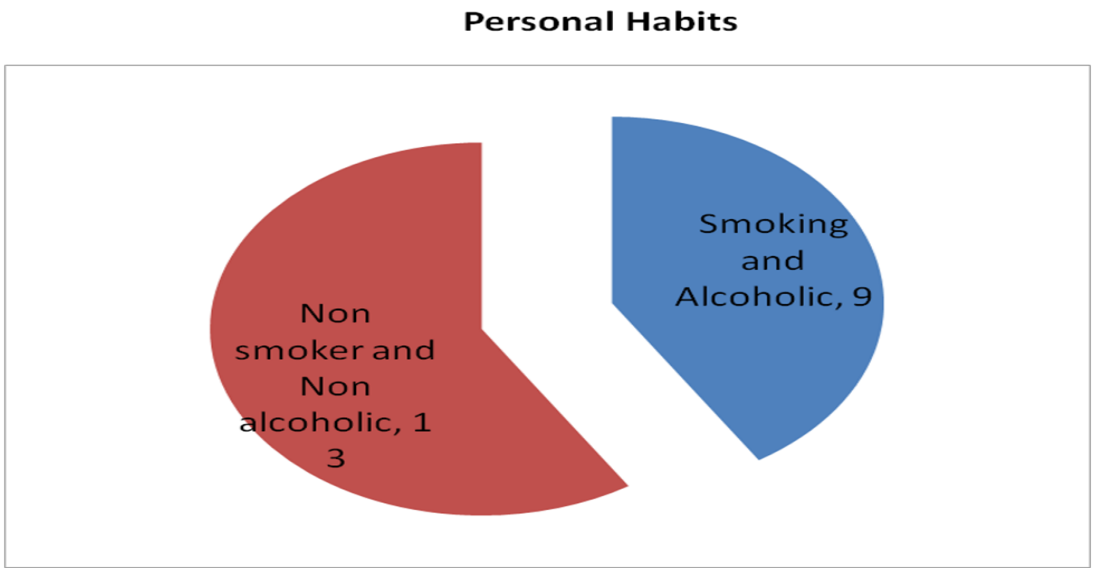
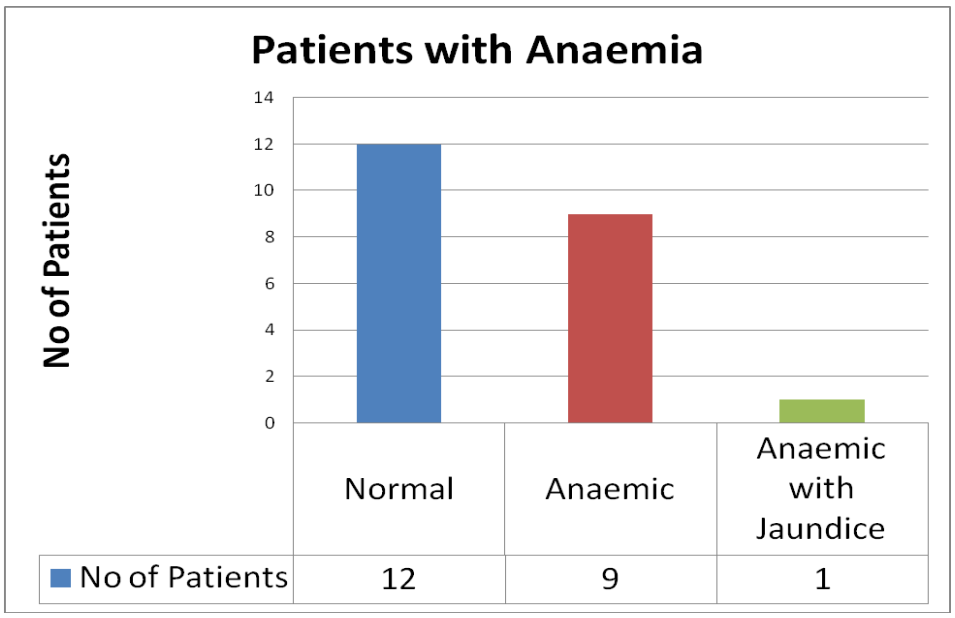


Table 14: Patients with Anaemia

General Examination	No of Patients
Normal	12
Anaemic	9
Anaemic with Jaundice	1

Graph 6: Patients with Anaemia

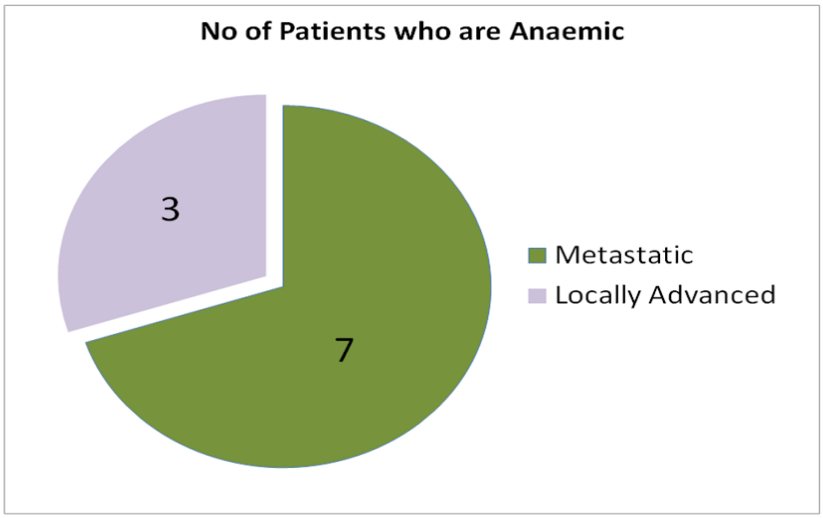


Out of 22 patients, 10 patients were anaemic.

Table 15: Stage of Disease vs Patients with Anaemia

Diagnosis	No of Patients who are Anaemic
Metastatic	7
Locally Advanced	3

Graph 7: Stage of Disease vs Patients with Anaemia



Out of 10 patients who are Anaemic, 7 patients were Metastatic and 3 patients were Locally Advanced

Table 16: Stage of Disease vs HB

Stage of Disease	<8 gms	8 to 11 gms	>11 gms
Metastatic	5	2	4
Locally Advanced	2	2	6

Graph 8: Stage of Disease vs HB

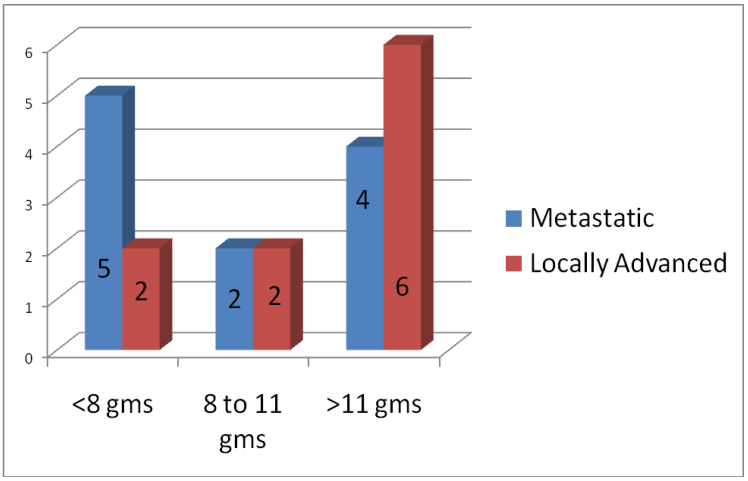


Table 17 – Clinical Findings

Local Examination	No of Patients
Epigastric Mass	7
No Significant finding	9
Ascites	2
Visible Gastric Peristalsis	4
Hepatomegaly	1

Graph 9 – Clinical Findings

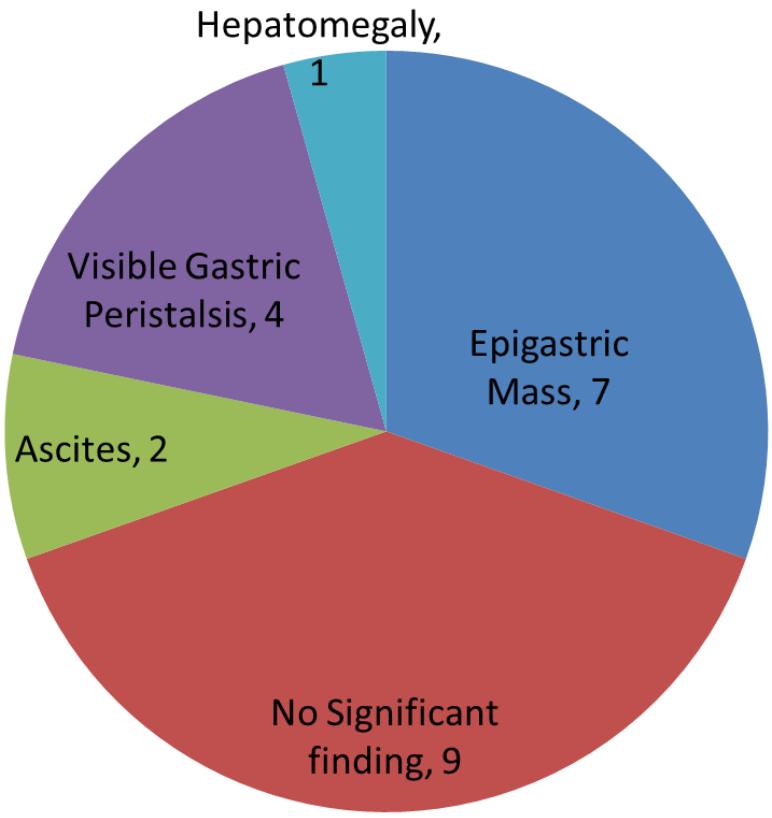


Table 18 – Mode of Presentation

Clinical Diagnosis	No of Patients
Epigastric mass for evaluation	5
Abdomen pain for evaluation	7
Gastric outlet obstruction	9
UGI bleed	1

Graph 10 – Mode of Presentation

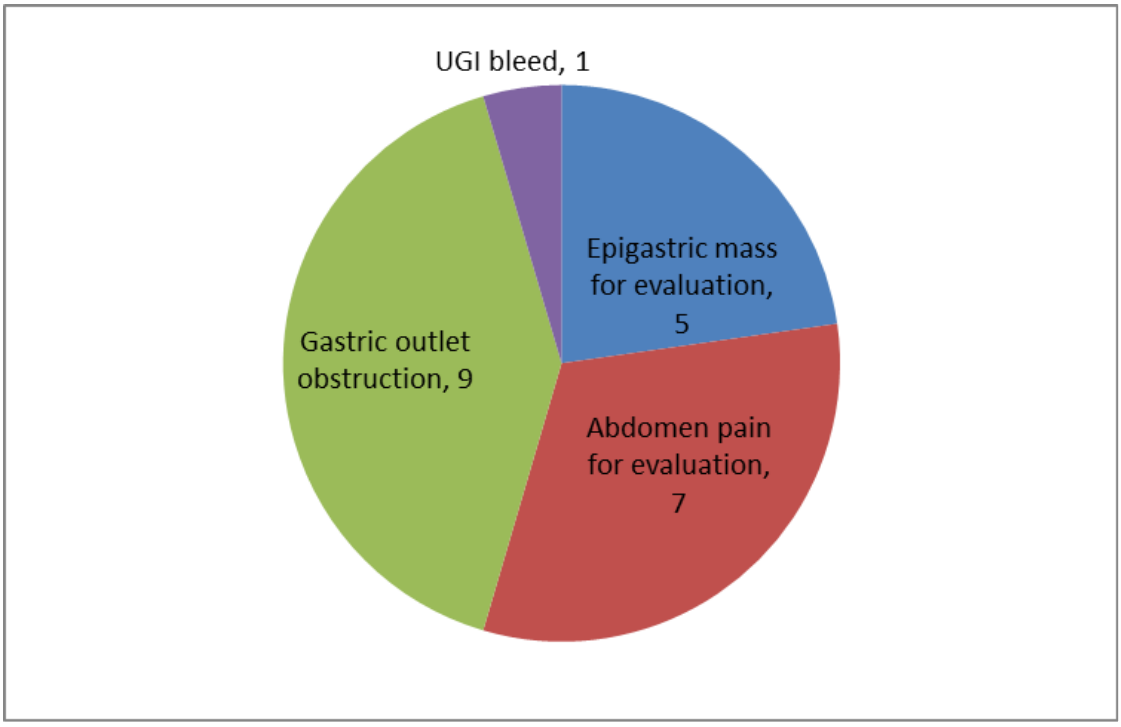


Table 19 – Blood group

Blood Group	No of Patients
A+	7
B+	5
AB+	2
O+	7
B-	1

Graph 11 – Blood group

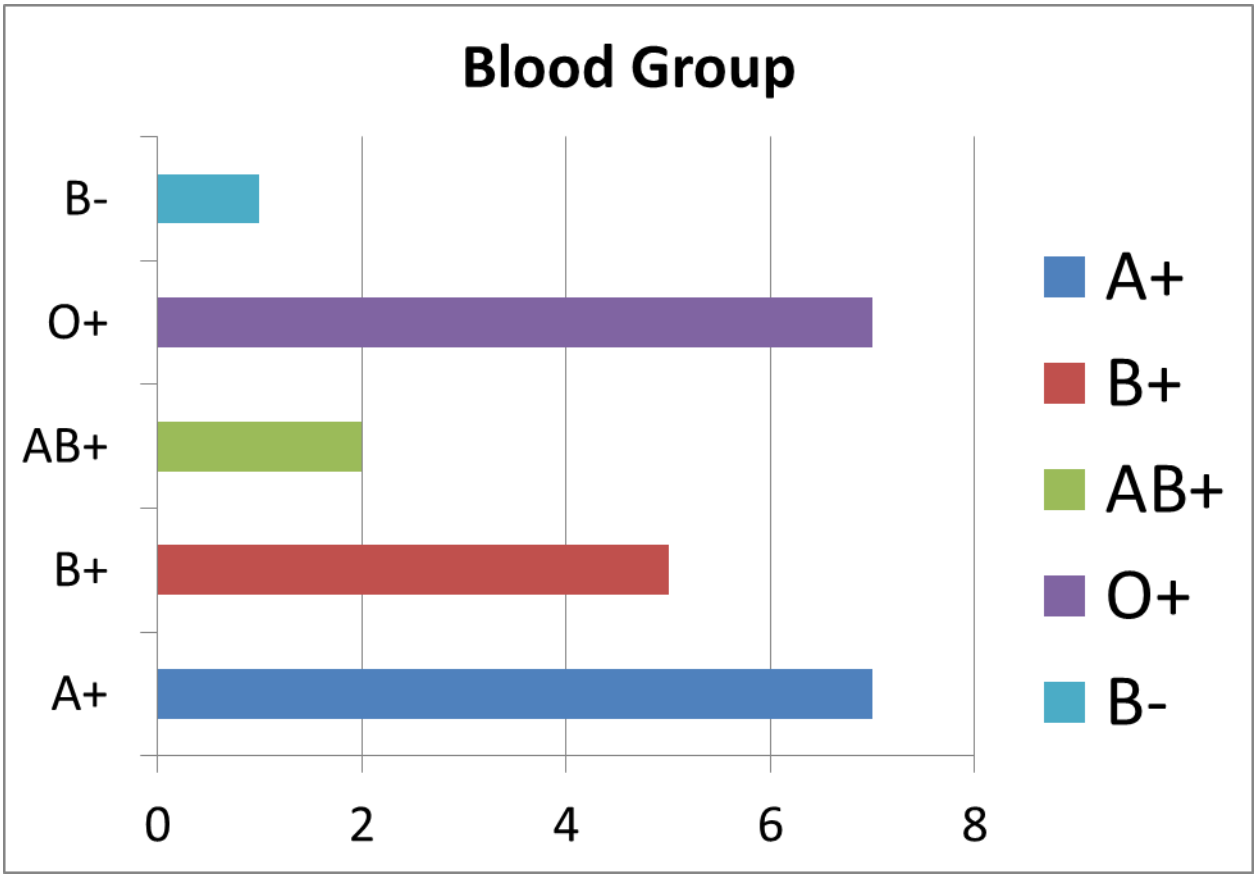


Table 20 – Upper GI Scopy

Site of growth	No of patients
Antro pylorus	16
Proximal body	1
Distal Body	4
Proximal Body & fundus	1

Graph 12 – Upper GI Scopy

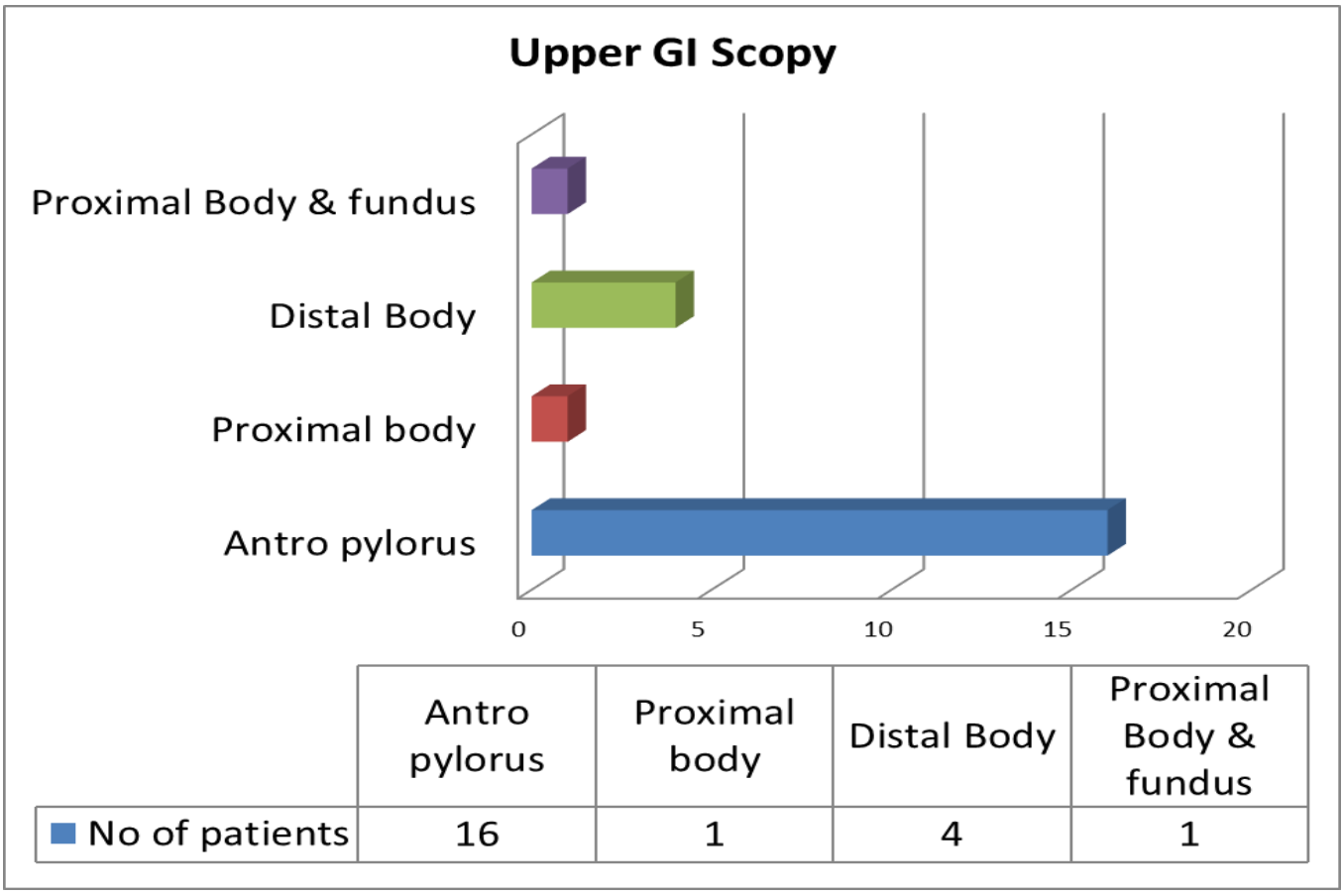


Table 21 – Histological Grade

Histological Grade	No of patients
Moderately Differentiated	11
Poorly Differentiated	9
Well differentiated	2

Graph 13 – Histological Grade

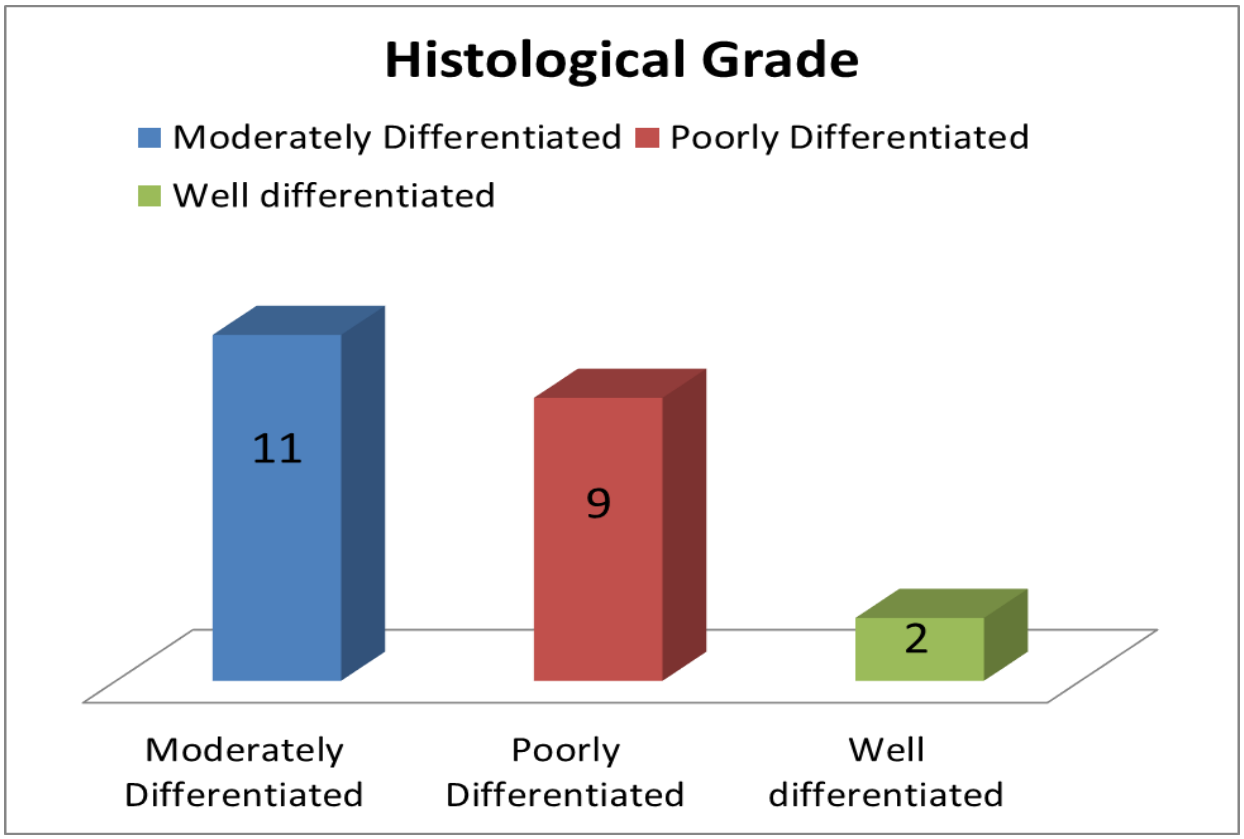


Table 22 – Histological Grade Vs Patients with Mets

Histological Grade	No of Patients with Metastatis
Moderately Differentiated	5
Poorly Differentiated	6
Well differentiated	0

Graph 14 – Histological Grade Vs Patients with Mets

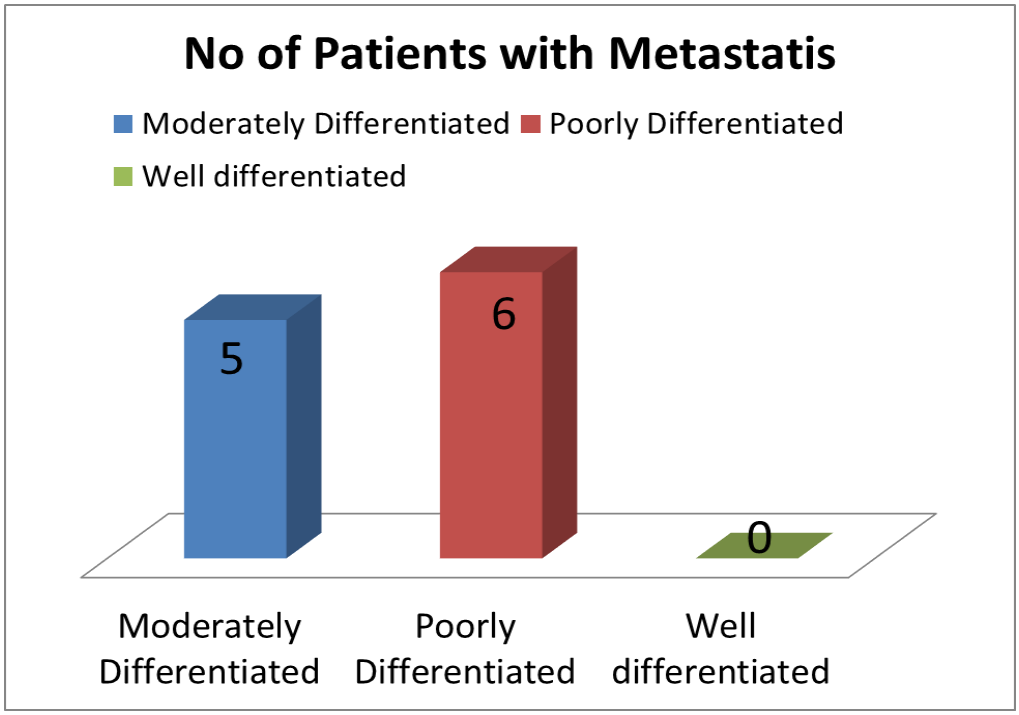


Table 23 – Presence of Mets in CECT

Presence of Mets	No of Patients
Peritoneal Mets	1
Liver Mets	7

Graph 15 – Presence of Mets in CECT

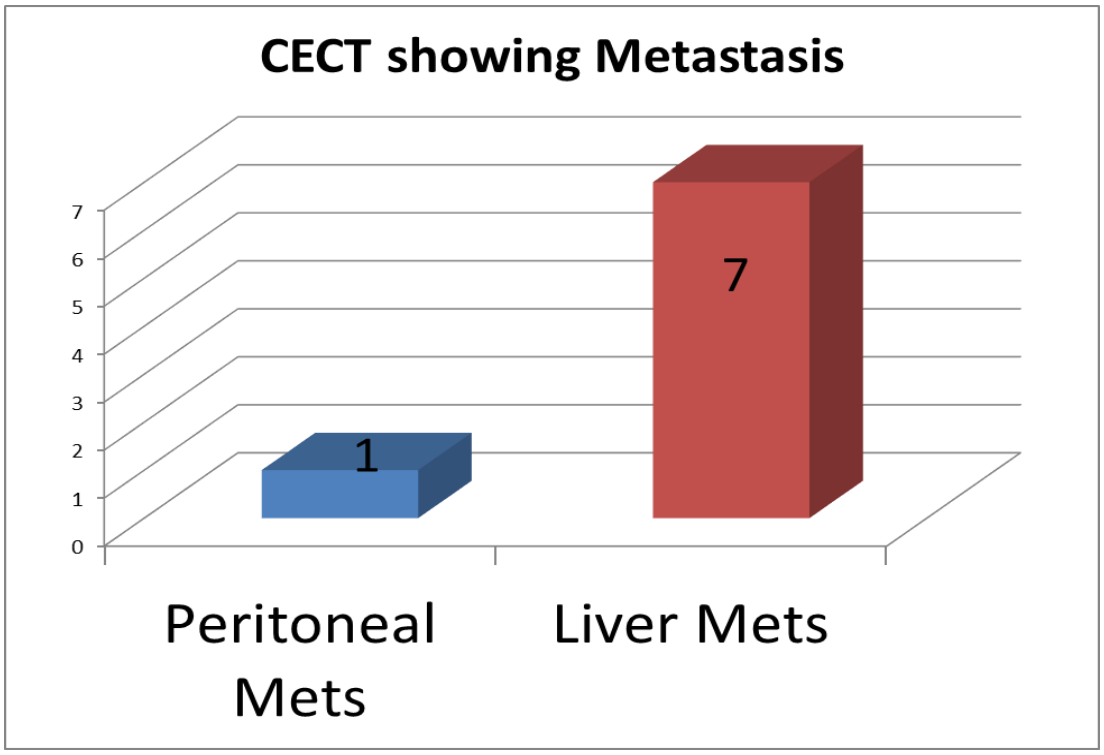


Table 24 – D-Lap/Laparotomy and Proceed findings

D-Lap findings	No of Patients
Peritoneal Mets present	2
Mets not present	12

Graph 16 – D-Lap/Laparotomy and Proceed findings

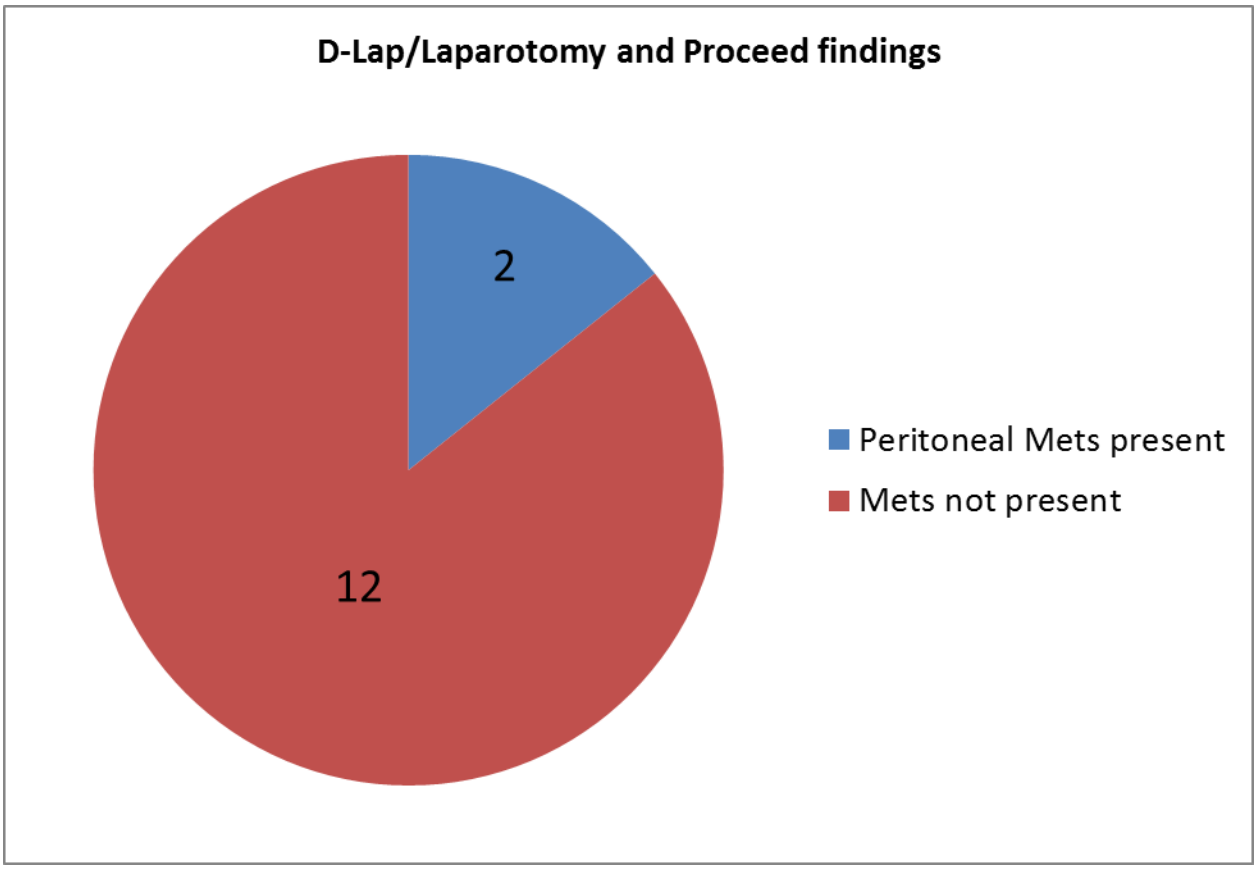


Table 25 - Wash Cytology in D-Lap negative patients

Wash Cytology in D-Lap negative patients	No of Patients
Positive for Malignancy	1
Negative for Malignancy	11

Graph 17 - Wash Cytology in D-Lap negative patients

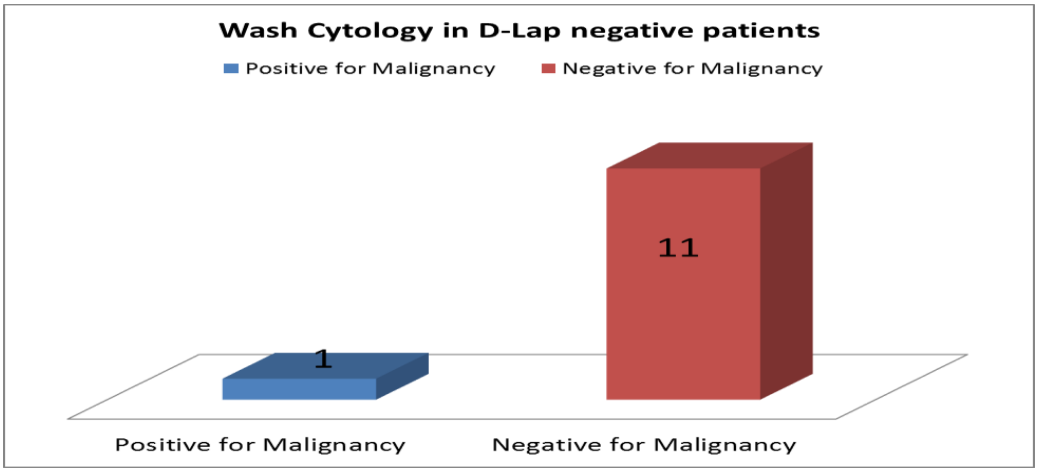


Table 26 - Peritoneal Wash cytology by D-Lap/Laparotomy

Peritoneal Wash cytology by D-Lap/Laparotomy	No of Patients
Laparotomy	10
D-Lap	3

Graph 18 - Peritoneal Wash cytology by D-Lap/Laparotomy

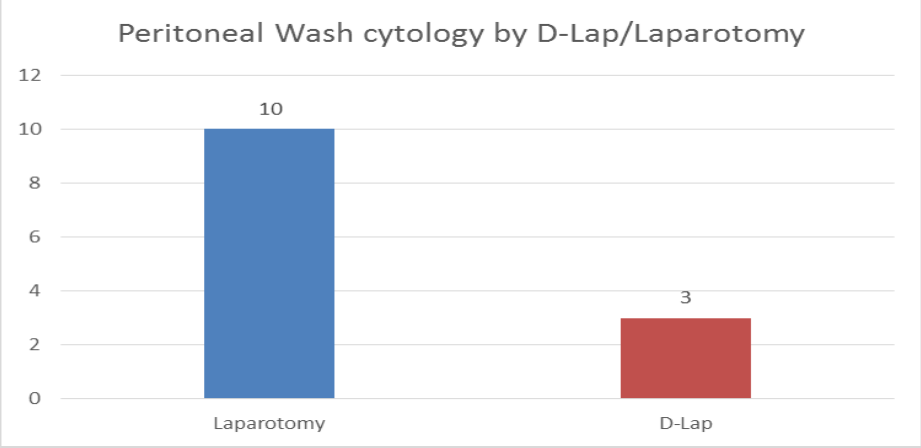


Table 27 – Final Diagnosis

Final Diagnosis	No of patients
Early	1
Locally Advanced	10

Graph 19 – Final Diagnosis

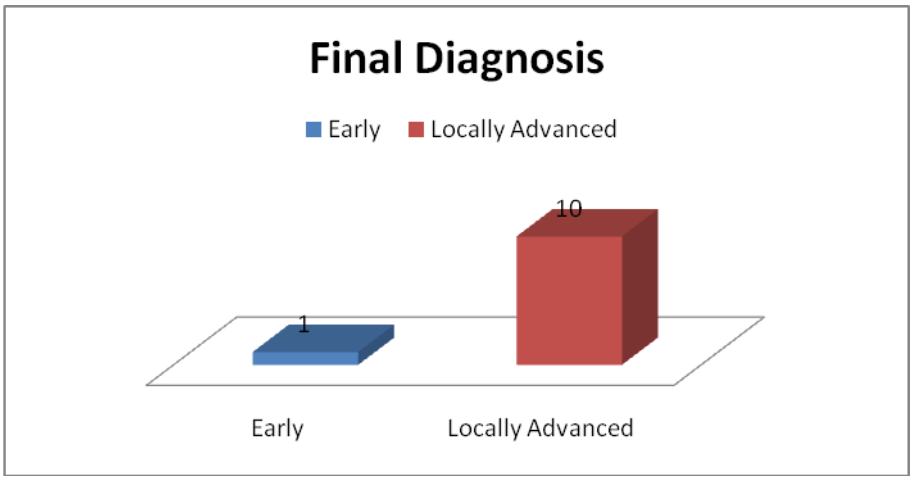


Table 27 – Diagnosis of Metastatic CA stomach

Method	Number of patients Diagnosed with Metastatic CA stomach
CECT	8
D-Lap	2
Wash cytology	1

Graph 19 – Diagnosis of Metastatic CA stomach

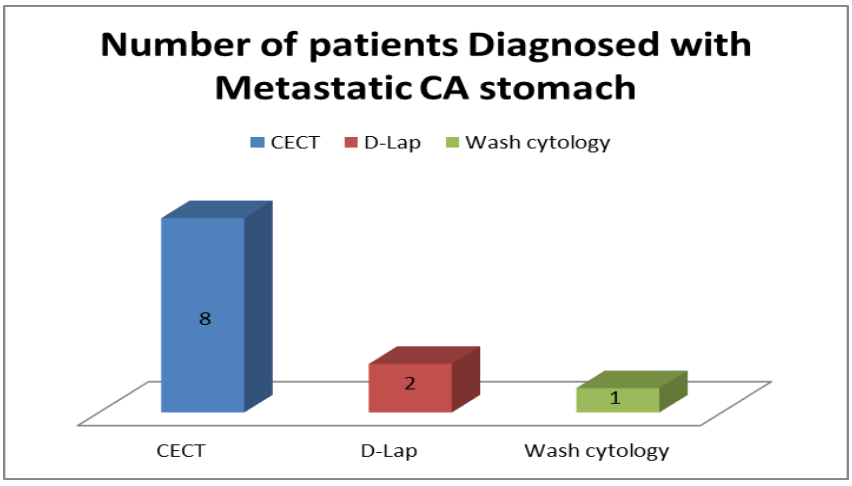
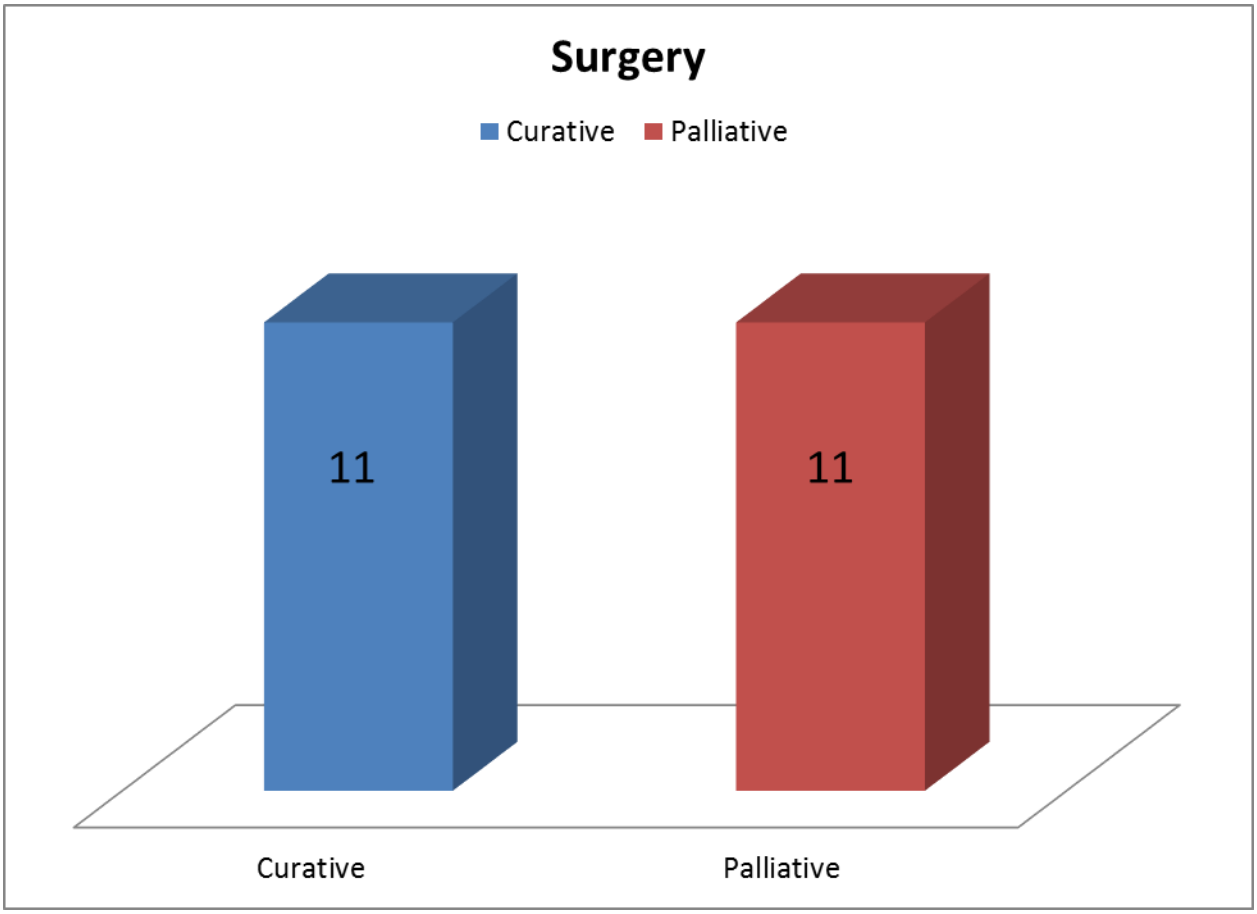


Table 28 – Surgery

Surgery	No of Patients
Curative	11
Palliative	11

Graph 20 – Surgery

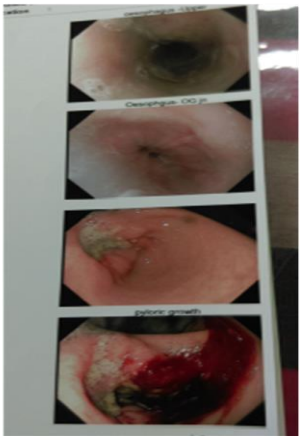


FIGURES RELATED TO MASTER CHART

UPPER GI SCOPY – taken outside
Mr. Ravichandran (Age - 46, IP Number - 1543679)

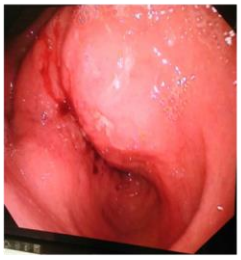
- Food stasis +
- Scope not passed beyond pylorus.

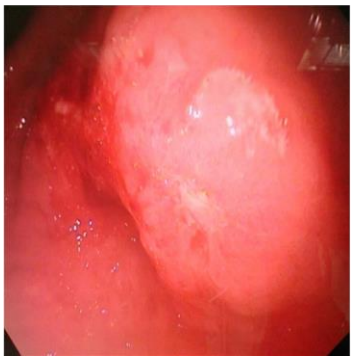
- IMPRESSION:
  - Antropyloric growth with gastric outlet obstruction.



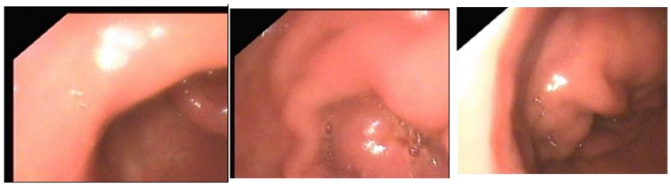
UPPER GI SCOPY – taken in Stanley Medical college and Hospital
Mr. Ramesh(Age - 37, IP Number - 1538402)

FINDINGS
- Antropyloric ulceroproliferative growth with outlet obstruction
- D1D2 not entered





UPPER GI SCOPY – taken in Stanley Medical college and Hospital
Mr.Dayalan (Age - 52, IP Number - 1607851)

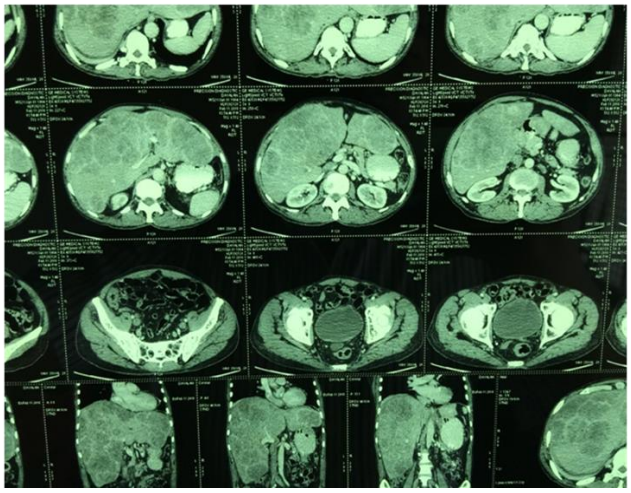


Borrmann type 2 growth in antrum extending circumferentially from anterior wall, posterior wall, greater curvature and part of lesser curvature

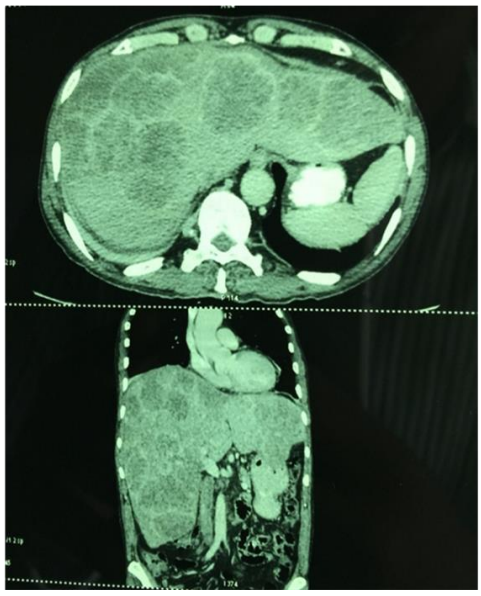
Multiple plaques in junction of middle and lower esophagus circumferentially

CECT abdomen & Pelvis showing Liver mets –

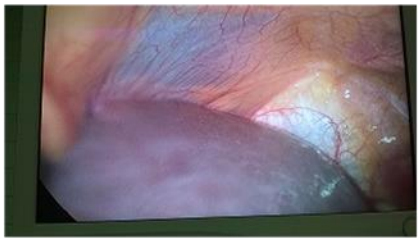
Mr.Dayalan (Age - 52, IP Number - 1607851)

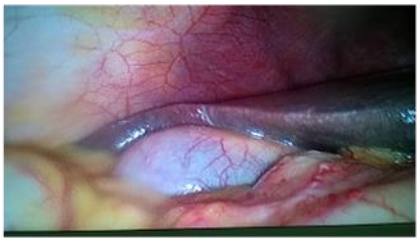


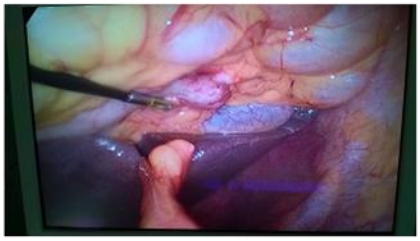
CECT abdomen & Pelvis showing Liver mets –
Mr. Govindan (Age – 77, IP Number – 1646573)

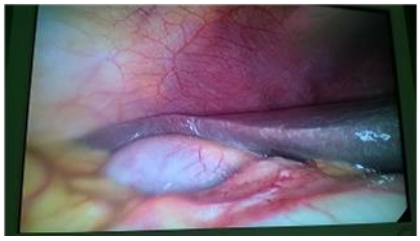


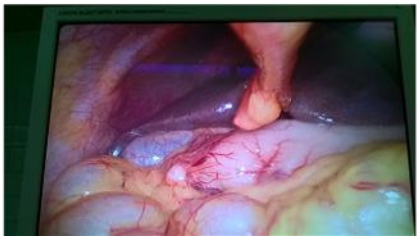
D-Lap photos of Mr. Ravichandran (Age - 46, IP Number - 1543679)



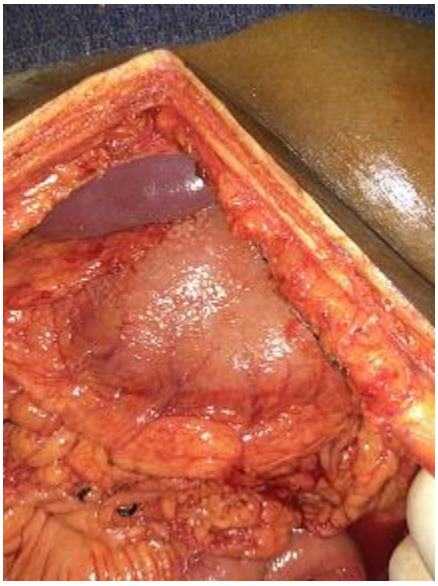




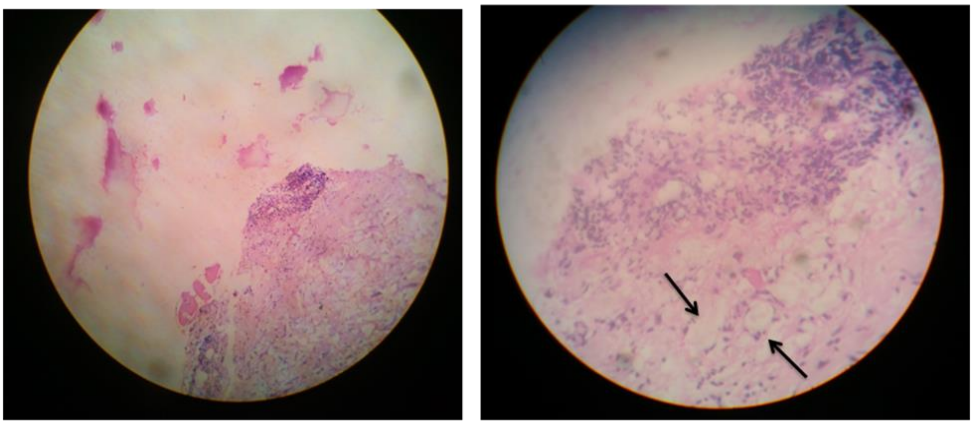




Laparotomy showing peritoneal mets–
Mrs. Aasai (Age – 40, IP Number – 1650877)



HPE slide of Wash cytology –
Mrs. Rasammal (Age – 70, IP Number – 1614844)



1. LOW POWER HIGH POWER

Discussion

DISCUSSION

The main purpose of staging laparoscopy in gastric cancer is to

1. Avoid unnecessary laparotomy in incurable metastatic patients.
2. Staging patients planned for pre operative treatments

Most common site of metastasis in patients with incurable metastatic disease is peritoneum. Conventional imaging techniques cannot detect peritoneal mets usually, but surgical laparoscopy is very accurate in detecting small intra abdominal metastasis. Studies have shown that 13 to 37% of patients with peritoneal deposits failed to be detected by contrast CT were picked up by staging laparoscopy.

In another study, 21% of the patients were found to have peritoneal metastasis. These patients were able to avoid unnecessary laparotomy/curative surgery.

Peritoneal dissemination can be early detected by cytological examination of the peritoneal wash cytology. Peritoneal wash fluid can predict survival and peritoneal relapse in patients with gastric cancer.

Usefulness of peritoneal cytology has been a subject of debate in literature. Survival of patients with positive cytology is worse as compared to those with negative cytology. In one study peritoneal cytology was positive in 11% of population studied (range being 4.4-11%) compared to our study, only one positive cytology patient out of 12 patients (D-Lap negative) examined, Positive cytology rate being 8.33%.

Hence a broad institutional based study should be done to analyze the significance of Peritoneal wash cytology during laparoscopy / laparotomy in unseen metastasis.

Conclusion

CONCLUSION

Peritoneal Wash cytology during Laparoscopy/Laparotomy though many studies have shown to upstage the disease process from locally Advanced to Metastatic.

Moreover, Positive cytology rate 4.4 – 11% reported in literature which reflects the heterogeneity of patient cohorts with variable disease severity, Experience of Pathology, Duration of sample retrieval to Sample Analysis & Differences in diagnostic criteria.

In my study, there was only one positive cytology patient out of 12 patients (D-Lap negative) examined. Positive cytology rate being 8.33%

Hence institutional based study should be done to establish the significance of Peritoneal Wash cytology in CA Stomach.

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3. Watt I, Stewart I, Anderson D, Bell G, Anderson JR (1989) Laparoscopy, ultrasound and computed tomography in cancer of the oesophagus and gastric cardia: a prospective comparison for detecting intra-abdominal metastases. Br J Surg 76: 1036-1039.
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5. Kinkel K, Lu Y, Both M, Warren RS, Thoeni RF (2002) Detection of hepatic metastases from cancers of the gastrointestinal tract by using noninvasive imaging methods (US, CT, MR imaging, PET): a meta-analysis. Radiology 224: 748-756.
6. Ang CW, Tan LC (2013) Peritoneal cytology in the staging process of gastric cancer: Do or Don't? J Gastroint Dig Syst 3: 156. Doi: 10.4172/2161-069x.1000156
7. Boku T, Nakane Y, Minoura T, Takada H, Yamamura M, et al. (1990) Prognostic significance of serosal invasion and free intraperitoneal cancer cells in gastric cancer. Br J Surg 77: 436-439.
8. Suzuki T, Ochiai T, Hayashi H, Hori S, Shimada H, et al. (1999) Peritoneal lavage cytology findings as prognostic factor for gastric cancer. Semin Surg Oncol 17: 103-107.

9. Role of staging laparoscopy with peritoneal lavage cytology in the treatment of locally advanced gastric cancer by Satoru Nakagawa, Atsushi Nashimoto and Hiroshi Yabusaki.SATORU NAKAGAVA, ATSUSHI NASHIMOTO,AND HIROSHI YABUSAKI The division of surgery,Niigata cancer center hospital,2-15-3 Kawagis hicho ,niigato 951-8566,japan
10. Burke EC ,Karpeh MS ,Conlon KC,Brennan MF,LAPAROSCOPY IN THE MANAGEMENT OF GASTRIC ADENO CARCINOMA
11. Lowy am, monsfield PF ,leach sd, Laparoscopic staging of gastric cancer. surgery 1996 ;119;611-4

Annexures

Patient Information Module & Informed Consent

PATIENT INFORMATION MODULE

You are being invited to be a subject in this study.

Before you participate in this study, I am giving you the following details about this trial, which includes the aims, methodology, intervention, possible side effects, if any and outcomes:

All Patients with proven adenocarcinoma of the stomach after diagnostic work-up will be included in this study. A detailed clinical history will be taken following a standardized proforma. A detailed clinical examination will be made and relevant basic investigations will be done at the time of admission. The results arising from this study will be analyzed and used for academic purposes. You will be given clear instructions at every step and you are free to ask/ clarify any doubts. Your identity will remain confidential. You are free to withdraw from this trial at any point of time, without any prior notice &/ or without any medical or legal implications.

I request you to volunteer for this study.

Thanking You,

Investigator's Sign

(Dr.G.RANGARAJAN)

Patient's Sign

(Name:)

INFORMED CONSENT

Name: Age/ Sex: IP:

I herewith declare that I have been explained in a language fully understood by me regarding the purpose of this study, methodology, proposed intervention, possible complications, if any.

I have been given an opportunity to discuss my doubts and I have received the appropriate explanation.

I understand that my participation in this study is completely voluntary and that I am free to withdraw from this study at anytime without any prior notice &/ or without having my medical or legal rights affected.

I permit the author and the research team full access to all my records at any point, even if I have withdrawn from the study. However my identity will not be revealed to any third party or publication.

I herewith permit the author and the research team to use the results and conclusions arising from this study for any academic purpose, including but not limited to dissertation/ thesis or publication or presentation in any level.

Therefore, in my full conscience, I give consent to be included in the study and to undergo any investigation or any intervention therein.

Patient's Sign

Investigator's Sign

(Dr.G.RANGARAJAN)

அரசு ஸ்டான்லி மருத்துவக் கல்லூரி, சென்னை - 600 001.

<u>பங்கு பெறுபவரின் ஒப்பம்</u>

ஆராய்ச்சியின் தலைப்பு	:	இரைப்பை புற்றுநோய் மற்றும் உள்ளுறை சுத்தம் உயிரணுவியல் முக்கியத்துவ ம் பற்றிய ஆய்வு
ஆராய்ச்சி நடைபெறும் இடம்	:	அரசு ஸ்டான்லி மருத்துவக் கல்லூரி, சென்னை - 1.
பங்கு பெறுபவரின் பெயரும் முகவரியும்	:	

நான், ... இந்த ஆராய்ச்சியின் விவரங்களை எனது சொந்த மொழியில் கூற அறிந்து கொண்டேன்.

இந்த ஆராய்ச்சியின் முழுவிவரங்களையும் நான் அறிந்து கொண்டேன். இந்த ஆராய்ச்சியில் நான் பங்குபெறும் போது எனக்கு ஏற்படும் நன்மை தீமைகளை முழுவதுமாக அறிந்து கொண்டேன்.

இந்த ஆராய்ச்சியின் போது எப்போது வேண்டுமானாலும் நான் விலகிக்கொள்ளலாம் என்பதயும், அதனால் எனக்கு கிடைக்கும் மருத்துவத்தில் எந்தவித மாற்றமோ பாதிப்போ இருக்காது என்றும் அறிவேன். இந்த ஆராய்ச்சியில் நான் பங்குபெறுவதற்காக நான் எந்தவித சன்மானமும் (பணமாகவோ, பொருளாகவோ) வாங்கமாட்டேன். இந்த ஆராய்ச்சியின் முடிவுகளை, என் அடையாளங்களை குறிப்பிடாமல் மருத்துவ இதழ்களில் வெளியிட எனக்கு எந்த ஆட்சேபனையும் இல்லை. இந்த ஆரய்ச்சியில் என் பங்கு என்ன என்பதை அறிவேன். இந்த ஆராய்ச்சிக்கு எனது முழுஒத்துழைப்பையும் தருவேன் என்று உறுதி அளிக்கிறேன்.

பங்கு பெறுபவரின் பெயரும் முகவரியும்:

பங்கு பெறுபவரின் கையொப்பம் / விரல்ரேகை :

தேதி:

ஆராய்ச்சி செய்பவரின் பெயரும் கையொப்பமும் :

Dr. கோ. ரங்கராஜன்

Proforma

PROFORMA

Investigator: **Dr.G.RANGARAJAN**, PG 2nd year – MS (General Surgery)

Guide: **Prof. Dr.LALITHKUMAR,** Chief, Unit S7

- NAME : SL. NO:
- AGE /SEX: IP NO:
- ADDRESS WITH CONTACT NUMBER:
- DATE OF ADMISSION: DATE OF DISCHARGE:

HISTORY OF PRESENTING ILLNESS:

PAST HISTORY: **PERSONAL HISTORY:**

GENERAL EXAMINATION:

PR: BP:

LOCAL EXAMINATION OF ABDOMEN:

SYSTEMIC EXAMINATION:

Respiratory System:

Cardio Vascular System:

Central Nervous System:

CLINICAL DIAGNOSIS:

INVESTIGATIONS:

Hematological	**Biochemical**
Hemoglobin:	BloodSugar:
TLC:	SerumElectrolytes:
DLC:	LFT: T. Bilirubin: DBR: SGOT: SGPT:
ALP:	
Platelet counts:	T. Protein: Serum Albumin:
Hematocrit:	RFT:
Coagulation Profile:	**Blood Group:**
ECG:	**Chest X ray:**

USG ABDOMEN & PELVIS:

Esophago gastro duodenoscopy:

CECT Abdomen/Pelvis:

Diagnostic Lap and peritoneal wash cytology:

FINAL DIAGNOSIS:

SURGERY: PALLIATIVE OR CURATIVE

TYPE OF SURGERY:

Master Chart

S. No	Name	Date of Admission	Age	Sex	IP Number	Presenting complaints					
						Abdomen Pain	Early Satiety	Vomiting	UGI Bleed	Dysphagia	LOA & LOW
1	Mr. Murugadoss, 44	16/8/2016	41-50	Male	1653964	Yes	No	No	No	No	Yes
2	Mr.krishnan, 70	16/2/2016	>60	Male	1608817	No	Yes	Yes	No	No	Yes
3	Mr.Dayalan, 52	10/2/2016	51-60	Male	1607851	Yes	Yes	Yes	Yes	No	Yes
4	Mr .Sathya, 55	1/9/2016	51-60	Male	1645846	No	No	Yes	Yes	No	Yes
5	Mrs. Parveen Banu, 37	6/8/2016	<=40	Female	1649569	Yes	No	No	Yes	No	Yes
6	Mr.Malik basha, 57	18/7/2016	51-60	Male	1644615	No	Yes	Yes	Yes	No	Yes
7	Mrs.Janaki, 52	11/7/2016	51-60	Female	1644925	Yes	No	No	Yes	No	Yes
8	Mr.Radhakrishnan, 60	12/7/2016	51-60	Male	1643172	Yes	No	No	No	No	No
9	Mr. Govindan, 77	15/6/2016	>60	Male	1646573	Yes	No	Yes	No	No	Yes
10	Mr. Ravichandran, 46	12/8/2015	41-50	Male	1543679	Yes	No	Yes	No	No	No
11	Mr. Saravanan, 53	12/7/2016	51-60	Male	1643020	Yes	Yes	No	No	No	Yes
12	Mr. Ramesh, 37	10/7/2015	<=40	Male	1538402	Yes	No	Yes	No	No	Yes
13	Mr. Srinivasan, 75	11/3/2016	>60	Male	1615509	Yes	Yes	No	No	No	No
14	Mrs. Rasammal, 70	8/3/2016	>60	Female	1614844	No	Yes	No	No	Yes	Yes
15	Mr. Jayavel, 38	11/8/2016	<=40	Male	1647200	No	No	Yes	No	No	No
16	Mr. Aasai, 40	20/8/2016	<=40	Male	1650877	Yes	No	Yes	No	No	Yes
17	Mr. Dhanasekar, 65	10/8/2016	>60	Male	1649139	No	No	Yes	No	No	Yes
18	Mrs. Gurulakshmi, 43	5/9/2016	41-50	Female	1655169	Yes	Yes	No	No	No	No
19	Mr. Madhayan, 55	20/9/2015	51-60	Male	1641377	No	No	Yes	No	No	Yes
20	Mr. Jayam, 38	14/8/2015	<=40	Male	1548200	Yes	No	Yes	No	No	Yes
21	Mr. Nagappan, 45	23/6/2015	41-50	Male	1511828	Yes	No	Yes	No	No	Yes
22	Mr. Kaliyaperumal, 65	5/4/2016	>60	Male	1670900	No	No	Yes	No	No	Yes

S. No	Name	Date of Admission	Age	Sex	IP Number	Presenting complaints						Personal History	General Examination	Local Examination	Clinical Diagnosis
						Abdomen Pain	Early Satiety	Vomiting	UGI Bleed	Dysphagia	LOA & LOW				
1	Mr. Murugadoss, 44	16/8/2016	41-50	Male	1653964	Yes	No	No	No	No	Yes	Mixed Diet with Smoke & Alcoholic	Normal	No Significant finding	Abdomen pain for evaluation
2	Mr.krishnan, 70	16/2/2016	>60	Male	1608817	No	Yes	Yes	No	No	Yes	Mixed Diet	Normal	Epigastric Mass	Gastric outlet obstruction
3	Mr.Dayalan, 52	10/2/2016	51-60	Male	1607851	Yes	Yes	Yes	Yes	No	Yes	Mixed Diet with Smoke & Alcoholic	Anemia with Jaundice	Ascites	Gastric outlet obstruction
4	Mr .Sathya, 55	1/9/2016	51-60	Male	1645846	No	No	Yes	Yes	No	Yes	Mixed Diet with Smoke & Alcoholic	Anaemic	Visible Gastric Peristalsis	Gastric outlet obstruction
5	Mrs. Parveen Banu, 37	6/8/2016	<=40	Female	1649569	Yes	No	No	Yes	No	Yes	Mixed Diet	Anaemic	Epigastric Mass	Epigastric mass for evaluation
6	Mr.Malik basha, 57	18/7/2016	51-60	Male	1644615	No	Yes	Yes	Yes	No	Yes	Mixed Diet	Anaemic	No Significant finding	UGI bleed
7	Mrs.Janaki, 52	11/7/2016	51-60	Female	1644925	Yes	No	No	Yes	No	Yes	Mixed diet but non alcoholic non smoker	Normal	Epigastric Mass	Epigastric mass for evaluation
8	Mr.Radhakrishnan, 60	12/7/2016	51-60	Male	1643172	Yes	No	No	No	No	No	Mixed diet but non alcoholic non smoker	Normal	No Significant finding	Abdomen pain for evaluation
9	Mr. Govindan, 77	15/6/2016	>60	Male	1646573	Yes	No	Yes	No	No	Yes	Mixed diet but non alcoholic non smoker	Anaemic	Hepatomegaly	Abdomen pain for evaluation
10	Mr. Ravichandran, 46	12/8/2015	41-50	Male	1543679	Yes	No	Yes	No	No	No	Vegetarian and non alcoholic & Smoking	Normal	No Significant finding	Gastric outlet obstruction
11	Mr. Saravanan, 53	12/7/2016	51-60	Male	1643020	Yes	Yes	No	No	No	Yes	Mixed Diet with Smoke & Alcoholic	Normal	No Significant finding	Abdomen pain for evaluation
12	Mr. Ramesh, 37	10/7/2015	<=40	Male	1538402	Yes	No	Yes	No	No	Yes	Mixed Diet with Smoke & Alcoholic	Anaemic	No Significant finding	Gastric outlet obstruction
13	Mr. Srinivasan, 75	11/3/2016	>60	Male	1615509	Yes	Yes	No	No	No	No	Mixed Diet	Normal	No Significant finding	Abdomen pain for evaluation
14	Mrs. Rasammal, 70	8/3/2016	>60	Female	1614844	No	Yes	No	No	Yes	Yes	Mixed Diet	Anaemic	Epigastric Mass	Epigastric mass for evaluation
15	Mr. Jayavel, 38	11/8/2016	<=40	Male	1647200	No	No	Yes	No	No	No	Mixed diet but non alcoholic non smoker	Normal	Epigastric Mass	Epigastric mass for evaluation
16	Mr. Aasai, 40	20/8/2016	<=40	Male	1650877	Yes	No	Yes	No	No	Yes	Mixed Diet with Smoke & Alcoholic	Normal	Visible Gastric Peristalsis	Gastric outlet obstruction
17	Mr. Dhanasekar, 65	10/8/2016	>60	Male	1649139	No	No	Yes	No	No	Yes	Mixed Diet with Smoke & Alcoholic	Anaemic	Ascites	Gastric outlet obstruction
18	Mrs. Gurulakshmi, 43	5/9/2016	41-50	Female	1655169	Yes	Yes	No	No	No	No	Mixed Diet	Normal	No Significant finding	Abdomen pain for evaluation
19	Mr. Madhayan, 55	20/9/2015	51-60	Male	1641377	No	No	Yes	No	No	Yes	Mixed diet but non alcoholic non smoker	Normal	Visible Gastric Peristalsis	Gastric outlet obstruction
20	Mr. Jayam, 38	14/8/2015	<=40	Male	1548200	Yes	No	Yes	No	No	Yes	Mixed Diet with Smoke & Alcoholic	Normal	No Significant finding	Abdomen pain for evaluation
21	Mr. Nagappan, 45	23/6/2015	41-50	Male	1511828	Yes	No	Yes	No	No	Yes	Mixed Diet with Smoke & Alcoholic	Anaemic	Visible Gastric Peristalsis	Gastric outlet obstruction
22	Mr. Kaliyaperumal, 65	5/4/2016	>60	Male	1670900	No	No	Yes	No	No	Yes	Mixed Diet	Anaemic	Epigastric Mass	Epigastric mass for evaluation

CECT - Abdomen & Pelvis						Diagnostic Lap							T N M Staging			
Site of growth	Perigastric fat stranding	Presence of Nodes	Presence of free fluid	Peritoneal mets	Presence of Liver mets	Done/Not Done	Presence of metastasis	Peritoneal Wash cytology by D-Lap/Laparotomy	Peritoneal Wash cytology	Final Diagnosis (Adeno Carcinoma of Stomach)	Surgery	Dissection	T	N	M	Staging
Antro Pylorus	Yes	Yes	No	No	No	Done	No	Laparotomy	Negative for Malignancy	Locally Advanced	Curative	D2	2	3	0	III
Antro Pylorus	Yes	Yes	No	No	No	Done	No	Laparotomy	Negative for Malignancy	Locally Advanced	Curative	D2	4	2	0	III
Antro Pylorus	Yes	Yes	Yes	No	Yes	Not Done	Not applicable	Not applicable	Not applicable	Metastatic	Palliative	Not Applicable	4	1	1	IV
Antro Pylorus	No	No	No	No	No	Done	No	Laparotomy	Negative for Malignancy	Locally Advanced	Curative	D2	3	1	0	III
Antro Pylorus	Yes	Yes	No	No	No	Done	No	Laparotomy	Negative for Malignancy	Locally Advanced	Curative	D1	3	2	0	III
Antro Pylorus	Yes	Yes	Yes	No	Yes	Not Done	Not applicable	Not applicable	Not applicable	Metastatic	Palliative	Not Applicable	3	2	1	IV
Proximal Body	Yes	Yes	Yes	Yes	No	Not Done	Not applicable	Not applicable	Not applicable	Metastatic	Palliative	Not Applicable	3	2	1	IV
Antro Pylorus	Yes	No	No	No	No	Done	No	Laparotomy	Negative for Malignancy	Locally Advanced	Curative	D2	3	1	0	III
Distal Body	Yes	Yes	No	No	Yes	Not Done	Not applicable	Not applicable	Not applicable	Metastatic	Palliative	Not Applicable	4	2	1	IV
Antro Pylorus	No	Yes	No	No	No	Done	No	D-Lap	Negative for Malignancy	Locally Advanced	Curative	D2	4	1	0	III
Distal Body	Yes	Yes	Yes	No	Yes	Not Done	Not applicable	Not applicable	Not applicable	Metastatic	Palliative	Not Applicable	4	3	1	IV
Antro Pylorus	Yes	Yes	No	No	No	Done	Yes	D-Lap	Positive for Malignancy	Metastatic	Palliative	Not Applicable	3	1	1	IV
Distal Body	No	Yes	No	No	No	Done	No	Laparotomy	Negative for Malignancy	Early	Curative	D2	3	1	0	II
Proximal Body & Fundus	Yes	Yes	No	No	No	Done	No	D-Lap	Positive for Malignancy	Metastatic	Palliative	Not Applicable	3	1	1	IV
Antro Pylorus	No	No	No	No	No	Done	No	Laparotomy	Negative for Malignancy	Locally Advanced	Curative	D2	4	0	0	III
Antro Pylorus	Yes	Yes	No	No	No	Done	Yes	Not applicable	Not applicable	Metastatic	Palliative	Not Applicable	3	1	1	IV
Antro Pylorus	Yes	Yes	Yes	No	Yes	Not Done	Not applicable	Not applicable	Not applicable	Metastatic	Palliative	Not Applicable	3	2	1	IV
Antro Pylorus	No	No	No	No	No	Done	No	Laparotomy	Negative for Malignancy	Locally Advanced	Curative	D2	3	0	0	III
Antro Pylorus	Yes	Yes	No	No	Yes	Not Done	Not applicable	Not applicable	Not applicable	Metastatic	Palliative	Not Applicable	4	2	1	IV
Distal Body	Yes	No	No	No	No	Done	No	Laparotomy	Negative for Malignancy	Locally Advanced	Curative	D2	3	1	0	III
Antro Pylorus	Yes	Yes	No	No	No	Done	No	Laparotomy	Negative for Malignancy	Locally Advanced	Curative	D2	4	1	0	III
Antro Pylorus	Yes	No	No	No	Yes	Not Done	Not applicable	Not applicable	Not applicable	Metastatic	Palliative	Not Applicable	4	1	1	IV