"A CLINICAL STUDY OF CARCINOMA STOMACH IN PATIENTS PRESENTING IN GMKMCH, SALEM"

Dissertation submitted to

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for the award of the degree of

M.S. GENERAL SURGERY BRANCH - I



GOVERNMENT MOHAN KUMARAMANGALAM MEDICAL COLLEGE, SALEM

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CERTIFICATE

This is to certify that this dissertation entitled "A CLINICAL STUDY OF CARCINOMA STOMACH IN PATIENTS PRESENTING IN GMKMCH SALEM" is a bonafidework done by Dr. T.KRISHNAKUMAR in the department of 'GENERAL SURGERY' in Government Mohan Kumaramangalam Medical College Hospital, Salem, from December 2011 – August 2013. This has been submitted in fulfilment of the award of M.S. Degree in General Surgery by the Tamil Nadu DR.M.G.R. Medical University, Chennai – 600032.

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Date: 20 /12/13

Place: SALEM

DECLARATION

I solemnly declare that this dissertation "A CLINICAL STUDY OF

CARCINOMA STOMACH IN **PATIENTS** PRESENTING

GMKMCH, SALEM" was prepared by me at Government Mohan

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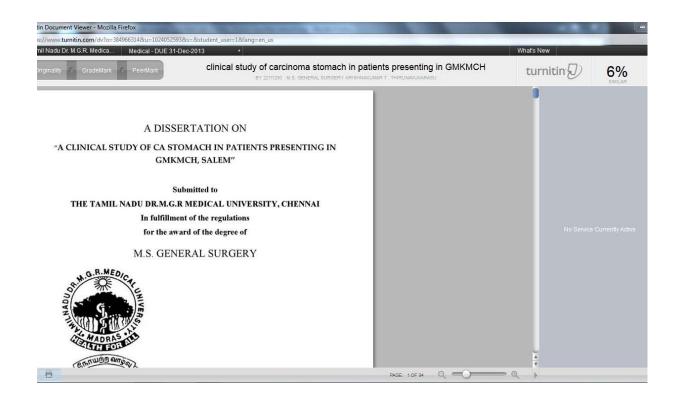
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ABSTRACT

BACKGROUND:

Second most common cancer related death worldwide. There is change in the site of occurance of gastric cancer, there is increase in occurance of proximal tumours than the distal tumours.

OBJECTIVES:

Study was undertaken in GMKMCH Salem, to study the prevalence, clinical features, association of risk factors, site of occurance and histopathology.

METHODS:

All patients with histopathology confirmed as stomach carcinoma was studied and details regarding clinical feautures, subsites, pathology, investigations and treatment modalities was collected.

RESULTS:

36 Patients with histology confirmation of stomach carcinoma was studied. There is a increase in male preponderance and old age. Association of risk factors was studied. Majority are present with distal tumours and in advanced stage.

INTERPRETATION AND CONCLUSION:

Stomach carcinoma is commonly seen in old age with male preponderance. Patients are mostly present in advanced stage. But the use of endoscopy early gastric cancers can be identified. Association of risk factors is well known and can be used in promodial prevention of disease. Despite of increase in proximal tumours in west, Distal tumours predominate in our study.

KEY WORDS

Stomach carcinoma, fundus, body, OG junction, antrum, gastrectomy, jejunostomy, chemotherapy, radiotherapy

AIMS AND OBJECTIVES OF THE STUDY

The Study was conducted based on following principles.

- ✓ To Study the anatomical location of tumour with respect to age and sex distribution.
- ✓ To Study the Etiology and Risk factors.
- ✓ To Study the classification of tumours based on morphology.
- ✓ To Study the symptoms and signs with respect to tumour location.
- To Study the various investigation modalities and its sensitivity and specificity.
- ✓ To Study the Histopathological type in relation to site of tumour.
- ✓ To Study the Surgical modalities of treatment.

INTRODUCTION

In the 21st century, Adenocarcinoma of stomach was one of the leading cause of cancer related death throughout the world. Adenocarcinoma stands second to lung cancer in causing cancer related deaths worldwide. Annually about 9,88,000 cases of the stomach was diagnosed world wide & estimated number of deaths was 4,50,000 .Stomach cancer has a geographical incidence due to diet modification, preparation of food & various environmental factors. Incidence of the carcinoma stomach is very low is India, but in most metropolitan cities stomach carcinoma was one of the 10 leading sites of cancer in both men & women. In India incidence of the stomach cancer is increasing due to migration to cities from rural population, lifestyle modification & increase in life expectancy.

One of the factor responsible for changing incidence rate is due to change in diet. In India there is diversity in diet unknown to most other countries, due to religious & cultural teachings in diet, executed for thousands of years, However role of Indian diet is causation or prevention of stomach cancer is little known, hence more attention is followed on some diet aspects such as vegetarianism, spices & food additives.

The stomach cancer has poor prognosis, except in some countries, poor prognosis due to multiple factors. Because of absence of definite risk factors, lack of symptoms specific & low incidence has contributed to delay in diagnosing at an earlier stage, leads to late diagnosis. But in Japan, since the stomach carcinoma is endemic, most of patients are diagnosed at earlier stage, this leads to increase in overall survival rates.

Overall incidence of stomach cancer is decreased over the past decades, the decrease is mainly due to the cancers arising below the esophagogastric junction. However the incidence of new cases of proximal gastric carcinomas arising is increasing. They are usually more aggressive and the treatment of such tumours is also very difficult.

The only definitive treatment for Gastric cancer is to remove the cancer both micro and macroscopically by surgical intervention. However even after complete curative treatment, disease is recurring in local or at distant region, in most of the patients. Hence now it is focusing on development of systemic and regional adjuvant therapies given pre and postoperatively.

EPIDEMIOLOGY:

Stomach cancer incidence is increasing in age. Peak incidence occurs in age between 60-80 years of age. In India age ranges from 35 – 55 yrs in south and 45-55 yrs in north. The disease shows a male preponderance in almost all countries. Two to four times higher in males than the females. The incidence of gastric cancer varies in various parts of the world. High incidence is seen in south eastasia, Europe and south America. Recent assessment of 5,56,000 deaths in cancer in India in 2010 based on National representative survey shows that Stomach cancer is the Second most fatal cancer in India.

REVIEW OF LITERATURE

HISTORY:

1st case of GASTRIC CANCER...1600 BC reported by Ebers papyrus.

Benign and malignant gastric ulcer reported by J.Cruveilhier in 1835.

Clinical picture of symptoms of gastric cancer reported by Bayle in 1839.

First Gastric resection for cancer. Jules Emiley Pean in 1879.

First successful subtotal resection with gastro duodenal anastamosis.

THEODER BILLROTH IN 22 JAN 1881.

First Total Gatrectomy.. Karl Schlatter in 1897.

ANATOMY

Stomach lies in the upper & left part of abdomen. It occupies epigastric, umbilical & hypogastric region.

- Stomach is divided into, two parts (cardiac & pyloric) division by line drawn downwards and to the left from Incisura angularis.

CARDIAC PART:-

Subdivided into Fundus & body

- 1. Fundus :-
 - Upper convex dome shaped region.
 - Situated above the horizontal line which was drawn at level of cardiac orifice.
- 2. Body:-
 - The portion that lies between the fundus and pyloric part is called body.

PYLORIC PART:-

- Subdivided into pyloric antrum and pyloric canal.

1. <u>Pyloric antrum:-</u>

- About 3 inches long
- Seperated from pyloric canal by inconstant sulcus
 Intermedius.

2. <u>Pyloric canal:-</u>

- 1 inch long, narrow and tubular curvatures of stomach.

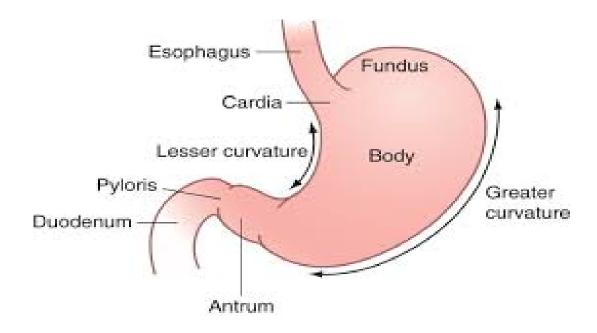
Greater curvatures:-

Convex, forms left border of stomach provides attachment to greater omentum, gastrosplenic and gastrophrenic ligaments.

Lesser curvature:

Concave, forms right border of stomach, provides attachment to lesser omentum. Incisuraangularis or angular notch is the most dependent part of lesser omentum.

PIC8. STOMACH AND ITS DIVISIONS



RELATIONS OF STOMACH:-

PERITONAL RELATIONS:-

Both anterior and posterior surfaces are lined by peritoneum At level of lesser curvature it meets &become lesser omentum. Near the cardiac end it gives attachment to form gastrosplenic ligament

Near cardiac end on posterior surface attachment for gastrophrenic ligament.

VISCERAL RELATIONS:-

Anterior part :-

- 1. Liver
- 2. The diaphragm
- 3. Anterior abdominal wall

Posterior Part :-

- 1. The Diaphragm
- 2. The left kidney
- 3. The left suprarenal gland
- 4. Pancreas
- 5. Splenic flexure of colon
- 6. Splenic artery

Blood supply:-

Blood supply of stomach is mainly from vessels arising from celiac plexus

Cystic artery

Hepatic artery

Hepatic artery

Aorta

Gastroduodenal artery

Head of pancreas

Right gastroepiptoic artery

Right gastroepiptoic artery

Splenic artery

Splenic artery

Splenic artery

Splenic artery

PIC 9. BLOOD SUPPLY OF STOMACH

LEFT GASTRIC ARTERY:

Arises directly from celiac axis course along lesser curvature. It divides is to anterior and posterior branch before reaching lesser curvature

Cardioesophagial artery arises from anterior branch. After giving this branch, it curves downwards and to right along the lesser curvature. During it descends it divides into anterior and posterior branch supplying anterior and posterior gastric wall.

RIGHT GASTRIC ARTERY;-

Small branch arises from Hepatic artery proper (51-60%) left Hepatic artery (29-41%), common Hepatic artery (3.5%). Anterior and posterior branches from this artery anastamose with infrapyloric vessels & branches from supraduodenal Ar and gives blood supply to distal gastric unit (ie Antrum, Pylorus, 2.5.cm of Ist part of duodenum) and anastamose – with left gastric artery.

RIGHT GASTROEPIPLOIC ARTERY:-

Branch of Gastroduodenal Artery or its Continuation. Can sometimes arises from superior pancreatico-duodenal Artery or Superior Mesentric Artery gives branches to Infrapyloric branch and continues along greater curvature within the gastrocolic ligaments. It gives origin to Single or Paired anterior, posterior gastric branches. It anastamose exclusively with left gastric artery branches. In about 75% of cases, clearly anatamoses with the left Gastroepiploic artery, but not as seen with the left Gastric artery. It runs in front of common bile duct & descends along posterior surface of pancreatic head.

LEFT GASTRO EPIPLOIC ARTERY:

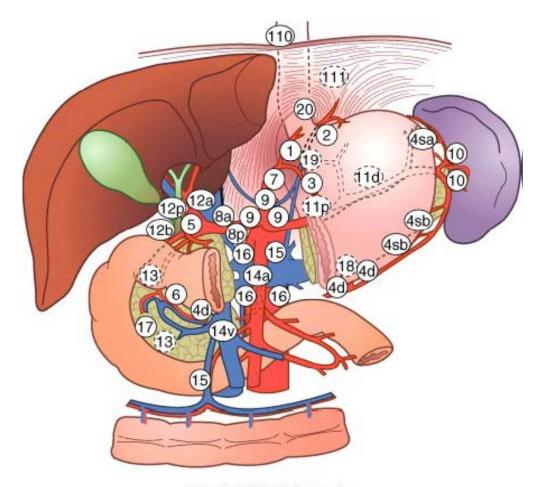
Largest branch of splenic artery, through the gastrosplenic ligament reach the stomach. It gives Anterior epiploic and Left epiploic branches, these branches along with the similar branches of Right Gastroepiploic, forms Arc of Barkow, along with branches from posterior epiploic and branches of Inferior Pancreatic Artery.

VENOUS SUPPLY OF STOMACH:-

They mainly accompany the arteries,

LYMPHATIC SUPPLY OF STOMACH:,-

Lymphatic Supply of stomach follows the blood supply and is divided into four zones. lymph from the upper lesser curvature drains into superior gastric group and it drains into left gastric and paracardial nodes .Lymph from antral segment of lesser curvature of stomach drains into suprapyloric group of nodes and into Right supra pancreatic nodes .Pancreatico lineal group of Lymph nodes drains from greater curvature and then into left Gastroepiploic and splenic nodes .Lymph along right gastroepiploic vessels drains into inferior gastric / subpyloric group of nodes. Lymph from all these 4 zones drains into celiac group of nodes and into thoracic duct.

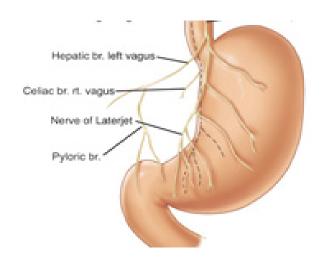


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NERVE SUPPLY OF STOMACH:-

Parasympathetic innervations to the stomach is through the vagus and acetylcholine is the neurotransmitter. Vagus through Anterior and posterior branches supplies the stomach.

PIC 11.NERVE SUPPLY OF STOMACH



Anterior vagus near the Gastro esophageal junction, gives branches to the lesser curvature, and continues as Anterior nerve of Latarget along the Lesser curvature.

Posterior vagus gives branches to celiac plexus and it continues similarly along posterior side as nerve of latarjet terminates near the incisura angularis as "crow foot" give branches to Antropyloric region.

Vagal nerves are more than 2 in 50% of patients .Near the esophageal Hiatus Criminal nerve of Grassi is the branch of posterior Vagus supplying posterior part of Fundus. It arises above the Oesophageal Hiatus and it is missed during Truncal or Highly selective Vagotomy (HSV)

The Sympathetic nerve supply of stomach is through T5-T10. It travels via splanchnic nerves to celiac ganglion. Nerves from the Celiac ganglion reaches the stomach through the pathway of blood vessels.

LAYERS OF STOMACH:-

SEROSA:-

Visceral peritoneum forms serosa, provides tensile strength to gastric anastamosis. Microscopic or gross peritoneal metastasis are common if the tumour penetrate the serosa.

MUSCULAR LAYER:

Also called as Muscularis externa, It consists of following layers.

- 1. Middle Circular layer:-
- It is complete and it is continuous with circular muscles of oesophagus and pylorus circular muscle.
- 2. Outer Longitudinal Layer.
- It is complete and it is continuous with. esophagus and duodenal Longitudinal Layer.
- 3. Inner Oblique Layer:-

It is incomplete.

Auerbach myentric plexus are rich network of autonomic ganglia occupying muscularis propria. Intestitial cells of cajal (Ice) which are pacemaker cells present in this Layer.

SUBMUCOSA:-

Lies deep to muscularis mucosa, consists of blood vessels, collagen, Lymphatics, Inflammatory cells, ganglion cells and Submucosal plexus of meissners. It is also called as Vascular layer.

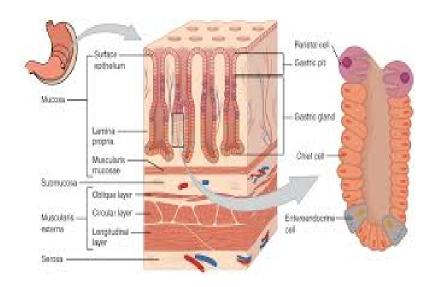
MUCOSA:-

It constitutes the Inner Layer, Lined by various types of Columnar epithelial cells. The epithelium, Lamina propria and the muscularis mucosa, together called as Mucosa. Simple columnar cells are present in cardia. Parietal (Oxyntic) and acid Secreating cells and chief pepsin - secreating cells are present in Fundus and body, Branched tubular gastric glands are present in cardia. Parietal (oxyntic) and acid secreting cells and chief pepsin secreating cells are present is fundus and body. Branched tubular gastric glands are present is lamina propria of fundus and body.

Esophagus
Pylorus
Duodenum
Mucosa
Submucosa
Muscle layers
Rugae
Serosa

PIC 12. LAYERS OF STOMACH

PIC 13.HISTOLOGY OF STOMACH



RISK FACTORS FOR CA. STOMACH

Acquired Factors:-

- High salt consumption
- High nitrate intake in dried, smoked foods,
- Poorly prepared food (smoked salt cured)
- Well water
- Lack of refrigeration
- Occupational
 - Rubber & coal workers

- Cigarette smoking
- H pylori infection
- Ebstein bar virus
- Prior gastric surgery for benign gastric ulcer disease.

GENETIC FACTORS:-

- Blood Group
- Pernicious anaemia
- Family History
- Hereditary Nonpolyposis colon cancer
- Li-Fraumeni syndrome

H-PYLORI INFECTION:-

- H pylori has strong association with gastric adenocarcinoma arising in antrum.
- Many studies demonstrates that H pylori is associated with double the risk of carcinoma mechanism by which H pylori acts is unclear however it produce chronic atrophic gastritis leads to a Low acid environment leading to Metaplasia and dysplasia

studies shows that only 5% of H pylori infected persons develops carcinoma occur 10 years .

- studies also shows that infection with cag A strains, produces more inflammation is stomach associated with increase risk of carcinoma.

PATHOLOGY:-

About 95% of all neoplasms are Adenocarinoma, other tumours are rare it includes squamous cell carcinoma, Adenoacanthoma, Carcinoid tumours and Leiomyosarcoma. There is increase is incidence of association between H pylori and mucosa associated lymphoid tissue lymphomas.

EARLY GASTRIC CANCER:-

Defined as Adenocarcinoma confined to mucosa and submucosa, irrespective of Lymphnode Status, About 70% of early gastic carers are well differentiated Adenocarcinoma about 30% are pooly differentiated About 95% have overall cure rate japanese classified based on this.

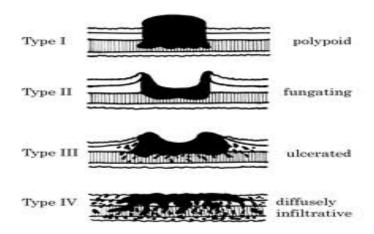
ADVANCED GASTRIC CARCINOMA:

Bormann divides Gastric carcinoma based on macroscopic appearance it invades muscularis termed as advanced Gastric carcinoma.

- Type I :- Polypoidal / fungating lesions.
- Type II :- Ulcerative lesions surrounded by elevated borders.
- Type III :-Ulcerative lesions infiltrating to gastric wall
- Type IV :Diffuse infiltrative lesions
- Type V : Unclassifiable cancers

Type iv when it involves entire stomach it is termed as Linitis plastica.

PIC14.BORRMANNS CLASSIFICATION



BORDERS HISTOLOGICAL CLASSIFICATION:-

According to differentiation of cells, independent of morphology type I [well differentiated] to Type 4 [anaplastic] type .

MING CLASSIFICATION:-

Classified into Expansive type which has favourable prognosis and infiltrating type, which has poor prognosis.

LEVEEN'S CLASSIFICATION:-

Proposed by Lauren in 1965.

Divided into two types Intestinal (53%) and Diffuse types (33%) based on Histology.

TAB 19. LAURENS CLASSIFICATION

INTESTINAL	DIFFUSE	
Environmental	Familial	
Gastric atrophy, Intestinal Metaplasia	Blood Group.A	
M> W	W>M	
Incidence Increase with age.	Occurs in Younger age.	
Formation of glands	Poorly differentiated, Signet ring cells.	
Spreads through Haematogenous	lymphatic/Transmural spread	
Microsatellite Instability	Decreased E- Cadherin	
APC gene mutation	P53, P16 Inactivation.	
P53, P16 Inactivation.		

INTESTINAL METAPLASIA:-

It is defined as process of replacing gastric mucosa with mucosa resembling small Intestine.

It is the intermediate step in development of stomach cancer in correa's model. It is due to the constant irritation of mucosa of stomach by risk factors such as H.Pylori. It is further classified into complete type I. Incomplete Type

II and Type III. The differentiation is based on mucin core protein (MUC).

Type III has Increased risk of progression to Ca. Stomach than Type I.

Moleucular alterations such as Overexpression of cyclo- oxygenase, D2 cyclins, P53 mutations, decreased P27 expression, Microsatellite. Instability, alterations in CDX1 x CDX2 can cause Carcinoma. Recently in patients having gastric remnants with dysplasia, Spasmolytic polypeptide, led to concept of Spasmolytic-polypeptides expressing. Metaplasia(SPEM) may be precursor to Gastric adenocarcinoma.

WHO CLASSIFICATION:-

- 1. Adenocarcinoma-
 - Papillary
 - Tubular
 - -Mucinous
 - -Signet ring cell.
- 2. Adenosquamous cell carcinoma
- 3. Squamous cell carcinoma
- 4. Undifferentiated carcinoma.

PREDISPOSING FACTORS:-

1. Gastric Polyps:-

- Malignancy is increased is polyps particularly Adenomas-Villous

2. Pernicious Anaemia:-

- Achlorhydria due to destruction of oxyntic mucosa by auto immune mechanism is major risk factor, this will leads to destruction of chief and parietal cells which develops antral and Intestinal metaplasia. 6% of Pt will develops ca stomach.

3. Mutations in CDHI:-

- E cadherin is calcium lon dependent adhesin moleucle present in tansmembrane responsible for epithelial cell interactions, so, when the expression is reduced as in cases of CDHI mutation, associated with tumour invasivenes.Reduced E-cadherin expression is noted in about 93% of carcinomas and it is associated with Undifferentiated, Diffuse type cancers therefore the persons who are carriers of these mutations are advised prophylactic gastrectomy.
- 4. p53 over expression seen is 50% patients
- 5. Menetiers disease
- 6. Hypogammaglobulinaemia..

CLINICAL PRESENTATION:-

Most patients are diagnosed is advanced stage of disease due to presence of vague, non specific symptoms, Anorexia, fatigue, Epigastric discomfort, usually signs of advanced disease weight loss is a common symptom, about 80% of patients have more than 10% decrease is body weight before confirming diagnosis location of tumour can cause specific symptoms presence of tumour in cardia presents with dysphagia. Diffuse infiltrative tumour presents with Early satiety due to loss of distensibility of Gastric wall. Antral carcinoma obstructing pylorus presents with persistent vomiting, Gastrointestinal bleeding presents is about 10% of patients Ascites, Jaundice, palpable mass indicates advanced or incurable disease symptoms sometimes related to spread of the disease Intestinal obstruction occurs is pt with diffuse peritoneal spread krukenberg's tumour (large ovarian mass) of peritoneal implant is the pelvis (Blumer's shelf) produces symptoms of Rectal obstruction. Sister mary joseph nodules is nodular metastasis is subcutaneous tissue around umbilicus.

SITE DISTRIBUTION:-

Ca stomach presents in both proximal and distal part. In Developing countries Distal gastric cancers predominate, also in blacks and peoples in Lower Socioeconomic status. Dietary factors and H.pylori are most common causes of distal tumours. Proximal cancers are common in developed countries

and also among whites and Higher Socioeconomic states. Obesity and Gastro oesophageal reflux disease are most common cause. Inspite of Increase in prevalence of proximal tumours, distal tumours are predominate in Japan. Reports from the west shows that there is a shift in site of occurance of malignancies of Gastric Cardia Increasing, which are contrary to Information from middle east and south asia.

INVESTIGATIONS:-

ENDOSCOPY:-

- Best method to diagnose Ca stomach is by directly visible the mucosa of the stomach and tissue biopsy can be taken for Histopathological diagnosis. Several biopsies are taken from ulcer. The diagnostic accuracy is increased if we add brush cytology to endoscopy.

ENDOSCOPIC ULTRASOUND:-

Its Main use is to stage the previously diagnosed stomach cancers. Used to evaluate T stage of the tumour and also about Surrounding lymphnodes. Accuracyof 90% in detecting T staging and for Nodal status it is 75%.

COMPUTED TOMOGRAPHY:-

CT Abdomen and pelvis is done to stage the patients. In case of tumours arising in proximal part, CT chest is also taken. It evaluates tumour penetration, perigastric lymph node states, liver, and other organs metastasis, and to detect peritoneal disease. Recently use of water as an oral contrast-Helical Hydro CT, Increase the efficacy of CT in detecting tumour site and staging.

4. PERITONEAL ANALYSIS:-

Occult metastasis can be identified by cytological analysis of peritoneal fluid their prognosis is similar to the patients with macroscopic or visceral peritoneal metastasis.

TUMOUR MARKERS:

About one third of patients with ca stomach has elevated carcinoembryonic antigen [CEA]. Sensitivity is low, but if the levels are raised it correlates with the staging. Other markers used are CA19-9, CA50, CA72.4. Combination of these markers increase the sensitivity. Most studies in tumour markers directed towards prognostic power of pre operative serum concentrations. Results of few studies reported that use of CEA, CA19-9, in follow up of the patients, suggesting that measurement of this markers is useful in early detection of recurrence.

POSITRON EMISSON TOMOGRAPHY:

Based on the uptake of 5 fluorodeoxy glucose radioactive material by cancerous cells. But in stomach carcinoma there are some drawbacks due to glucose transporter 1 major transport of FDG into tumour cells which is rarely

present in mucinous and signet ring type this will leads to false negative

imaging.

LAPROSCOPY:

Staging laproscopy is used in the pre treatment evaluation, of patients in

detecting CT occult metastatic disease. Since CT cannot diagnose metastasis of

less than 5mm and it also directly visualize peritoneal and visceral surfaces,

and for detecting metastasis peritoneal cytology and laproscopic USG can also

done.

STAGING:

American joint committee on cancer /International union against cancer

[AJCC/UICC] is the staging system currently in use.

PRIMARY TUMOUR

Tx: Primary tumour cannot be assessed

T0: no evidence of primary tumour

Tis: carcinoma in situ: intra epithelial tumour without invasion of the

lamina propria

T1: Tumour invades lamina propria or submucosa

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T2: Tumour invades the muscularis propria or the submucosa

T2a:Tumour invades muscularis propria

T2b:Tumour invades subserosa

T3: Tumour penetrates the serosa(visceral peritoneum) without invading

adjacent structures

T4: Tumour invades adjacent structures.

REGIONAL LYMPH NODES

Nx: Regional lymph nodes cannot be assessed

N0: No regional lymph node metastasis

N1:Metastasis in 1-6 regional lymph nodes

N2:Metastasis in 7-15 regional lymph nodes

N3:Metastasis in more than 15 regional lymph nodes.

DISTANT METASTASIS

Mx: Distant metastasis cannot be assessed

M0: No distant metastasis

M1: Distant metastasis.

AJCC STAGING

Stage 0 (Tis,N0,M0)

Stage 1a (T1, NO,MO)

Stage1b (T1,NI,MO;T2A,N0,M0;T2B,NO,MO)

StageI I (T 1,N2,MO;T2A,NI,MO;T2B,N1,M;T3,NO,MO)

StageIIIA (T2A,N2,MO;T2B,N2,MO;T3,NI,MO;T4,NO,MO)

StageIIIB (T3,N2,MO)

StageIV (T4,N1,MO; T4,N2,M0; T4,N3,MO; T1,N3,MO;T2,N3,M0;

T3,N3,M0; Any T, ANY N, M1)

JAPANESE STAGING:

Japanese classification of ca stomach was published in 1998.it gives

more detail information than the AJCC staging. On the basis of clinical,

surgical, pathological and final staging [prefixes c, s, p, f]. It also includes

classification system for early gastric cancer. It classifies lymph node into four

categories based on ha relation to primary tumour and anatomical location.

Group 1: perigastric lymph nodes (nodal station 1-6).

Group 2: lymph nodes along proximal left gastric artery(station 7),

common hepatic artery(station 8), celiac axis(station 9), splenic artery (station

11), proper hepatic artery (station 12).

Group 3: para aortic lymph nodes (station 16).

TUMOUR STAGE:T

T1: Tumour invasion of mucosa, and/or muscularismucosa, submucosa.

T2: Tumour invasion of muscularis propria or sub serosa.

T3: Tumour penetrates serosa.

T4: invasion of adjacent organs.

Tx: unknown.

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NODAL STAGE:N

N0: No evidence of lymph node metastasis .

N1: Metastasis to group 1 nodes.

N2: Metastasis to group 2 nodes.

N3: Metastasis to group 3 nodes.

Nx: unknown.

HEPATIC METASTASIS STAGE:H

H0: No liver metastasis.

H1: Liver metastasis.

H2: unknown.

PERITONEAL METASTASIS STAGE:P

P0: No peritoneal metastasis.

P1: Peritoneal metastasis.

Px: Unknown.

PERITONAL CYTOLOGY STAGE:CY

CY0: Benign or intermediate cells in cytology.

CY1: Malignant cells in cytology.

CY2: Cytology was not performed.

OTHER DISTANT METASTASIS(M):

M0: No other distant metastasis.

M1: Distant metastasis other than liver, peritonel and cytology measures.

M2: Unknown.

STAGE GROUPING:

T1	N0	N1	N2	N3
T2	IA	IB	II	
T3	IB	II	IIIA	
T4	II	IIIA	IIIB	IV
H1, P1, CY1, MI	IIIA	IIIB		

RESECTION CLASSIFICATION:

First described in 1994 by hermaneck.

R0: No gross microscopic or residual disease.

R1: Microscopic residual disease.

R2: Gross residual disease.

Some authors includes R staging as to complement the TNM staging, since survival can be increased by extent of resection of the tumour.

CLASSIFICATION OF OESOPHAGO GASTRIC JUNCTION TUMOURS:

Proposed by siewart and stein commonly known as SIEWART classification.

TYPE1: Adenocarcinoma of distal oesophagus arises intestinal metaplasia of oesophagus [Barrrets oesophagus], infiltrate into gastro oesophageal junction.

TYPE2: Adenocarcinoma of cardia, arises from the epithelium of cardia or from short segment with intestinal metaplasia at GE junction.

TYPE3: Adenocarcinoma of subcardial stomach, infiltrate the oesophago gastric junction or distal oesophagus from below.

PATTERNS OF SPREAD:

Direct spread:

It penetrates the muscularis propria and serosa and involves the liver, colon, pancreas.

Lymphatic spread:

Occurs by tumour penetration and emboli to the affected group of nodes, it is very extensive can appear in left supra clavicular nodes (Troissies sign). Nodal metastasis does not indicates systemic dissemination.

Haematogenous spread:

It first spread to the liver and then into the lungs and bone. It is uncommon in the absence of lymph node metastasis.

Trans peritoneal spread:

It indicates incurability occurs once serosa is breached and tumour involves the peritoneum, commonly manifests as ascites, in advanced disease peritoneal spread can be palpated rectally or abdominaly as 'shelf'. Involvement of ovaries (krukenberg tumour), or into umbilicus (sister josephs nodule).

SURGICAL TREATMENT:

EARLY GASTRIC CANCER:

Japanese classified early gastric cancer on the basis of endoscopic

appearance.

Type0I: Protruded type

Type0 IIa: Superficialy elevated.

Type0 IIb: Flat type

Type0 IIc: Superficialy depressed

Type 0 III: Excavated.

ENDOSCOPIC MUCOSAL RESECTION:

In 75% of cases of early gastric cancer complete resection can be

achieved by this technique. It involve injection of fluid in the submucosal space

and tumour is resected under endoscopic guidance. It should be offered only to

the patients with low metastatic potential, well differentiated Type IIa, Type IIc

lesions less than 3 cm in diameter.

LIMITED SURGICAL RESECTION:

Alternative to gastrectomy in patients with early gastric cancers. Patients

with small intra mucosal tumours (less than 3 cm) and with non ulcerated intra

mucosal tumours are candidates of this procedure. It includes gastrotomy with

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local excision performed with full thickness mural excision aided by intra

operative gastroscopy to localize the tumour.

GASTRECTOMY:

Patients having intra mucosal tumours with poor histological

differentiation or size more than 3cm which is penetrated the submucosa or

beyond are the candidates for gastrectomy with lymph node dissection. It

allows pathological staging and local theraphy in high risk patients.

PROXIMAL TUMOURS:

Surgical treatment of proximal tumour is based on the SIEWARTS

classification. They are usually advanced at the time of prognosis and

associated with poor prognosis.

Type I : oesophageal resection with marginal 10 cm clearence.

Type II: Oesophago gastrectomy.

Type III: Total gastrectomy with reconstruction.

Total gastrectomy had no survival benefit over proximal or distal

gastrectomy. If the tumour is completely removed with negative margins.

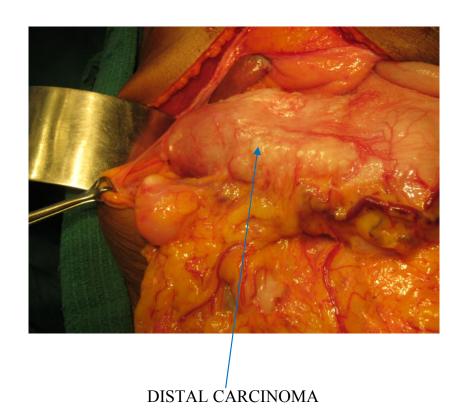
MIDBODY TUMOURS:

Treatment includes total gastrectomy with regional lymph adenectomy.

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DISTAL TUMOURS:

Treatment is distal subtotal gastrectomy with lymph adenectomy. Subtotal gastrectomy involves resection of three fourth of stomach includes majority of lesser curvature.

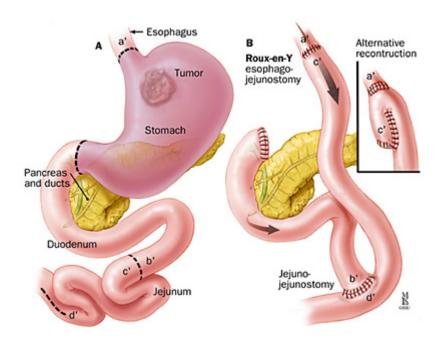


SURGICAL PROCEDURES:

TOTAL GASTRECTOMY:

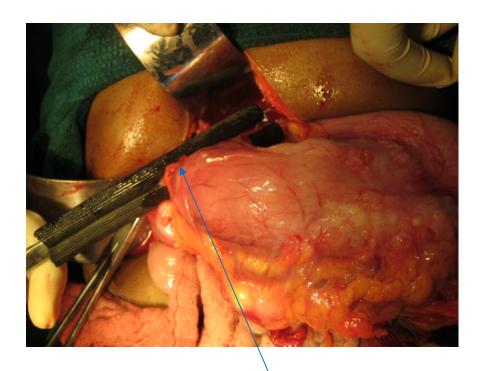
Indicated for proximal gastric tumours and extensive lesions Linitis plastic and for tumours where adequate marginal clearance of 4-6 cm is not possible by subtotal gastrectomy. Upper midline or bilateral roof top incision is made and thorough examination of abdominal organs is made. First step is detachment of greater omentum from transverse mesocolon along the avascular

plane about one cm from the bowel. The omentum and anterior leaf of transverse meso colon is removed along the stomach. On the right of dissection, right gastro epiploic vessels are ligated at the origin and sub pyloric nodes are excised. Then anterior layer of pancreatic capsule is removed and dissection is continued laterally where short gastric vessels are divided. Liver is retracted and gastro hepatic ligament is identified and is divided to access esophageal hiatus.



The peritoneal reflection on the liver is continued along the right side and lesser sac is entered to gain access to level 12 lymph nodes where tape is hold around hepatic artery where significant number of lymph nodes are excised. Right gastric artery arising from it are identified and ligated near its origin. Duodenum is fully kocherised to allow access for retro pancreatic and

hepato duodenal region. Duodenum is divided and closed to give access to infra pyloric group of lymph nodes .



DIVISION OF DUODENUM

Cephalad portion of stomach is elevated and it exposes celiac plexus and left gastric artery. Left gastric artery is divided near its origin to hold lesser curve arterial arcade and lymph nodes are removed. On the greater curvature remaining short gastric vessels are divided close to spleenic hilum and lymph nodes are removed en bloc. Stomach is fully mobilized and stay sutures are introduced in greater and lesser curvature and distal esophagus. Non crushing clamp is applied proximal to esophageal stay sutures and esophagus is divided. There are many options for reconstruction. Commonly used is roux en y loop reconstruction. Roux en y loop reconstruction is end to side esophago jejuno anastomosis. Jejunal loop is divided and distal end is brought through

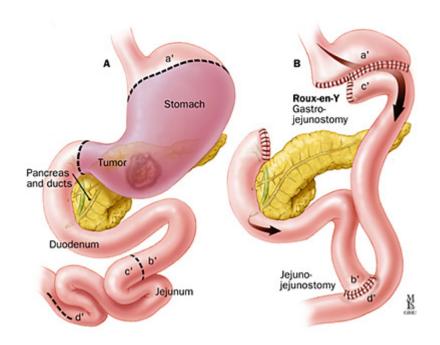
transverse meso colon and end to side anastomosis is done with single layer of mono filament absorbable sutures or with circular stapler. Proximal end of jejunum is anastomosed 40-50 cm downstream to reduce the reflux.

PROXIMAL GASTRECTOMY

Midline incision is made in abdomen. Esophageal hatus is identified by retracting the left lobe lateral segment of liver and diaphragmatic hiatus was incised. Retro peritoneal attachments to the diaphragm are incised on the left side and the esophagus is mobilized and retracted up and forwards and resected based on tumour extension. For extensive cardiac lesions spleen is mobilized and spleenectomy is done. For spleenic mobilization short gastric vessel are ligated close to spleenic hilum and resection is done forward alond posterior surface. It exposes celiac plexus and left gastric artery, lymph nodes along lesser curvature are dissected. Care should be taken not to de vascularise lesser curvature. Stomach is divided with GIA 60 OR TA stapler in oblique fashion.

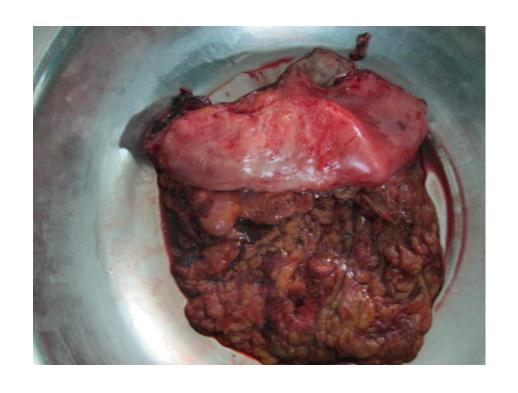
SUB TOTAL GASTRECTOMY

For tumours involving pylorus and distal one third of stomach, sub total gastrectomy with lymph node dissection (D2 D3 Lymphadenectomy) is done. It is having low peri operative morbidity and mortality. Surgical steps are similar to total gastrectomy except final step of dividing short gastric vessels which is not done. Once resection and lymphadenectomy in done, e construction is performed with Roux en y loop anastomosis or Billroth II.



EXTENT OF LYMPHADENECTOMY:

Japanese research society for ca stomach classified lymph nodes draining stomach into D1(stations 3 to 6), D2(stations 1,2,7,8,11), D3(station 9.10,12). Standard surgery for ca stomach in Asia is D2 gastrectomy involves extensive lymph node resection . (D1, D2). D2 resection removes peritoneum over the pancreas and anterior meso colon in addition to D2 resection.



RESECTED SPECIMEN OF CA STOMACH



CUT SURFACE

PALLIATIVE SURGERIES

It is done in case of advanced ca stomach to minimise the bleeding and to relieve gastric outlet obstruction.

Palliative gastrectomy

Anterior gastro jejunostomy

Laser ablation

Endoscopic placement of metallic stents

SURGICAL COMPLICATIONS:

Anastomotic leak (5-20%)

Dumping syndrome(10%)

Bleeding (0.5-6%)

Pulmonary complications (3-50%)

Infection (3-20%)

Renal complications(1-5%)

SURGICAL OUTCOME:

5 year survival rate of stomach cancer is analysed based on National cancer institute SEER data base classified based on the stage.

Stage IA - 70%

Stage IB - 57%

Stage IIA - 46%

Stage IIB - 33%

Stage IIIA - 20%

Stage IIIB - 14%

Stage IIIC – 9%

Stage IV - 4%

NEO ADJUVANT CHEMOTHERAPY:

Given before surgery. It reduces the tumour seedling during surgery. Drugs used are etoposide, epirubicin, cisplatin, 5- fluoro uracil, adriamycin and methotrexate. Many trials are conducted in evaluating neo adjuvant chemotherapy. But there is no decrease in operative morbidity and mortality with this treatment.

INTRA PERITONEAL CHEMOTHERAPY:

Intra peritoneal chemotherapy of mitomycin c along with hyperthermia is given to prevent high risk of peritoneal metastasis which was initial cause for treatment failure. Since about 50% gastric cancer patients who undergone curative resection have clinically evident peritoneal metastasis.

ADJUVANT CHEMO RADIOTHERAPY:

Combining 5 fluoro uracil (15 mg/kg/day bolus) for three days with 40 gy of external beam radiation. There is significant increase in survival when combined with radiotherapy alone. 5 fu act as a radio sensitizer. Patient who undergone radiotherapy have a 5 year survival rate of 20% vs 5% per cent survival rate in patients undergoing surgery alone. Loco regional recurrence also decreased from 54% to 39% in combined modality treatment.

TREATMENT OF ADVANCED DISEASE(STAGE IV)

SURGERY FOR PALLIATION:

Survival rate for patients with advanced gastric cancer are poor. It should provide symptomatic relief and minimize the morbidity operative mortality was 25% for gastro jejunostomy. 20% for palliative partial subtotal gastrectomy and 27% for proximal palliative gastrectomy. The most fatal complication following surgery is anastamotic leak.

CHEMOTHERAPY:

Multi drug regimens are now evaluated for gastric cancer. Combination therapy includes FAM(5FU,Adriamycin, methotrexate), FAMTX(5FU, adriamycin, methotrexate, etoposide), and 5 fluoro uracil, irinotecan, epirubicin cisplatin 5 fu, 5 fu irinotecan leucovarin, cisplatin plus irinotecan, 5 fu leucovarin oxaliplatin (FOLFOX).

RADIOTHERAPY:

Limited to palliate symptoms such as bleeding, pain which are secondary to tumour infiltration. No studies have evaluated the use of radiotherapy in locally recurrent or metastatic carcinoma of stomach.

TARGETED THERAPIES:

Bevacizumab:

It is the monoclonal antibody against vascular endothelial growth factor receptors (VEGF). It can be given along with cytotoxic chemotherapy even in primary gastric cancer.

Tyrosine kinase inhibitor:

Erlotinib and gefitinib, tyrosine kinase inhibitors are evaluated for gastro esophageal junction and gastric cancers. Epidermal growth factor receptor antibody cetuximab also under the study as a single or in combination with systemic chemotherapy.

METHODOLOGY

The present study was undertaken at Government Mohan Kumaramangalam Medical College Hospital Salem from period of and December 2011 to August 2013. It includes those patients presenting to the in and outpatient department GMKMCH, Salem.

MATERIALS

- ✓ Patients presenting to our hospital during the study period and who are found eligible for this study are included.
- ✓ 36 Cases of carcinoma stomach was studied during this period
- ✓ Details of 36 patients with carcinoma stomach was collected and followed based on various surgical treatment methods used.
- ✓ Cancer recurrence after surgery and benign lesion were excluded from the study.
- ✓ Age, Gender, Symptoms, Signs and Surgical treatments are recorded in pre structured proforma.

- ✓ Site of the tumours is classified based on following ,
 - 1. OG junction
 - 2. Fundus & Cardia
 - 3. Body
 - 4. Antrum
- ✓ Relationship of tumour marker (CEA) and its association were included in this study.
- ✓ Borrmans classification and WHO classification and its relation to site of Tumour were included in this study.

DATA ANALYSIS

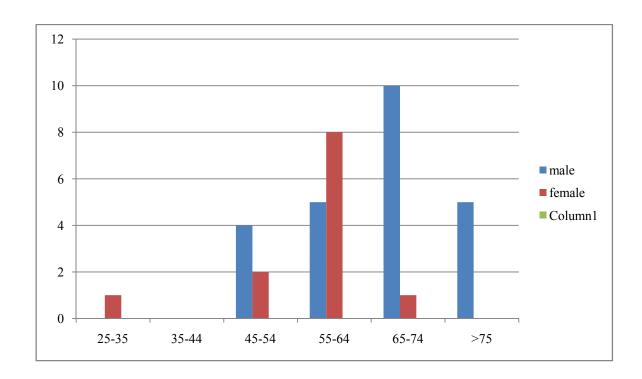
AGE PREVALENCE:

Carcinoma of stomach is disease of older age, and its incidence is increasing with increase in age. In our study maximum number of cases are seen between, 55-64 yrs, youngest is 33 and oldest is 74.

TABLE 1: AGE DISTRIBUTION

AGE	PRESENT STUDY							
	TOTAL	%	M	F				
< 25	0	0	0	0				
25-34-	1	77%	-	1				
35-44	4	11.11%	4	-				
45-54	7	19.44%	5	2				
55-64	18	50%	10	8				
65-74	6	16.66%	5	1				

PIC 1. AGE DISTRIBUTION

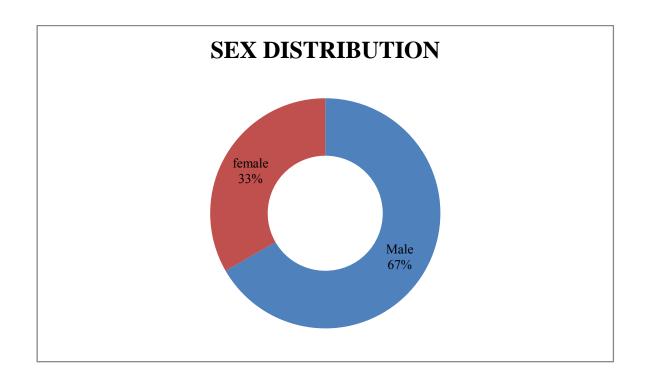


SEX DISTRIBUTION:-

Ca Stomach is common in males. In this study male female ratio is 3:1.

TABLE 2: SEX DISTRIBUTION

	PRESENT STUDY				
SEX	CASES	%			
MALE	24	66.66			
FEMALE	12	33.33			

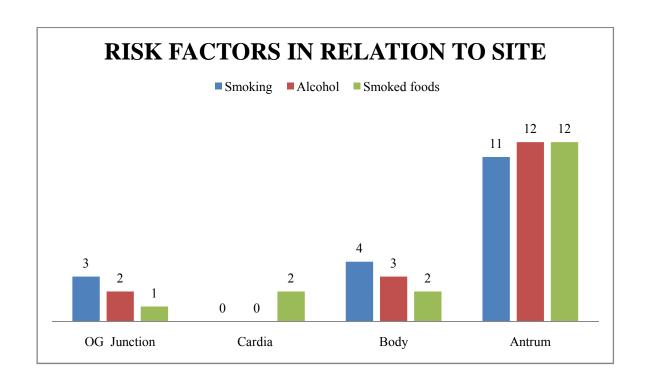


RISK FACTORS:-

TABLE 3: RISK FACTORS AND ITS RELATION TO SITE

SITE	SMOKING	ALCOHOL	SMOKED FOODS
OG junction	3	2	1
Cardia & Fundus	-	-	2
Body	4	3	2
Pyloric antrum	11	12	12
Linitis plastica	-	-	-

- ✓ In male 100% of OG junction tumours are associated with smoking and is antral tumour 68.75%, 100% of tumours in body are associated with smoking.
- ✓ Smoked food shows increase in association with antral growth.



SITE:-

In our study common site of Ca stomach is pyloric antrum . pyloric antrum constitutes 21 out of 36 cases.

Study was is concordance with cherian et al (2007) & swarman et al studies .

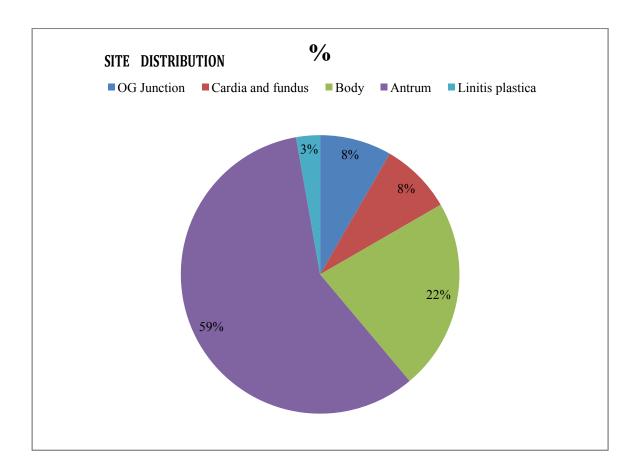
But the study was contradictory to increase in incidence of proximal growth in western countries

Finally this study indicates that there is increase in distal growth than the proximal growth in our region.

SITE DISTRIBUTION:

TABLE 4.SITE DISTRIBUTION

SITE	No of cases	%
OG Junction	3	8.33
Cardia and fundus	3	8.33
Body	8	22.22
Antrum	21	58.33
Linitis plastic	1	2.77



GENDER DISTRIBUTION ACCORDING TO SITE:

Based on gender distribution site are evaluated.

TABLE 5.GENDER DISTRIBUTION ACCORDING TO SITE

SITE	MALE	FEMALE	RATIO
OG Junction	3	0	3
Cardia and fundus	1	2	0.5
Body	4	4	1
Antrum	16	5	4.1
Linitis plastica	1	0	1

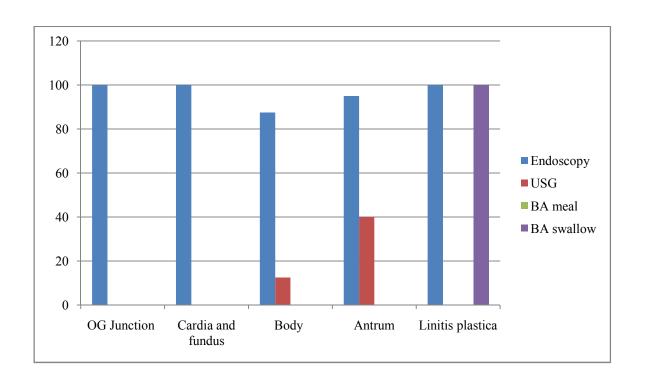
The ratio of male: female in cancers involving proximal stomach, body, antrum, OG junction was 0.5:1:4:1:3

This was not in concordance with study conducted by Cherian et al, ratio is 3:2:3:4 respectively

INVESTIGATIONS:

TABLE 6.INVESTIGATIONS

SITE	Endoscopy	USG	BA meal	BA swallow
OG Junction	100%	_	_	-
Cardia and fundus	100%	_	_	_
Body	87.5%	12.5%	_	_
Antrum	95%	40%	_	_
Linitis plastica	100%	_	_	100%



PIC 4.INVESTIGATIONS RELATED TO SITE

Endoscopy and biopsy was done in all cases

It shows 100% in detecting OG junction and proximal tumour growths, 87.5% in body and 95% in antrum

Ultrasound has similarity of detecting 12.5% of body and 40% of antral growth.

Carcino embryonic antigen:

12 cases are positive for carcino embryonic antigen

TOTAL CASES	CEA	Positive	Negative
36		12	24

About 33% of cases are positive for carcino embryonic antigen.

SYMPTOMS:

TABLE 7. SYMPTOMS

Symptoms	Total		Male=24		Female=12	
	Cases	%	Cases	%	Cases	%
Abdominal pain	28	77.77	20	83.33	8	66.66
Nausea/ vomiting	29	80.55	21	87.5	8	66.66
Weight loss	30	83.33	18	75	12	100
Anorexia	14	38.88	10	41.66	4	33.33
Early satiety	16	44.44	11	45.83	5	41.66
Dysphagia	12	33.33	11	45.83	1	8.3
Melena	6	16.66	4	35	2	1.66

Male patients commonly presents with abdominal pain and vomiting in about 80% of cases.

Female patients commonly presents with weight loss which is seen in 100% of cases.

Melena is common in males than the females.

PIC 5.SYMPTOM ANALYSIS IN MALE AND FEMALE:

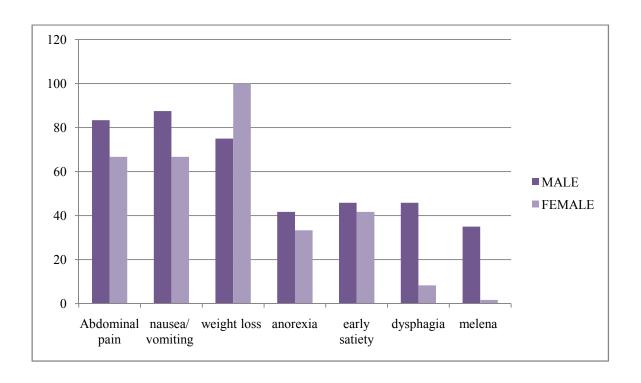


TABLE 8.COMPARISON OF SYMPTOMS WITH SITE OF TUMOUR:

		SITE							
SYMPTOM	Total	Total ANTRU		RUM (21) BODY(8)		FUNDUS (3)		OG JUNCTION (3)	
		CASES	%	CASES	%	CASES	%	CASES	0/0
Abdominal pain	28	20	95.2	4	50	3	100	1	33.3
Nausea/ vomiting	29	21	100	5	62.5	2	66.6	1	33.3
Weight loss	30	21	100	5	62.5	3	100	1	33.3
Anorexia	14	12	57.14	1	12.5	1	33.3		
Early satiety	16	12	57.14	3	37.5	1	33.3		
Dysphagia	12	5	23.80	1	12.5	2	66.6	2	66.66
Melena	6	2	9.5	2	25	2	66.6		

Antral growth predominantly presents with nausea, vomiting, weight loss and abdominal pain.

Dysphagia is reported in 24% of cases.

Growth in body presents with nausea, vomiting and weight loss predominantly.

Growth arising from fundus and OG junction has dysphagia, abdominal pain and weight loss as predominant symptoms.

SIGNS:

TABLE 9.SIGNS

CNAMPTOMO	TOTAL		MA	LE	FEMALE		
SYMPTOMS	CASES	%	CASES	%	CASES	%	
Anaemia	29	80.55	21	87.5	8	66.66	
Jaundice	7	19.44	6	25	1	8.33	
Lymphadenopathy	13	36.11	12	5.	1	8.33	
Mass	15	41.66	10	41.66	5	41.66	
VGP	21	58.33	17	70.83	4	33.3	
Ascites	8	22.22	5	20.83	3	25	

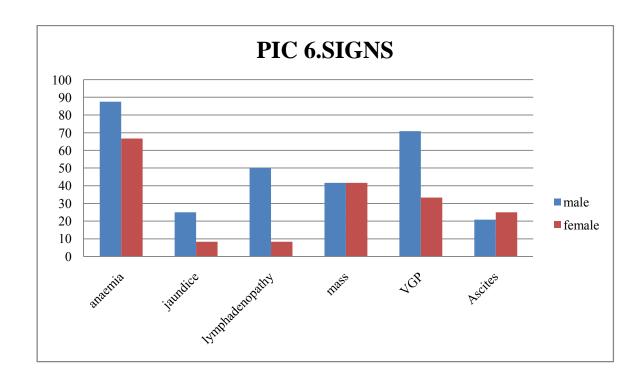
In both females and male, anaemia is the predominant sign.

Palpable mass was noted in about 41% of both male and female.

20% of patients presents with jaundice.

Visible gastric peristalsis is predominant in 70% of males and 33% of females.

Advanced stage of disease represented by ascites was noted in 20% of males and 25% of females.



COMPARISION OF SIGNS IN BOTH SEXES

TABLE 8. COMPARISON OF SIGNS WITH SITE OF TUMOUR:

		SITE							
SYMPTOM	Total	ANTRUM(21)		BODY(8)		FUNDUS(3)		OG JUNCTION (3)	
		CASES	%	CASES	%	CASES	%	CASES	%
Anaemia	29	19	90.47	4	50	3	100	3	100
Icterus	7	6	28.57	0	0	0	_	1	33.3
Mass	15	10	47.61	4	50	1	33.3	_	_
VGP	21	19	90.47	2	25	0	_	_	-
Ascites	8	5	23.80	2	25	0	_	_	_
Lymphadenopathy	13	9	42.85	2	25	1	33.3	1	33.3

Anaemia is present in 100% of proximal tumours.

In tumours arising from antrum, anaemia, VGP, followed by abdominal mass is major presentation.

In tumours arising in body, mass and anaemia are major presentation.

Preliminary Investigations:

Investigations	Total	Male	Female
Hb (gm%)	29	21	8
Elevated LFT	7	6	1
CXR	-	-	-

Among preliminary investigations Hb was reduced in 29 patients among which 21 are males and 8 are females.

Among liver function test deranged in 7 patients among which 6 are males and 1 female

DURATION OF SYMPTOMS:

	Total	<1 month	1-3 months	3-6 months	>6 months
Abdominal pain	28	20	4	4	-
Nausea/vomiting	29	6	6	14	3
Dysphagia	12	-	4	6	2
Weight loss	31	-	-	6	25

Among symptoms, abdominal pain presents maximally in less than 1 month duration, vomiting presents maximally in 3-6 month.

Dysphagia maximally in 3-6 months and weight loss commonly seen in age group >6 month.

MACROSCOPY:

TABLE 11.BORRMANNS CLASSIFICATION

BORRMANN	TOTAL		MALE (24)		FEMALE (12)	
TYPE	CASES	%	CASES	%	CASES	%
I	10	27.7	7	29.16	3	25
II	20	55.5	11	45.83	9	75
III	4	11.1	4	16.66	0	0
IV	2	5.5	0	0	2	16.66

Predominant macroscopic type was Borrmann type II followed by type I and type III.

In both males and females predominant type was type II

Females had higher percentage of advanced lesion.

STAGING:

Classified on the basis of AJCC Staging, the patient are categorized on this basis.

TABLE 12.STAGING

STAGE	TOTAL NO OF CASES	%
Ia	_	_
Ib	_	-
II	23	63.88
III a	5	19.44
III b	_	_
IV	8	16.66

About 63% of patients in this study are presented with stage II disease and about 16% are presented with stage IV disease.

STAGING AND ITS ASSOCIATION WITH SITE:

TABLE 13.STAGE IN RELATION TO SITE

	STAGE Ia	STAGE II b	STAGE II	STAGE IIIa	STAGE IIIb	STAGE IV
OG junction	_	_	1	2(66%)	-	
Cardia and fundus	_	_	3	0	-	
Body	_	_	3	3	-	2
Antrum	_	_	16(76%)	0	-	5
Linitis plastic	_	_	_			1

In this study, 63% of cases present with stage II disease.

In OG junction tumours 66% presents with stage III disease.

In antral disease 76% presents with stage II disease.

NODAL STAGING

STAGING	NO OF PATIENTS	%
NO	26	72.2
NI	6	16.6
N2	4	11.11
N3	_	_

METASTASIS

STAGING	NO OF PATIENTS	%
M0	28	77.77
M1	8	22.22

About 72% of patients present with NO status.

16% presents with N1 status and about 66.66 % presents with metastasis.

SURGICAL TREATMENT:

TABLE 16.SURGICAL MODALITIES

Total gastrectomy	6	16.66 %
Subtotal gastrectomy with Roux en y anastomosis	15	41.66 %
Palliative anterior gastro jejunostomy	6	16.66 %
Feeding jejunostomy	8	22.22 %
Lap sleeve gastrectomy	1	27 %

Total gastrectomy was done in 16.66% patients.

Subtotal gastrectomy was done in 41% patients

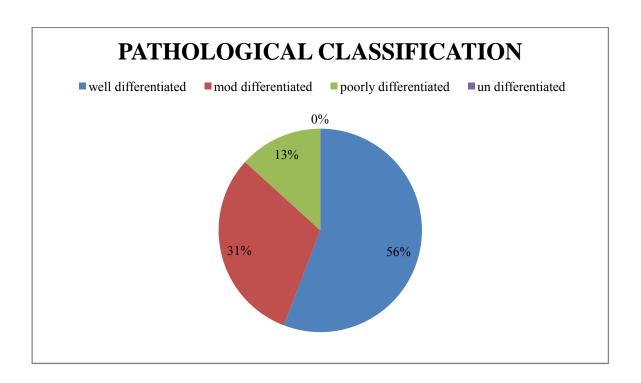
Palliative procedures such as anterior gastro jejunostomy done in 16%.

22% undergone feeding jejunostomy.

PATHOLOGY:

TABLE 17.PATHOLOGY

ТҮРЕ	TO	TOTAL MALE FEMA		MALE		EMALE
	CASES	%	CASES	%	CASES	%
Well differentiated adenocarcinoma	19	52.7	14	58.3	5	41.6
Moderately differentiated adeno carcinoma	11	30.5	7	29.16	4	33.3
Poorly differentiated adenocarcinoma	6	16.6	3	12.5	3	25
Undifferentiated adeno carcinoma	0	0	_	_	_	_



Majority are well differentiated adenocarcinoma with 52.7%.

In both males and females majority are well differentiated adenocarcinoma.

PATHOLOGY IN TUMOUR SITE:

TABLE 18.PATHOLGY IN RELATION TO TUMOUR SITE

DIFFEREN TIATION	TOTAL	ANTRUM	BODY	PROXIMAL	OG JUNCTION
Well	19	10	5	2	2
Moderately	11	7	2	1	_
Poorly	6	4	1	_	1
Undifferentiated	_	_	_	_	_

FOLLOW UP:

All patients are followed up after surgery patients of stage II disease are advised chemotherapy.

	FOLLOW UP	6 MONTHS	DEFAULT
STAGE II	23	10	13
STAGE III	5	1	4

OBSERVATION

Stomach cancer is increasing with increase in age maximum between the age group of 55-64 yrs.

Male has more preponderence than the females with the ratio of 2:1.

Incidence of Distal tumours is increasing than the proximal tumours.

Proximal tumours are increasing in associated with smoking.

Antral group of tumours predominantly presents with nausea, vomiting, abdominal pain and weight loss.

Proximal tumours commonly presents with anaemia seen in all the cases.

Female patients commonly presents with advanced stage of disease.

Majority of patients presents with stage II disease.

OG junction tumours commonly presents with stage III disease.

CONCLUSION

There is a increase in incidence of carcinoma stomach with increasing in age.

There is increase in incidence in males than the females.

Distal tumours continue to be more common than the proximal tumours.

Endoscopy is very useful modality in diagnosing carcinoma stomach even in early stages.

Most patients are presents in the advanced stage of disease indicating there is lack of proper screening for the disease.

Curative subtotal gastrectomy or distal gastrectomy is associated with good quality of life if the tumour is operable.

Preventive strategies offers best opportunity for control of disease such as Antioxidants, H.pylori therapy.

ANNEXURES

A CLINICAL STUDY OF GASTRIC CARCINOMA

PROFORMA

Case No:			
Name: Aş		Age:	Sex:
Occupation:		IP No	:
Symptoms:			
a. Abdominal Pain:		b. Nausea/ Vomitin	g
c. Weight loss:		d. Anorexia:	
e. Early satiety:		f. Jaundice:	
g. Dysphagia:		h. Malena:	
i. Other symptoms:			
Past History : Price	or gastric surgery		
Family History:			
Personal History:			
Tobacco use/Pan cl	hewing:		
Alcohol:			
GPE:			
Built :	Nourishment	: Hydrat	ion:
Pallor:	Icterus:	Lympl	nadenopathy:
Vitals: BP	PR:		

SYSTEMIC EXAMINATION:

P/A

INSPECTION:				
a. Distention/fullness:				
b. Dilated veins:				
c. Visible mass:				
d. Visible gastric peristalsis:				
e. Umblicus:				
f. Movement of abdominal quadrants:				
PALPATION:				
Mass				
a. site				
b. size				
c. shape				
d. surface				
e. borders				
f. consistency				
g. mobility				
h. movement with respiration				
i. succusion splash:				
j. organomegaly				
k. left supra clavicular nodes				

Over the ma	ss			
Liver span				
Shifting dullness				
AUSCULTATION				
P/R				
Other system Examination:				
RS :				
cvs :				
CNS :				
Investigations				
Routine :	Hemoglobin-	ESR-		
	Total count-	Differential count-		
	CT- BT-			
	Blood urea:			
Serum creatinine:				
	Random blood sugar:			
	Liver function tests:			
	Serum electrolytes:			
CEA:				
	Chest X –Ray:			

PERCUSSION:

Special investigations:			
Upper GI Endoscopy	· :		
USG Abdomen	:		
CT Scan Abomen	:		
Biopsy	:		
Treatment:			
Surgery	:		
Operative procedure	:		
Findings	:		
Post operative period	. :		
Examination of the sp	pecimen:		
Site of growth: pylor	rus/Antrum/Body/Fundus/Cardiooesophageal junction		
Type of growth: Polypoidal/Fungating			
U	lcerative with elevated/depressed edges		
D	iffuse/ Infiltrated		
U	nclassified		
Histopathology:			
Chemotherapy:			

ABBREVIATIONS

AP ABDOMINAL PAIN GJ **GASTROJEJUNOSTOMY** WL WEIGHT LOSS JJ JEJUNO JEJUNOSTOMY ES **EARLY SATIETY** FJ FEEDING JEJUNOSTOMY RY ROUX EN Y OJ OESOPHAGO JEJUNOSTOMY SER SEROSA INVOLVEMENT WD WELL DIFFERENTIATED MODERATELY DIFFERENTIATED MD PD POORLY DIFFERENTIATED GC **GREATER CURVATURE** V **VOMITING** LC LESSER CURVATURE DY **DYSPHAGIA**

FF

FREEFLUID

ANO ANOREXIA

STD SOFT TISSUE DENSITY

A ALCOHOL

PI PANCREATIC INVOLVEMENT

S SMOKING

ULC ULCERATIVE

DE DISTAL ESOPHAGUS

MAS MASS

PS PROXIMAL STOMACH

AN ANTRUM

BO BODY

M MALENA

J JAUNDICE

PYL PYLORES

PG PERIGASTRIC

PAN PANCREAS

ED EDEMATOUS

IRR IRREGULAR

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PATIENT CONSENT FORM

Study Title: Clinical Study of Carcinoma Stomach in patients presenting

in GMKMCH Salem.

Study Centre: Department Of General Surgery, GMKMCH Salem

Participant Name:

Age:

Sex:

I.P. No:

I confirm that I have understood the purpose of surgical procedure for

the above study. I have the opportunity to ask the question and all my questions

and doubts have been answered to my satisfaction.

I have been explained about the possible complications that may occur

during surgical and post-surgical procedure. I understand that my participation

in the study is voluntary and that I am free to withdraw at any time without

giving any reason.

I understand that investigator, regulatory authorities and the ethics

committee will not need my permission to look at my health records both in

respect to the current study and any further research that may be conducted in

relation to it, even if I withdraw from the study. I understand that my identity

will not be revealed in any information released to third parties or published,

unless as required under the law. I agree not to restrict the use of any data or

results that arise from the study.

I hereby consent to participate in this study for various surgical

procedures and their outcomes.

Time:

Date:

Signature / thumb impression of patient

Place:

Patient's Name:

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