

**“A CROSS SECTIONAL STUDY TO ASSESS THE VARIOUS
UPPER GASTROINTESTINAL ENDOSCOPIC FINDINGS IN
DYSPEPSIA PATIENTS ATTENDING TERTIARY LEVEL CARE
HOSPITAL”**

Dissertation submitted to

THE TAMILNADU

DR. M.G.R. MEDICAL UNIVERSITY

In partial fulfilment of the regulations for the award of the degree of

M.S. (GENERAL SURGERY) BRANCH – I



DEPARTMENT OF GENERAL SURGERY CHENGALPATTU

MEDICAL COLLEGE, CHENGALPATTU - 603001.

MAY – 2020

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MAY – 2020

DECLARATION

I, **Dr. S. SANTHOSH BABU**, solemnly declare that the dissertation titled “**A CROSS SECTIONAL STUDY TO ASSESS THE VARIOUS UPPER GASTROINTESTINAL ENDOSCOPIC FINDINGS IN DYSPEPSIA PATIENTS ATTENDING TERTIARY LEVEL CARE HOSPITAL**” is a bonafide work done by me at Chengalpattu Medical College, Chengalpattu during **2018 to 2019** under the guidance and supervision of **Prof. Dr. J. SELVARAJ, M.S**, Department of General surgery, Chengalpattu Medical College, Chengalpattu.

This dissertation is submitted to Tamilnadu Dr. M.G.R Medical University towards partial fulfilment of requirement for the award of **M.S. degree in General surgery (Branch -I)**.

I have not submitted this dissertation on any previous occasion to any University for the award of any degree.

Place: Chengalpattu

Dr. S. SANTHOSH BABU

Date:

Reg No: 221711261

CERTIFICATE

This is to certify that this dissertation entitled “**A CROSS SECTIONAL STUDY TO ASSESS THE VARIOUS UPPER GASTROINTESTINAL ENDOSCOPIC FINDINGS IN DYSPEPSIA PATIENTS ATTENDING TERTIARY LEVEL CARE HOSPITAL**” is the bonafide original work of **Dr. S. SANTHOSH BABU** in partial fulfilment of the requirements for M.S. Branch-I (General surgery) Examination of the Tamilnadu Dr. M.G.R. Medical University to be held in MAY – 2020, under the guidance of **Prof. Dr. J. Selvaraj M.S**, done during the period of 2018 to 2019.

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CERTIFICATE FROM THE GUIDE

This is to certify that the dissertation entitled “**A CROSS SECTIONAL STUDY TO ASSESS THE VARIOUS UPPER GASTROINTESTINAL ENDOSCOPIC FINDINGS IN DYSPEPSIA PATIENTS ATTENDING TERTIARY LEVEL CARE HOSPITAL**” submitted by the candidate **Dr. S. SANTHOSH BABU** is a record of original and bonafide work done by him under my guidance and supervision in the Department of General surgery, Chengalpattu Medical College, Chengalpattu during the tenure of his course in M.S. General Surgery from 2018 to 2019, submitted in partial fulfillment of the requirements for the award of M.S. degree in General surgery by The Tamilnadu Dr. MGR Medical University, Chennai-32.

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25

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TABLE OF CONTENTS

SL.NO	CONTENTS	PAGE NO.
1.	INTRODUCTION	1
2.	AIMS AND OBJECTIVES	2
3.	MATERIALS AND METHODS	3
4.	REVIEW OF LITERATURE	5
5.	OBSERVATIONS	60
6.	DISCUSSION	73
7.	CONCLUSION	75
8.	BIBLIOGRAPHY	77
9.	ANNEXURES	81
	1. Proforma	81
	2. Consent Form	83
	3. Master Chart	91

INTRODUCTION

Dyspepsia is constellation of upper abdominal symptoms that affect approximately a fourth of the population in industrialized countries. The Rome III criteria defined dyspepsia as 1 or more of the symptoms such as Postprandial fullness, Early satiety, Epigastric pain or burning. The evaluation and management of dyspepsia constitutes a significant clinical and economical burden.

Upper G.I. endoscopy is standard for diagnosis of structural disease in a patient with dyspepsia & is the investigation of choice for dyspepsia particularly where radiology has been negative. Advantage of negative endoscopy reduces patient anxiety & increases patient satisfaction. Initial endoscopy arm showed significant improvement in symptoms score, quality of life, reduction in use of PPI's. Endoscopy helps in early detection of carcinoma in dyspepsia.

Patient with alarm symptoms add significant increase in both GI cancer and mortality. Endoscopy helps in detection of cut-off age for carcinoma of upper GI tract in dyspepsia and outcome of various other alarming symptoms. So by early detection and treatment the outcome of patient may be better. This study is intended to study the various upper GI endoscopy findings in dyspeptic patients and association of other alarm symptoms with better outcome.

AIMS & OBJECTIVES OF THE STUDY:

Primary :

- To study the upper gastrointestinal endoscopic presentation of chronic dyspepsia.

Secondary :

- To study the age and sex distribution in patients presenting with chronic dyspepsia.
- To find out the common site of lesion in patients presenting with chronic dyspepsia.

MATERIALS AND METHODS

1. Study design: Cross sectional study
2. Period of study: One Year (2018 -2019)
3. Study population: Patients attending surgical outpatient department of Chengalpattu medical college with dyspeptic symptoms.
4. Sampling method: convenient sampling

5. Sample size:

Sample size was measured by using Z^2pq/d^2 .

Z-statistic 95% confidence interval,

Table value: 1.96

$p = 50\%$, $q = 100-p=50\%$,

confidence interval of 95%,and 20% relative precision. The calculated

minimum sample size is 96

So final sample size = 100

INCLUSION AND EXCLUSION CRITERIA INCLUSION CRITERIA:

1. Patients showing symptoms of dyspepsia for minimum of three months.
2. Patients who are willing to participate in the study

EXCLUSION CRITERIA:

1. Pregnant and lactating women.
2. Patients who are known cases of chronic pancreatitis and liver disease.
3. Age less than 18 years
4. Unwilling or unfit patients for endoscopy.

REVIEW OF LITERATURE

Epidemiology is the study of the distribution and determinants of disease and damage in human populations. Two phases, one descriptive ascertaining population with high and low incidence of known disease and other analytical which determines the reason for imbalanced distribution. In the case of dyspepsia, the hope is epidemiological research will lead to cause of the condition. The dyspepsia is the clinical syndrome will have different causative factors in different persons. The reasons for a particular person in same group developing dyspepsia are probably complex and will comprise genetic, environmental and psychological factors.

Major changes have happened in the tests and procedures for the evaluation of dyspepsia patients in the past 4 decades. With barium meal examination, it was very difficult to examine the duodenum and to discriminate between active ulceration and healed one. With the advantage of upper gastrointestinal endoscope it is possible to diagnose disease accurately in acute and scarring in stomach and duodenum. Environment reasons play important role in pathology of peptic ulcer and its inconsistent frequency.

Herring J et al. Studied 60 patients with dyspepsia by endoscopy. In those 60 patients, 70% were males while 30% were females. 82% of patients were in the age group of 30-50 years. The most common symptoms were epigastric pain then heartburn followed by flatulence. The findings in

oesophago duodenoscopy were normal in 50% patients. Also esophagogastritis, esophagogastrroduodenitis and carcinoma stomach were present in 2% of patients each. Histopathological examination done for all the atypical findings and confirmed. The conclusion of the study was that in most of the patients with dyspepsia, the endoscopic findings were found to be normal. The findings corroborated with the biopsy results.

Chadwick P et al. Studied 342 dyspeptic patients. Only 19% of patients in study found to be having significant findings in endoscopy. The clinical symptoms elicited in the history including the symptoms and signs did not correlate with the endoscopic findings. In 23% patients, biopsy proved the presence of H. pylori infection. The infection too did not correlate well with the significant findings in endoscopy. So they came to the conclusion that patients with dyspepsia had only few significant findings in endoscopy.

The presence of these lesions could not be reliably predicted using clinical data and H. pylori infection status. Finally the empirical anti ulcer treatment advised as the initial therapy before consideration of endoscopy in majority of patients.

Thomson A B R et al. did endoscopy in 1040 patients presented with dyspepsia within 10 days of referral. Clinically significant findings were made out in 58% of patients. Esophagitis was the most common finding (43%) and peptic ulcer was found to be the least (5.3%). Many patients had minimum 3 dyspepsia symptoms, almost 80% had six symptoms, and 50% had more than

8 symptoms. The patient's principal symptom did not correlate with the endoscopic findings. In patients with reflux type dyspepsia, the frequent finding was esophagitis. Conclusion is that the symptom did not substantiate the nature and clinically significant findings. The most common finding was found to be esophagitis.

Delaney et al. studied the cost-effectiveness of an earlier endoscopy weighed with routine management in dyspeptic patients, who were more than 50 years of age. If the cost of upper gastrointestinal endoscopy is low then it was found to be cost effective.

According to Hewson EG et al; the patients who presented with chest pain like angina had GERD. The hint shows that the pain is esophageal origin rather than cardiac were

Accompanied esophageal symptoms

Pain aggravated by food and in supine posture

Pain that lasted for days without cardiac deterioration

Pain relieved by antacids.

According to Singh S et al it was tough to distinguish between coronary angina and esophageal problem, because many had associated problems.

According to Brzana RJ and small PK non specific upper gastrointestinal symptoms like dyspepsia, nausea, bloating and indigestion may be present in patients with GERD. According to Fisher MJ et al singultus and hiccups could

be symptoms of GERD. Some may have water brash, filling of mouth by clear and salty fluid and this is attributed to secondary hypersalivation due to acid reflux. According to Mays EE et al pulmonary conditions particularly asthma associated with GERD.

In United States of America about four million people have peptic ulcer disease either duodenal or gastric and about 3,00,000 fresh cases are confirmed every year. American males have life time risk for developing ulcer disease is around 10% and females have 4%. With the complete review of literature and studies the patients who had dyspepsia are given reassurance by doing endoscopy and some required drug therapy, however the time limit for the reassurance is not very sure.

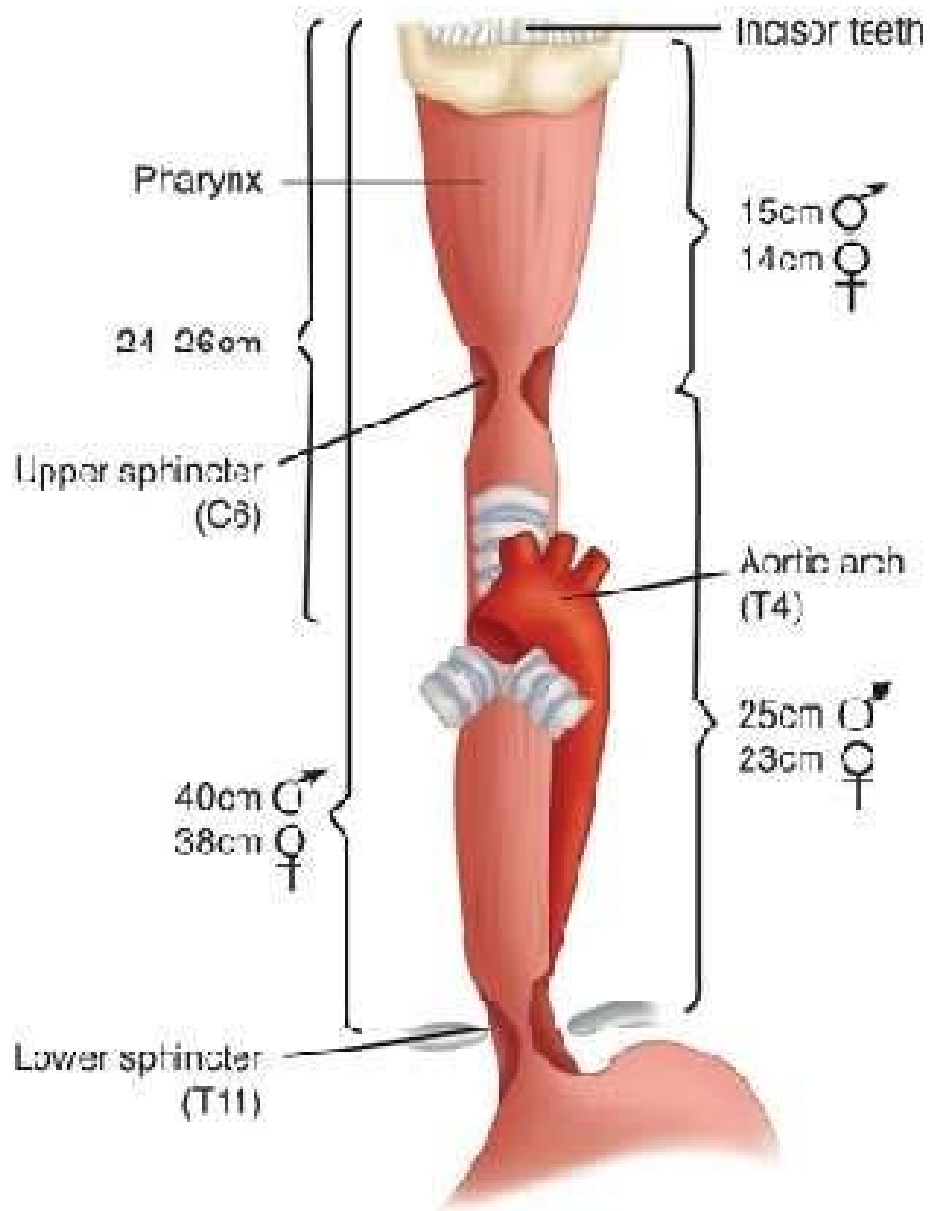
ANATOMY

ESOPHAGUS:

The esophagus, a soft muscular tube, allows food to pass between the pharynx and the stomach. It is about 25-30cm in length. The esophagus is a midline structure anterior to the spine and posterior to the trachea. From its origin at the cricoid cartilage in the neck opposite the fifth to sixth cervical vertebra, it passes into the thorax at the level of the sternal notch and travels caudally within the chest in the posterior mediastinum. It terminates in the abdomen at the esophagogastric junction opposite the twelfth thoracic vertebra. The esophageal hiatus of the diaphragm is at the level of the tenth thoracic vertebra.

Anatomically esophagus is divided into three parts:

- Cervical
- Thoracic
- Abdominal
- According to differing forms of motility (functionally) esophagus is divided into three zones:
 - Upper esophageal sphincter (UES)
 - Esophageal body
 - Lower esophageal sphincter (LES).



UPPER ESOPHAGEAL SPHINCTER (UES).

The high-pressure zone at the inlet of the esophagus is considered as UES. Anatomically it marks the end of a complex configuration of muscles that begin in the larynx and posterior pharynx and end in the neck. The pharyngeal constrictor muscles are three consecutive muscles that begin at the base of the palate and end at the crest of the esophagus. The superior and middle pharyngeal constrictor muscles, as well as the oblique, transverse, and posterior cricoarytenoid muscles, are immediately proximal to the UES and serve to anchor the pharynx and the larynx to structures in the mouth and palate. These muscles also aid in deglutition and speech, but are not responsible for the high pressures noted in the UES. The inferior pharyngeal constrictor muscle is the final bridge between the pharyngeal and esophageal musculature.

ESOPHAGEAL LAYERS:

The esophagus is comprised of two proper layers: the mucosa and the muscularis propria. It is distinguished from the other layers of the alimentary tract by its lack of a serosa. The mucosa is the innermost layer and consists of squamous epithelium for most of its course. The distal 1 to 2cm of esophageal mucosa transitions to cardiac mucosa or junctional columnar epithelium at a point known as the Z-line.

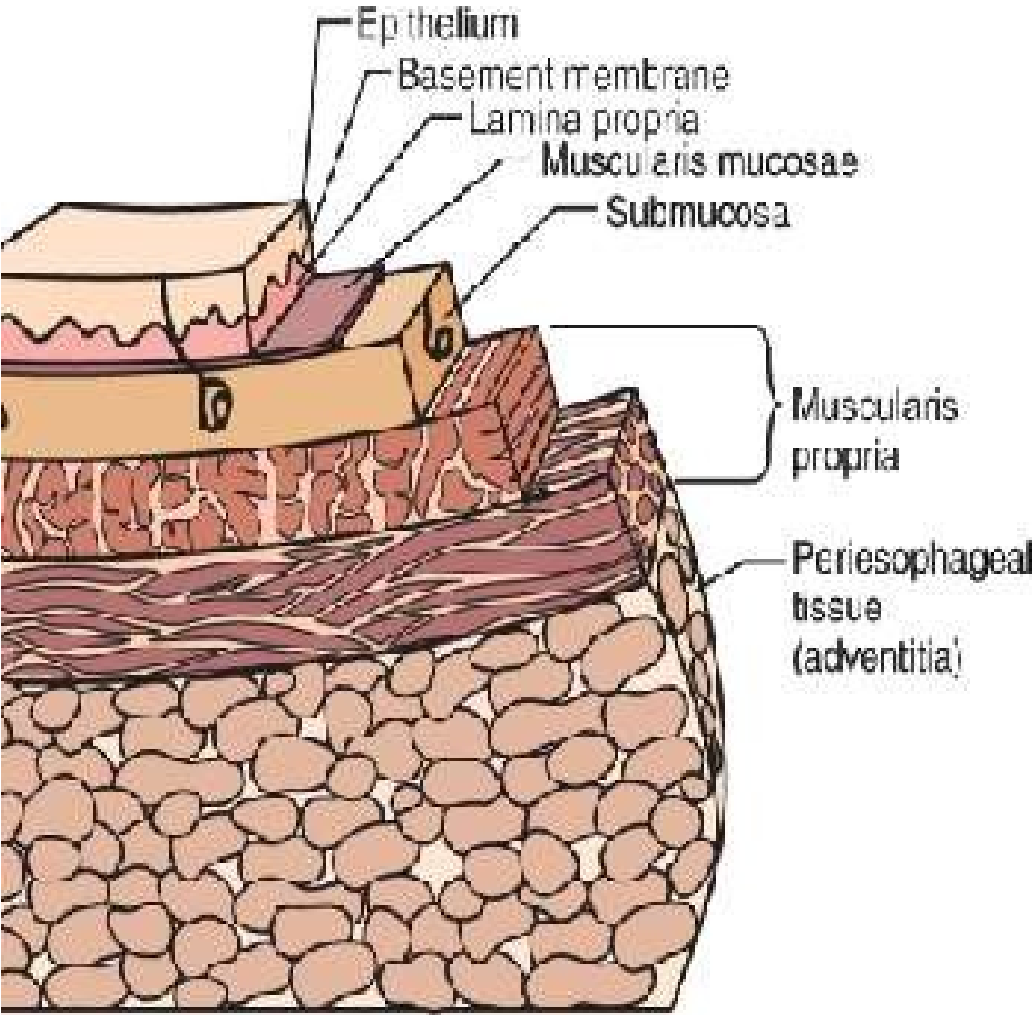
Within the mucosa, there are four distinct layers

1. Epithelium
2. Basement membrane
3. Lamina propria, and
4. Muscularis mucosae.

HISTOLOGY:

Enveloping the mucosa, directly abutting the submucosa, is the muscularis propria. Below the cricopharyngeus muscle, the esophagus is composed of two concentric muscle bundles: an inner circular and outer longitudinal. Both layers of the upper third of the esophagus are striated, whereas the layers of the lower two thirds are smooth muscle. The circular muscles are an extension of the cricopharyngeus muscle and traverse through the thoracic cavity into the abdomen, where they become the middle circular muscles of the lesser curvature of the stomach. The collar of Helvetius marks the transition of the circular muscles of the esophagus to oblique muscles of the stomach at the incisura (cardiac notch). Between the layers of esophageal muscle is a thin septum comprising connective tissue, blood vessels and an interconnected network of ganglia known as Auerbach's plexus. Enshrouding the inner circular layer, the longitudinal muscles of the esophagus begin at the cricoid cartilage and extend into the abdomen, where they join the longitudinal

musculature of the cardia of the stomach. The esophagus is then wrapped by a layer of fibroalveolar adventitia.



ESOPHAGEAL CONSTRICTIONS:

The esophageal silhouette resembles an hourglass. There are three distinct areas of narrowing that contribute to its shape. Measuring 14 mm in diameter, the cricopharyngeus muscle is the narrowest point of the gastrointestinal tract and marks the superior-most portion of the hourglass-shaped esophagus. Occurring just below the carina, where the left mainstem bronchus and aorta arch over the esophagus, the bronchoaortic constriction at the level of the 4th thoracic vertebra creates the center narrowing and measures 15 to 17 mm. Finally, the diaphragmatic constriction, measuring 16 to 19 mm, marks the inferior portion of the hourglass and occurs where the esophagus passes through the diaphragm. Between these three distinct areas of anatomic constriction are two areas of dilation known as the superior and inferior dilations. Within these areas, the esophagus resumes the normal diameter for an adult and measures about 2.5 cm.

LOWER ESOPHAGEAL SPHINCTER (LES)

The final phase of esophageal bolus transit occurs through the LES. Although this is not a true sphincter, there is a distinct high-pressure zone that measures 2 to 5 cm in length and generates a resting pressure of 6 to 26 mm Hg. The LES is located both in the chest and the abdomen. A minimum total length of 2 cm, with at least 1 cm of intra-abdominal length, is required for normal LES function. The transition from the intrathoracic to the intraabdominal sphincter is noted on a manometric tracing and known as the respiratory inversion point (RIP).

At this point, the pressure of the esophagus changes from negative to positive with inspiration and positive to negative with expiration. Peristaltic contractions alone do not generate enough force to open up the LES. Vagal-mediated relaxation of the LES occurs 1.5 to 2.5 seconds after pharyngeal swallowing and lasts 4 to 6 seconds. This flawlessly timed relaxation is needed to allow efficient transport of a food bolus out of the esophagus and into the stomach. A post-relaxation contraction of the LES occurs after the peristaltic wave has passed through the esophagus, allowing the LES to return to its baseline pressure, re-establishing a barrier to reflux.

STOMACH

Stomach is the most dilated part of the alimentary tract, extending from the cardiac end to the pyloric end. The stomach is sub-divided into;

1. Fundus
2. Body
3. Pyloric Antrum
4. Pyloric Canal

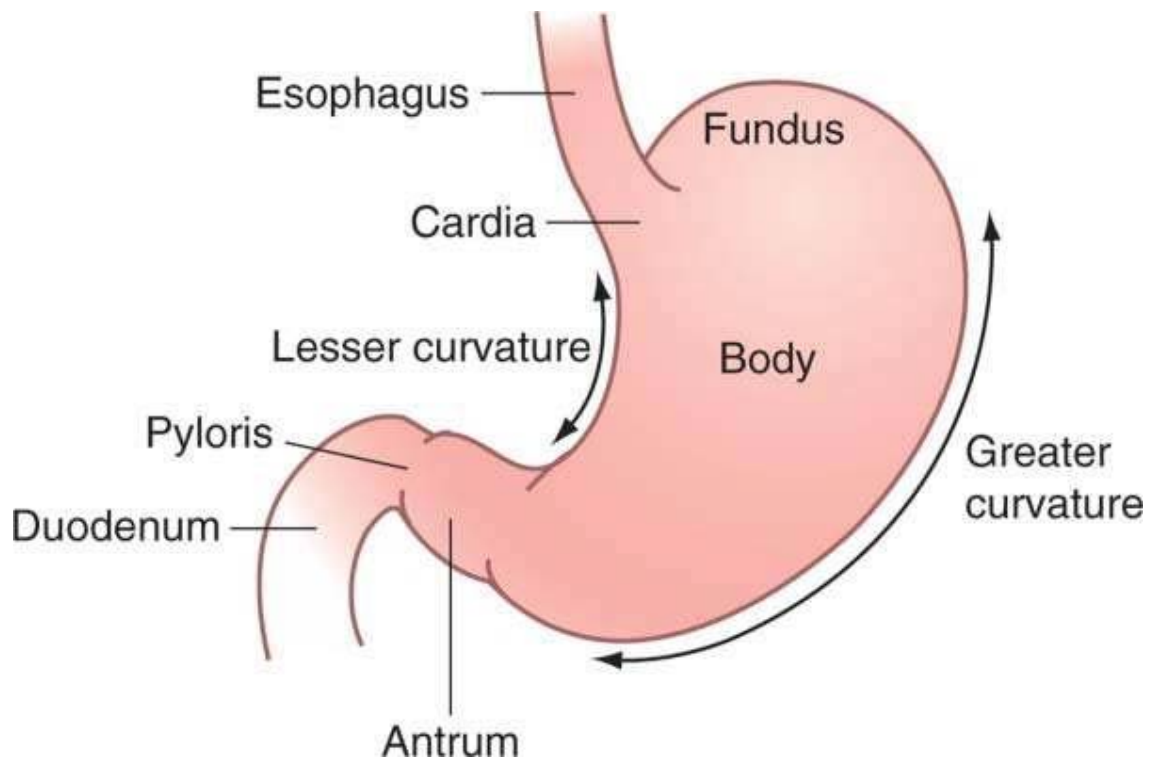
Fundus is the part which rises above the level of cardiac end of the stomach.

Body is that portion situated between the fundus and the level of incisura angularis in the lesser curvature of the stomach.

The pyloric part is situated below the body and consists of:

1. Pyloric antrum
2. Pyloric canal

It is in the pyloric antrum where *Helicobacter pylori* is most frequently colonized.



(FIGURE : 1)

Anatomy of stomach

Stomach wall has four basic layers:

1. Mucous membrane
2. Sub mucosa
3. Muscular layer
4. Serosa

HISTOLOGY

Damage to mucus layer exposes the stomach to gastric acid and active gastric enzymes and this is the basis of “Leaking Roof” hypothesis in the aetiology of peptic ulcers.

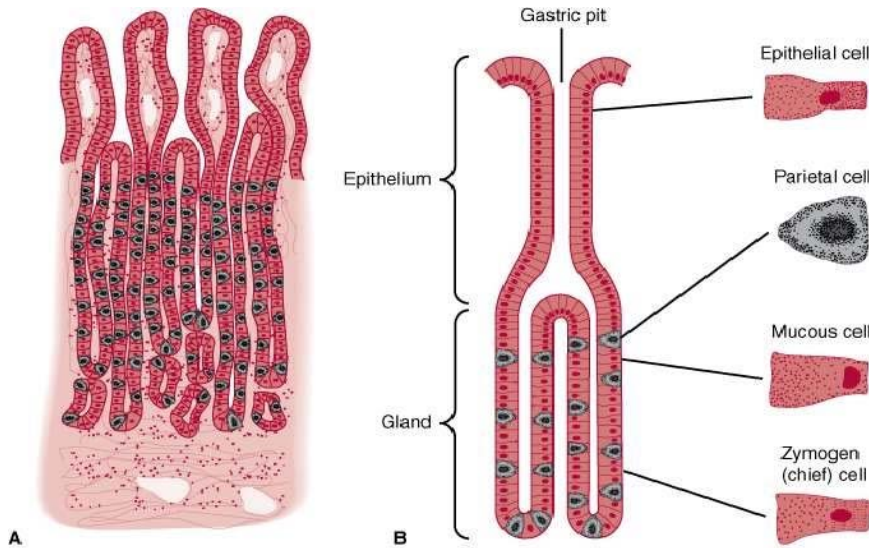
Gastric glands are three types:

- 1) Cardiac glands
- 2) Main gastric glands
- 3) Pyloric glands

Different cells in gastric glands

1. Cardiac glands:

These are either simple tubular or tubulo-alveolar type confined to small area near the opening of oesophagus. They contain mainly mucus secreting cells.



(FIGURE : 2)

Main gastric glands:

They are present in the fundus and body of the stomach and they open into Gastric pits. They contain the following cells.

- a.** Chief cells: They are numerous in the basal parts of the glands. They secrete digestive enzymes like pepsin.
- b.** Parietal cells (Oxyntic cells): They are numerous in the upper part of the gland. They are responsible for the secretion of hydrochloric acid and intrinsic factor.
- c.** Mucous neck cells: They are present near the upper end of the gland and secrete mucous. Their secretions are different from that of the surface mucous cells.
- d.** Endocrine cells: These include somatostatin secreting D-cells and histamine secreting enterochromaffin-like cells. These are scattered throughout the glands.
- e.** Gastrin secreting cells (G-cells): Although small in number, they play a vital physiological role. They occur either singly or in small clusters in the mid to deep sections of antral glands. They contain basilar cytoplasm densely packed with gastrin containing secretory granules. The apical or luminal surface of the G-cells is narrowed into small microvilli, which are thought to contain the receptors responsible for the amino acid and peptide stimulation for gastrin release.

- f. Undifferentiated cells: These are cells whose functions are not exactly known hence termed as undifferentiated cells.

3. Pyloric glands:

These are present in the antrum and pylorus. These extensively coiled glands are composed of endocrine, mucous and parietal cells. Mucous cells predominate in these glands.

PHYSIOLOGY

The gastric glands secrete about 2.5litres of gastric juice daily. The juice contains cations: Na^+ , K^+ , Mg^{2+} , H^+ Anions Cl^- , HPO_4^- , SO_4^- , Pepsins, Lipase, Mucus and Intrinsic factor. Parietal cells of gastric glands secrete hydrochloric acid and intrinsic factor.

Hydrochloric acid provides necessary pH for pepsin to start digestion of protein and also stimulates the secretion of bile and pancreatic juice. The mucosa of stomach is protected by various factors which includes bicarbonate ions secreted by surface mucous cells, surface mucus, mucosal blood flow, epithelial regenerative capacity and elaboration of prostaglandin.

The mucosal protection reinforced by surface cell's membrane potential and tight junctions stop the back diffusion of hydrogen ions and thereby protecting the epithelial damage. *Helicobacter pylori* colonizes the mucus layer of the stomach which provides the ecological niche in the antrum, which is conducive for its habitations.

The breakdown of mucus layer and damage to surface epithelial cells are the basis of “Leaking roof” hypothesis of the pathogenesis of *Helicobacter pylori*.

Regulation of gastric secretion:

Gastric motility and secretion are regulated by neural and hormonal mechanisms

a) The neural component: It comprises of;

1. Local autonomic reflexes involving cholinergic neurons.
2. Impulses from the CNS by the way of Vagus nerves.

b) The hormonal component: It involves various gastro intestinal hormones like gastrin, cholecystokinin and secretin.

Secretion of gastric juice has three interconnected phases:

1. Cephalic phase
2. Gastric phase
3. Intestinal phase

Cephalic phase:

Cephalic phase of gastric acid secretion acts by stimulating the vagal centre via the hypothalamus. Parietal and Chief cells are affected by direct cholinergic stimulation.

Gastric phase:

It starts by food entering and distending the stomach. Local and vasovagal distention reflexes stimulate the acid secretion of the stomach. Gastrin is released from the specialised „G“ cells of the antrum of stomach in response to food in the stomach and gastric distention. Gastrin then stimulates the acid secretion by the parietal cells in the body of the stomach.

Intestinal phase:

Gastric secretion is stimulated by food and its digestive products in the intestine. This may be due to stimulation of neuro-receptors and release of intestinal gastrin. In contrast acidification of the duodenum and the antrum results in inhibition of further acid secretion. This may be due to vagal inhibition or release of secretin or CCK-PZ (cholecystokininpancreozymin).

HISTORY & DEVELOPMENT OF ENDOSCOPY

In 19th Century, attempts were made to examine the interior of the upper GIT by reflecting light in to the body cavities through a hollow cylinder, but not possible until Thomas Edison's invention of the incandescent light bulb that it became possible in the late 1870's to perform rigid endoscopy. Progressively smaller lamps were developed that allowed insertion into the stomach through rigid endoscopes, but the nature of the light made it impossible to perform long and complex studies due to overheating of instruments.

In addition the difficulty in passing rigid instruments to the curvatures of the bowel permitted only limited examination of the upper GI tract. These procedures were mostly performed by surgeons, such as the 19th Century Polish surgeon Johann Von Mikulicz-Radecki.

The era of flexible endoscopy began with the introduction of the semirigid gastroscope by R.Schindler in 1936 by collaboration with the German physician Georg Wolf. The way to the development of a flexible fiberscope was initiated by Baird's demonstration in 1928 that light and images could be transmitted through a single glass or quartz fiber.

In 1950's when Van Heel and H.Hopkins and N.S.Kapany working independently, developed usable flexible glass fiber bundles that could transmit light across relatively longer distance and into the body cavities. The next phase of development took place in Ann Arbor at the University of

Michigan, Physicians H.M.Pollard and Basil Hirschowitz , C.Wilbur Peters in collaboration with Physics students Lawrence Curtis, designed the first completely flexible endoscope.

Hirschowitz and Curtis started working on this concept in 1955 by developing an instrument composed of a bundle of individual glass fibres that was in theoretical capable of transmitting light as well as images. They encountered numerous problems such as fiber “Crosstalk”, which differed the light, making interpretation of the images impossible. This led to the invention of a glass coating for the fibres for insulation and to the development of fiber scope.

The first controllable tip gastroscope was developed in 1962 was applied clinically first and then found an industrial application in the examination of jet engines. After trying the flexible gastroscope on himself, Hirschowitz first used it in a patient with a bleeding duodenal ulcer in February 1957. The diagnosis was successfully established, and the patient underwent operation based on Hirschowitz observations.

The first commercial fiberoptic endoscope made by American cystoscope makers Inc, Norwalk, CT. was first used in 1961 and the results were published in the Lancet in that year of may month. Once the development and wide spread use of fiberoptic upper GI endoscope became a routine practice, the therapeutic potential was established. Experimental studies, such

as those by W.D.Blackwood, S.Silivis, J.P.Papp, C.Sugawa and others demonstrated the feasibility and safety of endoscopic haemostasis. This has created the way for the use of endoscopes as vehicle for numerous accessories so that today endoscopic surgery includes methods of haemostasis, excision, ablation, dilatation, decompression, sclerosis and foreign body removal.

INSTRUMENTATION

Flexible endoscope come in a different variety of diameter and lengths, either direct- viewing or video. The primary endoscope used for upper GI endoscopy is a forward viewing zero-degree endoscope, whereas duodenoscope visualizes the GI tract at 90° to the shaft. Side viewing endoscope is primarily used to visualise the ampulla of Vater in duodenum and may also be used in the stomach.

All endoscopes are either video or fiberoptic and all have a control head. In the fiberoptic units, an eye piece is present for either direct visualization or for video attachment. The shaft of the endoscope is flexible at the distal tip, which has deflection capabilities ranging from 90- 240 in the up/down position and 100 in right or left directions. The diameter of the insertion tube can range from 5.5mm to the distal tip to 11mm for a therapeutic endoscope. The diameter of insertion tube for duodenoscope ranges from 11.5 to 12.5mm. The controls for maneuvering the deflection tip are located on the control head with a large inner knob producing up or down deflection and the smaller outer knob

producing a left or right deflection. Two depressable buttons are located adjacent to these deflection knobs. When pressed the top button produces suction that may be necessary during the examination. The lower button serves two additional functions. Air insufflations occurs by simple placement of a finger over the button without applying pressure. When this button is depressed a small amount of water is released from the tip of the endoscope that is useful for cleaning the tip during the examination if it becomes dirty. In the video endoscope, video control buttons on the top of the control head are used to freeze an image on the video screen or to save the image for printing.

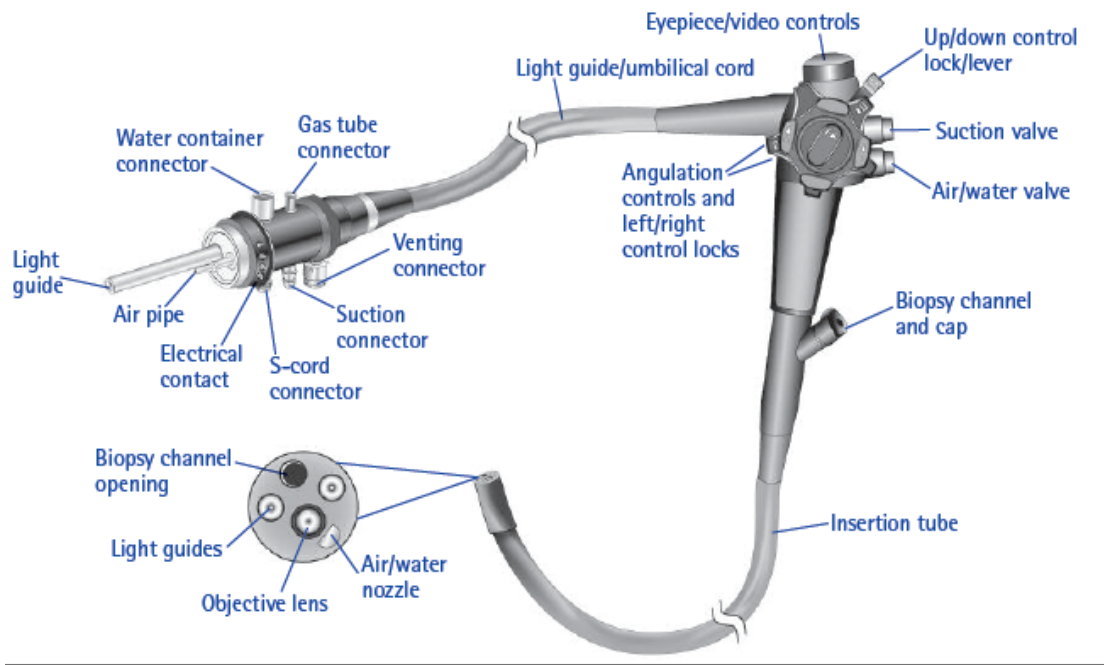
The flexible shaft is usually 110-120cms in length. This endoscope contain a working channel that varies between 2mm (paediatric endoscope) to 3.7mm (therapeutic endoscope). On the other hand the instrument channel in the duodenoscope varies from 3.2-4.2mm. Biopsy forceps, cytology brushes, or other diagnostic instruments are passed through the accessory channel. A double lumen therapeutic endoscope is also available for more

advanced therapeutic endoscopy. The flexible endoscope is connected to a light source that is either 300W Xenon arc lamp or a halogen- tungsten lamp. In addition, air and water pumps for insufflations, suction and irrigation are connected to the endoscope via the light source unit and controlled using the control buttons. If a video monitor is being used, this is also connected to the

endoscope through the light source.

Proper hand positioning and manipulation of the flexible endoscope is key to perform an efficient examination. Most endoscopists will hold the control head of the endoscope in the left hand, with the thumb on the up/down knob and the index and middle finger on the suction and air/water button. The thumb & index finger are then used to control the deflection tip during examination. The right hand of the endoscopist is used to hold the flexible shaft for insertion withdraw and rotation during the examination.

Parts of the endoscope : (FIGURE 3)



UPPER GASTROINTESTINAL ENDOSCOPY PROCEDURE:

PRINCIPLES:

Upper GI endoscopy plays a vital role in the examination of the upper GI tract. It provides both direct & complete visualization of the area and direct access for tissue sampling and/or therapeutic intervention. This should be mastered by any clinician with a special interest in diseases of the esophagus, stomach and duodenum.

PATIENT PREPARATION:

The procedure is explained to patient in simple terms. During the clinical evaluation , allergies, current medication and previous medical history are reviewed, the need for antibiotic prophylaxis is assessed. The patient should fast over night before the procedure. Out patients should be accompanied, particularly if intra venous sedation is to be used. Having a calm & relaxed patient avoids to some extent the need for sedation. A tense patient should not be submitted to endoscopy under simple topical anaesthesia. Proper sedation dictates the use of pulse oximetry and ECG.

A Lignocain gargle or spray is used for topical anaesthesia of the pharynx and hypopharynx. When needed, adequate sedation may be obtained with benzodiazepines (diazepam, midazolam). Pethedine hydrochloride may be added for relaxation and analgesia. This medication should be administered slowly in small doses until the desired level of sedation is obtained.

TECHNIQUE

INTRODUCTION OF THE ENDOSCOPE:

The patient lies in the left lateral decubitus position. Following appropriate topical anaesthesia, a mouth piece is positioned between upper and lower teeth. Endoscope is advanced, taking care to stay on the midline and at the interface between the tongue and hypopharyngeal mucosa. Tongue, uvula, epiglottis and cricoarytenoid cartilages are seen. Passing beside the midline, the cricoarytenoid cartilages are passed and the tip of the endoscope stops on the cricopharyngeus. Gentle local pressure while asking the patient to swallow allows the tip of the endoscope to pass into the cervical esophagus.

EXAMINATION OF ESOPHAGUS:

The instrument is advanced under direct vision, with the tip of the endoscope always in the center of the lumen. Using optimal insufflation keeps the lumen of the esophagus well distended. First hand inspection is important, because no trauma has caused by the manipulation or passage of the instrument.

Two rules should always be followed:

1. Endoscope must advanced with clear vision of the central lumen.
2. If direct vision is obscured or there are any doubts, the endoscope should be withdrawn.

Land marks distal to cricopharyngeal sphincter are extra luminal compression of left main bronchus, aortic arch and pulsations of left heart in the distal half. The gastro-esophageal mucosal junction is usually identified at 38-40cms from the incisors. This junction is usually serrated and readily identified by the color difference between the esophageal and gastric mucosa, called as Z line. The position of the esophageal hiatus in the diaphragm is identified by asking the patient to inhale deeply, the diaphragmatic hiatus during inspiration creates an imprint on the esophageal and gastric wall. The position of both the hiatus and the mucosal junction are recorded in order to document the possibility of a hernia or of a columnar lined esophagus.

PASSAGE IN TO THE STOMACH:

Gastro-esophageal junction should be observed for closed or widely patulous. Passage in to the gastric lumen is usually a simple manoeuvre that occurs without resistance. On entering the stomach, it becomes distended with air and this often causes discomfort to the patient. By dipping the end of the endoscope slightly down and towards the left, a view of greater curvature and of the posterior wall is obtained. Aspiration of all retained liquid is done to reduce the risk of aspiration & to allow proper examination of the stomach. A rotation movement of the tip of the instrument allows examination of the anterior and posterior walls of the body of the stomach. The lesser curvature down to the angulus and the greater curvature are viewed by the same position motion.

The most proximal part of both curvatures are better examined when using the J maneuver.

By rotating and angulating the tip, endoscope is advanced to assess the antrum. Prepyloric and pyloric ring observed directly, the passage through the pylorus being done under direct vision. When the pylorus yields, complete assessment of the first part of the duodenum is done as far as the superior duodenal angle.

While the tip of the endoscope lies along the distal lesser curvature and while the stomach is distended, rotation of the instrument is accomplished towards the greater curvature, complete 180 degree upwards angulation of the endoscope tip completes the J manoeuvre. The endoscope is pulled back while the stomach is distended, swinging of the retroflexed tip allows proper visualization of the stomach. Simultaneous rotation of the endoscope gives excellent view of the lesser curvature from the cardia to angulus. After straightening the tip endoscope is gently pulled back examining the esophagus again. Patients are encouraged to avoid drinking or eating for approximately 30mins after the procedure.

ENDOSCOPIC BIOPSY

Typical lesions routinely evaluated by biopsy are esophageal strictures, mass lesions, gastroduodenal ulcers, gastroduodenitis and polyps. Diagnostic yield increases when multiple specimens are taken of any suspicious lesion, if one suspects a malignancy, six biopsy specimens and cytology will increase

the diagnostic accuracy to better than 90-95%. Lesions arousing suspicion for being varices should not be biopsied, as this can lead to significant bleeding. The biopsy forceps is negotiated into the specified channel and after seeing the entry of forceps through the endoscope, the mouth of the forceps opened and introduced into the mucosa, closed and retrieved immediately taking the desired specimen.

Biopsy forceps containing a spike can be used to obtain multiple specimens without having to remove it from the endoscope. Biopsies for gastric ulcers should typically be taken in all four quadrants and at the base of the ulcer. The transition zone between the ulcer and surrounding mucosa is the area that most likely contains increased mitotic activity in malignant ulcers and therefore biopsy of this region improves diagnostic yield.

Biopsies of submucosal masses can have limited yield because the submucosal location is not easily reached. To increase yield several biopsies should be taken. Caution is the rule – because the area can become weakened and be at risk for perforation.

Esophageal stricture, which demonstrate dysplasia or malignant transformation should be biopsied. Polyps in the stomach or duodenum can be cancerous and should be sampled, either hot or cold biopsy forceps can remove diminutive polyps less than 5mm in diameter. Whereas a snare is best for larger polyps. The snare is placed at the base of a pedunculated polyp, and the polyp is removed in piecemeal fashion. Japanese investigators have developed

technique for lesion removal where by a suction apparatus is passed through the endoscope and lesion is grasped with suction. A snare is then placed around the base of the lesion & closed tightly and removal of the specimen is possible. If significant bleeding results, standard coagulation technique can be employed.

PROGRESS OF INTRAGASTRIC OBSERVATION THROUGH THE FIBEROSCOPE:

1. Simplification of the technique of intragastric observation based on direct vision.
2. Elimination of blind spots.
3. Regulation of various endoscopic conditions.
4. Advance in observing fine changes through close up observation.
5. Improvement of recording ability by aiming recording photography equipment.
6. Revolutionizing the technique of biopsy on direct vision through the use of fiberscope.
7. Progress in diagnosing cancer cells by viewing cells according to the direct vision method with the fiberscope.
8. Precise observation through the application of supplemental techniques, such as washing the lesion and applying a pigment solution.

DYSPEPSIA: Definition and prevalence

Dyspepsia (Dys – difficult, Pepse- digestion) is chronic or recurrent pain or discomfort in upper abdomen. Discomfort here refers to mild pain, upper abdominal fullness and early satiety. It can be accompanied by bloating, belching, nausea and heart burns. When patients have dyspeptic symptoms, but no underlying disease is found, the patient is said to have functional or idiopathic or non-ulcer dyspepsia.

Classification:

Classification of dyspepsia is based on the symptoms of the patient

Ulcer type – upper abdominal pain

Dysmotility type – unpleasant or troublesome non-painful sensation in the upper abdomen which might be associated with upper abdominal fullness, early satiety, bloatedness or nausea.

Reflux type

unclassified

40% of our general population suffers from dyspeptic symptoms, of which 5% get General practitioner consultation and 1% have their endoscopic study done.

In patients undergoing endoscopy, 40% have functional dyspepsia, 40% have GERD and 13% have some form of ulcer.

Features which suggest serious underlying diseases are:

- Age more than 55 years
- Family history of any upper GI malignancy
- Weight loss
- Upper GI bleeding
- Pain during swallowing
- Unexplained iron deficiency Anaemia
- Persistent Vomiting
- Lymphadenopathy
- Icterus

ETIOLOGY:

Etiology can be broadly classified into 2 main groups

- Structural abnormalities.
- Functional (Non ulcer) dyspepsia.

STRUCTURAL ABNORMALTIES:

- Hiatus hernia.
- Gastro-esophageal reflux disease (GERD).
- Barrett's esophagus.
- Peptic ulcer disease.
- Esophageal, gastric and duodenal cancer.

Hiatus hernia:

A hiatus hernia occurs when part of the stomach moves up into the chest through a defect in the diaphragm. It is a common problem occurring in 10% of people and the hernia rarely causes symptoms on its own. The presence of a hiatus hernia can cause weakness of the lower esophageal sphincter and this in turn can cause reflux of the acidic stomach contents into the esophagus. This causes the sensation of heartburn and patients with a hiatus hernia are more prone to heartburn than those without this defect. Nevertheless it is important to emphasise that not all patients with hiatus hernia have heartburn and some patients with heartburn do not have a hiatus hernia.

GASTRO-ESOPHAGEAL REFLUX DISEASE (GERD):

Gastro-esophageal reflux may occur when the pressure of the highpressure zone in the distal esophagus is too low to prevent gastric contents from entering the esophagus or when a sphincter with normal pressure undergoes spontaneous relaxation, not associated with a peristaltic wave in the body of the esophagus. GERD is often associated with a hiatus hernia. The most common presentation of patients with GERD is a longstanding heartburn and a shorter history of regurgitation. Heartburn, when typical, is a very reliable symptom. Heartburn is confined to the epigastric and retrosternal areas. It is identified as a caustic or stinging sensation. It does not radiate to the back and is not characteristically described as a pressure sensation.

BARRETT'S ESOPHAGUS:

It is metaplastic changes in the mucosa of the oesophagus as a result of gastroesophageal reflux disease .squamous epithelium in lower end of oesophagus is replaced by columnar epithelium. The endoscopic picture of barrett's metaplasia will be tongue like projection into the mucosa of oesophagus.

TYPES:

(1)Based on length

Long segment –metaplasia more than 3 cm

Short segment- metaplasia less than 3 cm(2)Histological types

Gastric type

Intestinal type

Junctional type

The diseased columnar epithelium is more prone for malignant transformation.

Regular endoscopic surveillance is essential for early detection of malignant transformation.

PEPTIC ULCER DISEASE (PUD)

Ulcer is caused by acid peptic digestion of the mucosa to variable depth either in mucosa containing acid secreting cells or in other sites. Peptic ulcer extends through the muscularis mucosa, an erosion is superficial to the muscularis mucosa. Although the name suggests an association with pepsin, it is the acid which is important for the occurrence of peptic ulcer. May be acute ulcers which are shallow and multiple or chronic which are single ,deep and scirrhous.

Common sites:

1. 1st part of duodenum
2. Lesser curve of stomach
3. Prepyloric and pyloric channel

Gastric ulcer: Seen commonly in late middle age and the incidence increases with age. Sex incidence is found to be equal.

Duodenal ulcer: Most common in middle age, more common in males. Male to female ratio was found to be 3:1. 10 - 20% of patients with a gastric ulcer may have concomitant duodenal ulcer.

Etiology

1. Helicobacter pylori infection
2. Endocrine – a) Zollinger-Ellison syndrome b) Cushing's syndrome
c) Parathyroid tumour - hypercalcemia
3. Genetic: cases with blood group „O“
4. Drugs : NSAIDs, aspirin, steroids
5. Smoking: a) Predispose to ulcer formation b) Increases the relapse rate after treatment.
6. Alcohol
7. Diet: irregular diet, spicy food and excessive intake of coffee and tea provoke the formation of peptic ulcer.
8. Emotional factors: anxiety, stress have always been incriminated to cause peptic ulcer.

Pathogenesis:

1. Loss of mucosal defense with hyperacidity
2. Gastric mucus is an important barrier that protects the gastric mucosa from the effects of acid and pepsin.
3. Decreased bicarbonate concentration
4. Decreased gastric mucosal prostaglandin production
5. Acid overproduction is an important factor for causing DU

H.pylori:

It is the most important factor in the development of peptic ulcer. Fifty percent of the world's population is infected with H. pylori, a major cause of chronic gastritis. Helicobacter also clearly has an etiologic role in the development of gastric lymphoma. H.pylori is a small curved, motile, Gram negative, microaerophilic rod with multiple polar flagellae. In stomach it remains close to the gastric mucus secreting cells. It hydrolyses urea ammonia increased gastrin.

ESOPHAGEAL AND GASTRIC CANCER:

Gastric and esophageal cancers are rare, accounting annually for 1% of deaths from all causes. Gastric cancer is on the decline, while esophageal cancer is on the increase. Gastric cancer may be declining because of the decreasing prevalence of *H.pylori*. Squamous cell carcinoma and adenocarcinoma account for 95% of all esophageal tumours. Traditionally squamous carcinoma was the most frequent lesion but in recent years adenocarcinoma has become the predominant disease. Adenocarcinoma of the esophagus is believed to originate from columnar metaplasia of the esophagus (Barrett's esophagus), providing a rationale for endoscopic screening of patient's with Barrett's esophagus.

Adenocarcinoma is responsible for over 95% of all gastric malignancies. Half of patients are inoperable at the time of diagnose and few of these survive five years, while of those undergoing operative treatment 20% are alive after 5 years. Overall 5 year mortality for this disease is therefore approximately 90%. Gastric neoplasia is strongly associated with *H.pylori* infection but as the vast majority of *H.pylori* infected individuals do not develop gastric carcinoma other environmental and genetic factors must be important.

McCarthy Dyspepsia Severity Score

In our study the severity of dyspepsia was measured by the score proposed by McCarthy. The symptoms evaluated consisted of a questionnaire including the frequency and severity of six dyspeptic symptoms.

The symptoms elicited were:

- a. Epigastric pain during day time.
- b. Epigastric pain during night.
- c. Nausea and vomiting.
- d. Anorexia.
- e. Early satiety.
- f. Regurgitation.

These symptoms were scored for severity and frequency from 0 to 4 as follows:

Frequency grade: (TABLE 1)

Frequency Grade	Score allotted
Absent	0
One per week	1
Several times per week	2

Severity grade: (TABLE 2)

Severity Grade	Score allotted
Absent	0
Present but not interfering with daily work of life	1
Present but interfering with daily work of life	2

So one can expect a maximum dyspepsia severity score of $6 \times 4 = 24$

and a minimum score of $6 \times 0 = 0$.

FUNCTIONAL (NON ULCER) DYSPEPSIA:

Functional gastrointestinal disorders include a variable combination of chronic or recurrent gastrointestinal symptoms that do not appear to be explained by structural or biochemical abnormalities. These functional disorders include symptoms attributed to dysfunction of the oropharynx, esophagus, stomach, small bowel, large bowel and biliary tract . These ulcer-like dyspepsia (presenting with ulcer like symptoms), dysmotility dyspepsia (symptoms include nausea, early satiety, bloating, and belching that suggest gastric stasis or small intestinal dysmotility), and reflux-like dyspepsia (heartburn or acid regurgitation accompanies upper abdominal pain or discomfort). Motility abnormalities may be important in a subset of dyspepsia patients but probably do not explain the symptoms in the majority.

OTHER CAUSES:

1. Biliary or pancreatic diseases.
2. Metabolic disturbances.
3. Irritable bowel disease.
4. Psychiatric diseases.

INVESTIGATIONS:

UPPER GI ENDOSCOPY: Endoscope is used to visualize the esophagus, stomach and proximal duodenum, if necessary therapeutic procedures can be performed. Endoscopy has now become the gold standard test for detecting esophageal, gastric and duodenal lesions.

TREATMENT

1. Reassurance.
2. Pharmacological treatment:
 - a) H₂ receptor blockers- Ranitidine 150mg bid.
 - b) Proton pump inhibitors- Omeprazole 20mg, Rabeprazole 20mg, Pantoprazole 40mg.
 - c) Antacids and alginates- Aluminium hydroxide, Magnesium trisilicate, Dimeticone and Peppermint oil.
 - d) Prostaglandin analogues- Misoprostol.
 - e) Prokinetics- Domperidone and Cisapride.

SURGICAL PROCEDURES:

The discovery of H.pylori and the development of powerful acid suppressive therapy have revolutionized the medical therapy of peptic ulcer and gastro esophageal reflux disease. This has made peptic ulcer surgery almost obsolete. Anti-reflux surgery is reserved for selected patients with documented acid reflux whose symptoms are unresponsive to medical therapy or who do not wish to take long term PPI treatment.

ANTI-REFLUX SURGERY

FUNDOPLICATION (OPEN OR LAPROSCOPIC APPROACH)

- a) Nissen fundoplication (360- degree wrap) - most common anti-reflux surgery.
- b) Partial anterior fundoplication.
- c) Partial posterior fundoplication.

ENDOSCOPIC THERAPY:

Recently, several endoscopic techniques have been developed for the treatment of GERD. These procedures have sparked significant interest because they each promise a mechanical treatment for reflux with less invasion than afundoplication. These techniques attempt to augment the LES by suturing, radiofrequency energy, Plexiglas injection or biocompatible polymer injection.

PEPTIC ULCER SURGERY

- a) **TRUNCAL VAGOTOMY:** Division of both vagus nerves

above the hepatic & celiac branches just above the GE junction. This procedure is usually combined with drainage procedure.

- Gastrojejunostom

- Pyloroplasty

b) **SELECTIVE VAGOTOMY**: Division of both vagus below the hepatic & celiac branches.

c) **HIGHLY SELECTIVE VAGOTOMY (HSV)**: Also called parietal cell or proximal gastric vagotomy. Severs vagal nerve supply to proximal 2/3rd of the stomach and preserves vagal innervation to the antrum and pylorus. Recurrence rate 5 to 10%.

GASTROJEJUNOSTOMY:

Anastomosis between proximal jejunum and the most dependant portion of greater curvature of the stomach. Anastomosis is antecolic / retrocolic, isoperistaltic, no loop, no tension. VAGOTOMY + ANTRECTOMY (This procedure has got the lowest recurrence rate < 2%).

Billroth I reconstruction (Gastroduodenostomy).

Roux-en-Y Gastrojejunostomy.

PYLOROPLASTY:

a) Heineke-Mikulicz pyloroplasty involves a longitudinal incision of the pyloric sphincter followed by a transverse closure. Most commonly performed pyloroplasty.

b) The Finney pyloroplasty is performed as a gastroduodenostomy with division of the pylorus.

c) The Jaboulay pyloroplasty differs from the Finney procedure in that the pylorus is not transected.

SURGERY FOR GASTRIC CANCER:

1. Endoscopic mucosal resection (EMR).
2. Endoscopic submucosal dissection (ESD).
3. Wedge resection.
4. Open gastrectomy (Partial/ subtotal).
5. Laparoscopically assisted gastrectomy (Partial/ subtotal).

LYMPH NODE LEVELS:

N 1 - Peri gastric nodes.

N 2 - Nodes along the vessels.

N 3 - Distant nodes.

EXTENT OF LYMPHADENECTOMY:

- D 1 Resection: Removal of tumour and N1 nodes.
- D 2 Resection: Removal of tumour and N1, N2 nodes also removes the peritoneal layer over the pancreas and anterior mesocolon.

SURGERY FOR ESOPHAGEAL CANCER: ESOPHAGECTOMY

- The Trans-hiatal Approach: The trans-hiatal esophagectomy is performed through an upper midline laparotomy and left cervical incision.
- The Ivor Lewis Approach: The trans-abdominal, trans-thoracic approach.
- Three-Field Esophagectomy: This approach is carried out through separate laparotomy, right thoracotomy, and cervical incisions.
- The Thoracoabdominal Approach: The left thoracoabdominal approach is probably the least utilized of all approaches to the esophagus.
- The Minimally Invasive Approach: A number of minimally invasive techniques to esophagectomy have been described. These include laparoscopic, hand-assisted, thoracoscopic and robotic-assisted esophagectomy.

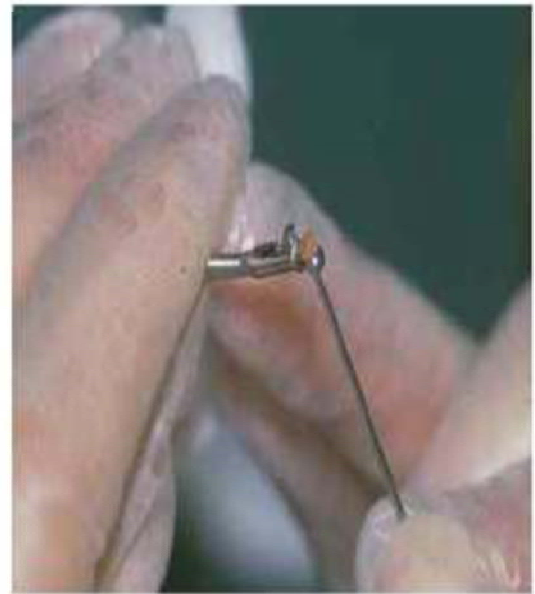
PROCEDURE:

All the patients in this study group, on inpatient and out patient basis underwent upper gastro- intestinal endoscopy under topical anesthesia. The patients were asked to fast for 12 hours prior to the procedure. Only a few patients were given 5-10mg diazepam intravenously for sedation. Lignocaine viscous or oral lignocaine sprays were given to the patient 5-10 minutes before the procedure for the local anaesthetic effect.

The upper gastro-intestinal endoscopy was conducted with Pentax, flexible, fibreoptic endoscope with patients in left lateral positions. The instrument is advanced under direct vision, with the tip of the endoscope in central lumen. Using the adequate insufflations to keep the lumen of the esophagus well distended. Esophagus was looked for any inflammatory changes, growth. The gastro-esophageal mucosal junction was identified at 38-40cms from the incisors. This junction is usually serrated and easily identified by the color difference between the esophageal and gastric mucosa, called as Z line.



Endoscopic biopsy forceps



Endoscopic biopsy piece being removed from the forceps

The position of the esophageal hiatus in the diaphragm is identified by asking the patient to inhale deeply, the diaphragmatic hiatus during inspiration creates an imprint on the esophageal and gastric wall. The position of both the hiatus and the mucosal junction are recorded in order to document the possibility of a hernia.

Gastro-esophageal junction should be looked for closed or widely patulous. On entering the stomach, endoscope slightly down and towards the left, a view of greater curvature and of the posterior wall is obtained. Aspiration of retained liquid is done completely to reduce the risk of aspiration and to allow clear examination of the stomach. A rotation movement of the tip of the instrument allows examination of the anterior and posterior walls of the body of the stomach.

The lesser curvature and the greater curvature are viewed by the same position motion. The most proximal part of both the curvatures are better examined by the J manoeuvre. Stomach was looked for inflammatory changes, ulcer, growth.

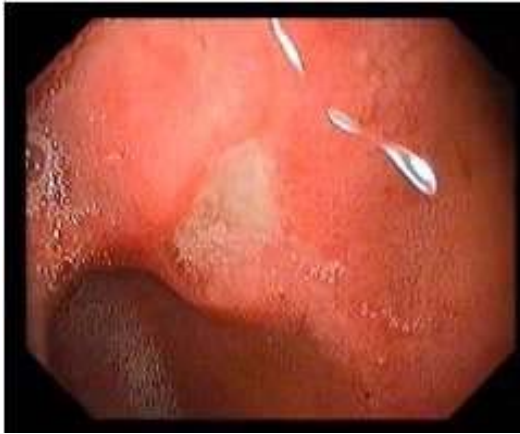
By rotating and angulating the tip endoscope is advanced to assess the antrum. Prepyloric and pyloric ring observed, the passage through the pylorus being done under direct vision. When the pylorus yields, complete assessment of the duodenum upto second part is done. Endoscopic biopsies were taken from the abnormal looking area, growth and the edge of the ulcer crater depending on the findings. Biopsy specimens were sent in formalin solution for histopathology. Each of the biopsy specimens were fixed in 10% buffered formalin and routinely processed to paraffin and 3 μ m sections cut.

Pentax fiber-optic upper G.I. scope used for the study : (FIGURE 4)



ENDOSCOPIC PICTURE AND CIRCUIT : FIGURE (5,6,7)

GASTRIC ULCER



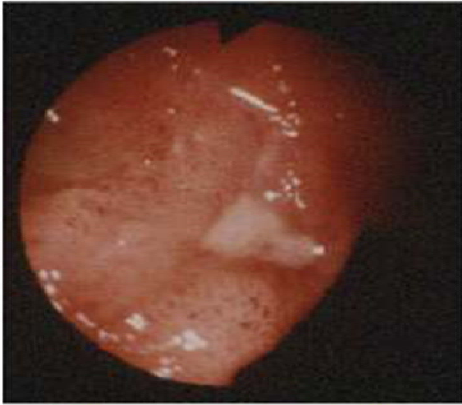
ANTROPYLORIC GROWTH



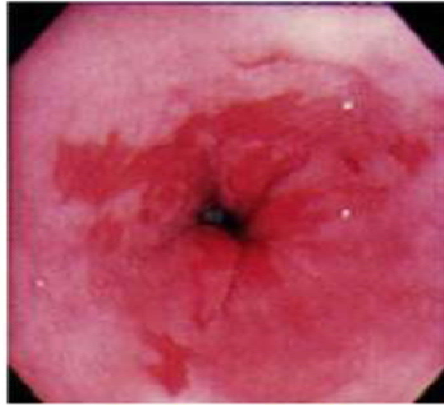
ENDOSCOPIC CIRCUIT



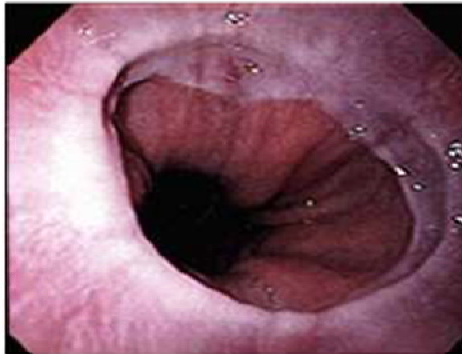
ENDOSCOPIC FEATURE OF DYSPEPTIC PATIENT



Endoscopic view of duodenal ulcer



Endoscopic view of Barrett's esophagus



Endoscopic view of Lax LES (hiatus hernia)

ENDOSCOPIC BIOPSY FORCEPS : (FIGURE 8)



OBSERVATION

(RESULTS)

(TABLE: 3) DISTRIBUTION OF AGE AND SEX OF THE STUDY

AGE(IN YEARS)	SEX		TOTAL
	MALE	FEMALE	
20-29	11	2	13
30-39	12	5	17
40-49	21	7	28
50-59	12	5	17
>60	22	5	27
TOTAL	78	24	102
MEAN	47.72 YEARS		
SD	14.67		

PARTICIPANTS

N = 102

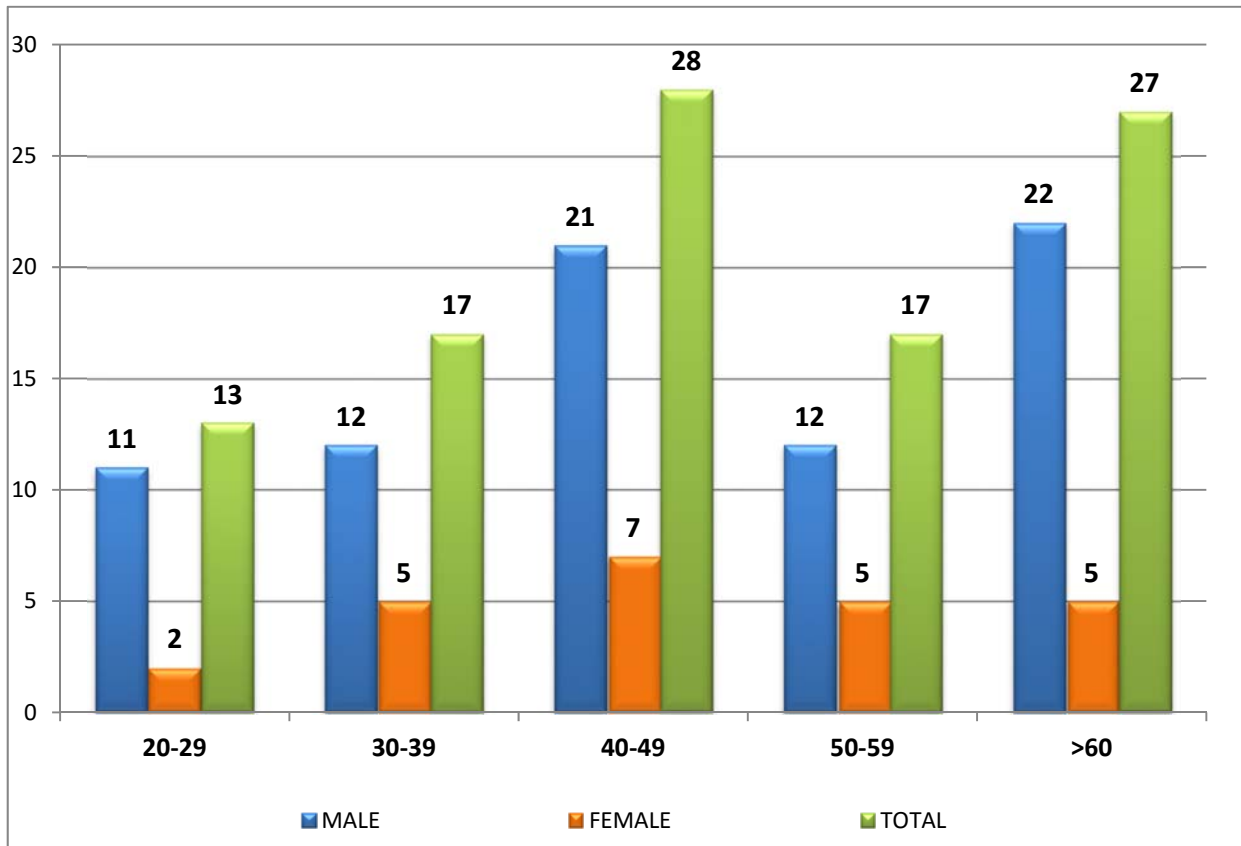


FIG. 9. DISTRIBUTION OF AGE AND SEX OF THE STUDY PARTICIPANTS

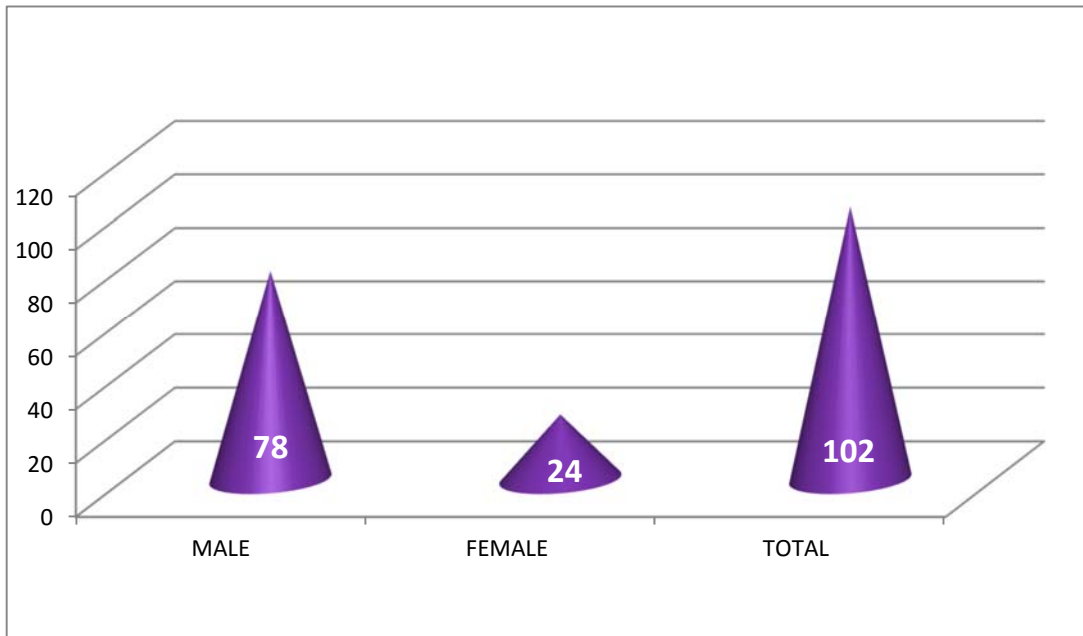
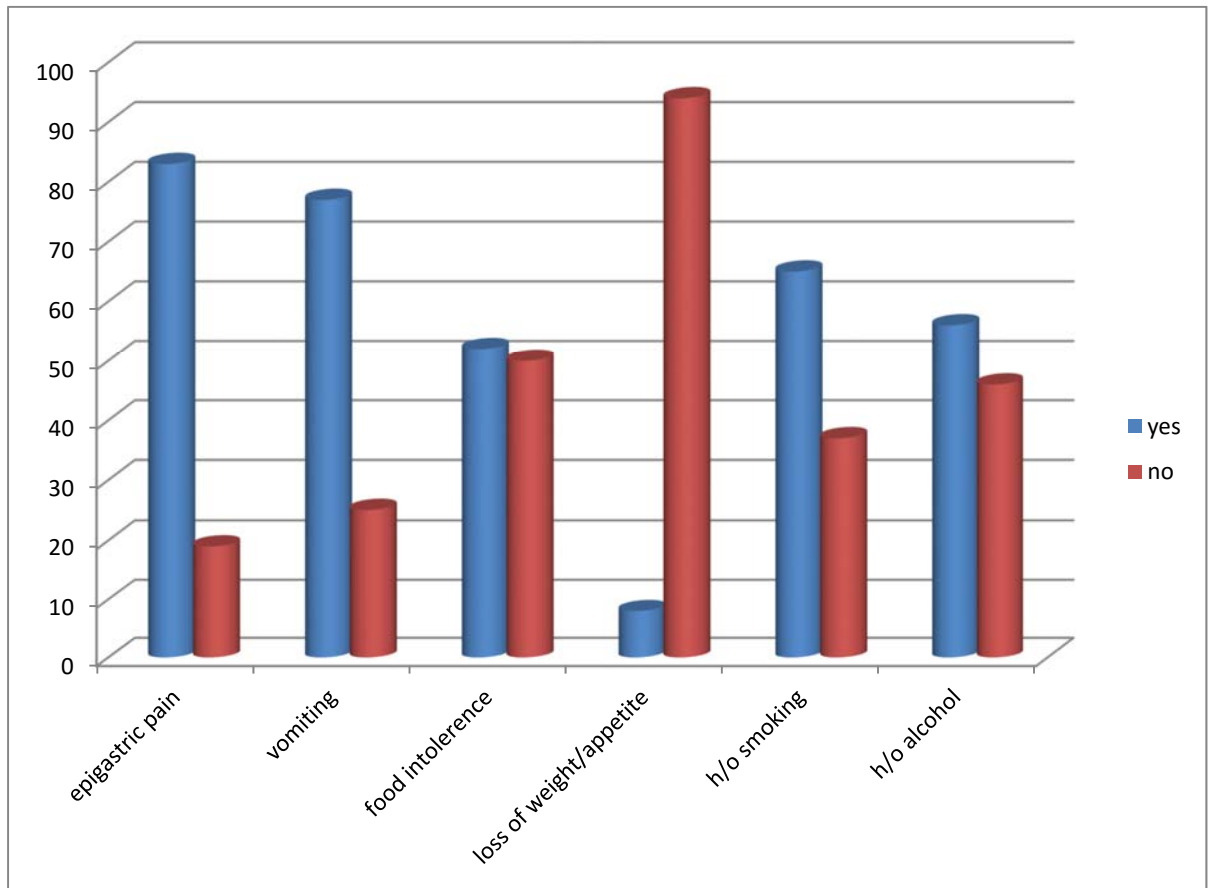


FIG 10. DISTRIBUTION OF AGE AND SEX OF THE STUDY PARTICIPANTS

(TABLE 4)**DISTRIBUTION OF CLINICAL FEATURES OF STUDY PARTICIPANTS**

CLINICAL FEATURES		MALE	FEMALE	TOTAL
EPIGASTRIC PAIN	YES	66	17	83
	NO	12	7	19
VOMITING	YES	56	21	77
	NO	22	3	25
FOOD INTOLERANCE	YES	40	12	52
	NO	38	12	50
LOSS OF WEIGHT/APPETITE	YES	7	1	8
	NO	71	23	94
H/O SMOKING	YES	65	0	65
	NO	13	24	37
H/O ALCOHOL	YES	53	3	56
	NO	25	21	46

(FIG 11) DISTRIBUTION OF CLINICAL FEATYRES OF STUDY PARTICIPANTS



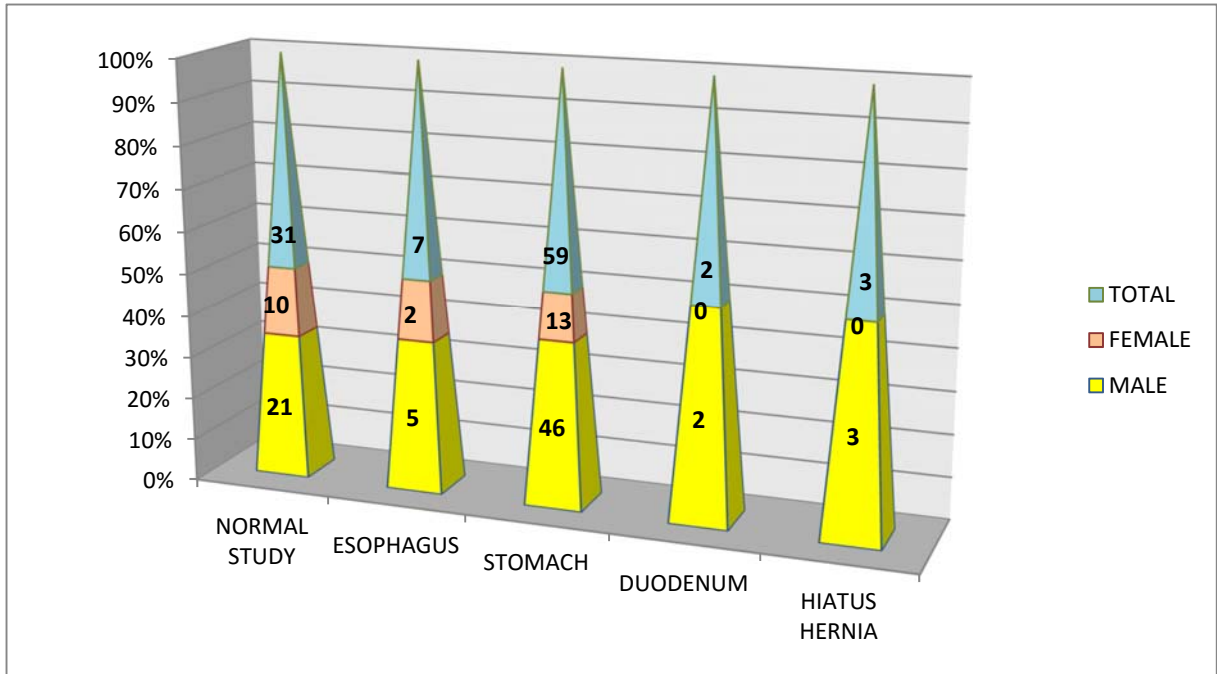
(TABLE 5) ENDOSCOPIC FINDINGS IN PATIENTS WITH DYSPEPSIA

FINDINGS	MALE	FEMALE	TOTAL
NORMAL STUDY	21	10	31
OESOPHAGITIS	0	1	1
GASTRITIS	38	11	49
DUODENITIS	3	1	4
GASTRIC ULCER	1	0	1
GROWTH/MALIGNANCY	8	1	9
HIATUS HERNIA	3	0	3
ESOPHAGEAL VARICES	4	0	4
TOTAL	78	24	102

(TABLE 6)

SITE OF LESION IN ENDOSCOPY PRESENTATION WITH DYSPEPSIA

SITE	MALE	FEMALE	TOTAL
NORMAL STUDY	21 (20.58%)	10 (9.80%)	31 (30.39%)
OESOPHAGUS	5 (4.90%)	2 (1.96%)	7 (6.86%)
STOMACH	46 (45.09%)	13 (12.74%)	59 (57.84%)
DUODENUM	2 (1.96%)	-	2 (1.96%)
HIATUS HERNIA	3 (2.94%)	-	3 (2.94%)



(FIG: 12) SITE OF LESION IN ENDOSCOPY PRESENTATION WITH DYSPEPSIA

(TABLE 7)-AGE WISE PRESENTATION OF DYSPEPSIA

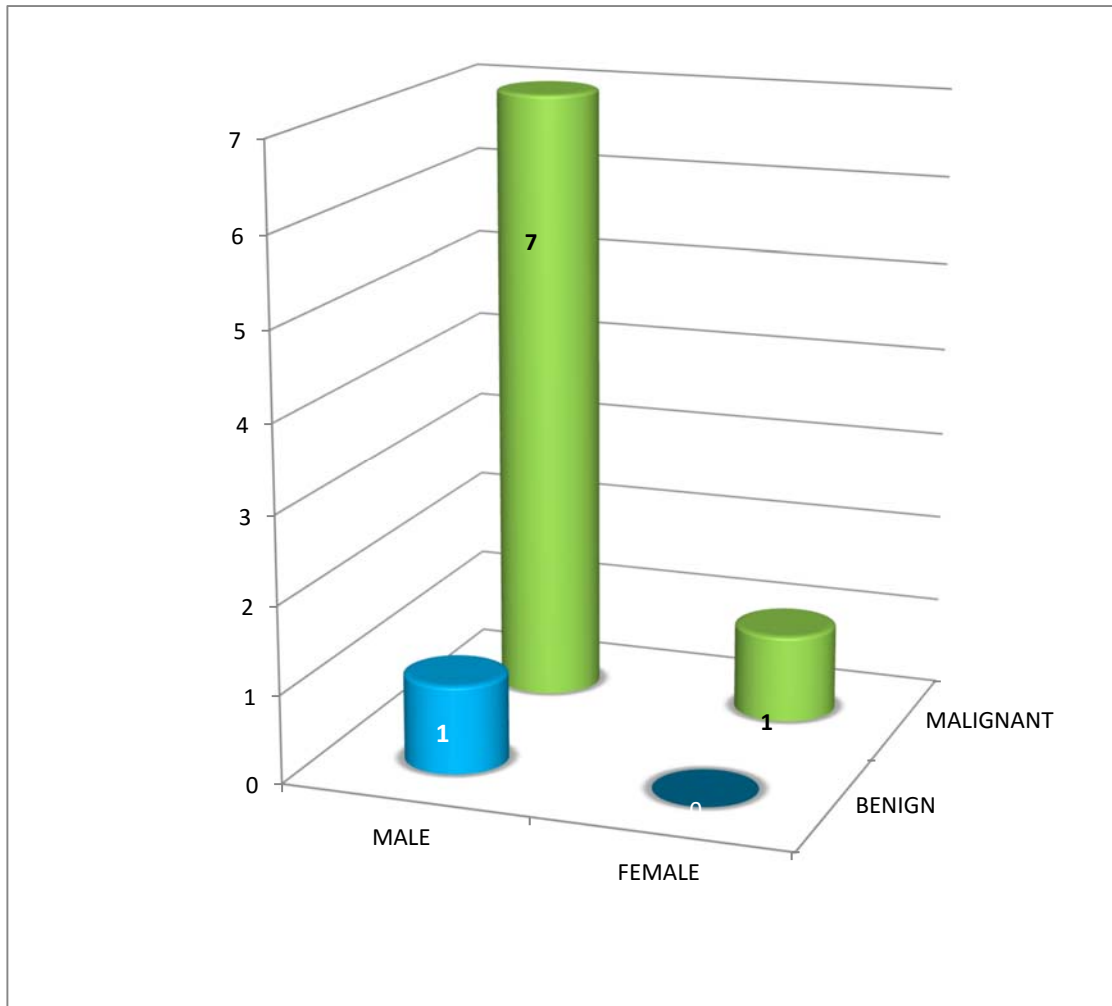
FINDINGS	20-29	30-39	40-49	50-59	>60
NORMAL	8	4	11	5	3
OESOPHAGITIS	-	-	1	-	-
GASTRITIS	3	9	13	8	16
DUODENITIS	-	2	1	1	-
GROWTH	-	1	-	3	5
HIATUS HERNIA	2	1	-	-	-
VARICES	-	1	2	-	1

(TAB 8)

GENDER WISE DISTRIBUTION OF BIOPSY FINDINGS OF THE GROWTH

GROWTH	BENIGN	MALIGNANT
MALE	1	7
FEMALE	0	1

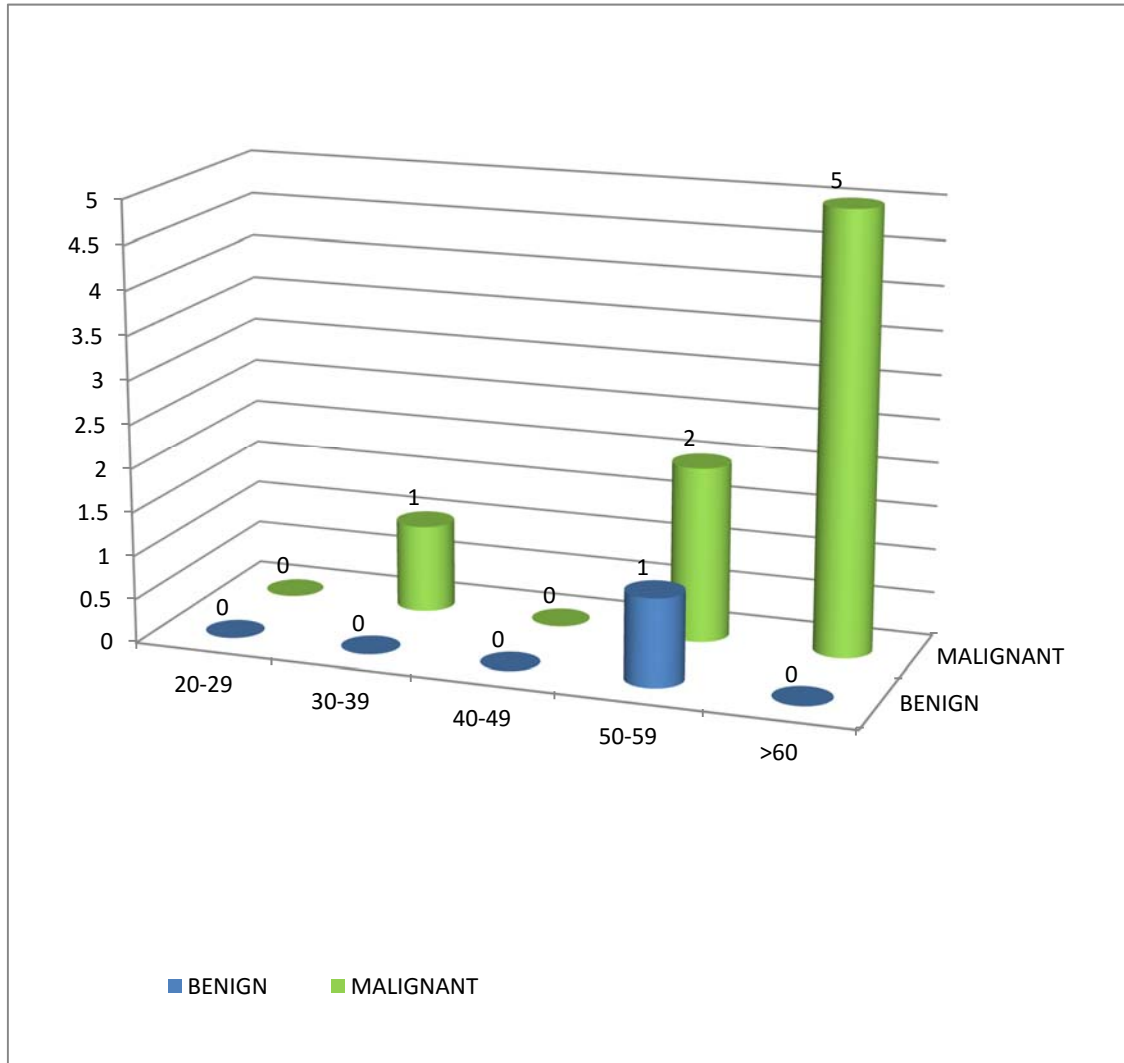
(FIGURE 13) – GENDER WISE DISTRIBUTION OF BIOPSY FINDINGS OF THE GROWTH



(TABLE 9) – AGE GROUP WISE DISTRIBUTION OF BIOPSY FINDINGS OF THE GROWTH

GROWTH	BENIGN	MALIGNANT
20-29	-	-
30-39	-	1
40-49	-	-
50-59	1	2
>60	-	5

(FIG 14) –AGE GROUP WISE DISTRIBUTION OF BIOPSY FINDINGS OF THE GROWTH



DISCUSSION

“A CROSS SECTIONAL STUDY TO ASSESS THE VARIOUS UPPER GASTROINTESTINAL ENDOSCOPIC FINDINGS IN DYSPEPSIA PATIENTS ATTENDING TERTIARY LEVEL CARE HOSPITAL” was conducted in Chengalpattu medical college hospital to study the endoscopic findings of dyspepsia and to detect esophageal and gastroduodenal carcinoma at early stages. After informed consent obtained 102 cases of dyspepsia were included in this study and were studied clinically as per the proforma from **2018 to 2019**. All the patients underwent upper gastrointestinal endoscopy and various findings were noted.

CLINICAL PRESENTATION:

Out of **102** patients, **83** patients had epigastric pain and discomfort and chief complaint of vomiting was present in **77** patients. The other complaints were food intolerance among **52** patients, and loss of weight and appetite among **8** patients. Similar study was conducted by Thomson A B R et al, in which the common presenting complaints were upper abdominal pain among **34.3%** and heart burns among **24.5%** and acid regurgitation among **13.3%**.

COMPARISON OF GENDER DISTRIBUTION

In this study **77** were male patients and **25** were female patients. The incidence of different presentations of dyspepsia were common in males compared to females. The male / female ratio in the studies conducted by Mustapha SK et al- 1.1:1 , Khan N et al – 2.3:1, Ziauddin- 1.6:1, respectively. In above studies also the majority of patients were males as observed in our study. In a population based study in Australia, female patient significantly outnumbered males in disorders includes functional dyspepsia.

COMPARISON OF VARIOUS ENDOSCOPIC FINDINGS:

In the present study, clinically significant endoscopic findings were observed in **71** patients out of which **48%** cases found to have Gastritis as their most common finding. The next common findings were growth, duodenitis and varices. The percentage of cases with gastritis in this study was higher than that observed in studies by Sarwar et al and Ziauddin.

COMPARISON OF INCIDENCE OF GASTRIC MALIGNANCIES:

In this study there were **8** patients with biopsy proven carcinoma, among them which **7** were male patients with carcinoma stomach and **1** were female Patient with periampullary growth. Gastric malignancies were common in older age groups.

CONCLUSION

From the present study of **“A CROSS SECTIONAL STUDY TO ASSESS THE VARIOUS UPPER GASTROINTESTINAL ENDOSCOPIC FINDINGS IN DYSPEPSIA PATIENTS ATTENDING TERTIARY LEVEL CARE HOSPITAL”**.

On endoscopic examination gastritis accounted for the majority of the cases. Incidence of malignancy in the present study was 8% out of which most common is gastric malignancies. Clinically significant endoscopic findings were observed in 71 patients with uninvestigated dyspepsia. Most patients presented with more than two dyspeptic symptoms and the symptom profile was not predictive of the endoscopic findings. Prevalence of large number of inflammatory lesions as a result of increased acid production and low incidence of malignancy in the study group suggests that the uninvestigated patients with dyspepsia may be initially managed medically with acid suppressive therapy.

SUMMARY

”A CROSS SECTIONAL STUDY TO ASSESS THE VARIOUS UPPER GASTROINTESTINAL ENDOSCOPIC FINDINGS IN DYSPEPSIA PATIENTS ATTENDING TERTIARY LEVEL CARE HOSPITAL” was conducted in **Chengalpattu medical college hospital** to know the various endoscopic findings in patients presenting with dyspepsia and early detection of oesophago gastroduodenal malignancy in these patients. 102 patients presenting with dyspepsia were evaluated.

The following were the observations:

1. Highest prevalence of dyspepsia among age group of 40-49years
2. Most common presenting complaint was epigastric pain and discomfort
3. Dyspepsia was more common in males (75%) when compared to females (25%)
4. Most common endoscopic finding was gastritis followed by normal study.
5. Malignancy was diagnosed in 8% patients with dyspepsia.
6. Stomach is the common site of lesion in patients presenting with dyspepsia
7. Gastritis, duodenitis and varices is common in males.
8. Malignancy is common among males than females.
9. Incidence of malignancy increases as the age advances.

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ANNEXURES

PROFORMA

Case serial number:

Name: Age Sex:

Occupation:

Complaints: Duration:

History of present illness:

a. Pain / Duration

b) Nature

c) Site

d) Radiation

e) Relation to food habits

f) Aggravating / Relieving factors

g) Periodicity 2.Nausea/ Vomiting

a) Number b) Contents c) Relation to food

d) Relation to pain.

3. Heart burn.

4. Food intolerance.

5. Indigestion.

6. Loss of weight and appetite.

Past History:

Treatment History: History of NSAIDs/ Corticosteroid usage

Personal History:

1. Diet: Vegetarian/Mixed.
2. Appetite: Good/Reduced.
3. Bowel habits: Frequency.
4. H/o smoking: Yes/no, duration, number/day.
5. H/o alcohol intake: Yes/no, duration, quantity/day.

General physical examination: Built: Well built/moderately built.

Nourishment: Well nourished/poorly nourished Pallor: Present/Absent

Per abdomen: Tenderness. Lump/Mass.

Free fluid. Organomegaly. Other systems: RS, CVS, CNS.

Clinical Diagnosis: Endoscopic findings : Other investigations if any:

INFORMED CONSENT FORM

Title of the Study : **“A CROSS SECTIONAL STUDY TO ASSESS
THE VARIOUS UPPER GASTROINTESTINAL ENDOSCOPIC
FINDINGS IN DYSPEPSIA PATIENTS ATTENDING TERTIARY
LEVEL CARE HOSPITAL”**

Name of the Participant:

_____.

Name of the Principal (Co-Investigator) :

_____.

Name of the Institution: Government Chengalpattu medical college and
Hospital

Name and address of the sponsor / agency (ies) (If any):

Documentation of the informed consent :

I _____ have read the
information in this form (or it has been read to me). I was free to ask any
questions and they have been answered. I am over 18 years of age and,
exercising my free power of choice, hereby give my consent to be included as

a participant “**A CROSS SECTIONAL STUDY TO ASSESS THE
VARIOUS UPPER GASTROINTESTINAL ENDOSCOPIC FINDINGS
IN DYSPEPSIA PATIENTS ATTENDING TERTIARY LEVEL CARE
HOSPITAL”**

(title of the study).

1. I have read and understood this consent form and the information provided to me.
2. I have had the consent document explained to me.
3. I have been explained about the nature of the study.
4. I have been explained about my rights and responsibilities by the investigator.
5. I have been informed the investigator of all the treatments I am taking or have taken in the past _____ months including any native (alternative) treatment.
6. I have been advised about the risks associated with my participation in this study.*
7. I agree to cooperate with the investigator and I will inform him/her immediately if I suffer unusual symptoms.*
8. I have not participated in any research study within the past _____month(s).*
9. I have not donated blood within the past _____months----add if the study involves extensive blood sampling.*

10. I am aware of the fact that I can opt out of the study at any time without having to give any reason and this will not affect my future treatment in this hospital.*
11. I am also aware that the investigator may terminate my participation in the study at any time, for any reason, without my consent.
12. I hereby give permission to the investigators to release the information obtained from me as result of participation in this study to the sponsors, regulatory authorities, Govt. agencies, and IEC. I understand that they are publicly presented.
13. I have understand that my identity will be kept confidential if my data are publicly presented.
14. I have had my questions answered to my satisfaction.
15. I have decided to be in the research study.

I am aware that if I have any question during this study, I should contact the investigator. By signing this consent form I attest that the information given in this document has been clearly explained to me and understood by me, I will be given a copy of this consent document.

Signature/left thumb impression of the participant

சுயஒப்புதல்படிவம்

ஆய்வுசெய்யப்படும் தலைப்பு :

**“A CROSS SECTIONAL STUDY TO ASSESS THE VARIOUS UPPER
GASTROINTESTINAL ENDOSCOPIC FINDINGS IN DYSPEPSIA PATIENTS
ATTENDING TERTIARY LEVEL CARE HOSPITAL”**

ஆய்வுசெய்யப்படும் இடம்:

பங்குபெறுபவரின் பெயர்:

பங்குபெறுபவரின் வயது: பங்குபெறுபவரின் எண் :

மேலேகுறிப்பிட்டுள்ள மருத்துவ ஆய்வின் விவரங்கள் எனக்கு

விளக்கப்பட்டுள்ளது. நான் இவ்வாய்வில் தன்னிச்சையாக பங்கேற்கின்றேன்.

எந்தகாரணத்தினாலோ, எந்த சட்டசிக்கலுக்கும் உட்படாமல் நான்

இவ்வாய்வில் இருந்து விலகிக்கொள்ளலாம் என்றும் அறிந்துகொண்டேன்.

இந்த ஆய்வுசம்பந்தமாகவோ, இதைசார்ந்து மேலும்

ஆய்வுமேற்கொள்ளும்போதும் இந்த ஆய்வில்பங்கு பெறும் மருத்துவர்,

என்னுடைய மருத்துவஅறிக்கைகளைபார்ப்பதற்கு என்

அனுமதிதேவைஇல்லை என அறிந்துகொள்கிறேன். இந்த ஆய்வின்

மூலம்கிடைக்கும் தகவலையோ, முடிவையோ பயன்படுத்திக்கொள்ள மறுக்க

மாட்டேன்.இந்த ஆய்வில் பங்குகொள்ள ஒப்புக்கொள்கிறேன். இந்த ஆய்வை

மேற்கொள்ளும் மருத்துவ அணிக்கு உண்மையுடன் இருப்பேன் என்று

உறுதியளிக்கிறேன்.

பங்கேற்பவரின் கையொப்பம்: சாட்சியாளரின் கையொப்பம்:

இடம்:

இடம்:

தேதி :

தேதி:

பங்கேற்பவரின் பெயர் மற்றும் விலாசம்:

ஆய்வாளரின் கையொப்பம்:

இடம்:

தேதி:

PATIENT CONSENT FORM

STUDY DETAIL:

**“A CROSS SECTIONAL STUDY TO ASSESS THE VARIOUS UPPER
GASTROINTESTINAL ENDOSCOPIC FINDINGS IN DYSPEPSIA
PATIENTS ATTENDING TERTIARY LEVEL CARE HOSPITAL”**

STUDY CENTER:

**CHENGALPATTU MEDICAL COLLEGE & HOSPITAL,
CHENGALPATTU**

PATIENT NAME:

PATIENT AGE:

IDENTIFICATION NUMBER:

I confirm that I have understood the purpose of 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 understand that my participation in the study is voluntary and that I am free to withdraw at anytime without giving any reasons, without my legal rights being affected.

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 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 agree to take part in the above study and to comply with the instructions given during the study and faithfully cooperative with the study team and to immediately inform the study staff if I suffer from any deterioration in my health or wellbeing or any unexpected or unusual symptoms.

I hereby give consent to participate in this study.

I hereby give permission to undergo complete clinical examination and diagnostic test.

Signature/Thumb impression:

Place:

Date:

Patient name and address:

Signature of the investigator:

Place:

Date:

Study investigator's name

KEY TO MASTER-CHART

OP.NO - Out patient number. IP NO – In patient number EP- Epigastric pain.

HB- Heart burn.

N/V- Nausea/ Vomiting. FI- Food intolerance.

IDG- Indigestion.

L W/A- Loss of Weight/ Appetite. NS- Normal Study

Es- Esophagitis. HH- Hiatus hernia. Gs- Gastritis.

GU- Gastric ulcer..

Ds- Duodenitis.

DU- Duodenal ulcer.

M- Male.

F- Female.

Y- Yes.

N- No.

S No.	PATIENT NAME	AGE	SEX	OP/IP No.	CLINICAL FEATURES						CLINICAL DIAGNOSIS	ENDOSCOPIC FINDINGS
					LW/ A	FI	VO	EP	H/A	H/S		
1	ELLAMAL	24	F	67754	no	yes	yes	No	no	no	DYSPEPSIA	BILE REFLUX GASTRITS
2	SUBRAMANI	56	M	97949	no	no	No	yes	no	yes	DYSPEPSIA	MULTIPLE FUNDAL POLYP- BIOPSY TAKEN
3	RANI	65	F	68284	yes	yes	yes	yes	yes	no	DYSPEPSIA	PERIAMPULLARY GROWTH - BIOPSY TAKEN
4	CHELLAMAL	48	F	68432	no	yes	No	yes	no	no	DYSPEPSIA	ANTRAL GASTRITIS
5	KUPPAN	70	M	68297	no	no	No	yes	yes	yes	DYSPEPSIA	EROSIVE GASTRITIS WITH LES

6	KUMAR	36	M	68437	no	no	No	No	yes	yes	DYSPEPSIA	DUODENITIS
7	SUNDARAM	62	M	68496	no	no	No	yes	no	no	DYSPEPSIA	NORMAL STUDY
8	SUDHAKAR	37	M	68383	no	no	yes	yes	no	yes	DYSPEPSIA	NORMAL STUDY
9	NAGAPPAN	75	M	01582	no	no	No	yes	no	yes	DYSPEPSIA	FUNDAL GASTRITIS
10	MURUGAN	32	M	26411	no	no	No	yes	no	yes	DYSPEPSIA	NORMAL STUDY
11	VIJAY	34	M	07959	no	yes	No	yes	yes	yes	DYSPEPSIA	PAN GASTRITIS
12	PADMAVATHY	57	F	04567	no	no	No	yes	no	no	DYSPEPSIA	NORMAL STUDY
13	SELVARAJ	54	M	73854	no	no	yes	yes	yes	yes	DYSPEPSIA	EROSIVE GASTRITIS
14	HARI	27	M	47534	no	no	No	yes	yes	yes	DYSPEPSIA	PAN GASTRITIS
15	KANNAN	70	M	67947	no	no	yes	yes	yes	yes	DYSPEPSIA	LAX PYLORUS- BILE REFLUX GASTRITS
16	MUTHU	60	M	30972	yes	yes	yes	yes	yes	yes	DYSPEPSIA	FUNDAL GROWTH-BIOPSY TAKEN
17	SHANMUGA	43	M	13749	no	no	yes	yes	yes	yes	DYSPEPSIA	BILE REFLUX GASTRITS
18	EGAMBARAM	75	M	12621	yes	yes	yes	yes	yes	yes	DYSPEPSIA	ANTRAL GROWTH – BIOPSY TAKEN

19	PARAMASIVAM	27	M	16251	no	no	No	yes	no	no	DYSPEPSIA	NORMAL STUDY
20	MURUGAN	45	M	10738	no	yes	No	No	no	yes	DYSPEPSIA	NORMAL STUDY
21	SIVAM	80	M	41811	no	yes	yes	yes	yes	yes	DYSPEPSIA	ESOPHAGEAL VARICES-GRADE III
22	SEKAR	40	M	17451	no	no	yes	yes	yes	yes	DYSPEPSIA	PAN GASTRITIS
23	BAVANI	45	F	53669	no	no	yes	No	no	no	DYSPEPSIA	BILE REFLUX GASTRITS
24	YUVARAJ	30	M	16971	yes	no	No	yes	yes	yes	DYSPEPSIA	LAX OG JUNCTION WITH GASTRITIS
25	DAMODHARAN	62	M	09834	no	no	yes	yes	yes	yes	DYSPEPSIA	BILE REFLUX GASTRITS
26	GOPAL	54	M	18860	no	no	yes	yes	no	yes	DYSPEPSIA	D 2 EROSION/ DUODENITIS
27	RANGAN	56	M	03208	no	no	yes	yes	yes	yes	DYSPEPSIA	ANTRAL GASTRITIS
28	MURUGESAN	75	M	03191	no	no	yes	yes	yes	yes	DYSPEPSIA	PAN GASTRITIS
29	SENTHIL	42	M	05424	no	no	yes	No	yes	no	DYSPEPSIA	NORMAL STUDY

30	PRAVEEN	29	M	15487	no	yes	No	yes	yes	no	DYSPEPSIA	NORMAL STUDY
31	PALANI	47	M	02999	no	no	yes	yes	yes	yes	DYSPEPSIA	ANTRAL GASTRITIS
32	GOVINDHARAJ	80	M	18347	no	yes	yes	yes	yes	yes	DYSPEPSIA	PAN GASTRITIS
33	RAJ	45	M	04715	no	yes	yes	yes	yes	yes	DYSPEPSIA	LAX PYLORUS BILE REFUX GASTRITIS
34	PREMA	65	F	20063	no	yes	No	yes	no	no	DYSPEPSIA	NORMAL STUDY
35	SARAVANAN	35	M	14319	no	no	yes	yes	yes	yes	DYSPEPSIA	BILE REFLUX GASTRITS
36	KARNAN	40	M	03661	no	no	yes	yes	yes	yes	DYSPEPSIA	NORMAL STUDY
37	ANADHAN	42	M	03549	no	yes	yes	yes	yes	yes	DYSPEPSIA	LOWER ESOPHAGEAL VARICES- GRADE II
38	LATHIKA	40	F	03095	no	no	yes	yes	no	no	DYSPEPSIA	BILE REFLUX GASTRITS
39	GANAPATHY	70	M	04769	yes	yes	yes	yes	yes	yes	DYSPEPSIA	ANTRO PYLORIC GROWTH - BIOPSY TAKEN
40	ALAMELU	40	F	46981	no	no	yes	No	no	no	DYSPEPSIA	NORMAL STUDY

41	MARIAMMAL	60	F	47857	no	yes	yes	yes	yes	no	DYSPEPSIA	NORMAL STUDY
42	SHANKARI	23	F	09154	no	no	yes	yes	no	no	DYSPEPSIA	NORMAL STUDY
43	DHINESH	23	M	26467	no	no	No	yes	yes	yes	DYSPEPSIA	NORMAL STUDY
44	SURESH	27	M	25918	no	yes	yes	yes	yes	no	DYSPEPSIA	HIATUS HERNIA
45	JAYA	30	F	26450	no	yes	yes	No	no	no	DYSPEPSIA	NORMAL STUDY
46	PERUMAL	55	M	05920	yes	yes	yes	yes	yes	yes	DYSPEPSIA	LESSER CURVATURE GROWTH - BIOPSY TAKEN
47	SANTHAMOORTHY	53	M	06083	no	no	yes	yes	no	yes	DYSPEPSIA	BILE REFLUX GASTRITS
48	JAYAGOPAL	65	M	73181	no	no	yes	No	yes	yes	DYSPEPSIA	BILE REFLUX GASTRITS
49	JEYARAJ	50	M	06524	no	yes	yes	No	yes	yes	DYSPEPSIA	BILE REFLUX GASTRITS
50	PREM	45	M	06077	no	yes	yes	yes	yes	yes	DYSPEPSIA	ESOPHAGEAL VARICES-GRADE III
51	MALLIGA	55	F	26377	no	yes	yes	No	no	no	DYSPEPSIA	LAX OG JUNCTION WITH ANTRAL GASTRITIS
52	KALAISELVI	32	F	34099	no	no	yes	yes	no	no	DYSPEPSIA	ANTRAL GASTRITIS

53	AREENA	45	F	06537	no	yes	yes	No	yes	no	DYSPEPSIA	NORMAL STUDY
54	PANDIAN	60	M	06185	no	no	No	yes	yes	yes	DYSPEPSIA	ANTRAL GASTRITIS WITH D 1 EROSION
55	RANJITH	38	M	09018	no	yes	yes	No	no	no	DYSPEPSIA	NORMAL STUDY
56	SATHASIVAM	68	M	93187	no	yes	yes	yes	yes	yes	DYSPEPSIA	PAN GASTRITIS
57	CHAKRAVARTHY	53	M	35171	no	yes	yes	yes	yes	yes	DYSPEPSIA	ANTRAL GASTRITIS
58	MOHANDASS	36	M	95817	no	yes	No	yes	yes	yes	DYSPEPSIA	ANTRAL GASTRITIS
59	MURUGAN	46	M	68321	no	yes	No	yes	no	yes	DYSPEPSIA	NORMAL STUDY
60	GOTHANDAM	29	M	29987	no	yes	No	yes	no	yes	DYSPEPSIA	BILE REFLUX GASTRITS
61	SELVI	39	F	47871	no	yes	yes	yes	no	no	DYSPEPSIA	LAX OG JUNCTION WITH GASTRITIS
62	SUGANYA	50	F	04091	no	no	yes	yes	no	no	DYSPEPSIA	PAN GASTRITIS
63	ARJUNAN	40	M	05109	no	yes	yes	No	no	yes	DYSPEPSIA	NORMAL STUDY
64	GOPAL	60	M	41087	no	yes	yes	No	no	yes	DYSPEPSIA	ANTRAL GASTRITIS

65	NAGAPPAN	47	M	08261	no	no	No	yes	yes	yes	DYSPEPSIA	DEFORMED PYLORUS WITH BILE REFLUX GASTRITIS
66	JAYACHANDRAN	56	M	07908	no	yes	No	yes	no	no	DYSPEPSIA	NORMAL STUDY
67	THAMBIRAM	25	M	09953	no	no	No	yes	yes	no	DYSPEPSIA	NORMAL STUDY
68	GOWRI	75	F	09592	no	no	yes	yes	no	no	DYSPEPSIA	BILE REFLUX GASTRITIS
69	SARAVANAN	45	M	55457	no	no	yes	No	yes	yes	DYSPEPSIA	BILE REFLUX GASTRITIS
70	LALITHA	62	F	48236	no	yes	yes	yes	no	no	DYSPEPSIA	ANTRAL GASTRITIS
71	EGAMBARAM	63	M	11040	no	yes	yes	yes	yes	yes	DYSPEPSIA	ANTRAL GASTRITIS
72	JAYA	31	F	64486	no	no	yes	yes	no	no	DYSPEPSIA	ANTRAL GASTRITIS
73	SARASWATHY	40	F	11221	no	no	yes	No	no	no	DYSPEPSIA	LAX LES WITH ESOPHAGITIS
74	RAJA	53	M	11261	no	no	No	yes	yes	yes	DYSPEPSIA	BILE REFLUX GASTRITIS
75	GNANAVEL	28	M	67149	no	no	yes	yes	yes	yes	DYSPEPSIA	NORMAL STUDY
76	SUBURAJ	35	M	05568	no	yes	yes	yes	yes	yes	DYSPEPSIA	HIATUS HERNIA
77	SELVAM	32	M	07183	no	yes	yes	yes	yes	no	DYSPEPSIA	ESOPHAGEAL VARICES-GRADE III

78	KARTHI	70	M	11310	no	no	yes	yes	yes	yes	DYSPEPSIA	DEFORMED PYLORUS WITH GASTRIC ULCER
79	RAMU	42	M	12076	no	yes	yes	yes	yes	yes	DYSPEPSIA	PAN GASTRITIS
80	NARASIMAN	45	M	68776	no	no	yes	yes	no	yes	DYSPEPSIA	PAN GASTRITIS
81	RAMAN	41	M	01376	no	yes	yes	yes	no	no	DYSPEPSIA	NORMAL STUDY
82	KAMALA	40	F	12524	no	yes	yes	yes	no	no	DYSPEPSIA	NORMAL STUDY
83	PAKKIRI	67	M	77154	no	yes	yes	No	yes	yes	DYSPEPSIA	ANTRAL GASTRITIS
84	KARTHICK	38	M	90194	no	yes	yes	No	no	yes	DYSPEPSIA	ANTRAL GASTRITIS
85	MUNUSAMY	65	M	13609	no	no	yes	yes	no	yes	DYSPEPSIA	BILE REFLUX GASTRITS
86	KATHIR	28	M	40558	no	yes	yes	No	yes	no	DYSPEPSIA	HIATUS HERNIA
87	KUPPAN	45	M	04431	no	yes	yes	yes	no	yes	DYSPEPSIA	LAX OG JUNCTION WITH DUODENITIS
88	NELSAN	46	M	01058	no	yes	yes	yes	yes	yes	DYSPEPSIA	ANTRAL GASTRITIS
89	NAGARAJAN	60	M	40338	no	yes	yes	yes	no	yes	DYSPEPSIA	ANTRAL GASTRITIS

90	ARUMUGAM	50	M	40787	yes	yes	yes	yes	yes	yes	DYSPEPSIA	ANTRAL GROWTH - BIOPSY TAKEN
91	GANESH	39	M	18672	no	yes	No	yes	no	yes	DYSPEPSIA	GROWTH IN BODY OF STOMACH - BIOPSY TAKEN
92	MANI	50	M	40321	no	no	yes	yes	no	no	DYSPEPSIA	NORMAL STUDY
93	KANNIYAPAN	63	M	42851	no	yes	yes	yes	yes	yes	DYSPEPSIA	BILE REFLUX GASTRITS
94	RAJAVEL	28	M	43081	no	no	yes	yes	yes	yes	DYSPEPSIA	NORMAL STUDY
95	KAMALA	58	F	43026	no	no	yes	yes	no	no	DYSPEPSIA	NORMAL STUDY
96	SARASWATHY	50	F	42704	no	yes	yes	yes	no	no	DYSPEPSIA	NORMAL STUDY
97	BABY	35	F	91102	no	no	yes	yes	no	no	DYSPEPSIA	BILE REFLUX GASTRITS
98	DHAYALAN	47	M	48418	no	yes	yes	yes	no	yes	DYSPEPSIA	ANTRAL GASTRITIS
99	SELVAM	66	M	48918	yes	yes	yes	yes	yes	yes	DYSPEPSIA	LESSER CURVATURE PROLIFERATIVE GROWTH - BIOPSY TAKEN
100	JOHN	23	M	49018	no	no	yes	yes	no	no	DYSPEPSIA	NORMAL STUDY

101	JAYACHANDRAN	40	M	53672	no	yes	yes	yes	yes	yes	DYSPEPSIA	NORMAL STUDY
102	MOHAN	43	M	77019	no	no	yes	yes	yes	no	DYSPEPSIA	NORMAL STUDY