"A CLINICAL STUDY OF UPPER GASTROINTESTINAL ENDOSCOPY FINDINGS IN PATIENTS PRESENTING WITH DYSPEPSIA"

BY

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Dissertation submitted to the
TAMIL NADU DR.MGR MEDICAL UNIVERSITY
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for the degree of

MASTER OF SURGERY IN
GENERAL SURGERY

Under the guidance of
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April 2013

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This is to certify that this dissertation titled "**A CLINICAL STUDY OF UPPER GASTROINTESTINAL ENDOSCOPY FINDINGS IN PATIENTS PRESENTING WITH DYSPEPSIA"** is a bonafide work of Dr. **D.R.SIVAKUMAR**, and has been prepared under my guidance,

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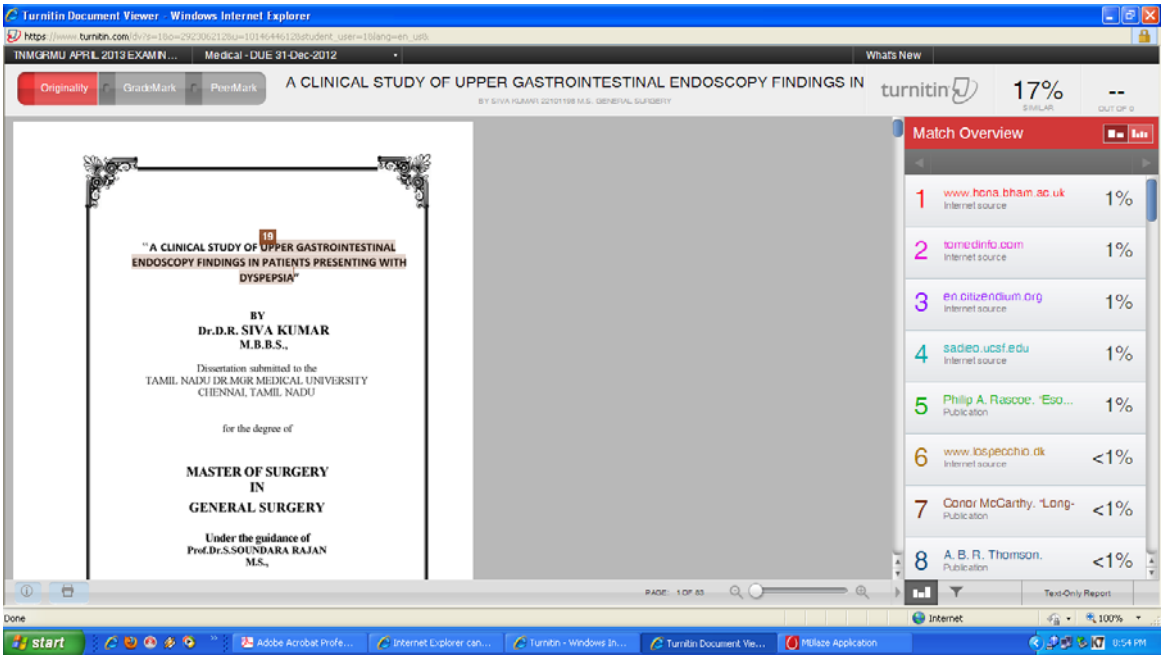
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INTRODUCTION

Dyspepsia is affecting about 25% of general population in developed nations and it is a major cause for medical visits. New patients comprise about 10% of population every year. Dyspepsia majorly affects quality of life and it is a major burden in view of social costs. Directly the expenses are for laboratory tests, medical consultation and drugs and indirectly by absence from work.

Dyspepsia refers to spectrum of diseases and heterogeneous group of symptoms confined to upper abdomen. Dyspepsia is a vague term used to explain upper abdominal collection of symptoms like indigestion, fullness, early satiety (not able to complete the meals), bloating, belching, nausea, epigastric discomfort or pain and anorexia. Indigestion is very common in general population; almost all have had indigestion at some time in their lifetime. Sometimes patients will include constipation and undigested food particles in the stool.

Rome II working team defined dyspepsia as discomfort or pain in upper abdomen. Central abdominal pain is considered to be a vital symptom. Pain which is present in other regions or associated to defecation is not considered.

Non ulcer dyspepsia, this description comprises a group of symptom complex simulating peptic ulcer in patients who have no provable or objective evidence of an ulcer. Based on analysis of problems individuals with non ulcer dyspepsia categorized into two types.

Pseudo ulcer syndrome—with classic symptoms of ulcer disease

Functional dyspepsia—with post prandial fullness, belching and bloating, occasionally associated with pyloroduodenal irritability and prolonged gastric emptying. Usually this functional component is attributed to uncoordinated motor activity and afferent hyper reactivity.

Gastro-oesophageal reflux disease is a condition, defined as abnormal entering of gastric juice into oesophagus and causes symptoms due to tissue damage. The principal pathophysiological problem is the presence of unusual amount of gastric juice in the lumen of oesophagus. Symptoms thought to suggest of gastro-oesophageal disease , such as heart burn or regurgitation are very much prevalent in general population and many individuals do not seek medical advice. The presence of symptoms doesn't correlate well with the tissue damage. For instance the significant problem like Barrett's oesophgus, even in early adenocarcinoma, can occur without symptoms.

Gastro-oesophageal reflux disease is most commonly treated by physicians, this is substantiated by amount of revenue recorded by many pharmacteutical company. The symptoms are due to failure of protective antireflux mechanisms. A clear understanding of the normal anatomy and physiology of esophagus is mandatory to decide the surgical and medical management.

OBJECTIVES

1. To study the endoscopic presentation of dyspepsia.
2. Early detection of esophagogastroduodenal carcinoma.
3. To study the age and sex prevalence in patients presenting with dyspepsia.
4. To study the common site of lesion in patients presenting with dyspepsia.

REVIEW OF LITERATURE

Epidemiology is the study of the distribution and determinants of disease and damage in human populations. So there are 2 phases, one descriptive ascertaining population with high and low incidence of known disease and the other analytical which determines the reason for imbalanced distribution. In the case of dyspepsia, the hope is that epidemiological research will lead to cause of the condition. The dyspepsia, 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 comprise genetic, environmental and psychological factors.

Major changes have happened in the tests and procedures available for the evaluation of dyspepsia patients in the last 40 years. With barium meal examination, it was difficult to examine the duodenum and to discriminate between active ulceration and healed one. With the advent of upper gastrointestinal endoscope it is possible to diagnose disease accurately, both acute and scarring, in both stomach and duodenum.

Environmental reasons play major role in pathogenesis of peptic ulcer and its inconsistent frequency.

Chadwick P et al. evaluated 342 dyspeptic patients. Only 19% of patients found to be having significant findings in endoscopy. The clinical symptoms elicited in the history including the ominous symptoms and signs

did not correlate with the endoscopic findings. In 23% patients, biopsies proved the presence of H. pylori infection. The infection too did not correlate well 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.

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 in 90% of cases, heartburn in 72% and flatulence in 70% cases. The findings in oesophago duodenoscopy were normal in 50% patients. The findings included esophagitis in 12% of patients, gastric ulcer in 10% of patients, duodenal ulcer in 8% of patients, gastritis in 8% of patients and duodenitis in 4% of patients; while esophagogastritis , esophagogastroduodenitis and carcinoma stomach were present in 2% of patients each. Histopathological examination done for all the atypical findings and confirmed. The conclusion was that in most of the patients with dyspepsia, the endoscopic findings were found to be normal. The frequent abnormal findings in endoscopy were esophagitis, gastric ulcer, duodenal ulcer and gastritis. The findings corroborated with biopsy results.

Thomson A B R et al. did endoscopy in 1040 adult patients presented with unevaluated dyspepsia within 10 days of referral. Clinically significant findings were made out in 58% (603/1040) of patients. Esophagitis was the most identified 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. According to the predominent symptom, 463 (45%) patients had ulcer-type, 393 (38%) had reflux-type and 184 (18%) had dysmotility-type dyspepsia. 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 key symptom did not substantiate in predicting the nature and clinically significant findings. The most common finding was found to be esophagitis. Empirical therapeutic test with anti ulcer regimen can be considered before the endoscopy.

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

According to Harding SM et al; the patients who presented with aspiration and extraesophageal(pulmonary), regurgitation was found to be the prominent one.

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

- Accompanied esophageal symptoms
- Pain aggravated by food and in supine position
- 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

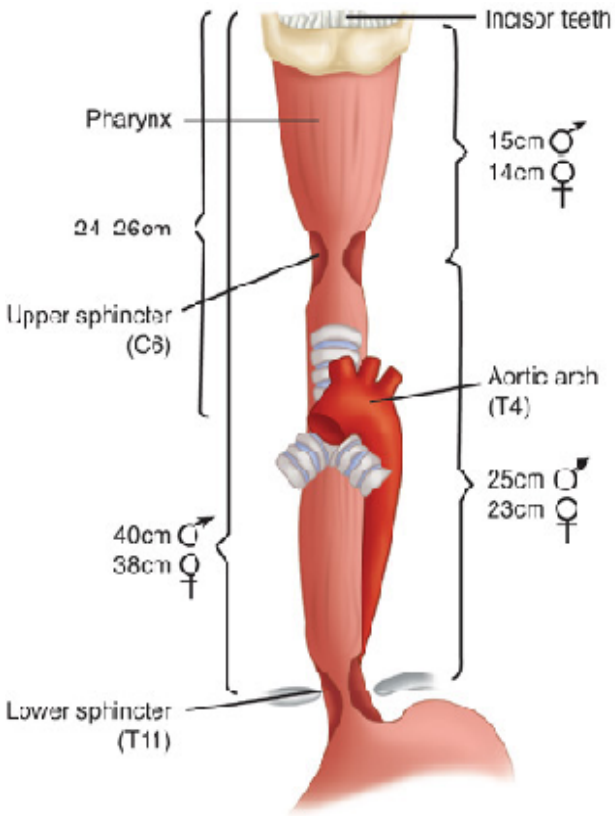
confirmed every year. American males have life time risk for developing ulcer disease is around 10% and females have 4%.

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.



Anatomy of esophagus

Anatomically esophagus is divided into three parts:

- Cervical
- Thoracic
- Abdominal

Function divides the esophagus according to its differing forms of motility into the following three zones

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.

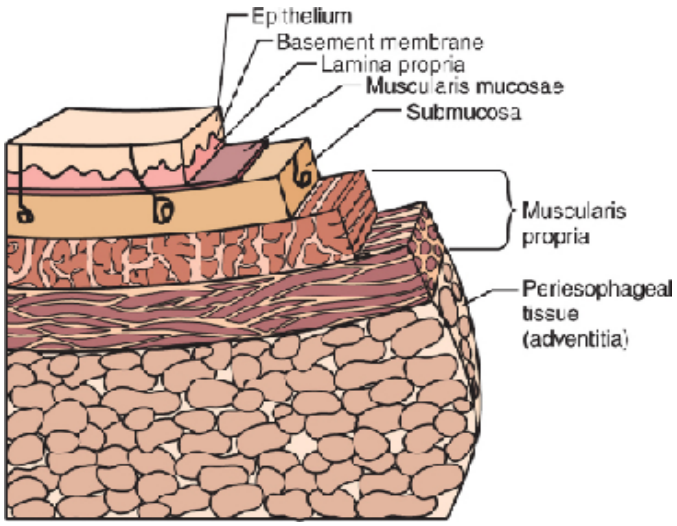
ESOPHAGEAL LAYERS:

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 2

cm 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 main-stem bronchus and aorta abut 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 intra-abdominal 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;

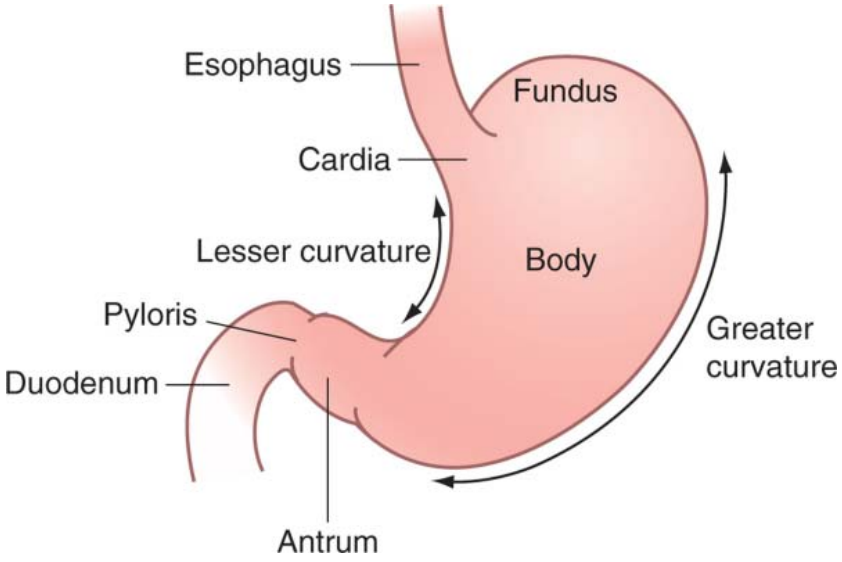
1. Fundus
2. Body
3. Pyloric Antrum
4. Pyloric Canal

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.

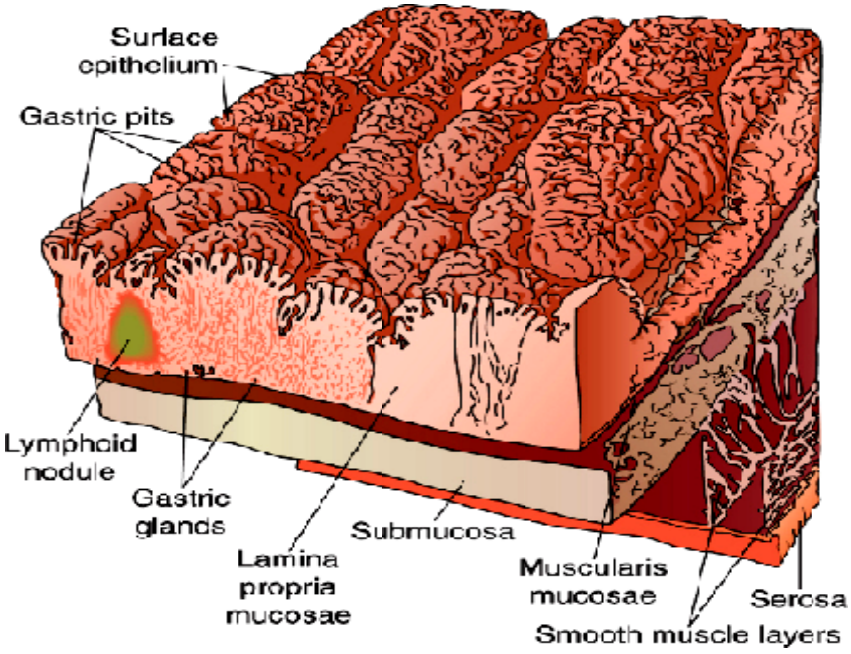


Anatomy of stomach

Stomach wall has four basic layers:

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

HISTOLOGY



The Helicobacter pylori colonizes in the mucous layer of the gastric antrum and is important in relevance to its possible etiology of peptic ulcer disease.

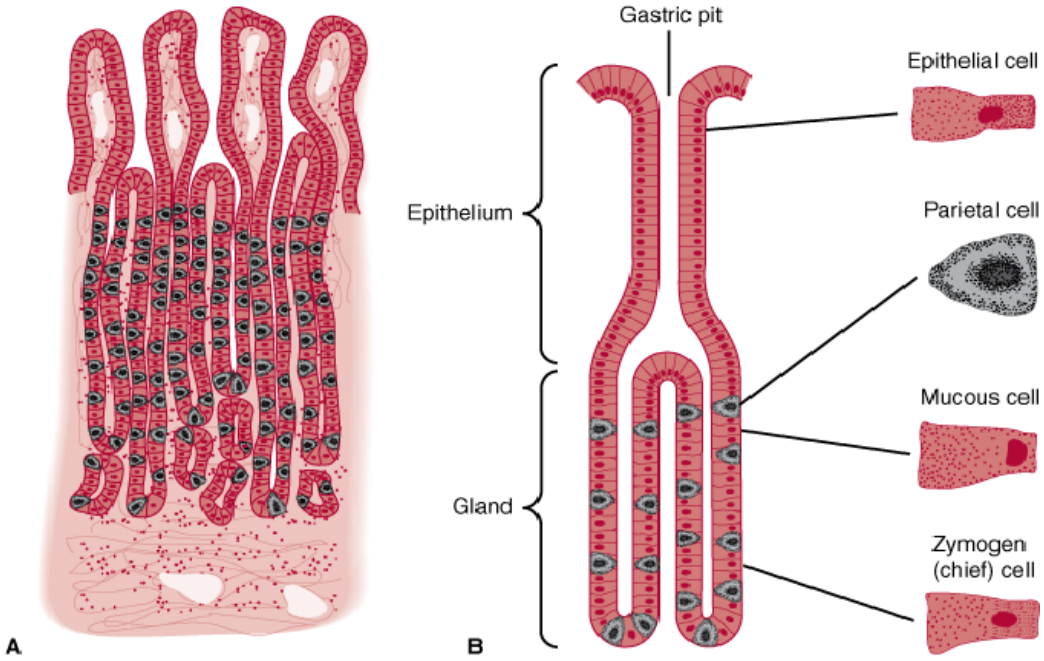
Mucous membrane:

It is smooth and soft. To the naked eye, it appears as numerous folds (rugae) which disappear when stomach is distended. These rugae are most prominent towards the body and greater curvature and are less apparent in the antrum. The lining epithelium is a single layer of columnar cells which secrete mucus and are called “surface mucous cells”. This surface epithelium dips into the lamina propria to form gastric pits. The mucosa is covered by a thick mucus layer, secreted from surface mucous cells. The mucus acts as a lubricant and protects the stomach against its own acid and enzymes.

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.

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 aminoacid 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^{2-},SO_4^{2-}, 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

As early as the 19th Century, attempts were been made to examine the interior of the upper GIT by reflecting light in to the body cavities through a hollow cylinder, but it was not until Thomas Edison's invention of the incandescent light bulb that it became possible in the late 1870's to perform rigid endoscopy.

Nevertheless 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 or complex studies due to overheating of the instruments. In addition the inability to adapt 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 semi rigid gastroscope by R.Schindler in 1936 through work developed in collaboration with the German physician Georg Wolf. The way to the development of a flexible fiberscope was paved 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 in the Netherlands and H.Hopkins and N.S.Kapany in England working independently, developed usable flexible glass fiber bundles that could transmit light across relatively long 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 clinically usable, 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.

Along the way, then 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 and in contrast to the most landmark inventions, was first applied clinically 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 commercially produced fiberoptic endoscope made by American cystoscope makers Inc, Norwalk, CT. was first used in 1961 and the results were published in the Lancet in may of that year.

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 paved the way for the use of endoscopes as vehicle for numerous accessories so that today endoscopic surgery include methods of haemostasis, excision, ablation, dilatation, decompression, sclerosis and foreign body removal.

INSTRUMENTATION

Flexible endoscope come in a variety of diameter and lengths, either direct- viewing or video. The primary endoscope used for upper GI endoscopy is a zero degree, forward viewing endoscope, where as duodenoscope visualizes the GI tract at 90º to the shaft. Side viewing

endoscope is primarily used in the duodenum to visualise the ampulla of Vater, but they may also be used in the stomach.

All endoscopes are either video or fibreoptic 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, especially 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 between 5.5mm to the distal tip to 11mm for a therapeutic endoscope. The diameter of insertion tube for duodenoscope ranges between 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.

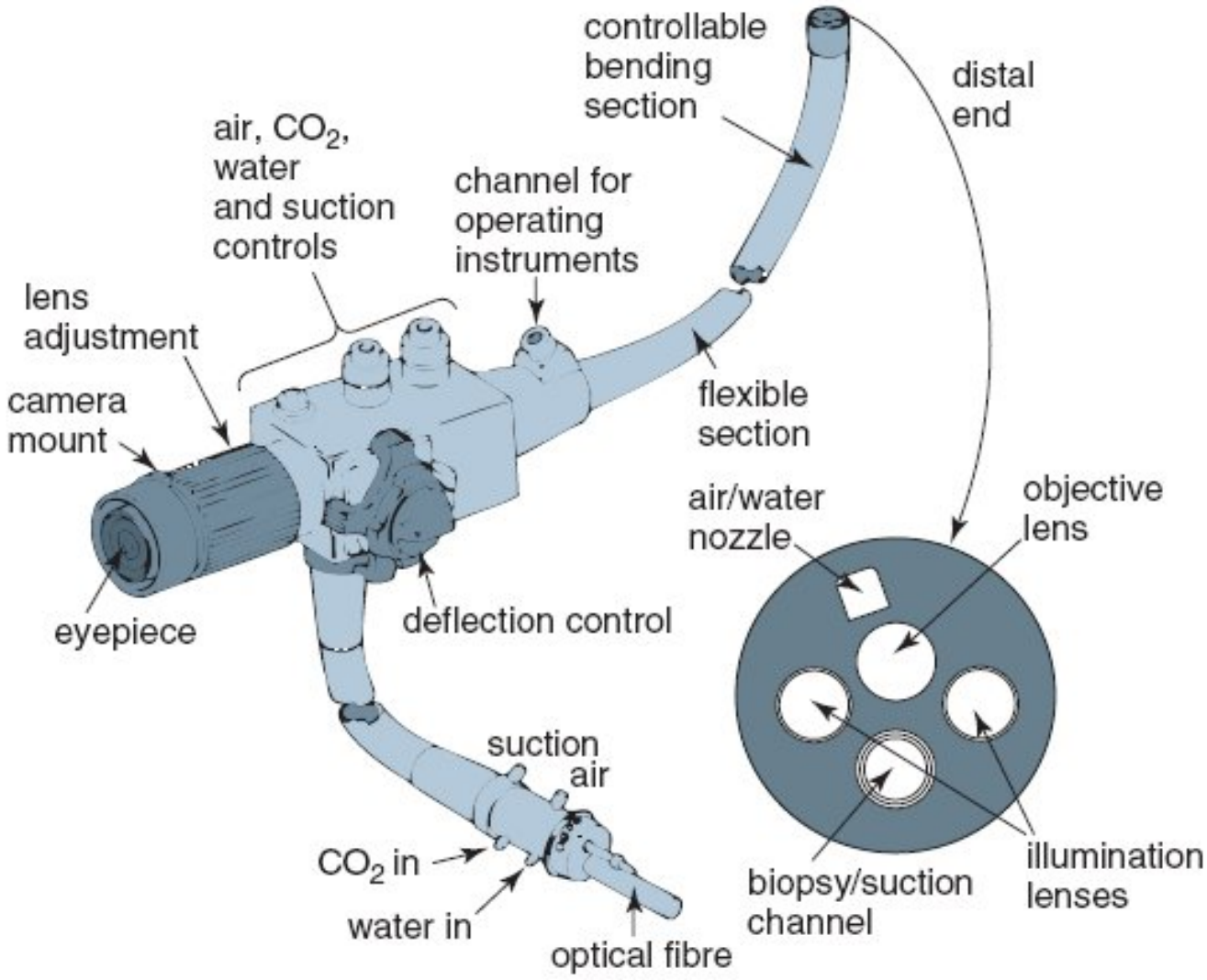
Biopsy forceps, cytology brushes, or other diagnostic instruments are passed through the accessory channel. A double lumen therapeutic endoscope is also available for more advanced therapeutic endoscopy.

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

UPPER GASTROINTESTINAL ENDOSCOPY

PROCEDURE:

PRINCIPLES: Upper GI endoscopy plays a dominant 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 postioned 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 centerof 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 discomfprt 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 upper GI malignancy
- Weight loss(unexplained)
- Upper GI bleeding
- Pain during swallowing

- Unexplained Anemia due to iron deficiency
- Vomiting which is persistent
- 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 in to 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 high-pressure 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 long-standing 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) Cushings 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:

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

Severity grade:

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 6x4 = 24 and a minimum score of 6x0 = 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.

Functional dyspepsia is a heterogeneous syndrome. It can be grouped into symptomatic clusters. 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 perform. Endoscopy has now become the gold standard test for detecting esophageal, gastric and duodenal lesions.

TREATMENT

1. Reassurance.
2. Pharmacological treatment:
    - **a)** H 2 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 a fundoplication. 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%.

GASTROJEUJUNOSTOMY:

Ananstomosis 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.

METHODOLOGY

A prospective clinical study was undertaken at Tirunelveli medical college hospital, tirunelveli to know the various upper gastro-intestinal endoscopic findings in patients presenting with dyspepsia The study was conducted from march 2011 to October 2012. The patient selection was by convenience sampling.

Dyspeptic patients were included in this study with their informed consent.

A detailed clinically history was elucidated, followed by careful clinical examination, which were recorded as per the proforma. All the patients included in the study underwent upper gastrointestinal endoscopy and the findings were noted.

The inclusion and exclusion criterias were as follows:

Inclusion criteria:

1. Patients above 13 years of age.
2. Patients showing symptoms of dyspepsia for 4 or more than 4 weeks.
3. Patients with uncomplicated and uninvestigated dyspepsia.

Exclusion criteria:

1. Patients below 10 years of age.
2. Pregnant and lactating women.

3. Patients on proto-pump inhibitors.
4. Patients who are known cases of chronic pancreatitis and liver disease.
5. Patients on NSAID's for more than one month duration.
6. Unwilling or unfit patients for endoscopy.

PROCEDURE:

All the patients in this study group, on inpatient 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 optimal 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 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. Gastro-esophageal junction should be observed 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 all retained liquid is done to reduce the risk of aspiration and 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 the curvatures are better examined when using 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 directly, the passage through the pylorus being done under direct vision. When the pylorus yields, complete assessment of the duodenum is done upto second part.

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, routinely processed to paraffin and 3 μm sections cut.

Pentax fiber-optic upper G.I. scope used for the study


SONY
PENTAX LH-150PC
PENTAX
MBS
SERVOSTABILIZER
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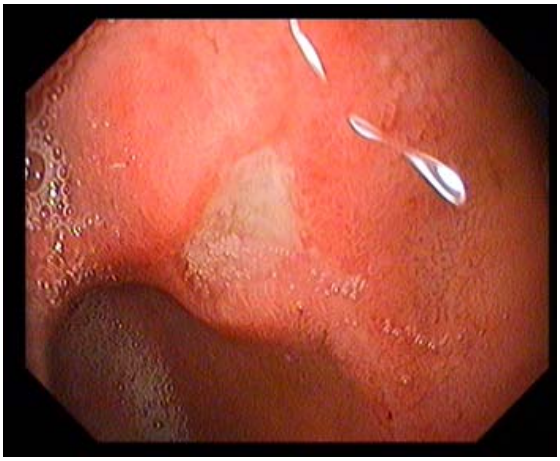
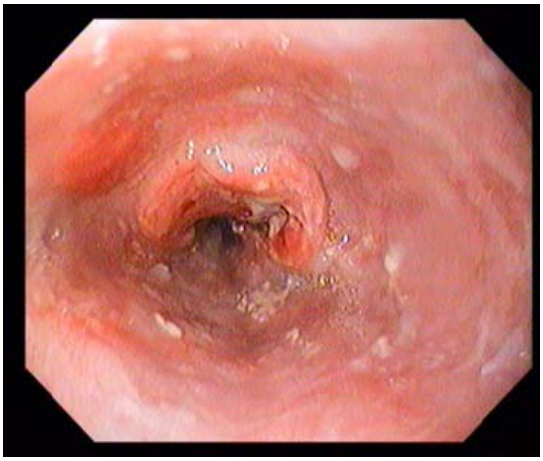
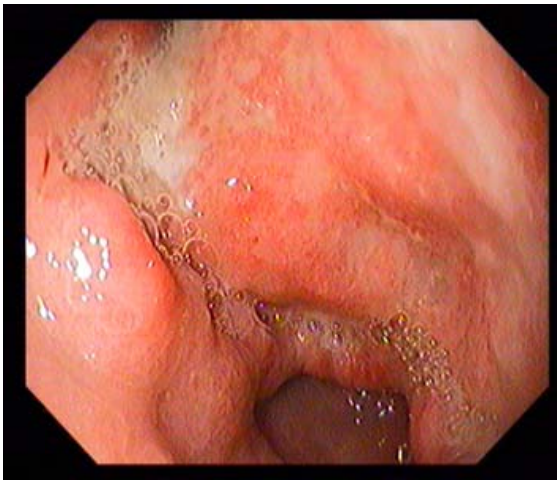


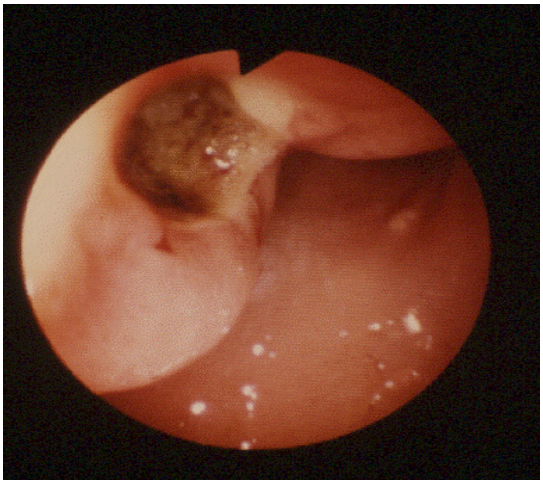
Endoscopic view of an gastric ulcer.



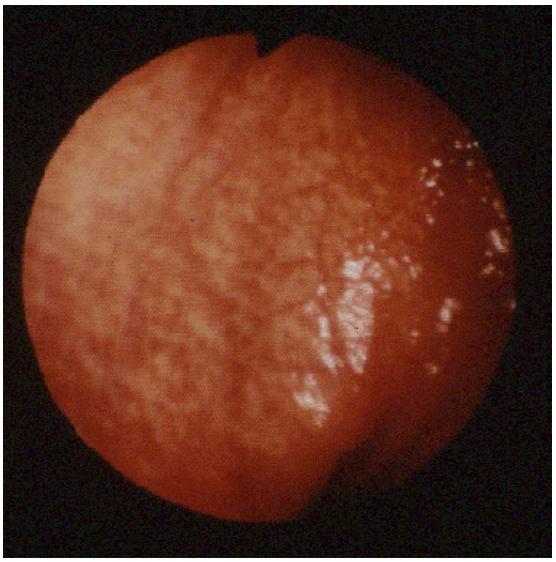
Endoscopic view of esophageal carcinoma



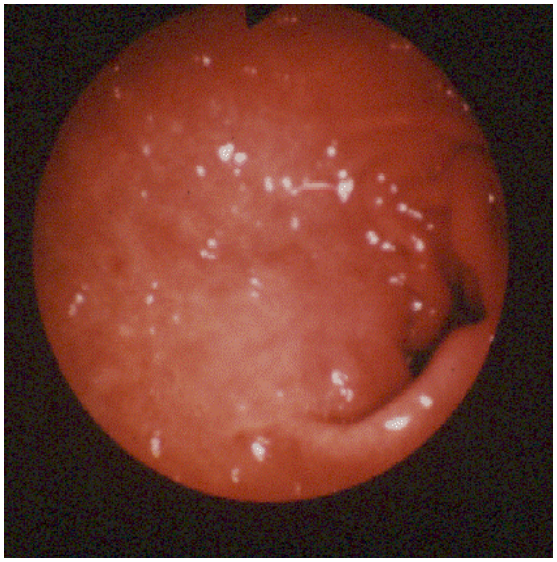
Endoscopic view of carcinoma stomach with ulcer



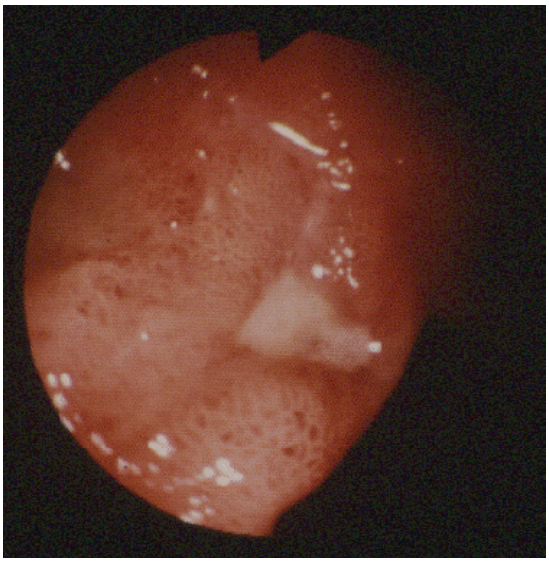
Endoscopic view of antral gastric ulcer



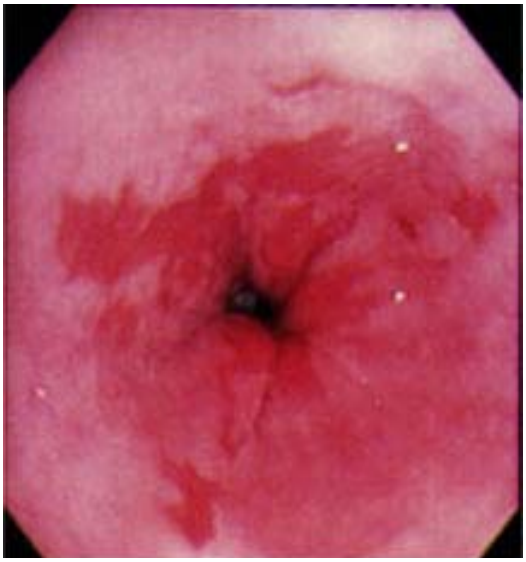
Endoscopic view of chronic antral gastritis



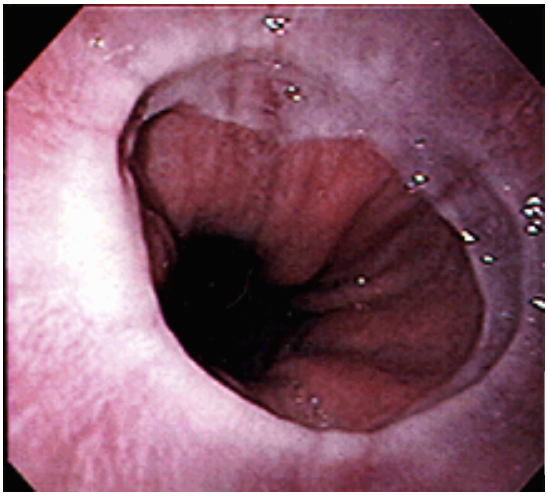
Endoscopic view of bile reflux gastritis



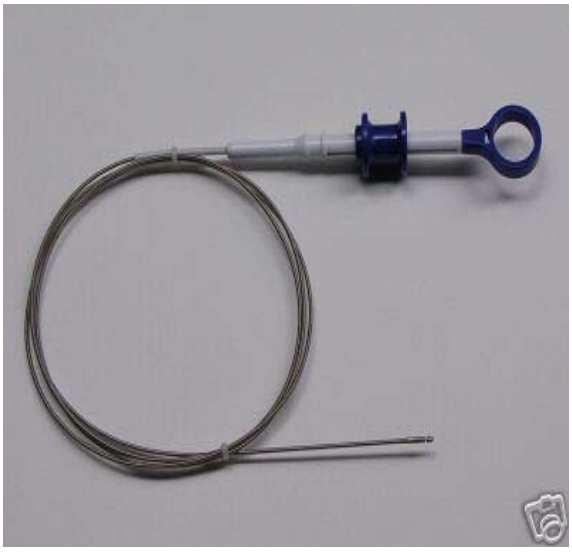
Endoscopic view of duodenal ulcer



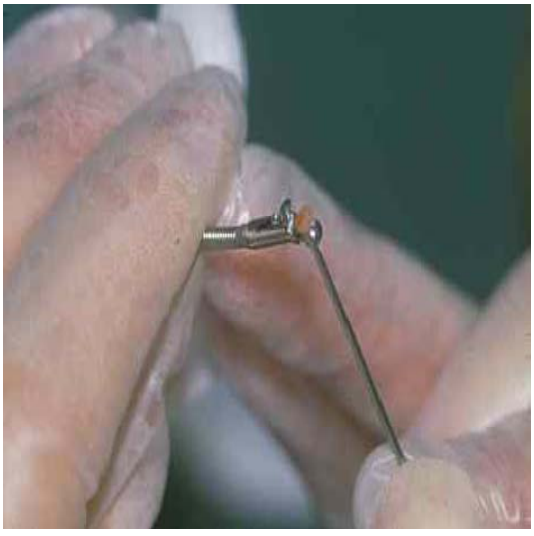
Endoscopic view of Barrett's esophagus



Endoscopic view of Lax LES (hiatus hernia)



Endoscopic biopsy forceps

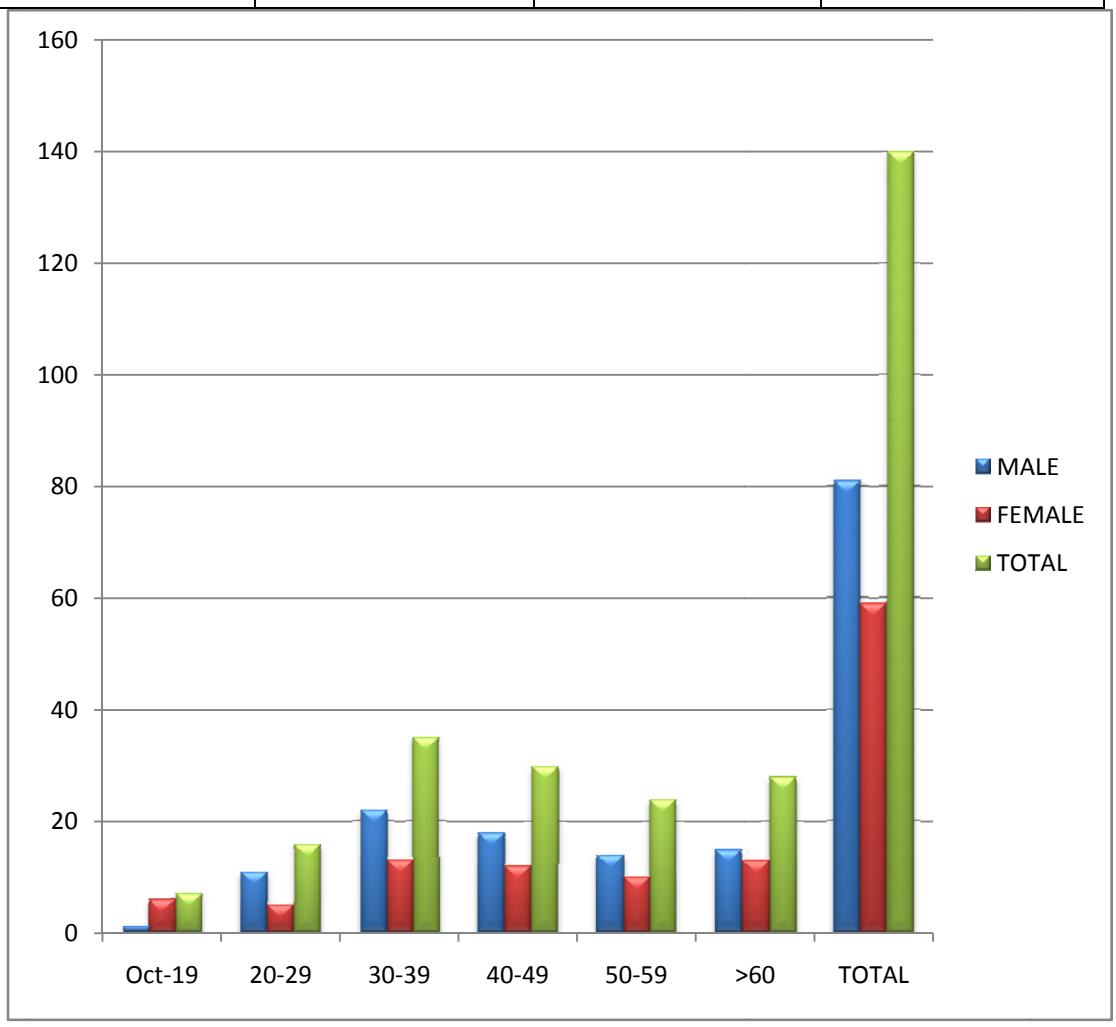


Endoscopic biopsy piece being removed from the forceps

RESULTS

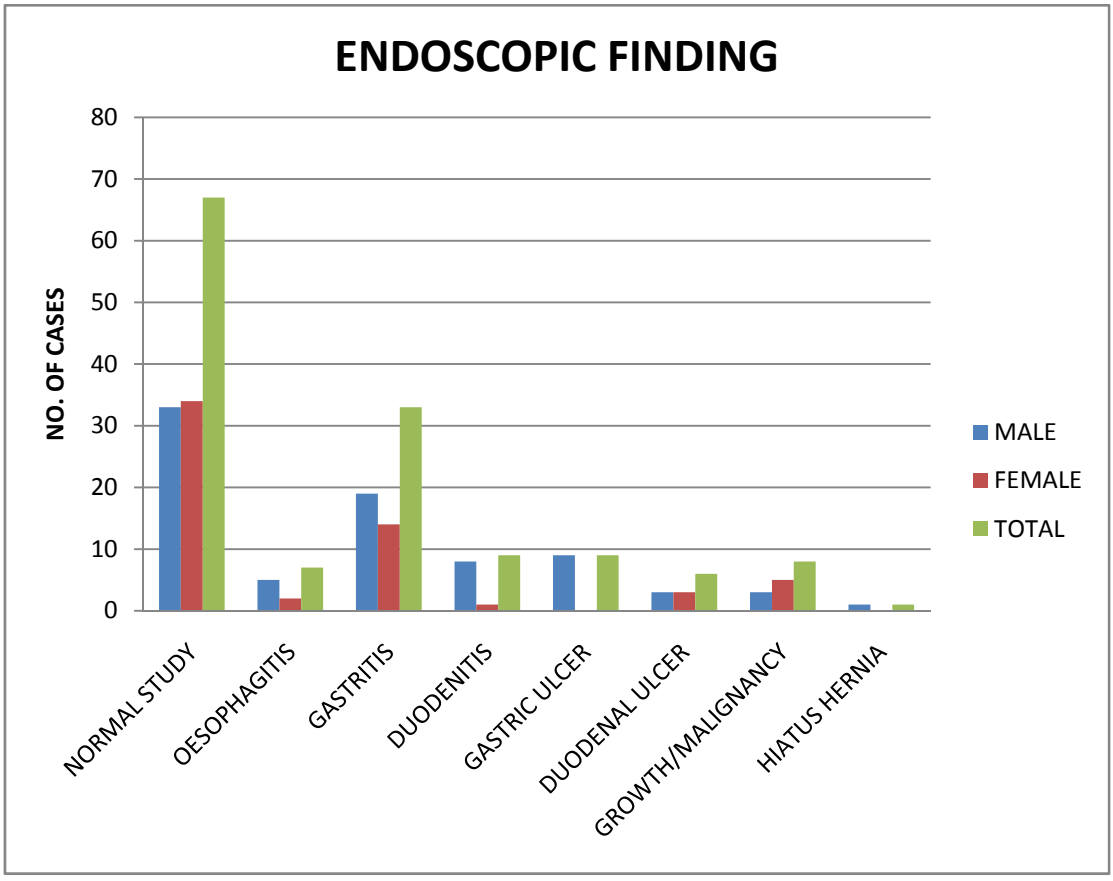
AGE AND SEX PREVALENCE IN PATIENTS PRESENTING WITH DYSPEPSIA

AGE/SEX	MALE	FEMALE	TOTAL
10-19	1	6	7
20-29	11	5	16
30-39	22	13	35
40-49	18	12	30
50-59	14	10	24
>60	15	13	28
TOTAL	81	59	140



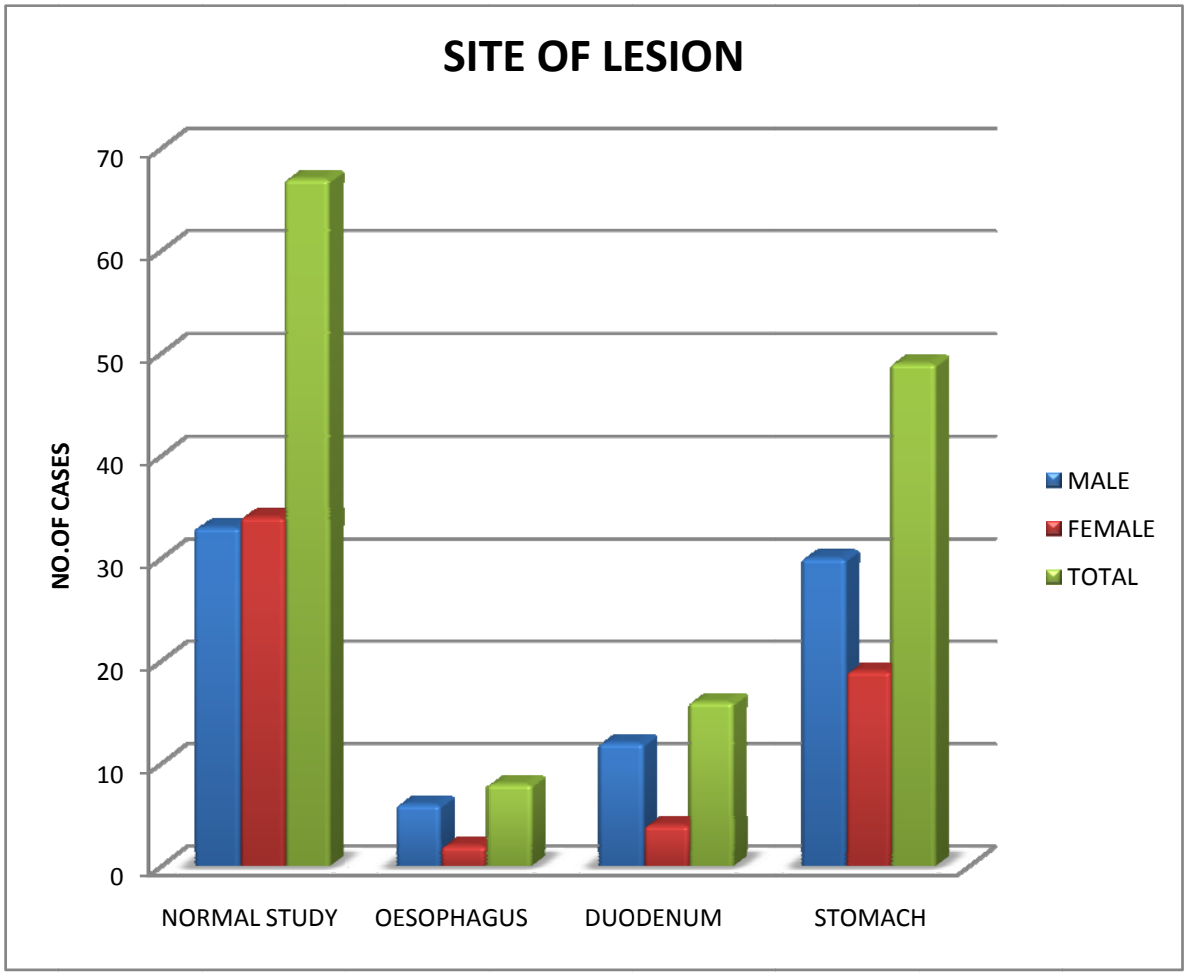
ENDOSCOPIC FINDING IN PATIENTS WITH DYSPEPSIA

FINDINGS	MALE	FEMALE	TOTAL
NORMAL STUDY	33	34	67
OESOPHAGITIS	5	2	7
GASTRITIS	19	14	33
DUODENITIS	8	1	9
GASTRIC ULCER	9	0	9
DUODENAL ULCER	3	3	6
GROWTH/MALIGNANCY	3	5	8
HIATUS HERNIA	1	0	1
TOTAL	81	59	140



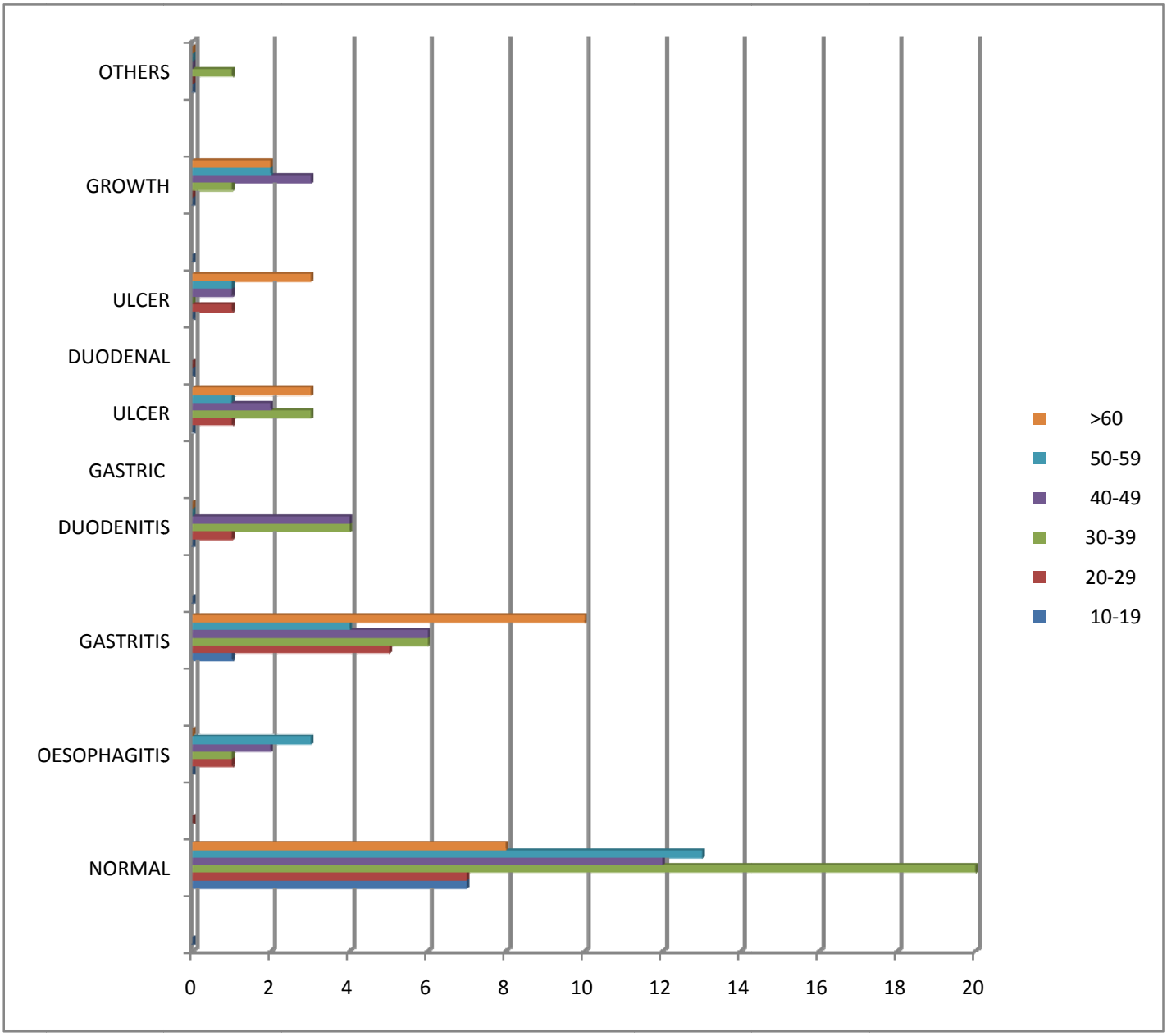
SITE OF LESION IN ENDOSCOPY PRESENTING WITH DYSPEPSIA

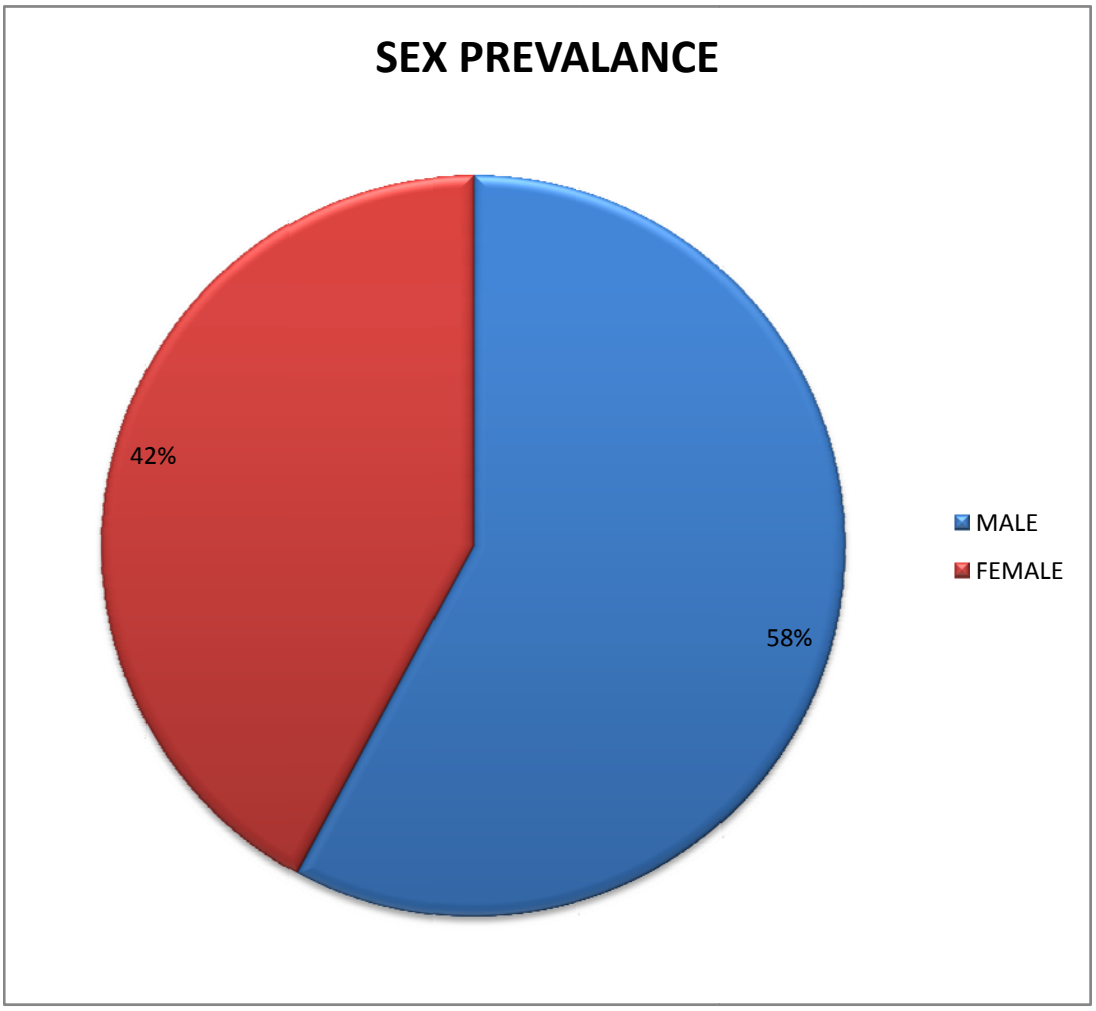
SITE	MALE	FEMALE	TOTAL
NORMAL STUDY	33	34	67
OESOPHAGUS	6	2	8
STOMACH	30	19	49
DUODENUM	12	4	16



AGE WISE PRESENTATION OF DYSPEPSIA

FINDINGS	10-19	20-29	30-39	40-49	50-59	>60
NORMAL	7	7	20	12	13	8
OESOPHAGITIS	0	1	1	2	3	0
GASTRITIS	1	5	6	6	4	10
DUODENITIS	0	1	4	4	0	0
GASTRIC ULCER	0	1	3	2	1	3
DUODENAL ULCER	0	1	0	1	1	3
GROWTH	0	0	1	3	2	2
OTHERS	0	0	1	0	0	0


OTHERS
GROWTH
ULCER
DUODENAL
ULCER
GASTRIC
DUODENITIS
GASTRITIS
OESOPHAGITIS
NORMAL
0
2
4
6
8
10
12
14
16
18
20
>60
50-59
40-49
30-39
20-29
10-19


SEX PREVALANCE
42%
58%
MALE
FEMALE

DISCUSSION

A prospective clinico-pathological study entitled "A Clinical study of various findings in upper gastro-intestinal endoscopy in patients presenting with dyspepsia" was undertaken in Tirunelveli medical college hospital to study the endoscopic findings of dyspepsia and to detect esophagogastroduodenal carcinoma at early stages.

After informed consent 140 cases of dyspepsia were included in the study and were studied clinically as per the proforma from march 2011 to October 2012. All the patients underwent upper gastro-intestinal endoscopy and various findings were noted.

CLINICAL PRESENTATION:

Out of 140 patients, 112 (78.9%) patients had epigastric pain and discomfort as their chief complaint where as nausea and vomiting was present in 100 (72.8%) patients. The other complaints were heart burn 85 (59.9%), food intolerance 70(49.3%), indigestion 65(45.8%) and loss of appetite and weight 47(33%).

Similar study was conducted by Thomson A B R et al, in which the common presenting complaints were upper abdominal pain (34.3%), heart burn (24.5%) and acid regurgitation (13.3%), the observations were comparable with that of the present study.

COMPARISON OF GENDER DISTRIBUTION

In this study 58% were male patients, 42% were female patients. The incidence of different presentations of dyspepsia were common in males compared to females. Only the incidence of esophagogastritis was more in female patients.

The male / female ratio in the studies conducted by Khan N et al – 2.3:1, Ziauddin- 1.6:1, Mustapha SK et al- 1.1:1 respectively. In these studies also the majority of patients were males as observed in our study.

In a population based study in Australia, female adults significantly out numbered males in most functional gastrointestinal disorders includes functional dyspepsia.

COMPARISON OF VARIOUS ENDOSCOPIC FINDINGS:

In the present study, clinically significant endoscopic findings were observed in 73 patients accounting for 52.14%. Gastritis was by far the most common finding (23.6%). The next common findings were duodenitis, and gastric ulcer accounting for 6.4% each.

The percentage of cases with gastritis in this study was higher than that observed in studies by Sarwar et al and Ziauddin. The percentage of patients with GERD was nearly equal to that observed by Sarwar et al.

Table 13. Comparison of common endoscopic findings in various studies

SI.No	Name of the study	Gastritis
1	Sarwar et al.[39]	13%
2	Ziauddin[40]	18%
3	Present study	23.6%

COMPARISON OF INCIDENCE OF GASTRIC MALIGNANCIES:

In this study there were 8 patients with carcimona stomach accounting for 5.7%, among them which 3 were male patients and 5 were female patients. Gastric malignancies were common in older age groups. Incidence of gastric malignancies observed by various authors are as follows:

Table 16. Comparison of incidence of gastric malignancies

Sl.NO	NAME OF STUDY	PERCENTAGE OF GASTRIC MALIGNANCIES
1	Chadwick P et al.[5]	1%
2	Khan N et al.[6]	3%
3	Ziauddin[40]	4%
4	Present study	5.7%

The incidence of gastric malignancy in these studies is comparable with that observed in the present study.

CONCLUSION

From the present study of “A clinical study of various findings in upper gastro-intestinal endoscopy in patients presenting with dyspepsia ”.

On endoscopic examination gastritis accounted for the majority of the cases. Incidence of malignancy in the present study was observed to be 5.7% (gastric malignancies).

Clinically significant endoscopic findings were observed in 52.14% of patients with uninvestigated dyspepsia. Most patients presented with a complex of three or more 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 prospective clinico-pathological study was undertaken in Tirunelveli medical college hospital to know the various endoscopic findings in patients presenting with dyspepsia and early detection of oesophagogastroduodenal malignancy in these patients.

140 patients presenting with dyspepsia were evaluated.

The following were the observations:

1. Highest prevalence of dyspepsia in the age group of 30-39years
2. Most common presenting complaint was epigastric pain and discomfort
3. Dyspepsia was more common in males (58%) when compared to females(42%)
4. Most common endoscopic finding was normal study followed by gastritis
5. Malignancy was diagnosed in 5.7% patients with dyspepsia.
6. Stomach is the common site of lesion in patients presenting with dyspepsia
7. Gastritis, duodenitis and gastric ulcer is common in males while malignancy/growth is more common in females presenting with dyspepsia.
8. 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
a) 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.
Deep tender spot.
Lump/Mass.
Free fluid.
Organomegaly.

Other systems:

RS, CVS, CNS.

Clinical Diagnosis:

Endoscopic findings:

Other investigations if any:

MASTER CHART

S No.	PATIENT NAME	AGE	SEX	OP/IP No.	CLINICAL FEATURES						CLINICAL DIAGNOSIS	ENDOSCOPIC FINDINGS
					EP	HB	N/V	FI	IDG	LW/A		
1	VALLITHAI	65	F	53088	Y	Y	Y	Y	Y	N	DYSPEPSIA	DU
2	MANI	28	M	53837	Y	N	Y	Y	N	N	DYSPEPSIA	GU
3	KARTHIK	45	M	55946	Y	Y	Y	Y	Y	N	DYSPEPSIA	Ds
4	RAJASEKAR	25	M	27602	Y	N	Y	Y	N	N	DYSPEPSIA	Gs
5	SUNDARI	52	F	57631	N	Y	N	N	Y	Y	DYSPEPSIA	NS
6	PEER MOHAMED	48	M	7323	Y	Y	Y	N	Y	N	DYSPEPSIA	Ds
7	MASANAM	72	M	35553	N	N	Y	Y	N	Y	DYSPEPSIA	GU
8	PADMAVATHY	66	F	9908	Y	Y	N	Y	Y	N	DYSPEPSIA	Gs
9	PETCHIAMMAL	43	F	4081	N	Y	Y	N	Y	Y	DYSPEPSIA	GROWTH

10	**MOHAMED SALIM**	**32**	**M**	**41124**	**Y**	**Y**	**N**	**Y**	**Y**	**N**	**DYSPEPSIA**	**GU**
11	**PANDIAN**	**45**	**M**	**13718**	**Y**	**N**	**N**	**Y**	**Y**	**N**	**DYSPEPSIA**	**Es**
12	**BANUMATHY**	**55**	**F**	**20023**	**N**	**N**	**Y**	**N**	**Y**	**Y**	**DYSPEPSIA**	**NS**
13	**MADASAMY**	**59**	**M**	**21069**	**Y**	**Y**	**N**	**Y**	**Y**	**N**	**DYSPEPSIA**	**Es**
14	**PERUMAL**	**55**	**M**	**21100**	**Y**	**N**	**Y**	**Y**	**Y**	**N**	**DYSPEPSIA**	**GU**
15	**KRISHNAMMAL**	**60**	**F**	**22692**	**Y**	**N**	**Y**	**N**	**N**	**Y**	**DYSPEPSIA**	**NS**
16	**THAVIDHU**	**70**	**M**	**22037**	**Y**	**N**	**Y**	**N**	**Y**	**Y**	**DYSPEPSIA**	**NS**
17	**GANGADHARAN**	**50**	**M**	**22729**	**Y**	**Y**	**Y**	**Y**	**N**	**N**	**DYSPEPSIA**	**NS**
18	**ESAKKIAMMAL**	**18**	**F**	**23384**	**Y**	**N**	**N**	**Y**	**Y**	**N**	**DYSPEPSIA**	**NS**
19	**SHANMUGATHAI**	**46**	**F**	**81698**	**Y**	**N**	**Y**	**Y**	**N**	**N**	**DYSPEPSIA**	**NS**
20	**MUTHULAKSMI**	**45**	**F**	**22302**	**Y**	**Y**	**Y**	**N**	**N**	**N**	**DYSPEPSIA**	**NS**
21	**POOSAM**	**30**	**M**	**26073**	**Y**	**N**	**Y**	**N**	**Y**	**Y**	**DYSPEPSIA**	**NS**
22	**MUTHU ESAKKI**	**30**	**F**	**23727**	**Y**	**N**	**Y**	**N**	**N**	**N**	**DYSPEPSIA**	**NS**
23	**VEEMAN**	**46**	**M**	**26315**	**Y**	**Y**	**N**	**Y**	**Y**	**N**	**DYSPEPSIA**	**GU**

24	**ANNATHAI**	**16**	**F**	**27462**	**Y**	**N**	**Y**	**Y**	**N**	**N**	**DYSPEPSIA**	**NS**
25	**RATHINA PAUL**	**67**	**M**	**109317**	**Y**	**N**	**Y**	**N**	**Y**	**N**	**DYSPEPSIA**	**GU**
26	**RASOOL BEEVI**	**36**	**F**	**28706**	**Y**	**Y**	**Y**	**N**	**N**	**N**	**DYSPEPSIA**	**NS**
27	**MUNIAPPAN**	**43**	**M**	**112095**	**Y**	**Y**	**N**	**N**	**Y**	**Y**	**DYSPEPSIA**	**NS**
28	**PANDI**	**50**	**M**	**29818**	**Y**	**Y**	**Y**	**N**	**N**	**N**	**DYSPEPSIA**	**NS**
29	**EUACHI**	**27**	**M**	**111384**	**Y**	**N**	**Y**	**Y**	**N**	**N**	**DYSPEPSIA**	**NS**
30	**KASIPANDI**	**50**	**M**	**30734**	**Y**	**Y**	**Y**	**N**	**Y**	**Y**	**DYSPEPSIA**	**Gs**
31	**SUNDAR**	**35**	**M**	**31249**	**Y**	**N**	**Y**	**N**	**Y**	**Y**	**DYSPEPSIA**	**NS**
32	**SHANMUGAVADIVU**	**60**	**F**	**31722**	**Y**	**N**	**N**	**N**	**Y**	**Y**	**DYSPEPSIA**	**GROWTH**
33	**MARIAPPAN**	**23**	**M**	**27989**	**Y**	**Y**	**N**	**Y**	**N**	**N**	**DYSPEPSIA**	**Gs**
34	**MURUGAN**	**27**	**M**	**31235**	**Y**	**Y**	**Y**	**N**	**N**	**Y**	**DYSPEPSIA**	**NS**
35	**LAKSHMI**	**55**	**F**	**33888**	**Y**	**N**	**Y**	**N**	**Y**	**N**	**DYSPEPSIA**	**GROWTH**
36	**JEEMNA**	**19**	**F**	**35359**	**Y**	**Y**	**N**	**N**	**Y**	**N**	**DYSPEPSIA**	**NS**
37	**SIVAGURUNATHAN**	**55**	**M**	**136352**	**Y**	**N**	**Y**	**N**	**N**	**Y**	**DYSPEPSIA**	**NS**

38	**EASWARAN**	**52**	**M**	**129474**	**N**	**Y**	**N**	**Y**	**N**	**Y**	**DYSPEPSIA**	**NS**
39	**SUNDARAJAN**	**39**	**M**	**36736**	**N**	**Y**	**Y**	**N**	**Y**	**N**	**DYSPEPSIA**	**Gs**
40	**SURESH**	**19**	**M**	**38058**	**N**	**Y**	**N**	**Y**	**N**	**N**	**DYSPEPSIA**	**NS**
41	**AYYADURAI**	**38**	**M**	**37975**	**Y**	**N**	**Y**	**N**	**Y**	**N**	**DYSPEPSIA**	**GROWTH**
42	**SEKAR**	**34**	**M**	**37886**	**Y**	**Y**	**Y**	**N**	**N**	**N**	**DYSPEPSIA**	**NS**
43	**PATTU**	**60**	**F**	**37268**	**N**	**Y**	**N**	**Y**	**Y**	**N**	**DYSPEPSIA**	**NS**
44	**ANGAMMAL**	**41**	**F**	**38554**	**N**	**Y**	**Y**	**N**	**Y**	**N**	**DYSPEPSIA**	**GROWTH**
45	**ESAKKIMUTHU**	**30**	**M**	**38551**	**Y**	**N**	**Y**	**Y**	**N**	**N**	**DYSPEPSIA**	**NS**
46	**LAKSHMI**	**60**	**F**	**140632**	**Y**	**N**	**Y**	**Y**	**N**	**Y**	**DYSPEPSIA**	**NS**
47	**THANGARAJ**	**60**	**M**	**38669**	**N**	**N**	**Y**	**Y**	**Y**	**N**	**DYSPEPSIA**	**Gs**
48	**GURUNATHAN**	**65**	**M**	**40079**	**Y**	**Y**	**N**	**Y**	**N**	**N**	**DYSPEPSIA**	**Gs**
49	**SINGARAJ**	**33**	**M**	**39651**	**N**	**N**	**Y**	**N**	**Y**	**Y**	**DYSPEPSIA**	**NS**
50	**RAVI**	**26**	**M**	**47346**	**Y**	**Y**	**N**	**Y**	**N**	**N**	**DYSPEPSIA**	**Ds**
51	**ARUNACHALAM**	**70**	**M**	**41562**	**Y**	**Y**	**Y**	**N**	**N**	**N**	**DYSPEPSIA**	**GU**

52	**LAKSHMIAMMAL**	**80**	**F**	**42261**	**N**	**N**	**Y**	**Y**	**Y**	**Y**	**DYSPEPSIA**	**NS**
53	**SHAHUL HAMEED**	**55**	**M**	**42483**	**Y**	**N**	**N**	**Y**	**Y**	**Y**	**DYSPEPSIA**	**DU**
54	**YOGAMMAL**	**60**	**F**	**4240**	**Y**	**N**	**Y**	**Y**	**N**	**N**	**DYSPEPSIA**	**NS**
55	**SELVI**	**43**	**F**	**42836**	**Y**	**Y**	**Y**	**N**	**N**	**N**	**DYSPEPSIA**	**GROWTH**
56	**KANAGARAJ**	**58**	**M**	**45537**	**Y**	**Y**	**Y**	**N**	**N**	**Y**	**DYSPEPSIA**	**GROWTH**
57	**MARIAPPAN**	**57**	**M**	**45023**	**N**	**Y**	**Y**	**Y**	**N**	**N**	**DYSPEPSIA**	**Es**
58	**PERIYASAMY**	**68**	**M**	**44988**	**Y**	**Y**	**N**	**Y**	**N**	**N**	**DYSPEPSIA**	**Gs**
59	**PETCHIMUTHU**	**39**	**M**	**47164**	**Y**	**Y**	**Y**	**N**	**N**	**N**	**DYSPEPSIA**	**NS**
60	**ANANDHARAJ**	**17**	**M**	**47380**	**N**	**Y**	**Y**	**N**	**Y**	**N**	**DYSPEPSIA**	**Gs**
61	**JEYARANI**	**19**	**F**	**47429**	**N**	**Y**	**Y**	**N**	**Y**	**Y**	**DYSPEPSIA**	**NS**
62	**KALIMUTHU**	**36**	**M**	**48010**	**Y**	**Y**	**N**	**N**	**N**	**Y**	**DYSPEPSIA**	**NS**
63	**SANKAR**	**48**	**M**	**47933**	**Y**	**Y**	**N**	**Y**	**Y**	**N**	**DYSPEPSIA**	**Ds**
64	**GANAPATHYAMMAL**	**58**	**F**	**49079**	**N**	**Y**	**Y**	**N**	**Y**	**N**	**DYSPEPSIA**	**Gs**
65	**AAMINA BEGAM**	**37**	**F**	**191513**	**Y**	**Y**	**Y**	**N**	**N**	**N**	**DYSPEPSIA**	**NS**

66	**ANNATHAI**	**40**	**F**	**50699**	**Y**	**Y**	**N**	**N**	**Y**	**N**	**DYSPEPSIA**	**NS**
67	**MUTHUKRISHNAN**	**46**	**M**	**51929**	**N**	**Y**	**Y**	**N**	**Y**	**N**	**DYSPEPSIA**	**NS**
68	**MANDIRAN**	**45**	**M**	**51873**	**Y**	**N**	**Y**	**Y**	**N**	**N**	**DYSPEPSIA**	**NS**
69	**AMMAPONNU**	**45**	**F**	**53172**	**Y**	**Y**	**N**	**Y**	**N**	**N**	**DYSPEPSIA**	**DU**
70	**PARAMASIVAM**	**45**	**M**	**52753**	**Y**	**N**	**Y**	**Y**	**N**	**N**	**DYSPEPSIA**	**NS**
71	**KANNAN**	**30**	**M**	**52163**	**Y**	**N**	**Y**	**N**	**Y**	**Y**	**DYSPEPSIA**	**NS**
72	**MARIAPPAN**	**33**	**M**	**56531**	**Y**	**Y**	**Y**	**N**	**N**	**Y**	**DYSPEPSIA**	**Es**
73	**MUTHUPETCHI**	**30**	**F**	**222176**	**Y**	**Y**	**Y**	**Y**	**N**	**N**	**DYSPEPSIA**	**NS**
74	**CHANDRAN**	**31**	**M**	**57767**	**Y**	**Y**	**Y**	**N**	**N**	**N**	**DYSPEPSIA**	**GU**
75	**UDHAYAKUMAR**	**35**	**M**	**57803**	**Y**	**N**	**Y**	**Y**	**N**	**N**	**DYSPEPSIA**	**Ds**
76	**ARUMUGAM**	**43**	**M**	**59361**	**N**	**Y**	**Y**	**Y**	**N**	**N**	**DYSPEPSIA**	**NS**
77	**RAJAKUMARI**	**48**	**F**	**61574**	**Y**	**Y**	**Y**	**N**	**N**	**N**	**DYSPEPSIA**	**Gs**
78	**KANDAN**	**70**	**M**	**61047**	**Y**	**Y**	**N**	**Y**	**Y**	**N**	**DYSPEPSIA**	**NS**
79	**MURUGAN**	**33**	**M**	**2046**	**Y**	**N**	**N**	**Y**	**Y**	**Y**	**DYSPEPSIA**	**NS**

80	**CHELLAPPA**	**70**	**M**	**4154**	**Y**	**N**	**Y**	**Y**	**Y**	**N**	**DYSPEPSIA**	**Gs**
81	**PADMAVATHI**	**26**	**F**	**4273**	**N**	**Y**	**Y**	**N**	**Y**	**Y**	**DYSPEPSIA**	**NS**
82	**RAMALAKSHMI**	**45**	**F**	**3623**	**Y**	**Y**	**Y**	**N**	**N**	**N**	**DYSPEPSIA**	**NS**
83	**RAHMATH NISHA**	**35**	**F**	**4614**	**Y**	**N**	**Y**	**Y**	**N**	**Y**	**DYSPEPSIA**	**NS**
84	**MURUGAN**	**38**	**M**	**4723**	**Y**	**N**	**Y**	**Y**	**N**	**N**	**DYSPEPSIA**	**Gs**
85	**NAGESHWARI**	**27**	**F**	**17665**	**Y**	**N**	**Y**	**Y**	**Y**	**Y**	**DYSPEPSIA**	**Ds**
86	**MARIAPPAN**	**31**	**M**	**2913**	**Y**	**N**	**Y**	**N**	**Y**	**Y**	**DYSPEPSIA**	**NS**
87	**RAMAIAH**	**65**	**M**	**24204**	**Y**	**Y**	**Y**	**N**	**N**	**N**	**DYSPEPSIA**	**GROWTH**
88	**ANITHA**	**34**	**F**	**9150**	**Y**	**N**	**Y**	**Y**	**N**	**Y**	**DYSPEPSIA**	**Gs**
89	**MARIYAM BEEVI**	**70**	**F**	**12130**	**N**	**N**	**Y**	**Y**	**Y**	**Y**	**DYSPEPSIA**	**Gs**
90	**EDWARD**	**25**	**M**	**15681**	**Y**	**Y**	**N**	**Y**	**N**	**N**	**DYSPEPSIA**	**NS**
91	**MARIMUTHU**	**32**	**M**	**53104**	**Y**	**Y**	**N**	**Y**	**N**	**N**	**DYSPEPSIA**	**Ds**
92	**MANIAMMAL**	**45**	**F**	**19183**	**N**	**Y**	**Y**	**Y**	**N**	**N**	**DYSPEPSIA**	**Es**
93	**PUSHPAM**	**50**	**F**	**17297**	**Y**	**Y**	**N**	**Y**	**N**	**Y**	**DYSPEPSIA**	**NS**

94	**MADASAMY**	**60**	**M**	**19533**	**Y**	**Y**	**Y**	**N**	**N**	**N**	**DYSPEPSIA**	**DU**
95	**SELVIMENAKA**	**39**	**F**	**20966**	**N**	**N**	**Y**	**Y**	**Y**	**Y**	**DYSPEPSIA**	**Gs**
96	**VASANTHI**	**22**	**F**	**24176**	**Y**	**Y**	**N**	**Y**	**N**	**N**	**DYSPEPSIA**	**Gs**
97	**VIGNESHWARAN**	**30**	**M**	**25624**	**N**	**Y**	**Y**	**Y**	**N**	**N**	**DYSPEPSIA**	**NS**
98	**SANTHANAMARI**	**55**	**F**	**32389**	**Y**	**Y**	**N**	**Y**	**Y**	**N**	**DYSPEPSIA**	**Gs**
99	**DEVIKA**	**25**	**F**	**87602**	**Y**	**Y**	**N**	**Y**	**N**	**Y**	**DYSPEPSIA**	**NS**
100	**SIVAGAMI**	**65**	**F**	**35834**	**Y**	**Y**	**Y**	**N**	**N**	**N**	**DYSPEPSIA**	**NS**
101	**SIVAKUMAR**	**20**	**M**	**46749**	**Y**	**Y**	**Y**	**N**	**Y**	**N**	**DYSPEPSIA**	**NS**
102	**MURUGAN**	**46**	**M**	**46813**	**Y**	**N**	**Y**	**N**	**Y**	**Y**	**DYSPEPSIA**	**GU**
103	**PALANIRAJ**	**42**	**M**	**43076**	**Y**	**Y**	**Y**	**N**	**Y**	**N**	**DYSPEPSIA**	**Ds**
104	**ARUMUGASAMY**	**47**	**M**	**160410**	**Y**	**N**	**Y**	**N**	**Y**	**Y**	**DYSPEPSIA**	**NS**
105	**VIJAYASHREE**	**32**	**F**	**46612**	**Y**	**N**	**Y**	**Y**	**N**	**Y**	**DYSPEPSIA**	**NS**
106	**CHELLAMMAL**	**65**	**F**	**43431**	**N**	**Y**	**Y**	**Y**	**N**	**N**	**DYSPEPSIA**	**NS**
107	**ESWARAN**	**63**	**M**	**37234**	**Y**	**Y**	**Y**	**N**	**N**	**N**	**DYSPEPSIA**	**Gs**

108	**PATTAMMAL**	**50**	**F**	**38480**	**N**	**Y**	**Y**	**N**	**Y**	**Y**	**DYSPEPSIA**	**NS**
109	**SENTHIL**	**32**	**M**	**41496**	**Y**	**N**	**Y**	**N**	**Y**	**Y**	**DYSPEPSIA**	**Ds**
110	**KANAGAMANI**	**45**	**F**	**100743**	**Y**	**Y**	**N**	**Y**	**Y**	**N**	**DYSPEPSIA**	**NS**
111	**MANIKANDAN**	**21**	**M**	**98236**	**Y**	**N**	**Y**	**Y**	**N**	**N**	**DYSPEPSIA**	**Gs**
112	**GOMATHINAYAGAN**	**57**	**M**	**89856**	**Y**	**Y**	**Y**	**N**	**N**	**Y**	**DYSPEPSIA**	**NS**
113	**ANWAR**	**36**	**M**	**33234**	**Y**	**N**	**Y**	**Y**	**N**	**N**	**DYSPEPSIA**	**Gs**
114	**SARASWATHI**	**50**	**F**	**35240**	**N**	**N**	**N**	**N**	**N**	**N**	**DYSPEPSIA**	**NS**
115	**AMAICHAR**	**46**	**F**	**91225**	**Y**	**N**	**Y**	**N**	**Y**	**Y**	**DYSPEPSIA**	**Gs**
116	**SELVAKUMAR**	**41**	**M**	**19658**	**Y**	**Y**	**N**	**Y**	**N**	**Y**	**DYSPEPSIA**	**Gs**
117	**SORNAMMAL**	**59**	**F**	**19678**	**Y**	**N**	**Y**	**N**	**Y**	**Y**	**DYSPEPSIA**	**NS**
118	**KARUPPAIAH**	**70**	**M**	**22279**	**Y**	**Y**	**Y**	**N**	**N**	**N**	**DYSPEPSIA**	**NS**
119	**BABU**	**37**	**M**	**24718**	**Y**	**Y**	**Y**	**N**	**N**	**N**	**DYSPEPSIA**	**Gs**
120	**SAGUNTHALA**	**19**	**F**	**25537**	**Y**	**Y**	**N**	**N**	**Y**	**N**	**DYSPEPSIA**	**NS**
121	**RAJALAKSHMI**	**33**	**F**	**25488**	**Y**	**Y**	**N**	**Y**	**N**	**N**	**DYSPEPSIA**	**NS**

122	**MURUGAN**	**54**	**M**	**12443**	**N**	**Y**	**Y**	**N**	**Y**	**N**	**DYSPEPSIA**	**Gs**
123	**PERUMALAMMAL**	**45**	**F**	**122955**	**Y**	**N**	**Y**	**Y**	**Y**	**N**	**DYSPEPSIA**	**Gs**
124	**CHELLAMMAL**	**65**	**F**	**12630**	**N**	**Y**	**Y**	**Y**	**N**	**N**	**DYSPEPSIA**	**Gs**
125	**KUMAR**	**40**	**M**	**15947**	**Y**	**N**	**Y**	**N**	**Y**	**Y**	**DYSPEPSIA**	**NS**
126	**MUTHUSAMY**	**43**	**M**	**18033**	**Y**	**Y**	**Y**	**N**	**N**	**N**	**DYSPEPSIA**	**Gs**
127	**MARIMUTHU**	**32**	**M**	**53104**	**Y**	**Y**	**Y**	**N**	**N**	**N**	**DYSPEPSIA**	**Ds**
128	**KANAGA**	**21**	**F**	**54811**	**Y**	**Y**	**N**	**N**	**Y**	**N**	**DYSPEPSIA**	**Es**
129	**JEGANMOHAN**	**33**	**M**	**28419**	**N**	**Y**	**Y**	**Y**	**N**	**N**	**DYSPEPSIA**	**HH**
130	**SUDALAIKANI**	**65**	**F**	**25074**	**Y**	**Y**	**N**	**N**	**Y**	**N**	**DYSPEPSIA**	**Gs**
131	**SHAHUL HAMEED**	**53**	**M**	**94385**	**Y**	**Y**	**Y**	**N**	**N**	**N**	**DYSPEPSIA**	**Es**
132	**ISMAIL MEERA**	**18**	**F**	**9422**	**Y**	**N**	**Y**	**Y**	**Y**	**N**	**DYSPEPSIA**	**Gs**
133	**PERUMAL**	**53**	**M**	**11451**	**Y**	**N**	**Y**	**Y**	**N**	**Y**	**DYSPEPSIA**	**NS**
134	**SULOCHANA**	**65**	**F**	**11284**	**N**	**Y**	**N**	**Y**	**N**	**Y**	**DYSPEPSIA**	**DU**
135	**KANAGAVALLI**	**24**	**F**	**5317**	**Y**	**Y**	**N**	**Y**	**N**	**N**	**DYSPEPSIA**	**NS**

136	**GANESH**	**45**	**M**	**38557**	**Y**	**Y**	**Y**	**N**	**N**	**N**	**DYSPEPSIA**	**Gs**
137	**MAHARAJA**	**21**	**M**	**6094**	**Y**	**N**	**Y**	**N**	**Y**	**N**	**DYSPEPSIA**	**Gs**
138	**REGITHA**	**33**	**M**	**6467**	**Y**	**Y**	**Y**	**N**	**N**	**N**	**DYSPEPSIA**	**NS**
139	**GNANVELAMMAL**	**55**	**F**	**4462**	**Y**	**Y**	**N**	**Y**	**N**	**N**	**DYSPEPSIA**	**NS**
140	**MAYILAMMAL**	**30**	**F**	**5875**	**Y**	**N**	**Y**	**N**	**Y**	**N**	**DYSPEPSIA**	**Gs**

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.