A STUDY OF CLINICAL PROFILE OF PATIENTS WITH HIATUS HERNIA

Submitted by

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Dissertation submitted to

B.L.D.E (Deemed to be University)'s
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In partial fulfilment of the requirements for the degree of

MASTER OF SURGERY

In

GENERAL SURGERY

Under the guidance of

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LIST OF ABBREVIATIONS

BMI	Body Mass Index
GI	Gastro-intestinal
GERD	Gastro Esophageal Reflux Disease
GEJ	Gastro Esophageal Junction
T11	11^{th} Thoracic Vertebra
C6	6^{th} Cervical Vertebra
LES	Lower Esophageal Sphincter
tLESR	Transient Lower Esophageal Sphincter Relaxation
HRM	High Resolution Manometry
PEH	Para-esophageal Hernia
COPD	Chronic Obstructive Pulmonary Disease
CLE	Columnar-Lined Epithelium
EMS	Esophageal Motility Study
DES	Diffuse Esophageal Spasm
RIP	Respiratory Inversion Point
PPI	Proton Pump Inhibitor
pH	Potential of Hydrogen
KG	Kilogram
m^2	Meter square
CM	Centimeter
N	Number
OPD	Out-patient Department
IPD	In-patient Department
SD	Standard Deviation

ABSTRACT

AIMS AND OBJECTIVES:

To study the patient clinical profile which includes age, sex, BMI, abdominal girth, abdominal tone, the dietary habits, symptomatology and endoscopy findings of the patients with hiatus hernia.

METHODS:

This is a prospective cross-sectional observational study of 115 patients presenting with Hiatus hernia on upper GI scopy in B.L.D.E. (D.U)'S Shri B.M. Patil Medical College Hospital.

RESULTS:

Out of 115 patients, 29.6% belonged to the age group of 21-30 years, 69.6% were males and 67% had normal range of BMI. Most common presenting symptoms were epigastric pain (52.2%) and heartburn (26.1%). The most common clinical diagnosis made before upper GI endoscopy was GERD (gastro-esophageal reflux disease) (41.7%), followed by gastritis (23.5%). Reflux esophagitis (74.8%) was most commonly associated finding with hiatus hernia.

CONCLUSION:

According to older studies, hiatus hernia was considered to be a disease of old age, females and obese individuals. But not much studies have been done regarding the incidence of hiatus hernia in recent times. In this study, we concluded that incidence of hiatus hernia is more common in males, young adults and even non-obese individuals. Hiatus hernia is not significantly associated with lifestyle changes like smoking or other habits and food consumption. This in contradiction to the older studies done on hiatus hernia and signifies the changing trends of incidence of hiatus hernia.

TITLE OF THE TOPIC

STUDY OF CLINICAL PROFILE OF PATIENTS WITH HIATUS HERNIA

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INTRODUCTION

Hiatal hernia is a condition in which parts of the abdominal contents, mainly the gastro-esophageal junction (GEJ) and the stomach, are proximally displaced above the diaphragm through the oesophageal hiatus into the mediastinum. The laxity of phreno-oesophageal ligament is the main pathology behind Hiatus Hernia[1].

Hiatus hernia is one of the most prevalent defects in the gastrointestinal tract in the western world, and an upcoming concern in India. Although few instances of hiatus hernia are congenital, the hernias usually appear later in life and are considered to be acquired. Hiatus hernia has been considered to be a disease of the elderly and predominantly seen in women, but recent incidental findings suggest its increased incidence in young adults[1].

Although many data are available in the literature on prevalence of hiatus hernia, not much data has been published on the incidence of hiatus hernia. In our experience, we are routinely finding that the disease is much more prevalent in thin, young individuals and even in male patients. Hence, we propose to take up this study to evaluate the clinical profile of patients diagnosed with hiatus hernia.

AIM OF THE STUDY

To study the clinical profile of patients presenting with hiatus hernia.

OBJECTIVES OF THE STUDY

To study-

- The patient clinical profile which includes age, sex, BMI, abdominal girth, abdominal tone.
- The dietary habits of the patients with hiatus hernia.
- The symptomatology with which the patient presents.
- Endoscopy findings such as length between the incisors and lower oesophageal sphincter.
- The length of gastric mucosal extension into the oesophagus.

RESEARCH HYPOTHESIS

To study the changing trends in Hiatus Hernia.

REVIEW OF LITERATURE

Hiatal hernia is a condition in which parts of the abdominal contents, mainly the gastro-esophageal junction (GEJ) and the stomach, are proximally displaced above the diaphragm through the oesophageal hiatus into the mediastinum[1].

Oesophageal hiatus is an elliptically shaped opening most commonly formed by elements of the right diaphragmatic crus that encircles the distal portion of the oesophagus in a sling-like fashion[1]. Normally, the distal portion of the oesophagus is anchored to the oesophageal hiatus by the phreno-oesophageal ligament (also called the fascia of Laimer) that is formed by the fusion of endothoracic and endo-abdominal fascia. This ligament is essential in maintaining the competence of GEJ and preventing the migration of GEJ and/or stomach into the posterior mediastinum by sealing off potential spaces between the oesophageal hiatus and the distal portion of the oesophagus[1].

During normal swallows, the oesophageal body shortens due to the contraction of the longitudinal muscles and the phreno-oesophageal ligament is stretched. As a result, GEJ and a small part of the stomach is proximally displaced through the oesophageal hiatus[1]. At the end of each swallow, the migrated segment is brought to its normal position by the elastic recoil of the phreno-oesophageal ligament[1].

The laxity of phreno-oesophageal ligament is the main pathology behind Hiatus Hernia. The actual prevalence of the hiatus hernia in overall population is not known. Women are four times more likely to develop the hernias than men[2]. The incidence increases with advancing age.

ANATOMY OF ESOPHAGUS:

The esophagus is a muscular tube that starts at the inferior border of the cricoid cartilage in the neck and traverses the chest, ending as it enters the stomach in the upper abdomen. The esophagus is anchored superiorly to the cricoid cartilage and inferiorly to the diaphragm[2].

Its length from the cricoid cartilage at C6 to the gastric orifice at T11 ranges from 22 to 28 cm. The length of the esophagus varies with the height of the patient. The esophageal bed lies in the posterior mediastinum along the ventral surface of the vertebra. On endoscopy, the upper esophageal sphincter is approximately 15 cm from the incisors, the carina at 25 cm, and the lower esophageal sphincter at 38 to 40 cm in men and 36 to 38 cm in women[2].

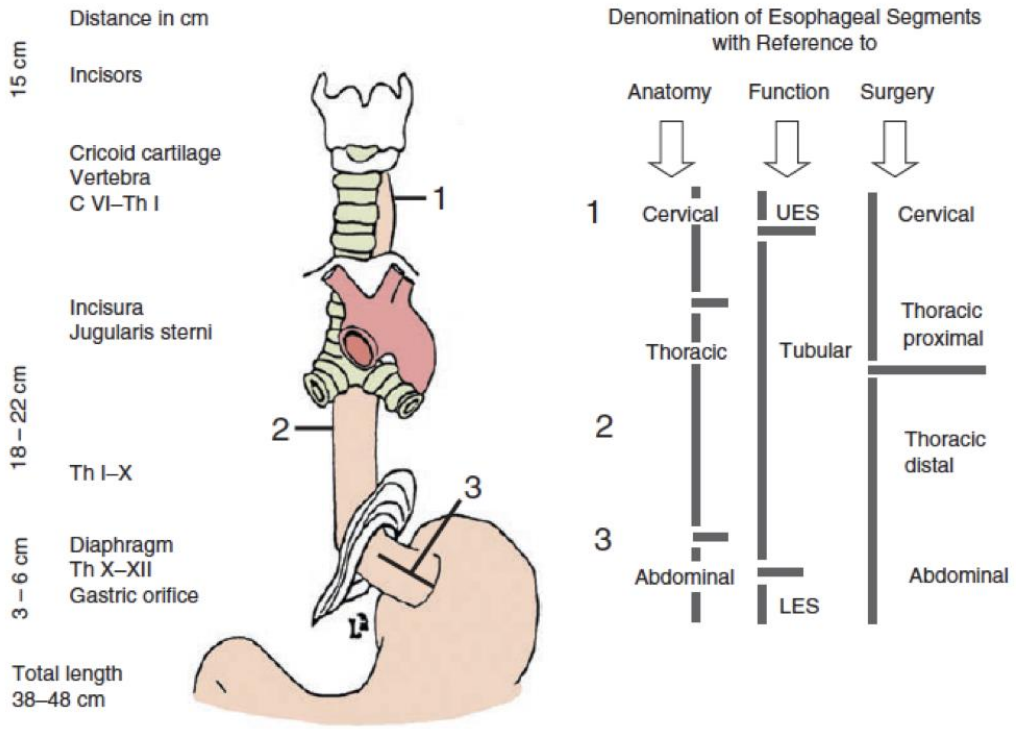


FIGURE 1

While the cervical esophagus is flattened by surrounding structures, the thoracic esophagus is more round due to negative intrathoracic pressure. Once the esophagus enters the abdomen, it becomes more flattened again as a result of positive intraabdominal pressure. The overall diameter of the esophagus is approximately 2.5 cm^2.

There are three anatomic narrowings of the esophagus. The narrowest part of the gastrointestinal tract is at the cricopharyngeal constriction at the upper esophageal sphincter, where the esophagus is 1.5 cm in diameter. In the superior

mediastinum, the aortic arch, left atrium, and left mainstem bronchus compress the left anterolateral aspect of the esophagus around 22 cm from the incisors. The third area of narrowing is at the lower esophageal sphincter[2].

The esophageal wall is composed of four layers, including an inner squamous mucosal layer, submucosa, muscularis propria, and adventitia. The mucosal layer consists of the squamous epithelium, lamina propria, and muscularis mucosa, while the muscularis propria consists of an inner circular and outer longitudinal layer of muscle[2].

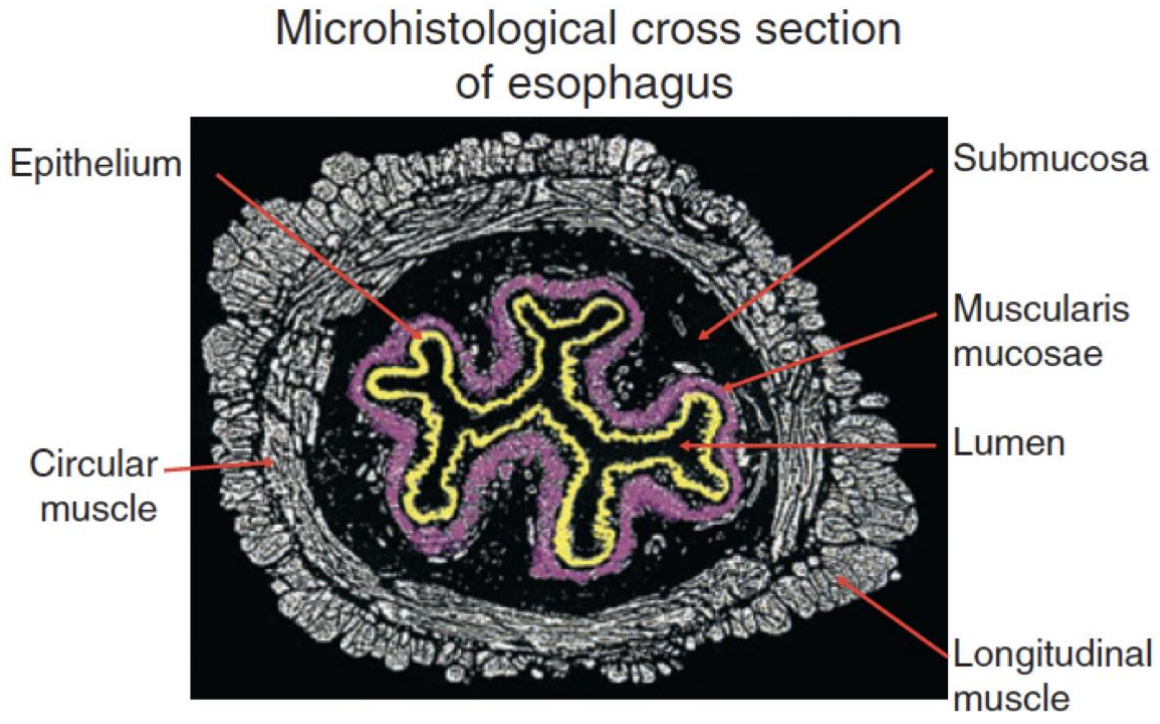


FIGURE 2

GASTROESOPHAGEAL JUNCTION:

The location of the gastroesophageal junction remains controversial and can vary depending on the criteria used, which can be based on histologic, endoscopic, or surgical findings[2].

Histologically, the gastroesophageal junction has been defined as the point where there are no longer any submucosal esophageal glands or the proximal extent of the gastric oxyntic glands[2].

Endoscopically, the junction has been defined as the squamocolumnar junction, or Z-line, and the proximal extent of the gastric mucosal folds[2].

Surgically, the gastroesophageal junction has been identified as the peritoneal reflection on the stomach and the junction of the tubular esophagus and the stomach[2].

LOWER ESOPHAGEAL SPHINCTER:

Approximately 3 cm proximal to the gastroesophageal junction, the circular muscle layer increases in thickness. Although there is no palpable circular sphincter at the gastroesophageal junction, the circular fibers become short muscular clasps along the lesser curvature. Along the greater curvature and the angle of His, the circular fibers become oblique gastric sling fibers. This muscular arrangement extends 3 to 4 cm above the gastroesophageal junction

and 1 to 2 cm onto the stomach, forming a high-pressure zone allowing the lower esophageal sphincter to be identified on manometry[2].

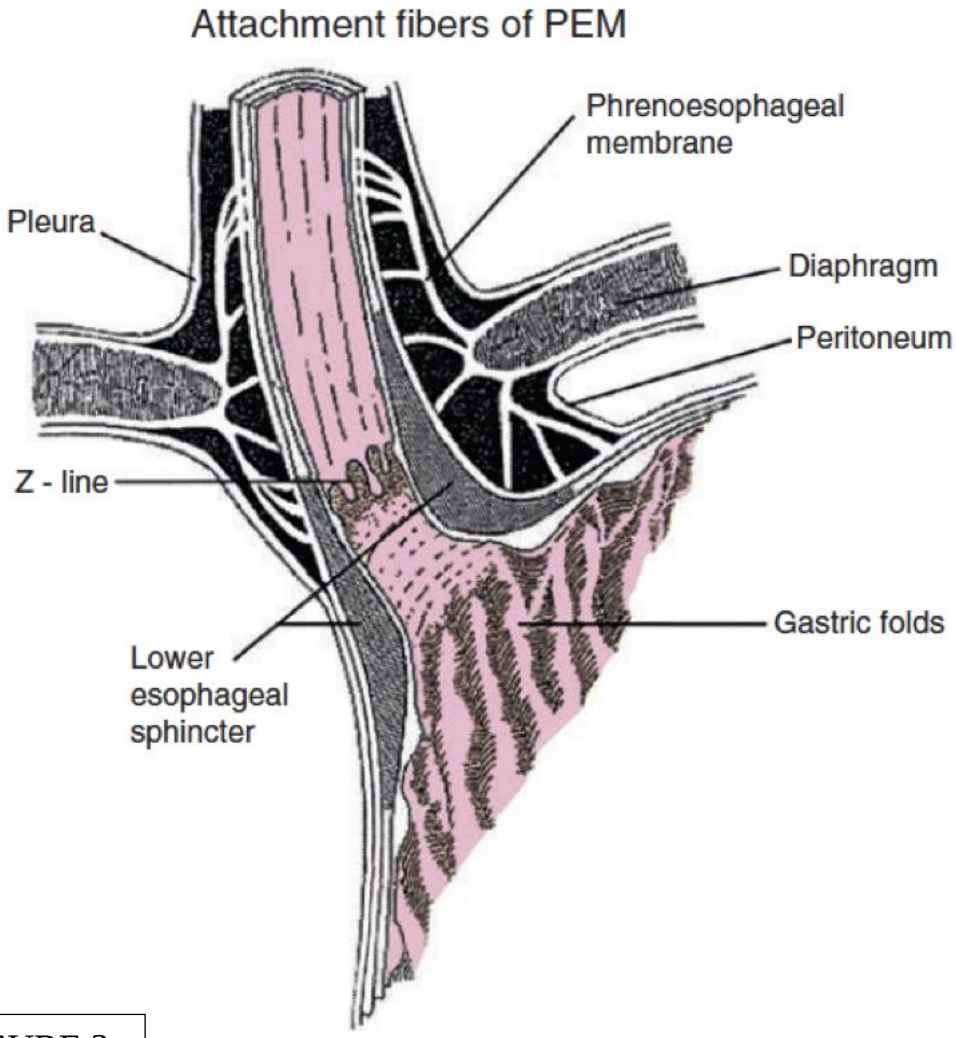


FIGURE 3

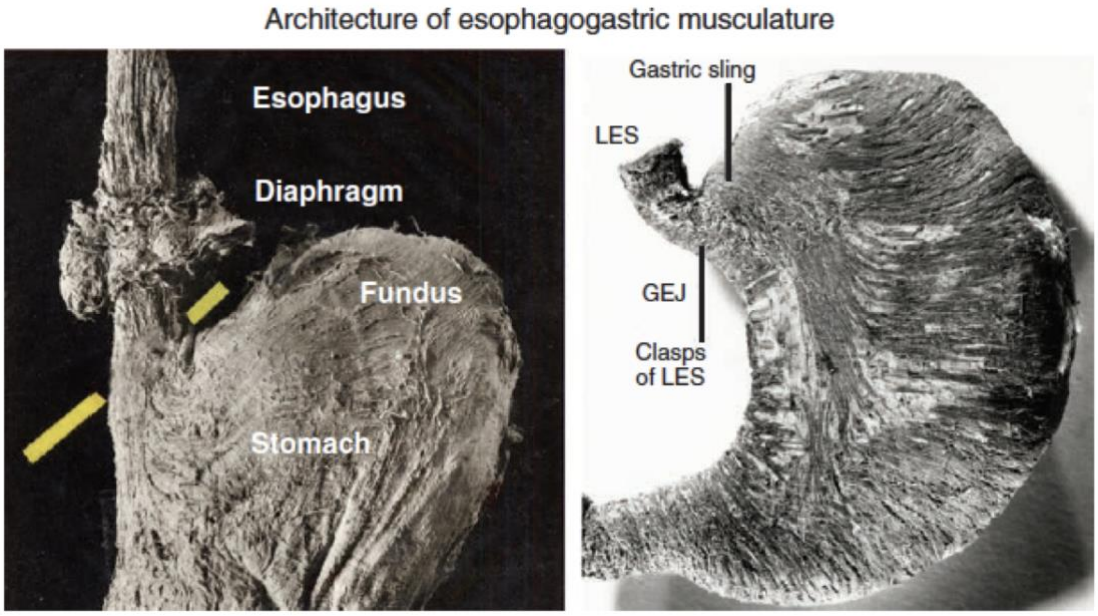


FIGURE 4: The dry muscle fiber specimen shows the muscle orientation at the gastroesophageal junction (GEJ) with the outer, longitudinal muscle (left) and the inner, circular fibers (right). Along the lesser curvature, the circular fibers become short muscular clasps, while the circular fibers become oblique gastric sling fibers along the gastric curve at the angle of His, forming a functional and anatomic lower esophageal sphincter (LES).

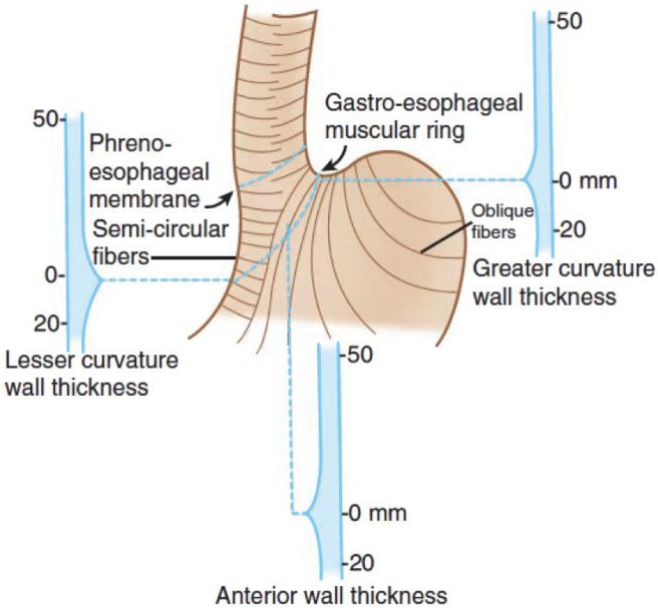


FIGURE 5: Wall thickness and orientation of fibers on microdissection of the cardia. At the junction of the esophageal tube and gastric pouch, there is an oblique muscular ring composed of an increased muscle mass inside the inner muscular layer. On the lesser curve side of the cardia, the muscle fibers of the inner layer are oriented transversely and form semi-circular muscle clasps. On the greater curve side of the cardia, these muscle fibers form oblique loops that encircle the distal end of the cardia and gastric fundus. Both the semi-circular muscle clasps and the oblique fibers of the fundus contract in a circular manner to close the cardia.

DIAPHRAGM:

The left and right edges of the esophageal hiatus most commonly originate from the deep and superficial layers of the right crus, while the left crus forms one edge of the aortic hiatus. The phreno-esophageal membrane, also known as Laimer or Allison membrane, arises from the sub-diaphragmatic fascia and attaches the esophagus to the diaphragm. The phreno-esophageal membrane is composed of two sheaths. One sheath passes superior from the diaphragm for 2 to 4 cm along the distal esophagus, and its fibers insert into the submucosa of the esophagus. The other sheath extends inferiorly across the cardia and blends into the gastric serosa, muscle, dorsal mesentery, and the gastro-hepatic ligament. The phreno-esophageal membrane allows the esophagus to move dynamically relative to the diaphragm[2].

ABDOMINAL ESOPHAGUS:

The abdominal esophagus is 3 to 6 cm in length. The abdominal esophagus angulates to the left after passing through the diaphragmatic hiatus. The esophagus ends as it enters the cardia along the lesser curvature of the stomach[2].

The abdominal esophagus receives blood supply from small arteries derived from the left gastric artery and the inferior phrenic artery. Venous drainage is through the left gastric vein. Intra-esophageal veins are located in a

subepithelial plexus located in the lamina propria. In the distal esophagus, this venous plexus connects with the portal system[2].

The esophagus passes through the esophageal hiatus at the level of the 10th thoracic vertebra. The crura originate from the anterior aspect of the first three to four lumbar vertebrae. As the esophagus passes through the hiatus, the left lobe of the liver lies anteriorly. The aorta is posterior to the esophagus and passes through its own hiatus in the diaphragm. The inferior vena cava lies lateral and inferior to the right crus[2].

FIGURE 6

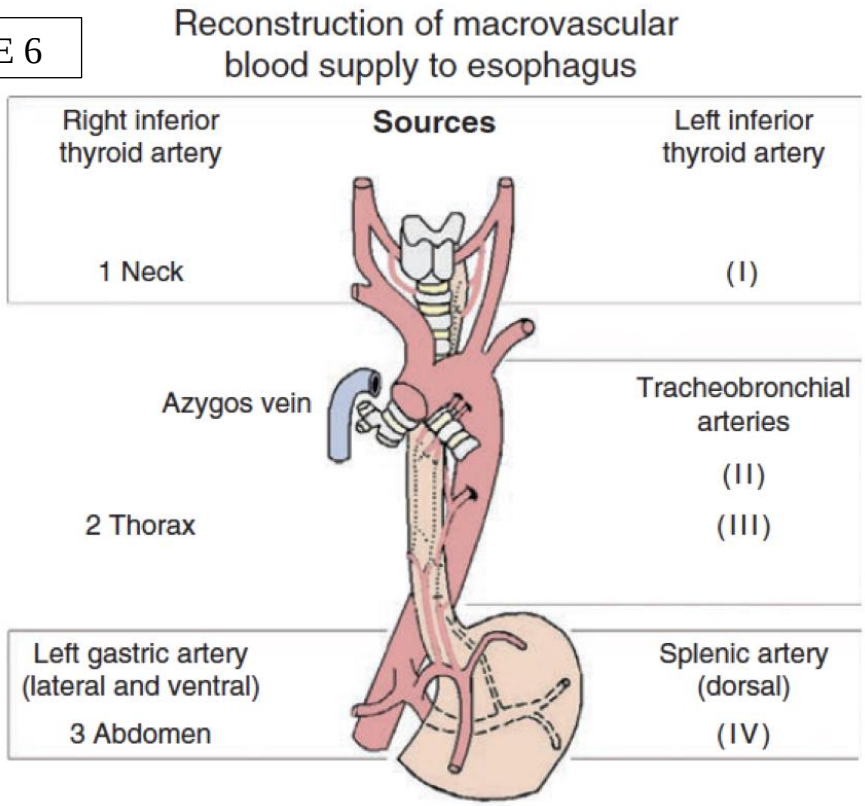
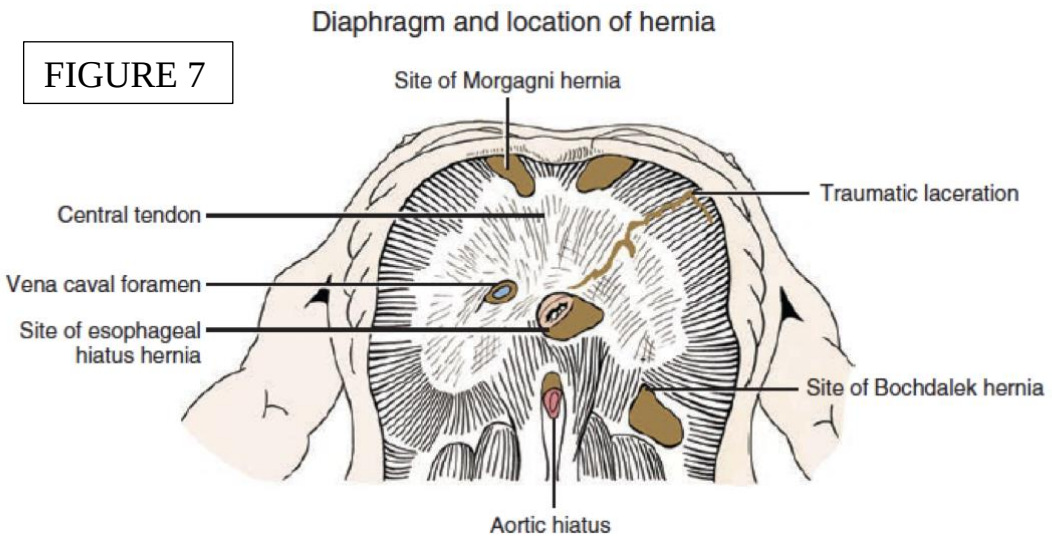


FIGURE 7



LOWER ESOPHAGEAL SPHINCTER IN HEALTH:

PHYSIOLOGIC FUNCTION AND ANATOMIC STRUCTURE:

The lower esophageal sphincter (LES) represents the lower boundary of the esophageal tube. It cannot be identified from inside the abdomen as a visible anatomic area at the lower end of the esophagus because there is no muscular thickening of the esophageal wall or special identifiable muscular structure. It can be roughly visualized by endoscopy and radiographic studies, but it can be best demonstrated and assessed quantitatively with esophageal manometry[2].

The major function of the LES is closure of the gastric reservoir to prevent reflux of gastric contents back into the esophagus. This is important because gastric juice can be toxic for the esophageal mucosa. However, the LES cannot exclusively function as a one-way valve just for ingested liquid and food to pass

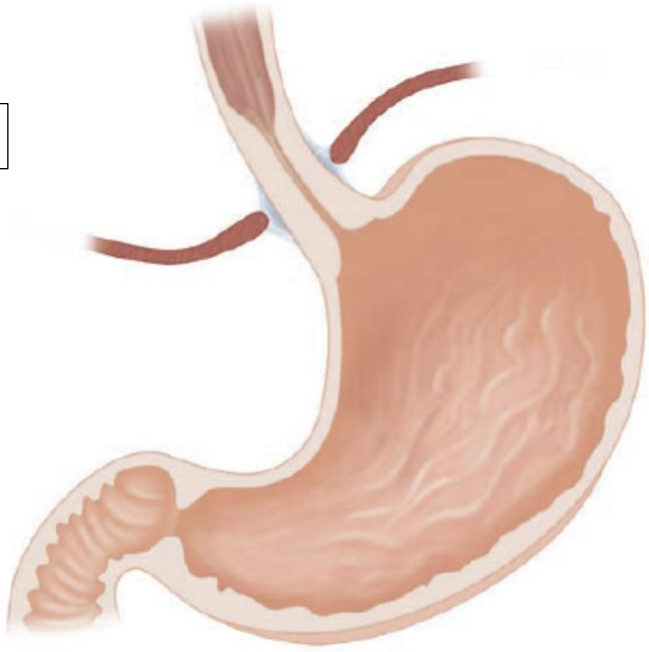
into the stomach, because it must also allow for selective retrograde passage of gas, usually ingested air, from the stomach[2].

In healthy individuals, these mechanisms prevent an overload of air in the stomach and small bowel with subsequent substantial discomfort for the individual. Excessive intragastric air can be vented through the LES back into the esophagus and outside trans-orally. In several diseases, particularly those with partial or total obstruction of a normal gastric emptying or small bowel paralysis, vomiting can be an important mechanism to solve the problem. Thus, the physiologic LES must allow for this retrograde emptying of the stomach[2].

The anatomic structure of the LES was best studied by Liebermann-Meffert. A detailed analysis of fiber specimens shows, in the distal esophagus and proximal stomach, several different bundles of muscle fibers that create this high-pressure zone. Although in the esophageal corpus there is a distinct two-layer formation of longitudinal and circular muscles, at the gastroesophageal junction, there are the so-called semi-circular muscular claps toward the lesser curvature. On the left side of the cardia at the angle of His, gastric sling fibers create the lower end of the LES, forming the left part of the high-pressure zone. These structural elements create a certain thickening at the LES with an asymmetric structural and functional high-pressure zone. Vagal branches regulate the neurologic function of the LES[2].

The second important anatomic structure that is responsible for the lower boundary of the esophagus is the diaphragm and its crura, the diaphragmatic arch at the hiatal opening, and the phreno-esophageal membrane, fixing the distal esophagus in its position within the hiatal opening[2].

FIGURE 8



Inspiration creates a negative pressure in the thoracic environment and an increased positive pressure environment in the abdominal cavity. Additional body activity of a person will further increase intra-abdominal pressure. The phreno-esophageal membrane and the esophagus itself, filling the hiatal gap, are under constant pressure changes because of the positive intra-abdominal pressure environment and the negative thoracic pressure environment during respiration[2].

In healthy individuals, the phreno-esophageal membrane is not just a circular ligament, because the hiatal opening or gap is not a circular structure. Depending on the size of the individual, this transition zone of the distal esophagus through the hiatal opening has a length of 1 to 3 cm^2.

The main body of the crura develop their muscle structure from a caudal posterior position toward the ventral and more cranial position to unify at the hiatal arch, thus surrounding the LES in an almost circular fashion, usually with an open posterior part on the aorta. The posterior part of the opening is represented by the aorta, on which the crura are also fixed partially or sometimes completely[2].

The latter carries a certain risk because clinical observations have shown that quite often in young reflux patients, the left crus is shorter than the right one, and its insertion is weakened by spreading out on the anterior aspect of the aorta, which limits its stable fixation point posteriorly[2].

In healthy individuals, the structures at the hiatal gap have to keep the cardia in its position over decades despite a constant tendency to wear out secondary to respiratory movements, coughing, and pressure increases from the abdominal and the thoracic environments, while still allowing for some flexibility such as movements of the esophagus during swallowing. Given this situation, it is not surprising that over decades, the system becomes insufficient in some people.

The tissue in some people weakens and structures elongate, finally resulting in sliding of structures within the hiatal opening and into the chest, thus creating a sliding hiatal hernia[2].

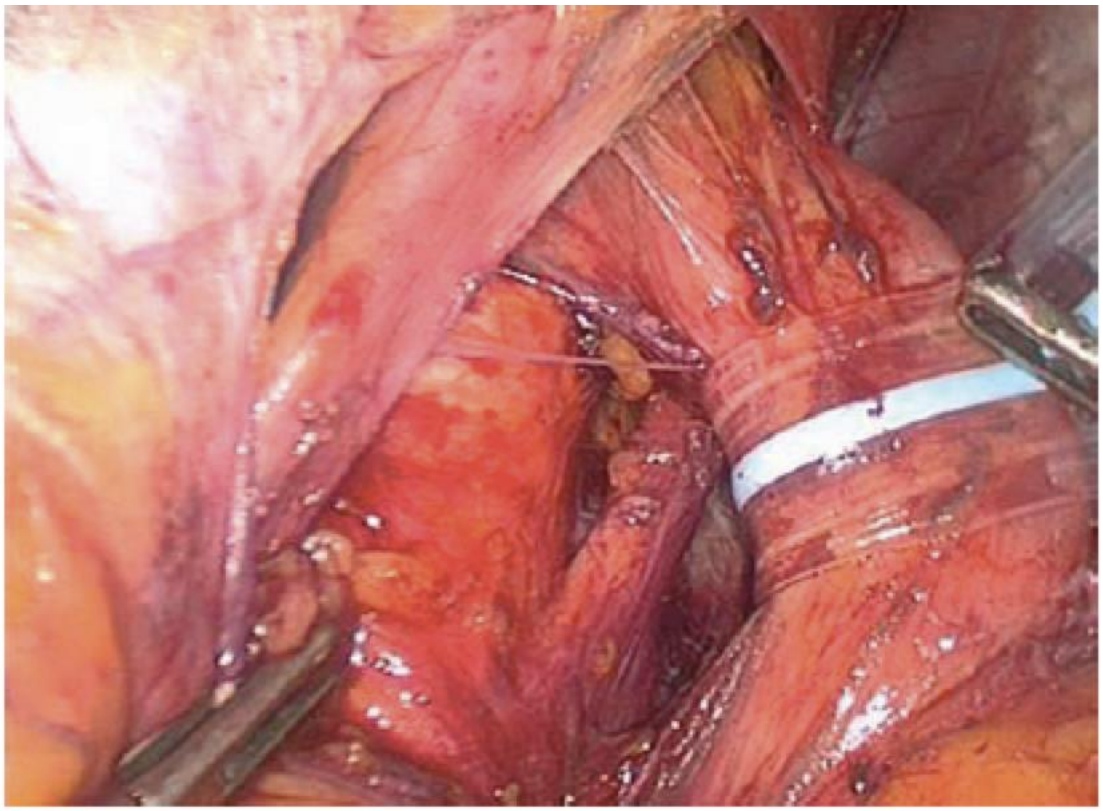


FIGURE 9: Laparoscopic view of the hiatal gap and the crura in a young gastroesophageal reflux disease patient, showing the shortage of the left crus, causing insufficient fixation of the lower esophageal sphincter by the phreno-esophageal membrane.

FUNCTIONAL CHARACTERISTICS OF THE LOWER ESOPHAGEAL SPHINCTER AND THE ANTI-REFLUX BARRIER:

The major functional finding at the distal esophagus is a high-pressure zone. Over decades, traditional esophageal manometry was performed by perfusion catheters with multiple radially oriented recording openings. Over the past 40 years there have emerged two major concepts regarding the quantitative manometric description of the anti-reflux barrier. One is a more mechanical description by the surgical DeMeester school. The second was developed mainly by gastroenterologists Dent and Dodds, and invokes a more dynamic concept involving transient lower esophageal sphincter relaxations (tLESR)[2].

Following the DeMeester school, the LES can be characterized by three manometrically assessed components: the overall length of the high-pressure zone, the sphincter pressure, and the sphincter position, expressed by the intra-abdominal length of the high-pressure zone[2].

The quantitative assessment of the sphincter was based on the rather limited measurements provided by traditional perfusion manometry. The perfusion catheter usually consisted of five radially oriented side holes, through which water was perfused and pressure changes were recorded initially on paper and later on computer software. Considering the complex movements and subtle pressure changes as well as positional changes of LES during breathing and

swallowing, the harvest of data using the early water-perfused systems was very limited[2].

In healthy individuals, the high-pressure zone in the distal esophagus can be measured from the thoracic part of the distal esophagus through the hiatal opening to the intra-abdominal part of the esophagus and the proximal stomach. In the normal physiologic condition, this high-pressure zone is 3 to 5 cm long and has a mean pressure of 14 mm Hg depending on the size of the individual. The LES must create an effective pressure (resistance) over a certain length to fulfil its function of preventing substantial reflux of gastric juice into the esophagus. It should be emphasized that the shorter the overall length, the higher the LES pressure must be to maintain its barrier function[2].

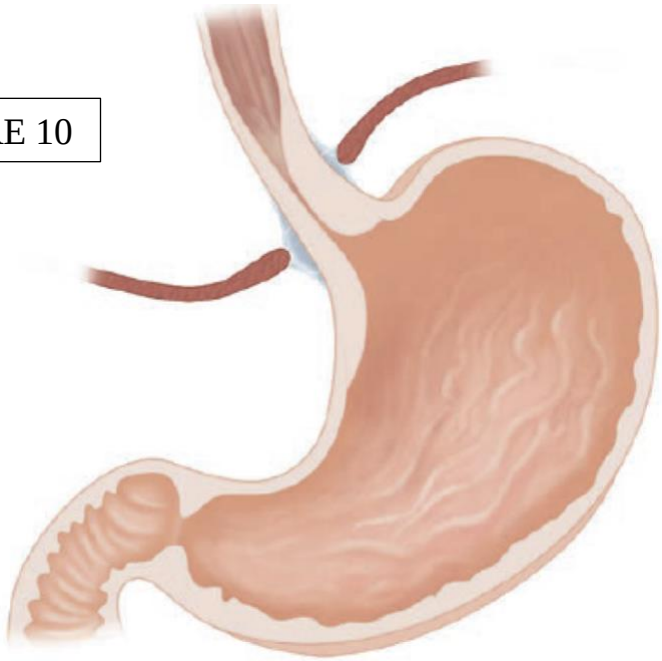
A critical component of overall length is the length of the LES exposed to positive intra-abdominal pressure, or the intra-abdominal length. The longer the intra-abdominal segment of the LES, the better it can adapt to changing intra-abdominal pressures to maintain sphincter competence and prevent reflux of gastric juice into the esophagus. Many publications have shown that the measurement of these three characteristics of the LES is clinically relevant. As GERD severity worsens, as assessed by the presence of symptoms, esophagitis, and the presence of complications such as stricture or Barrett esophagus, an increasing number of deficiencies in overall length, intra-abdominal length, and pressure can be demonstrated. Therefore, it can be clearly stated that the

mechanical measurable components of the LES play a major role in the function of the anti-reflux barrier and these criteria can be used for decision making and assessment of disease severity[2].

The gastroenterologists Dent and Dodds described the concept of tLESR as a significant mechanism of gastro-esophageal reflux in normal individuals and in patients with GERD. These tLESRs are identified by manometric sleeve technology systems as further developed by Dent, which allow for pressure measurement over the complete length of the high-pressure zone. It is emphasized that these relaxations of the high-pressure zone occurred without previous swallowing and without pharyngeal contractions. They also showed that these relaxations are coordinated with crural diaphragmatic relaxations. Based on these findings, tLESR became accepted in gastroenterology as the major mechanism of gastro-esophageal reflux[2].

However, there is controversy regarding this phenomenon because studies in healthy subjects and patients showed that tLESRs were most severe in the postprandial phase. This may indicate that LES relaxations could be associated with gastric filling and/or gastric distention. Further controversies around the concept of the tLESR arose when manometric studies showed that postprandial distention of the stomach caused sphincter shortening. Radiographic studies supported this finding[2].

FIGURE 10



This figure demonstrates the enlargement of the gastric fundus by overfilling of the fundus, which is a common phenomenon in Western societies as a result of overeating. Excessive filling of the gastric fundus will pull the distal part of the high-pressure zone apart because of fundic accommodation, thus shortening the LES even in healthy persons[2].

The shorter the overall length of the high-pressure zone, the higher the pressure must be for the LES to remain competent. If the stomach is filled by food and/or ingested air, sphincter shortening can occur. With progressive shortening and loss of intra-abdominal sphincter length, the LES pressure becomes unable to maintain competency, and once the intra-gastric pressure reaches the LES residual pressure, the LES gives away and will manometrically appear as

tLESRs because the LES pressure drops to zero during opening of the sphincter. Thus, critics of the tLESR concept argue that tLESRs may in fact represent transient LES shortenings resulting from gastric distention. This would explain why tLESRs increase in the postprandial phase and why, in patients with advanced reflux disease and a destroyed LES, there is not a substantial increase in tLESRs as one would otherwise expect if the tLESRs were the cause of the reflux[2].

With the advent of high-resolution manometry (HRM), the number of pressure detecting recording sites has increased tremendously. Current HRM technology has 36 recording site levels that are 1 cm apart on the probe, with circumferential recording at each level. This technology allows for an integral assessment of pressure changes of the complete esophageal corpus, the proximal and distal high-pressure zones, as well as pressure changes of the surrounding structures such as the diaphragm and its respiratory-dependent changes. This new technology has clinical advantages because it allows for the assessment of complex pressure profiles in the physiologic situation as well as in pathologic circumstances[2].

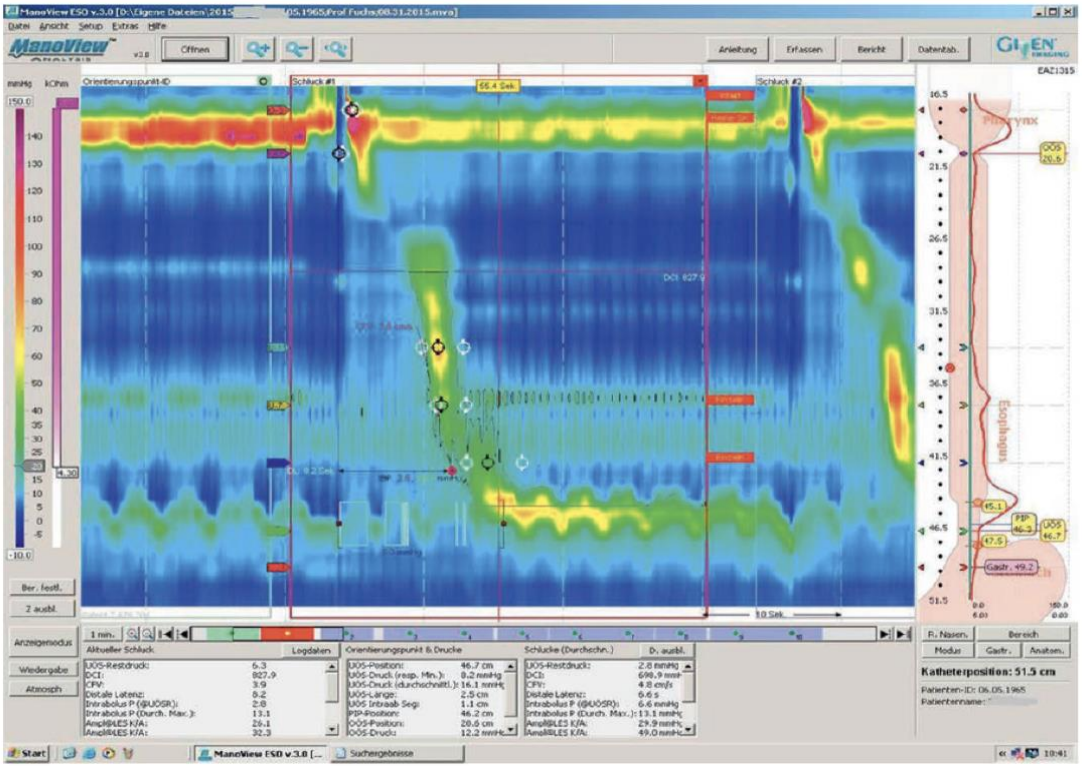


FIGURE 11: High-resolution manometry of the esophagus: demonstration of the esophageal body and lower esophageal sphincter in a healthy individual, showing swallowing and relaxation of the lower esophageal sphincter.

The above figure demonstrates a physiologic LES area with a pressure profile of the complete anti-reflux barrier, that is, the LES and the diaphragmatic pressure influences are combined in the gastro-esophageal junction high pressure zone[2].

LOWER ESOPHAGEAL SPHINCTER IN DISEASE:

The most frequent form of failure of the LES is weakening or incompetence leading to increased gastro-esophageal reflux. This phenomenon is usually accompanied by structural changes of the hiatal architecture and the cardia. The weakening of the sphincter and the weakening of the phreno-esophageal ligament with changing position of the sphincter within the diaphragm can result in a total loss of anti-reflux barrier function[2].

LOWER ESOPHAGEAL SPHINCTER IN GASTRO-ESOPHAGEAL REFLUX DISEASE:

In patients with GERD, the high-pressure zone and the other structural components of the anti-reflux barrier such as the phreno-esophageal membrane can weaken over time. Very often, a sequence of changes occurs over many years, leading to progressively worsening gastro-esophageal reflux. There is a high prevalence of GERD in Western industrial societies. One can speculate that the initial problem is overeating, which enlarges the fundus and effaces the intra-abdominal portion of the LES. This exposes the effaced portion of the LES to the acid pocket in the stomach after a meal, with subsequent injury to the distal portion of the LES. Over time the damage gradually leads to loss of the intra-abdominal length of the LES, and progressively more effacement until the sphincter completely loses competence and frank GERD develops[2].

In addition, increased intra-abdominal pressure because of intra-abdominal fat leads to weakening of the supporting structures of the esophagus within the hiatal opening. Combined with tissue weakening as a result of age, the initially strong phreno-esophageal membrane becomes a loose fatty hernia sac, which facilitates a temporary sliding of the LES in the chest. Later, the LES will move to a permanent position in the lower mediastinum[2].

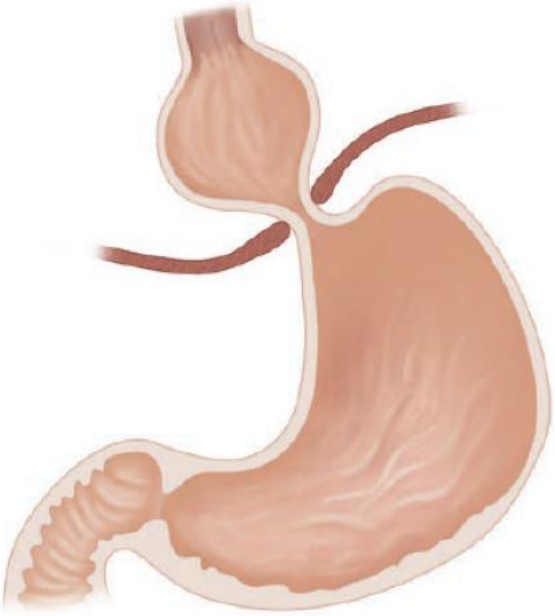


FIGURE 12: demonstration of the gastroesophageal junction and the alterations as a result of a hiatal hernia. The phreno-esophageal membrane has deteriorated from a firm ligament into a floppy and fatty hernia sac.

Many publications have shown the loss of LES competence in GERD and the relationships among disease severity, the presence of its complications, and the increasing incidence of LES incompetence in these patients[2].

Patients with advanced GERD typically have a hiatal hernia and an incompetent LES on manometry. Using HRM, the LES and the pressure influences from the

diaphragm are displayed. In someone without a hiatal hernia, these pressures are superimposed to form one gastro-esophageal junction pressure[2].

When a hiatal hernia is present, the LES and the hiatal structure separate and the separate pressure influences of the LES and the diaphragm become evident. This allows for the description of a hiatal hernia in the HRM pressure profile. These findings can be important for therapeutic decision making regarding further medical treatment or a laparoscopic anti-reflux procedure[2].

The manometric finding of a loss of mechanical sphincter function has a prognostic component. Kuster showed that patients with GERD and an incompetent sphincter on manometry have a higher probability of having GERD problems after 10years, which could be an argument for earlier surgical therapy[2].

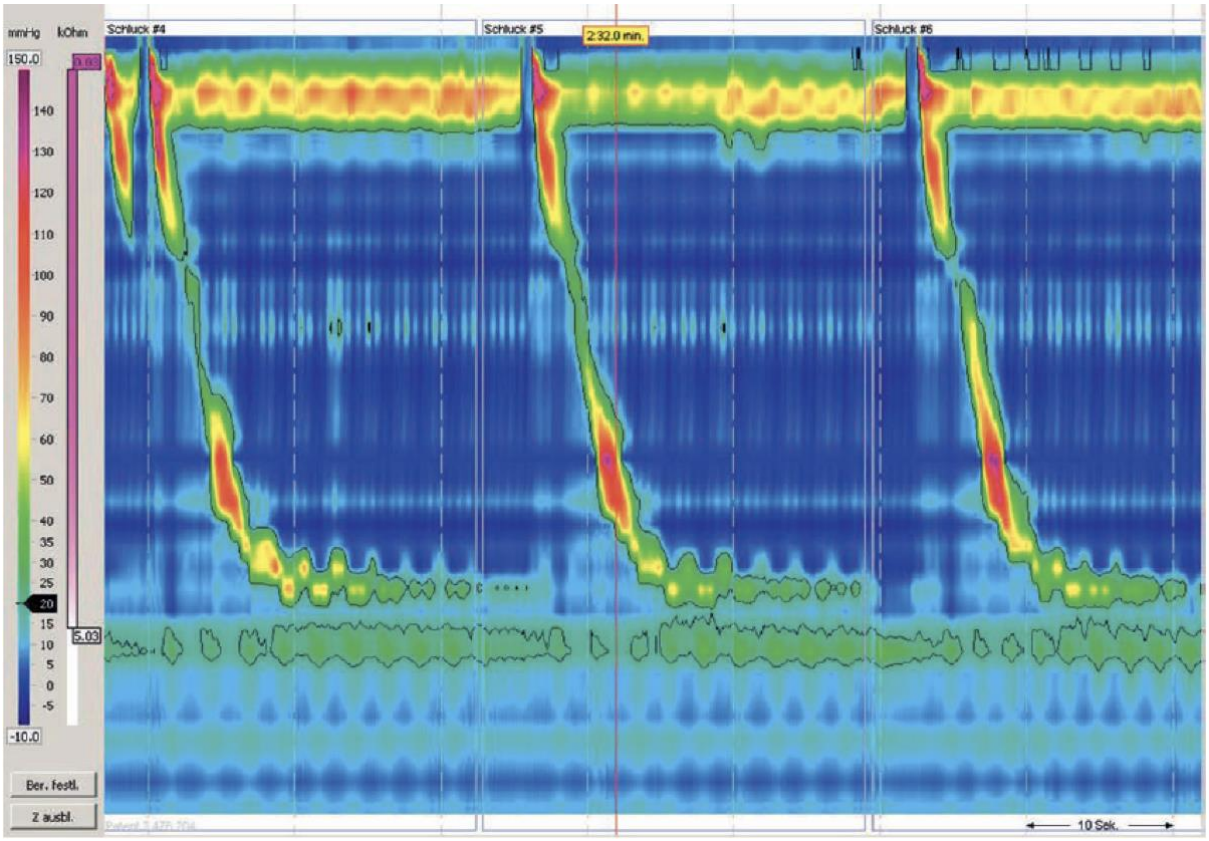


FIGURE 13: High-resolution manometry in a patient with gastroesophageal reflux disease and hiatal hernia. The weak lower esophageal sphincter can be noted as well as the separation of the remaining pressure level of the lower esophageal sphincter and the diaphragmatic structures.

HIATUS HERNIA:

DEFINITION and CLASSIFICATION:

Hiatal hernias are herniation of the stomach or other abdominal organs into the chest through the esophageal hiatus in the diaphragm[3].

There are traditionally four types of hiatal hernias:

- Type I is defined as a migration of the GEJ (gastro-esophageal junction) into the chest secondary to an attenuated phreno-esophageal ligament. The "sliding" (type I) hernia and is the most common type of hiatal hernia[3].

- True para-esophageal hernias (PEH), or type II hiatal hernias, occur when the gastric fundus herniates anterior to the esophagus while the GEJ remains in the abdomen[3].

- Type III hiatal hernias are a combination of types I and II, in which both the GEJ and the gastric fundus herniate into the chest[3].

- Type IV hiatal hernias occur when not only the stomach, but other abdominal organs such as the colon, also herniates into the chest through the esophageal hiatus. Because of the large size of the defect and extent of herniation, the stomach may undergo rotation within the hernia sac. Most commonly, this consists of organo-axial volvulus in which the stomach rotates along the axis of the organ. This type of volvulus results in the greater curvature being flipped

upward and at a higher position in the mediastinum than the lesser curvature. The stomach can also rotate along the axis of its mesentery (meso-axial volvulus). Meso-axial volvulus is associated with a higher risk of gastric ischemia because of the twisting of the mesentery, which can compromise venous return and gastric blood flow[3].

Of note, PEH (types II-IV) account for only about 14% of all hiatal hernias, with about 90% of all PEH being of the type III variety[3].

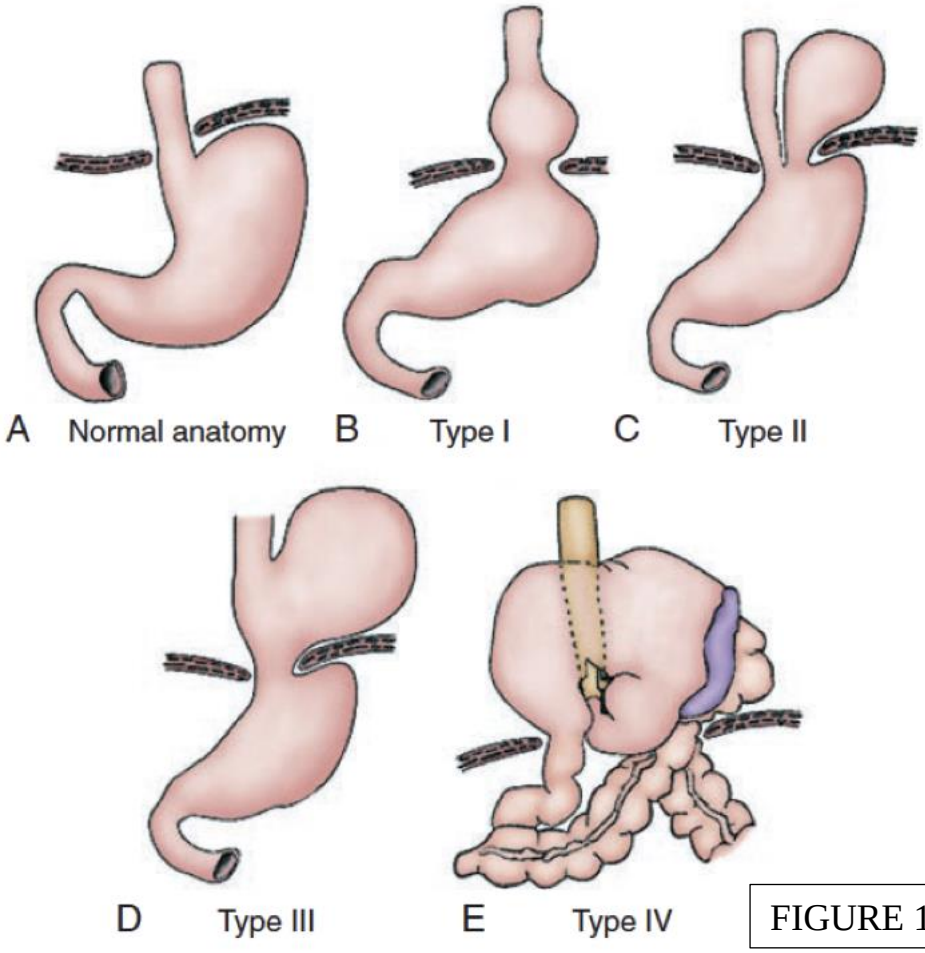


FIGURE 14

PREVALENCE:

The actual prevalence of para-esophageal hernias is not known. The most common hiatal hernia is type I, which accounts for up to 95% of all hiatal hernias. Para-esophageal hernias may account for up to 14% of all hiatal hernias, and the majority of paraesophageal hernias are of the type III variety. The incidence of para-esophageal hernias increases with age. Para-esophageal hernias tend to develop on the left anterior aspect of the esophageal hiatus. Women are more likely to develop paraesophageal hernias compared to men, and kyphosis is a risk factor[2].

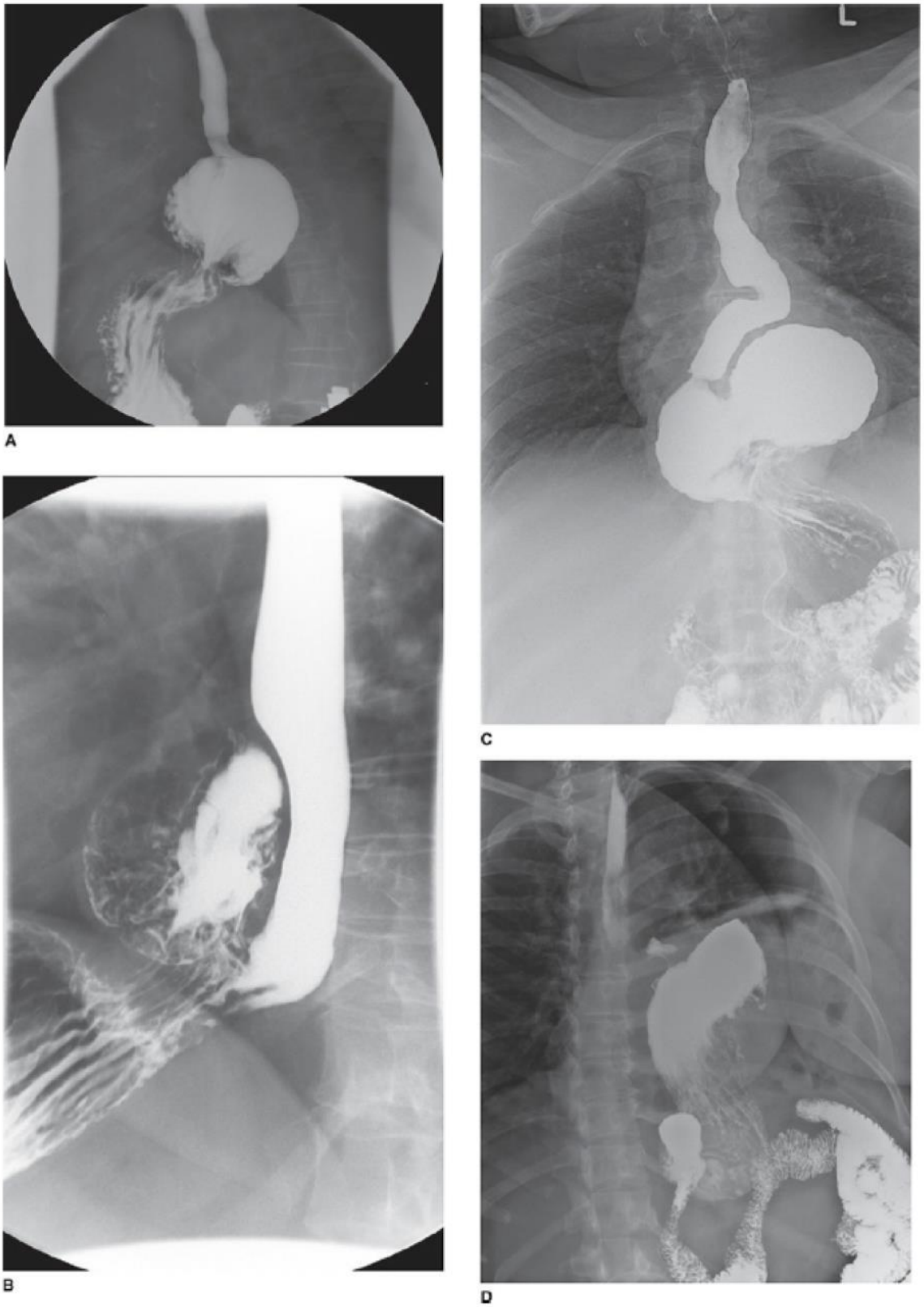


FIGURE 15: Barium swallow that illustrates the 4 types of hiatal hernias. A. Type I sliding hernia. B. Type II paraesophageal hernia (PEH). C. Type III PEH. D. Type IV PEH with intrathoracic stomach; note the bowel gas in the left chest, which is due to herniated colon.

CLINICAL PRESENTATION:

Patients with PEH may be completely asymptomatic, but more often, they have a variety of symptoms depending on the extent of the herniation. There may be a longstanding history of hiatal hernia that has been managed medically[3].

Postprandial fullness, discomfort, and pain, especially after eating a larger meal, are some of the most common symptoms associated with PEH. These symptoms occur because the herniated segment does not empty properly, and therefore, with a larger meal, the stomach becomes distended, which leads to discomfort[3].

Typical heartburn and regurgitation symptoms of gastro-esophageal reflux disease (GERD) may be present but are less common, likely because these patients may still have a competent lower esophageal sphincter[3].

Patients may also have dysphagia either because of associated esophageal dysmotility or because the large hernia compresses the distal esophagus[3].

Approximately 25% of patients present with anemia from gastrointestinal bleeding. Anemia in this setting is typically due to Cameron lesions, which are superficial erosions or ulcerations in the proximal stomach that can be secondary to constriction of the stomach at the hiatal defect and friction from movement across the hiatus, which can lead to occult blood loss[3].

Up to 20% of patients with PEH present with gastric volvulus. While gastric volvulus can be asymptomatic, it can also develop acutely and lead to gastric outlet obstruction with acute onset of chest or epigastric pain, nausea, and emesis. In some cases, the volvulus may lead to gastric ischemia and strangulation[3].

ETIOLOGY:

Factors contributing to increased intra-abdominal pressure like obesity, pregnancy, chronic constipation, chronic obstructive pulmonary disease (COPD) with chronic coughing, and strenuous jobs with significant amount of weight lifting. Ageing is also a significant risk factor[2].

PATHOPHYSIOLOGY:

Abnormalities in the phreno-esophageal membrane may lead to hiatal hernia formation. The elastic fibers of the phreno-esophageal membrane are replaced by inelastic fibers with ageing[2].

Eliska et al. believe that abnormal anchorage of the phreno-esophageal membrane, along with increasing adipose tissue between the membrane and the cardia, may lead to the development of a hiatal hernia[2].

Mittal reported that the phreno-esophageal membrane and its esophageal attachments may also contribute to the function of the lower esophageal

sphincter, although hiatal hernia experiments in cats found no effect on lower esophageal sphincter function with complete disruption of the phreno-esophageal membrane and positioning the cardia in the chest[2].

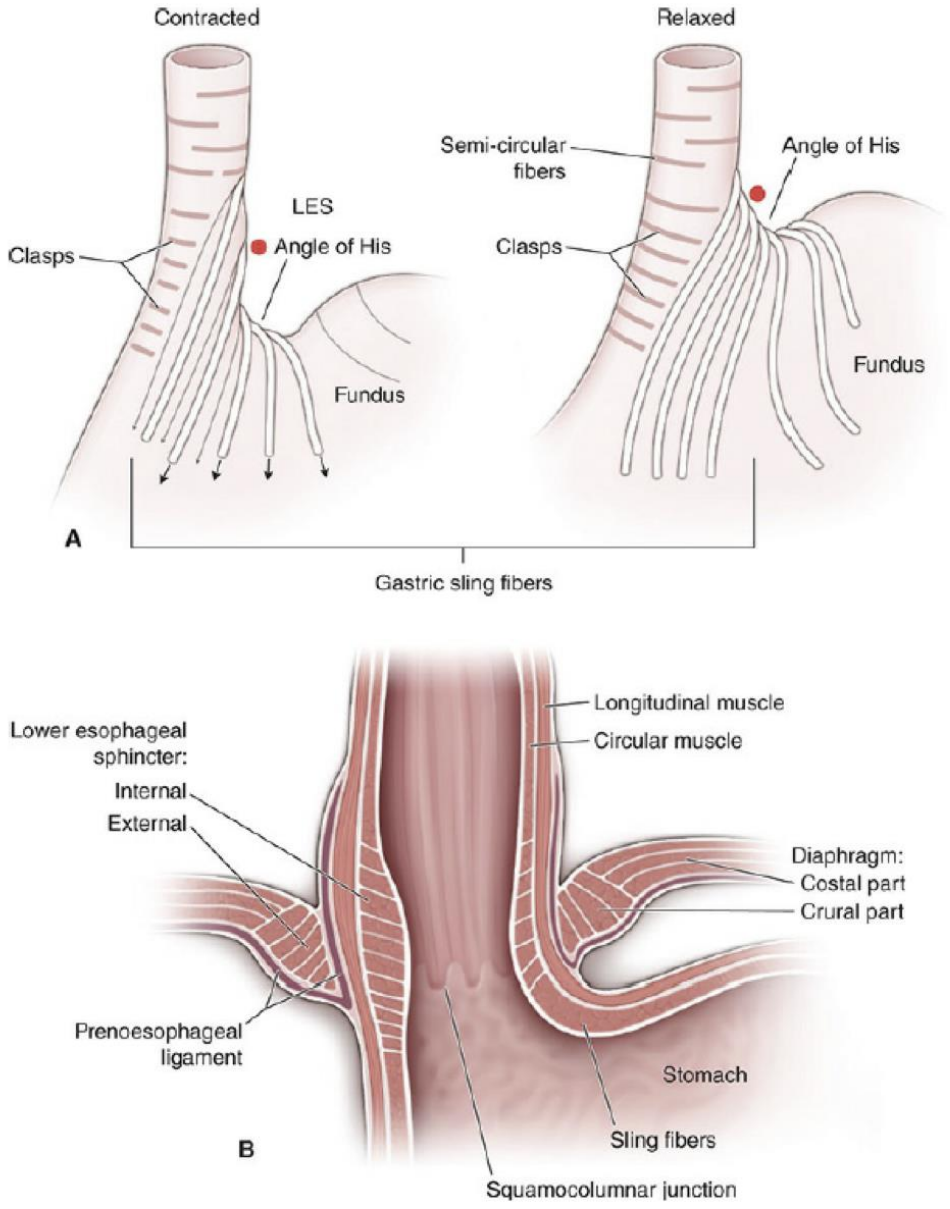


FIGURE 16: A. The clasp and sling muscle fibers that make up the lower esophageal reflux barrier in the contracted and relaxed state, respectively. B. The anatomic relationship of the gastroesophageal reflux junction, the phreno-esophageal ligament and the diaphragm.

INVESTIGATIONS:

ENDOSCOPIC EVALUATION:

The first diagnostic test in patients with suspected esophageal disease is usually upper gastrointestinal endoscopy. This allows assessment and biopsy of the mucosa of the stomach and the esophagus, as well as the diagnosis and assessment of obstructing lesions in the upper gastrointestinal tract[4].

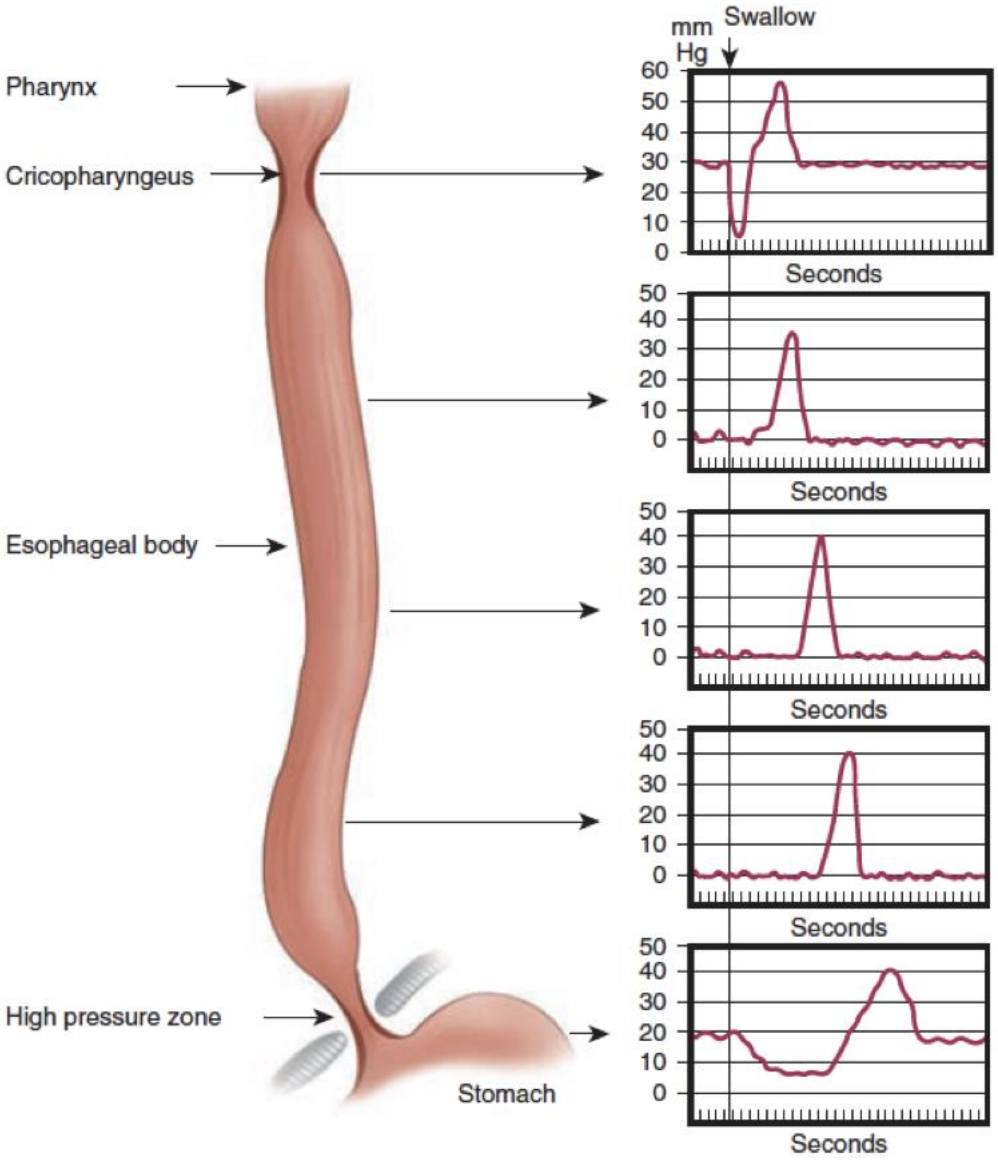


FIGURE 17: Intraluminal esophageal pressures in response to swallowing.

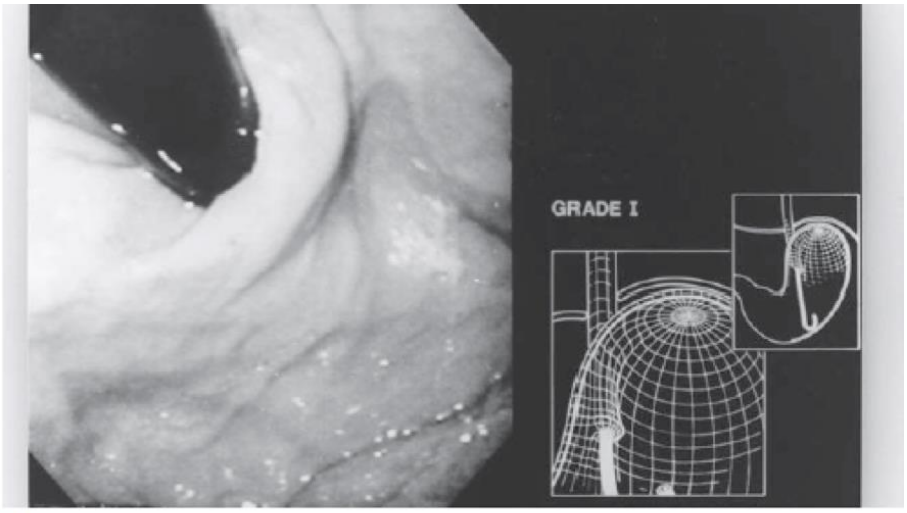
In any patient complaining of dysphagia, esophagoscopy is indicated, even in the face of a normal radiographic study. For the initial endoscopic assessment, the flexible fiber-optic esophagoscope is the instrument of choice because of its technical ease, patient acceptance, and the ability to simultaneously assess the stomach and duodenum. Rigid endoscopy is now only rarely required, mainly for the dis-impaction of difficult foreign bodies impacted in the esophagus, and few individuals now have the skill set and experience to use this equipment[4].

When GERD is the suspected diagnosis, particular attention should be paid to detecting the presence of esophagitis and Barrett's columnar-lined esophagus (CLE). It should be remembered that gastro-esophageal reflux is not always associated with visible mucosal abnormalities, and patients can experience significant reflux symptoms, despite an apparently normal endoscopy examination[4].

Abnormalities of the gastroesophageal flap valve can be visualized by retroflexion of the endoscope. Hill has graded the appearance of the gastroesophageal valve from I to IV according to the degree of unfolding or deterioration of the normal valve architecture. The appearance of the valve correlates with the presence of increased esophageal acid exposure, occurring predominantly in patients with grade III and IV valves[4].

A hiatal hernia is endoscopically confirmed by finding a pouch lined with gastric rugal folds lying 2 cm or more above the margins of the diaphragmatic crura, identified by having the patient sniff. A hernia is best demonstrated with the stomach fully insufflated and the gastro-esophageal junction observed with a retroflexed endoscope. A prominent sliding hiatal hernia frequently is associated with increased esophageal exposure to gastric juice[4].

When a para-esophageal hernia (PEH) is observed, particular attention is taken to exclude gastric (Cameron's) ulcers or gastritis within the pouch. The intra-gastric retroflex or J manoeuvre is important in evaluating the full circumference of the mucosal lining of the herniated stomach[4].



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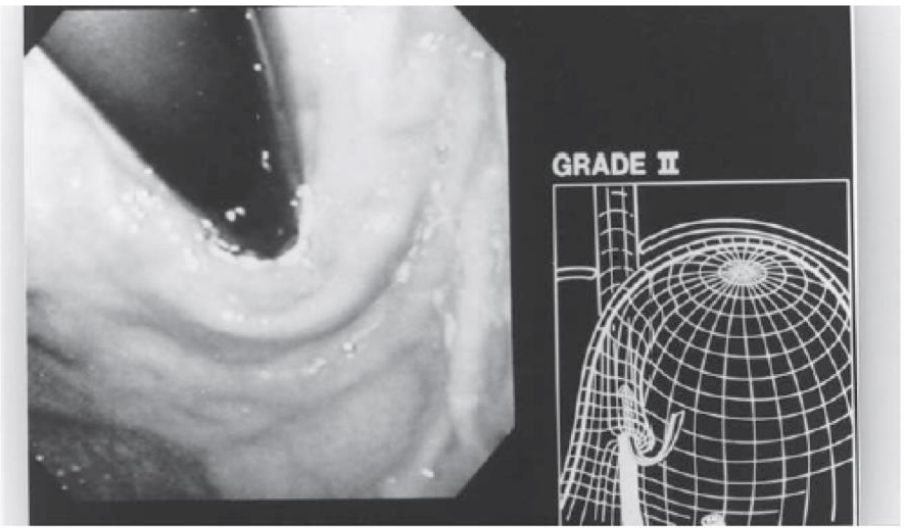

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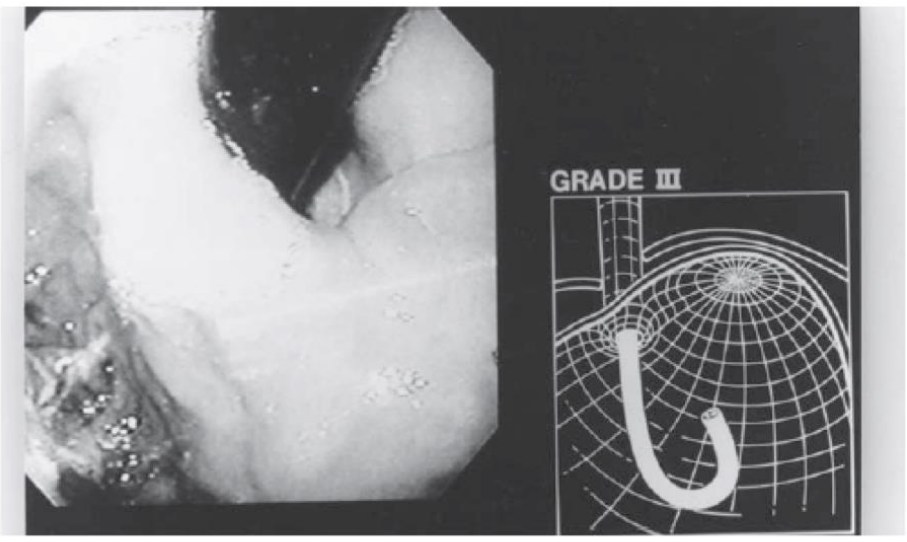

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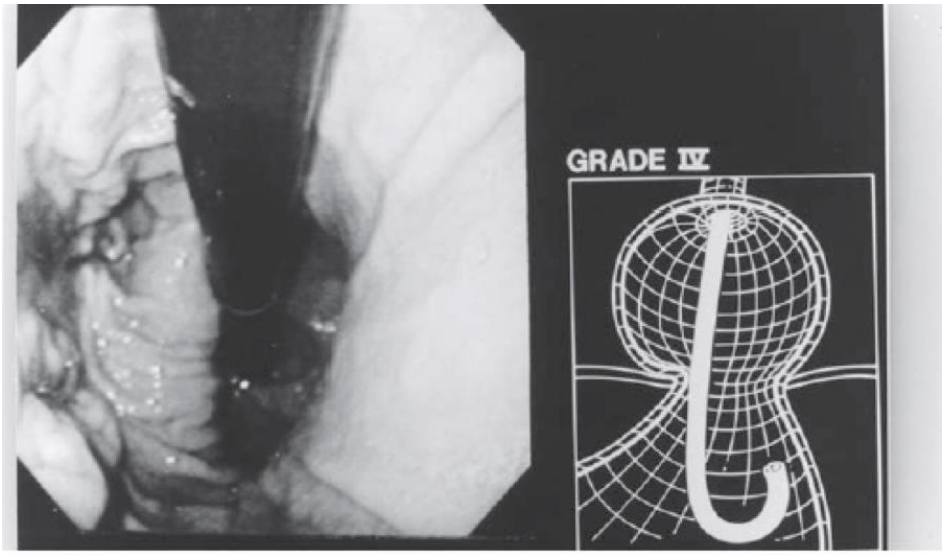


D

FIGURE 18: A. Grade I flap valve appearance. Note the ridge of tissue that is closely approximated to the shaft of the retroflexed endoscope. It extends 3 to 4 cm along the lesser curve. B. Grade II flap valve appearance. The ridge is slightly less well defined than in grade I and it opens rarely with respiration and closes promptly. C. Grade III flap valve appearance. The ridge is barely present, and there is often failure to close around the endoscope. It is nearly always accompanied by a hiatal hernia. D. Grade IV flap valve appearance. There is no muscular ridge at all. The gastroesophageal valve stays open all the time, and squamous epithelium can often be seen from the retroflexed position. A hiatal hernia is always present.

RADIOGRAPHIC EVALUATION:

Barium swallow evaluation is undertaken selectively to assess anatomy and motility. The anatomy of large hiatal hernias is more clearly demonstrated by contrast radiology than endoscopy, and the presence of coordinated esophageal peristalsis can be determined by observing several individual swallows of barium traversing the entire length of the organ, with the patient in the horizontal position[4].

Hiatal hernias are best demonstrated with the patient prone because the increased intra-abdominal pressure produced in this position promotes displacement of the esophago-gastric junction above the diaphragm. The density of the barium used to study the esophagus can potentially affect the accuracy of the examination. Esophageal disorders shown clearly by a full-column technique include circumferential carcinomas, peptic strictures, large esophageal ulcers, and hiatal hernias. Lesions extrinsic but adjacent to the esophagus can be reliably detected by the full-column technique if they contact the distended esophageal wall[4].

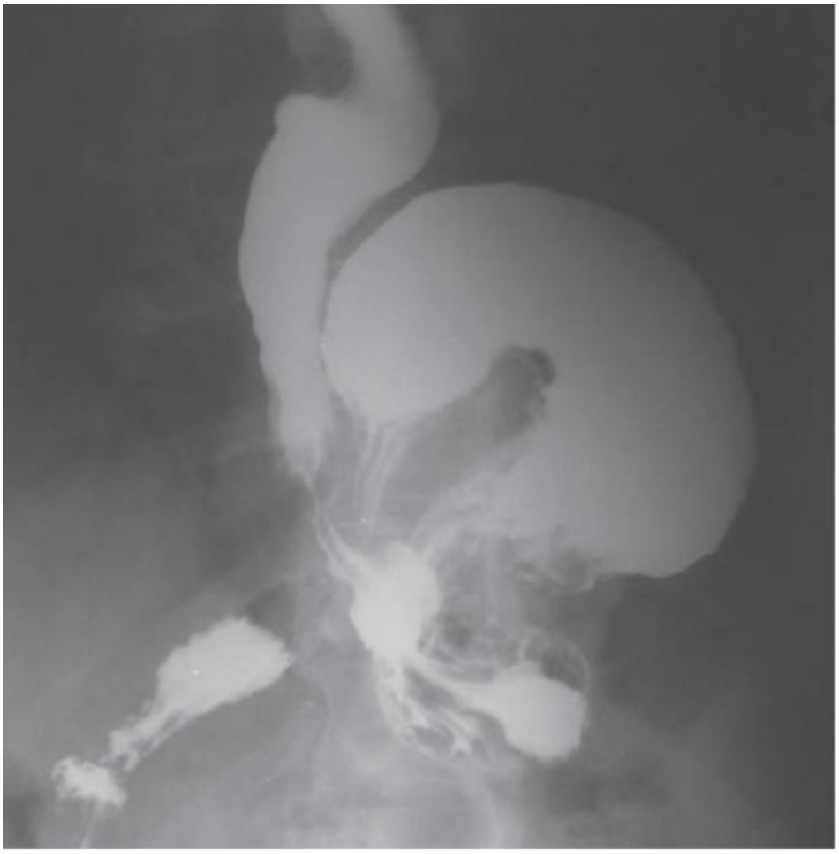


FIGURE 19: Radiogram of an intrathoracic stomach. This is the end stage of a large hiatal hernia, regardless of its initial classification.

ESOPHAGEAL MOTILITY:

Esophageal motility is a widely used technique to examine the motor function of the esophagus and its sphincters. The esophageal motility study (EMS) is indicated whenever a motor abnormality of the esophagus is suspected on the basis of complaints of dysphagia, odynophagia, or noncardiac chest pain, and the barium swallow or endoscopy does not show a clear structural abnormality[4].

EMS is particularly necessary to confirm the diagnosis of specific primary esophageal motility disorders (i.e., achalasia, diffuse esophageal spasm [DES], nutcracker esophagus, and hypertensive LES). It also identifies nonspecific esophageal motility abnormalities and motility disorders secondary to systemic disease such as scleroderma, dermatomyositis, polymyositis, or mixed connective tissue disease[4].

In patients with symptomatic GERD, manometry of the esophageal body can identify a mechanically defective LES and evaluate the adequacy of esophageal peristalsis and contraction amplitude. EMS has become an essential tool in the preoperative evaluation of patients before anti-reflux surgery, guiding selection of the appropriate procedure based upon the patient's underlying esophageal function and excluding patients with achalasia who can be misdiagnosed with gastro-esophageal reflux when clinical and endoscopic parameters alone are used for diagnosis[4].

EMS is performed using electronic, pressure-sensitive transducers located within the catheter, or water-perfused catheters with lateral side holes attached to transducers outside the body. The traditional water perfused catheter has largely been replaced by high resolution manometry (HRM). From these measurements, the pressure, abdominal length, and overall length of the sphincter are determined. To account for the asymmetry of the sphincter, the pressure profile is repeated with each of the five radially oriented transducers,

and the average values for sphincter pressure above gastric baseline, overall sphincter length, and abdominal length of the sphincter are calculated[4].

A mechanically defective sphincter is identified by having one or more of the following characteristics: an average LES pressure of <6 mmHg, an average length exposed to the positive-pressure environment in the abdomen of 1 cm or less, and/or an average overall sphincter length of 2 cm or less[4].

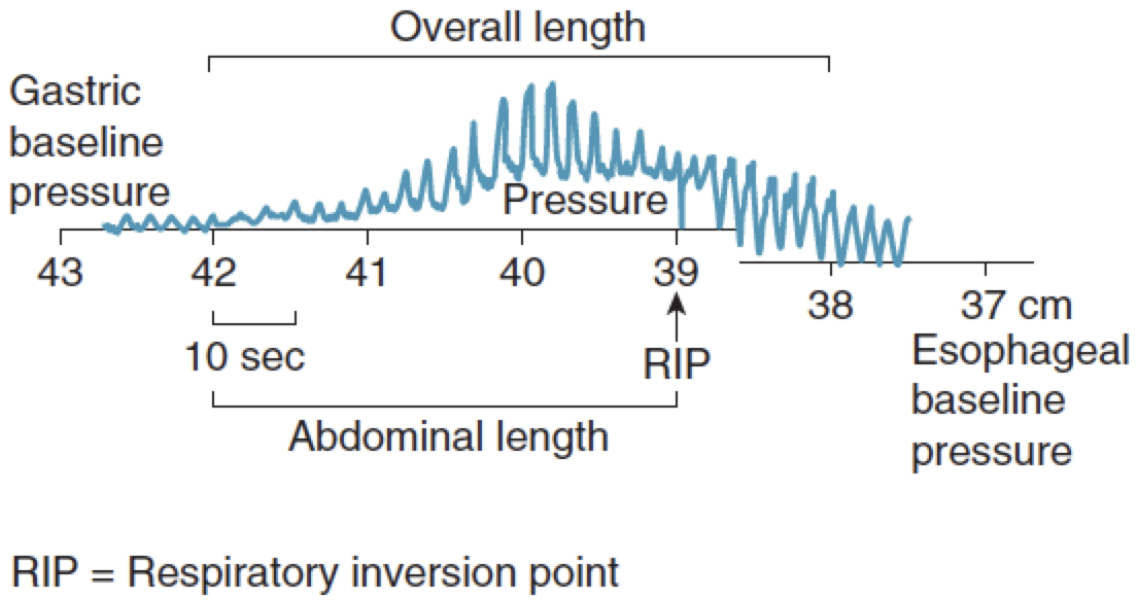


FIGURE 20: Manometric pressure profile of the lower esophageal sphincter. The distances are measured from the nares.

HIGH RESOLUTION MANOMETRY:

Esophageal manometry was introduced into clinical practice in the 1970s and, until recently, has changed little. In 1991, Ray Clouse introduced the concept of improving conventional manometry by increasing the number of recording sites and adding a three-dimensional assessment. This “high-resolution manometry” is a variant of the conventional manometry in which multiple, circumferential recording sites are used, in essence creating a “map” of the esophagus and its sphincters[4].

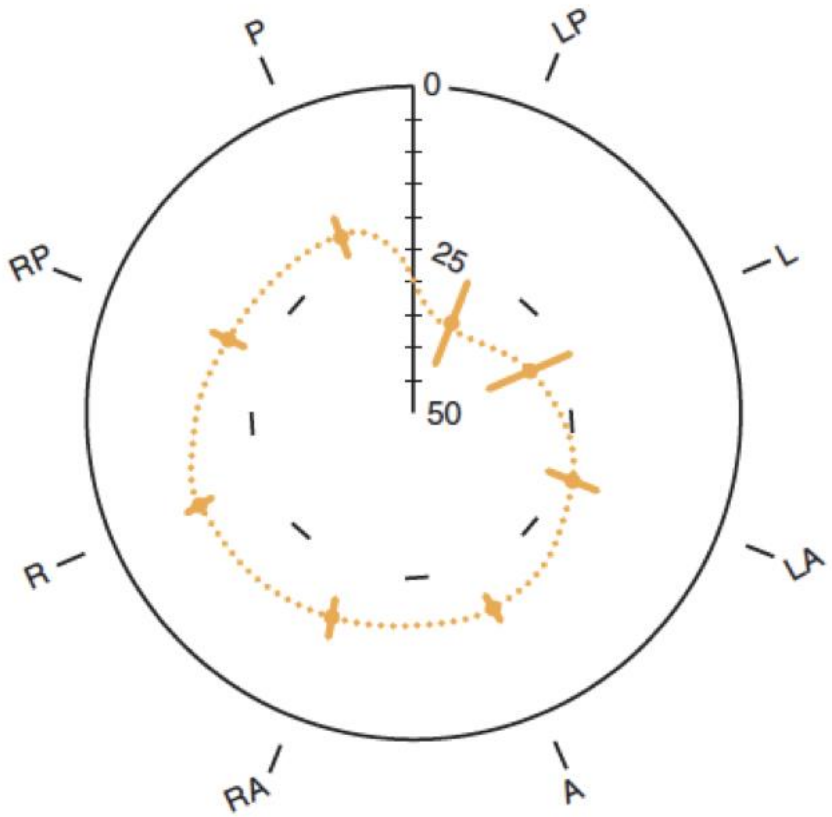


FIGURE 21: Radial configuration of the lower esophageal sphincter. A = anterior; L = left; LA = left anterior; LP = left posterior; P = posterior; R = right; RA = right anterior; RP = right posterior.

High-resolution catheters contain 36 miniaturised pressure sensors positioned every centimetre along the length of the catheter. The vast amount of data generated by these sensors is then processed and presented in traditional linear plots or as a visually enhanced spatiotemporal video tracing that is readily interpreted[4].

The function of the esophageal body is assessed with 10 to 15 wet swallows. Amplitude, duration, and morphology of contractions following each swallow are visually displayed. The relationship of the esophageal contractions following a swallow is classified as peristaltic or simultaneous. The data are used to identify motor disorders of the esophagus. The position, length, and function of the lower esophageal sphincter (LES) are demonstrated by a high-pressure zone that should relax at the inception of swallowing and contract after the water or solid bolus passes through the LES[4].

Simultaneous acquisition of data for the upper esophageal sphincter, esophageal body, LES, and gastric pressure minimizes the movement artefacts and study time associated with conventional esophageal manometry. This technology significantly enhances esophageal diagnostics, bringing it into the realm of "image"-based studies. High-resolution manometry may allow the identification of focal motor abnormalities previously overlooked. It has enhanced the ability to predict bolus propagation and increased sensitivity in the measurement of pressure gradients[4].

ESOPHAGEAL IMPEDANCE:

Newer technology introduced into the clinical realm a decade ago allows measurement of esophageal function and gastro-esophageal reflux in a way that was previously not possible. An intraluminal electrical impedance catheter is used to measure GI function. Impedance is the ratio of voltage to current, and is a measure of the electrical conductivity of a hollow organ and its contents[4].

Intraluminal electrical impedance is inversely proportional to the electrical conductivity of the luminal contents and the cross-sectional area of the lumen. Air has a very low electrical conductivity and, therefore, high impedance. Saliva and food cause an impedance decrease because of their increased conductivity. Luminal dilatation results in a decrease in impedance, whereas luminal contraction yields an impedance increase. Investigators have established the impedance waveform characteristics that define esophageal bolus transport. This allows for the characterization of both esophageal function, via quantification of bolus transport, and gastro-esophageal reflux[4].

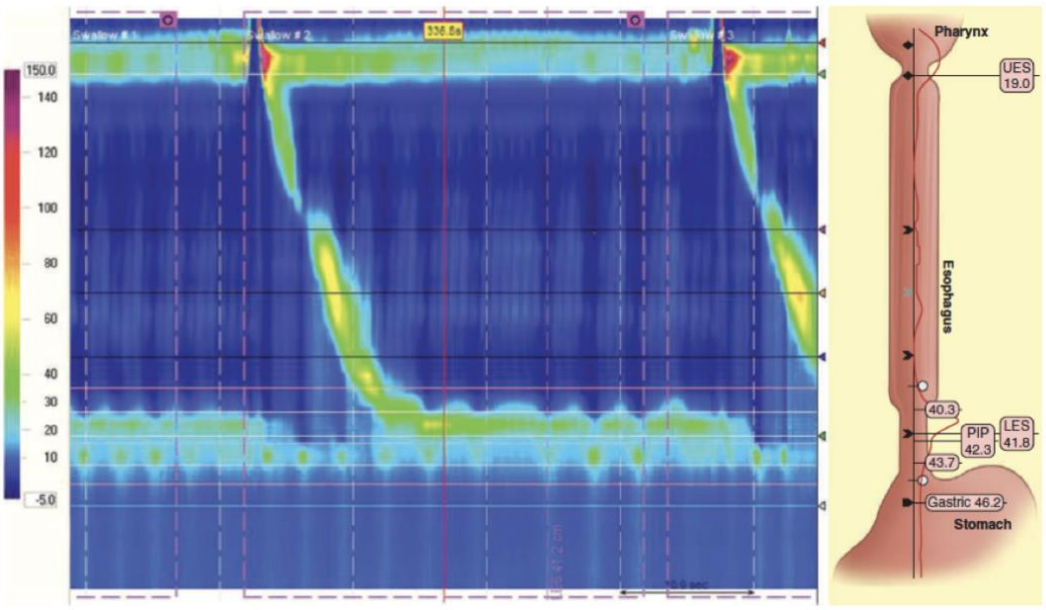


FIGURE 22: Normal high-resolution manometry motility study. Pressure measurements are recorded with color coding (red = high; blue = low). LES = lower esophageal sphincter; PIP = pressure inversion point; UES = upper esophageal sphincter.

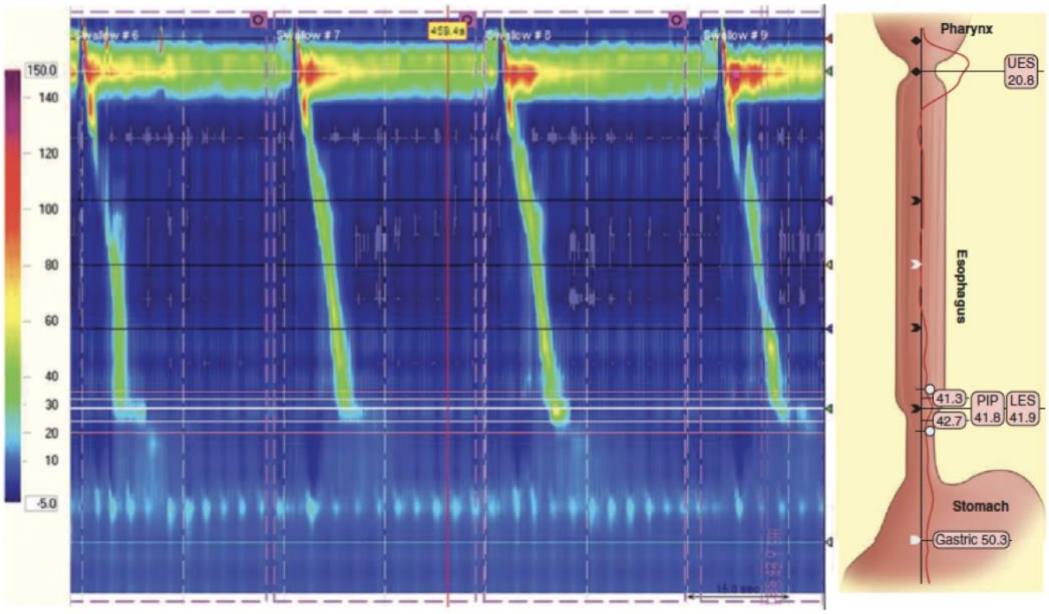


FIGURE 23: High-resolution manometry motility study in patient with mechanically defective lower esophageal sphincter. Note the absence of lower esophageal sphincter tone. Pressure measurements are recorded with color coding (red = high; blue = low). LES = lower esophageal sphincter; PIP = pressure inversion point; UES = upper esophageal sphincter.

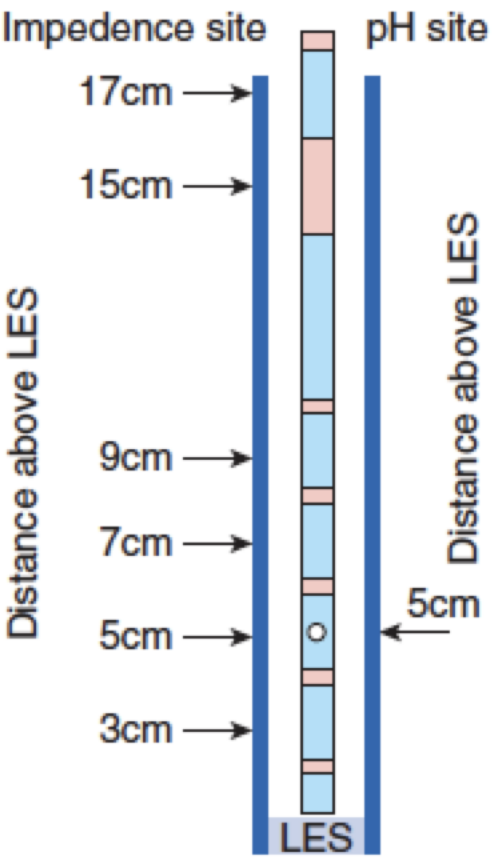


FIGURE 24: Esophageal impedance probe measures electrical resistance between evenly spaced electrodes. LES = lower esophageal sphincter.

Esophageal impedance has been validated as an appropriate method for the evaluation of GI function and is used selectively for the diagnosis of gastro-esophageal reflux. It has been compared to cine-radiography showing that impedance waves correspond well with actual bolus transport illustrated by radiography. Bolus entry, transit, and exit can be clearly identified by impedance changes in the corresponding measuring segments[4].

Studies comparing standard esophageal manometry with impedance measurements in healthy volunteers have shown that esophageal impedance correlates with peristaltic wave progression and bolus length[4].

TWENTY-FOUR-HOUR pH MONITORING:

It is the historical gold standard for diagnosing and quantifying gastro-esophageal reflux, and has some significant limitations. With 24-hour ambulatory pH testing, reflux is defined as a drop in the pH below 4, which effectively "blinds" the test to reflux occurring at higher pH values. Furthermore, in patients with persistent symptoms on proton pump inhibitor (PPI) therapy, pH monitoring has limited use as it can only detect abnormal acid reflux (pH <4), the occurrence of which has been altered by the anti-secretory medication[4].

Given that PPI anti-secretory therapy is highly effective in neutralizing gastric acid, the question of whether persistent symptoms are a result of persistent acid reflux, non-acid reflux, or are not reflux related becomes a key issue in surgical decision making. Until recently, this differentiation could not be made. Detection of both acid and non-acid reflux has potential to define these populations of patients and thus improve patient selection for anti-reflux surgery[4].

Multichannel intra-luminal impedance technology allows the measurement of both acid and non-acid reflux, with potential to enhance diagnostic accuracy. Using this technology, Balaji and colleagues showed that most gastro-esophageal reflux remains despite acid suppression[4].

Impedance pH may be particularly useful in evaluating patients with persistent symptoms despite PPI treatment, patients with respiratory symptoms, and postoperative patients who are having symptoms that are elusive to diagnosis[4].

MANAGEMENT:

CONSERVATIVE MANAGEMENT:

Initially tried as a first line management for asymptomatic patients, patients with small sliding hiatus hernias, immunocompromised state, old age, patients unfit for surgery.

It includes lifestyle modifications such as:

- Consumption of food at regular timings and intervals
- Reducing the consumption of oily or spicy foods
- Abstinence from alcohol, smoking etc

Medical management includes treatment with proton pump inhibitors and barrier forming compounds such as oxetacaine, sucralfate etc. mainly to treat the reflux symptoms and the associated esophagitis.

SURGERY:

Indications:

- GERD refractory to medical therapy, dysphagia, early satiety, postprandial chest or abdominal pain, postprandial shortness of breath, aspiration, chronic anemia (Cameron erosions), or vomiting.
- Bleeding esophagitis
- Esophageal ulceration
- Esophageal stricture
- Nocturnal overflow of gastric contents into the tracheo-bronchial tree
- Large hiatus hernias
- Obstruction or incarceration

The goal of the surgery is prevention of recurrence and elimination of gastro-oesophageal reflux symptoms. Surgical repair can be done by laparotomy, thoracotomy or by laparoscopy (most common)[2].

Laparoscopic hernia repair:

Performed via trans-abdominal approach under general anesthesia with patient in lithotomy position. It has an advantage over open repairs of being relatively safe, with less post-operative complications and reduced hospital stay time.

Relative contraindications to laparoscopic PEH repair include conditions that might preclude or increase the risk of all laparoscopic surgery, such as portal hypertension, significant hematologic clotting disorders, and contraindications to surgery in general, such as inadequate cardiovascular function or the inability to tolerate general anesthesia[2].

Open repairs:

It can be performed trans-abdominally or trans-thoracically.

Open trans-abdominal approach is used in patients who have had a limited number of upper abdominal procedures in the past[2].

The trans-thoracic approach is used in patients who have failed at least two previous trans-abdominal procedures, a history of midline abdominal incisional hernia repair with mesh, history of abdominal abscess, infection, contamination, significant elevated body mass index (BMI) (>40), have a true shortened esophagus, have associated esophageal dysmotility disorders, have a complex type IV PEH[2].

Surgical technique:

To optimize repair durability and to ensure long-term symptom resolution, PEH repair requires strict attention to several key elements[2]

(1) complete reduction of the hernia sac and contents[2]

(2) careful preservation of the anterior and posterior vagus nerves[2]

(3) mobilization of the gastro-esophageal fat pad, resection of excess hernia sac, and identification of the GEJ[2]

(4) recognition and management of a shortened esophagus[2]

(5) extensive mediastinal mobilization and performance of a Collis gastroplasty when necessary for shortened esophagus[2]

(6) preservation of crural integrity with absolute requirement that the peritoneal lining of the crural muscle bodies remain intact[2]

(7) closure of the hiatal defect without tension[2]

(8) consideration of mesh reinforcement of the primary crural closure[2] and

(9) addition of a full or partial fundoplication, or in select cases addition of a gastropexy[2].

MATERIALS AND METHODS

SOURCE OF DATA:

- All patients visiting Shri B.M.Patil Medical College, Hospital and Research centre, Vijayapur between November 2018 to June 2020.

METHOD OF COLLECTION OF DATA:

- This is a prospective cross-sectional observational study of patients presenting with Hiatus hernia in B.L.D.E. (D.U)'S Shri B.M. Patil Medical College Hospital.
- The period of study is from November 2018 to June 2020.
- All patients in both OPD and IPD will be included in the study.
- A pretested structural proforma will be used to collect relevant information for each individual patient selected.
- Detailed history of each individual patient will be taken.
- Endoscopy report of each individual patient selected will be included.
- Any other investigation will be done if required based on history and other complaints.
- Written informed consent will be obtained from all the patients with detailed explanation of the procedure going to be performed on them, the risk factors and complications involved and the advantages and disadvantages of the same.

- Cases will be selected consequently following the inclusion and exclusion criteria.

INCLUSION CRITERIA:

All patients diagnosed of having hiatus hernia on upper GI endoscopy.

EXCLUSION CRITERIA:

All patients undergoing upper GI endoscopy, but not having hiatus hernia.

RESEARCH HYPOTHESIS: To study the changing trends in Hiatus Hernia.

STUDY DURATION: November 2018 to June 2020.

SAMPLING

TOTAL SAMPLE SIZE: 115

With 95% level of confidence and margin of error ±10%, a sample size of 115 subjects will allow the study of clinical profile of patients with hiatus hernia.

Statistical analysis:

The following formula is used to estimate the sample size for a cross-sectional study of clinical profile of the patients with hiatus hernia:

$$n=\frac{Z^2 p(1-p)}{d^2}$$

Where Z= z statistic at 5% level of significance

d= Margin of error

p= Anticipated prevalence rate

- All characteristics will be summarised descriptively.
- For continuous variables, the summary statistics of N, mean, standard deviation (SD) will be used. For categorical data, the number and percentage will be used in the data summaries and data will be analysed by Chi square test for association, comparison of means using t-test, ANOVA and diagrammatic presentation.

INVESTIGATIONS / INTERVENTIONS:

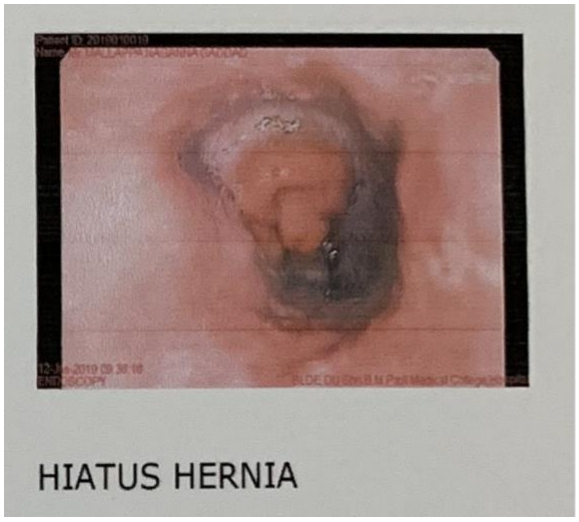
Investigations or interventions required in this study are routine standardized procedures.

There are no animal experiments involved in this study.

These routine investigations are will be done:

- Complete blood picture.
- Tests to detect infection with Human Immunodeficiency Virus and Hepatitis B virus (in accordance with Universal Safety Precautions).
- Upper GI Endoscopy.

UPPER GI ENDOSCOPY REPORTS AND HIATUS HERNIA:



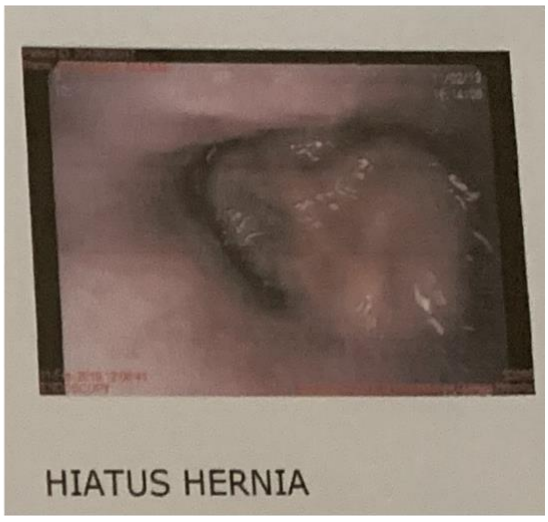


FIGURE 25, 26

BLDE DU Shri.B.M.Patil Medical College,Hospital and RC
ASHRAM ROAD
Vijayapur - 586103
Phone No: 08352262770

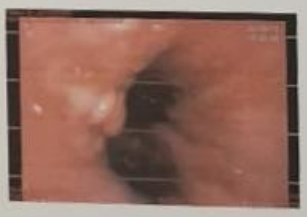
Video Endoscopy Report

Scope Used	:Pentex EPK - P
Indication	: HEMETEMESIS
Premedication	: Xylocaine Spray
Oesophagus	: REFLUX OESOPHAGITIS WITH HIATUS HERNIA.
Stomach	: MULTIPLE EROSIONS NOTED IN THE FUNDUS, BODY OF STOMACH WITH COATING OF MUCOSA WITH FRESH BLOOD.
Proximal Duodenum	: 1st and 2nd Part - Visualized -Normal .No ulcer or stenosis. Normal mucosa, shape , lumen folds. Ampulla of vater appears - Normal.

Endoscopy Conclusion : EROSIVE GASTRITIS WITH REFLUX OESOPHAGITIS WITH HIATUS HERNIA .

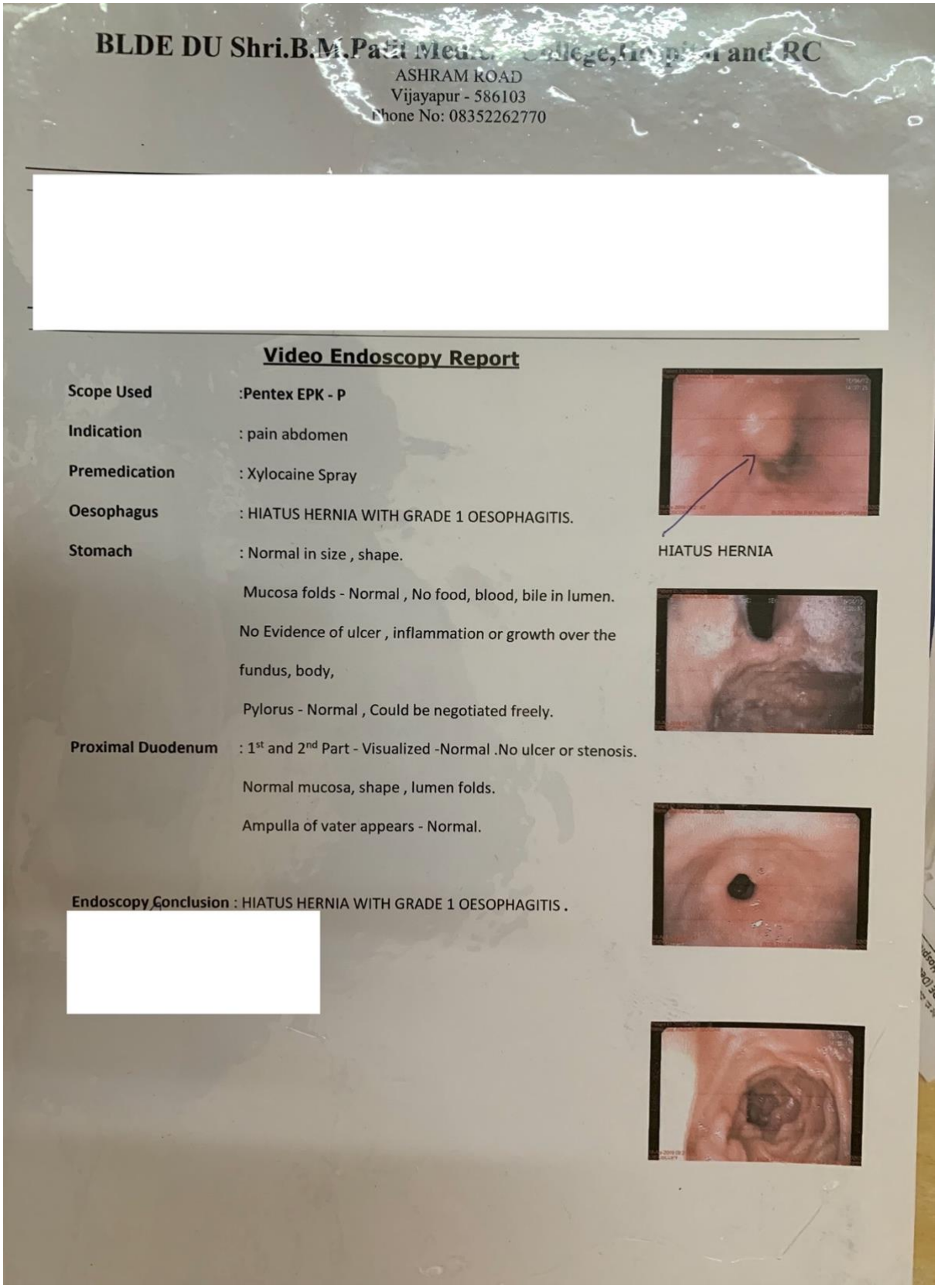
HIATUS HERNIA

EROSIONS



EROSIONS

FIGURE 27



BLDE DU Shri.B.M.Patil Medical College,Hospital and RC
ASHRAM ROAD
Vijayapur - 586103
Phone No: 08352262770

Video Endoscopy Report

Scope Used	**:Pentex EPK - P**
Indication	: pain abdomen
Premedication	: Xylocaine Spray
Oesophagus	: HIATUS HERNIA WITH GRADE 1 OESOPHAGITIS.
Stomach	: Normal in size , shape. Mucosa folds - Normal , No food, blood, bile in lumen. No Evidence of ulcer , inflammation or growth over the fundus, body, Pylorus - Normal , Could be negotiated freely.
Proximal Duodenum	: 1st and 2nd Part - Visualized -Normal .No ulcer or stenosis. Normal mucosa, shape , lumen folds. Ampulla of vater appears - Normal.

Endoscopy Conclusion : HIATUS HERNIA WITH GRADE 1 OESOPHAGITIS .

HIATUS HERNIA

FIGURE 28

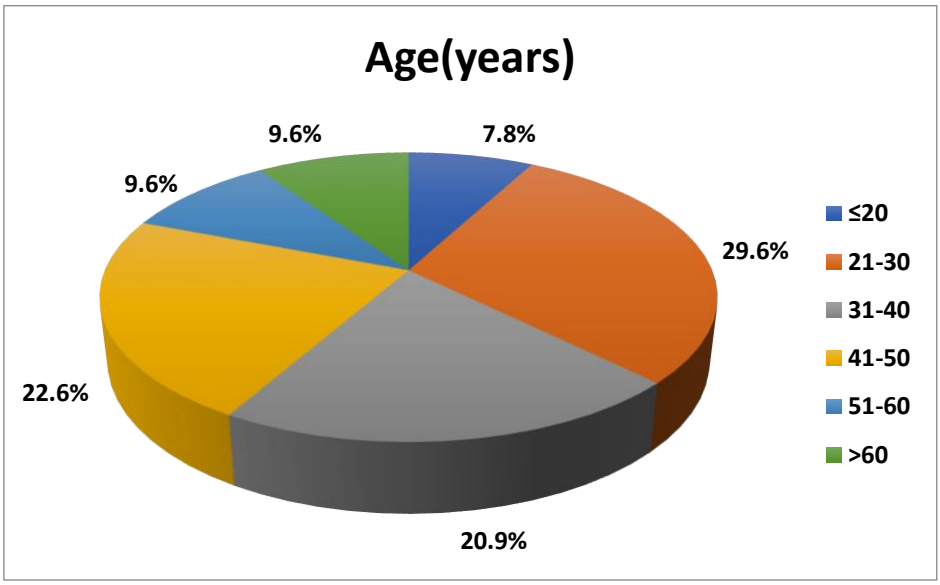
RESULTS

Table 1: Distribution of Cases according to Age:

Age(years)	N	Percent
≤20	9	7.8
21-30	34	29.6
31-40	24	20.9
41-50	26	22.6
51-60	11	9.6
>60	11	9.6
Total	115	100

Descriptive Statistics	Min	Max	Mean	SD
Age(years)	9	85	39.0	15.3

Figure 29: Distribution of Cases according to Age:



The aforementioned tables show a total of 115 patients, out of which most of the patients (29.6%) belonged to age group of 21-30 years, and the youngest patient was a 9 year old male and the oldest patient was an 85 year old female. Mean age was 39.0.

Table 2: Distribution of Cases according to Sex:

SEX	N	Percent
Male	80	69.6
Female	35	30.4
Total	115	100

The above table shows the distribution of sex. The maximum number of patients were male, that is 80 out of 115 patients (69.6%), while there were only 35 females (30.4%).

Figure 30: Distribution of Cases according to Sex:

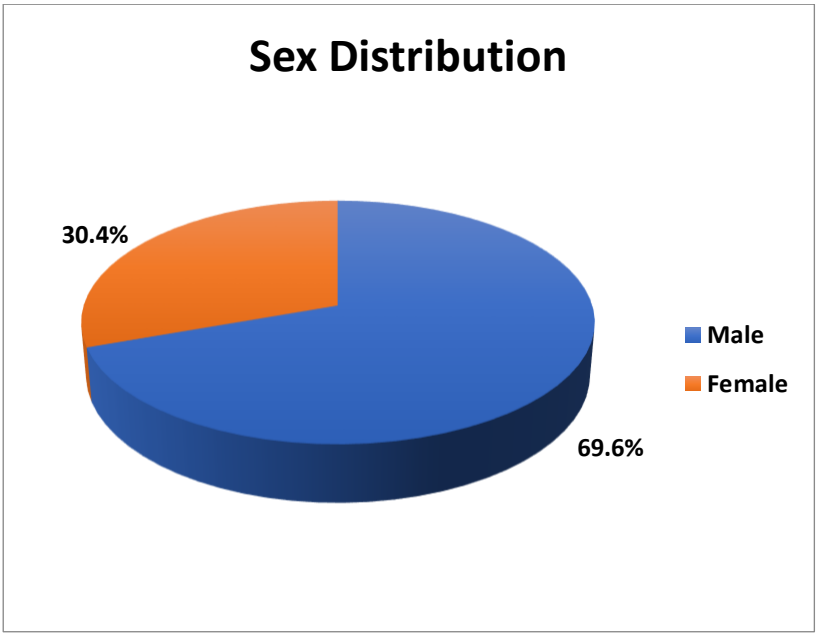


Table 3: Association of Age and Sex:

Age(years)	Male		Female		p value
	N	%	N	%	
≤20	6	7.5%	3	8.6%	0.999
21-30	23	28.8%	11	31.4%	
31-40	17	21.3%	7	20.0%	
41-50	18	22.5%	8	22.9%	
51-60	8	10.0%	3	8.6%	
>60	8	10.0%	3	8.6%	
Total	80	100.0%	35	100.0%	

Figure 31: Association of Age and Sex:

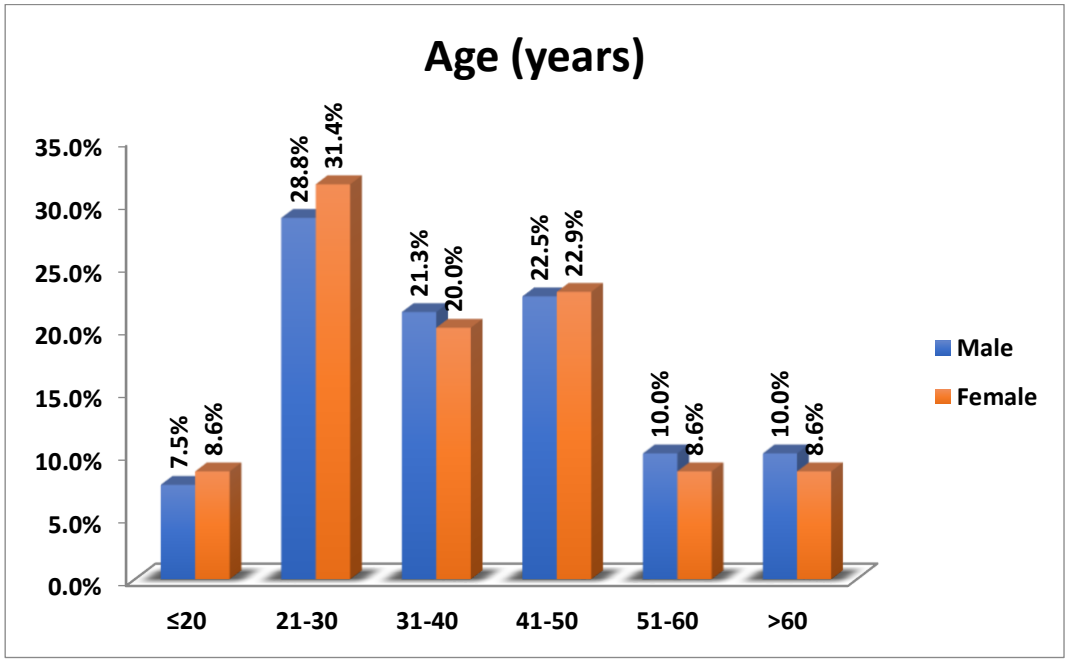
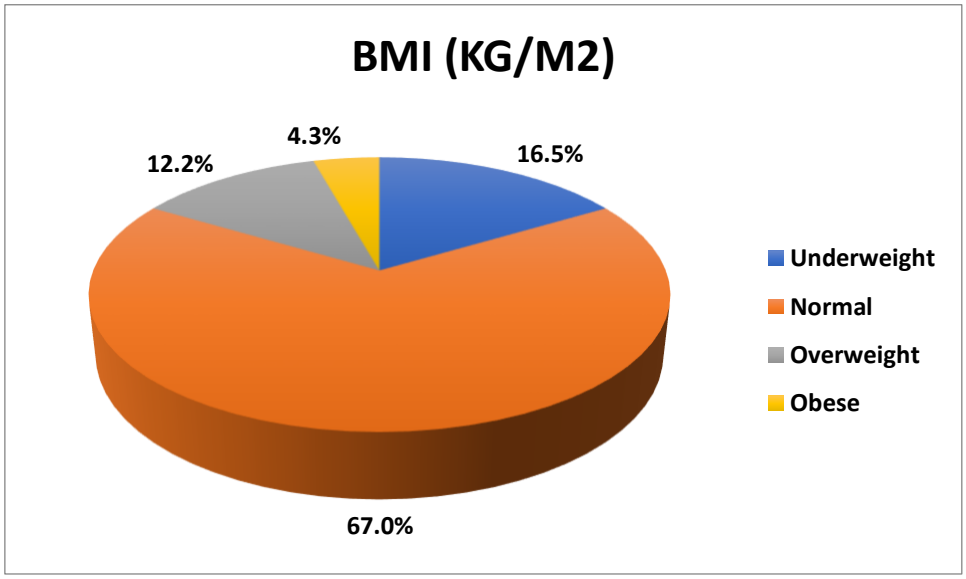


Table 4: Distribution of Cases according to BMI:

BMI (KG/M2)	N	Percent
Underweight	19	16.5
Normal	77	67
Overweight	14	12.2
Obese	5	4.3
Total	115	100

Descriptive Statistics	Min	Max	Mean	SD
BMI (KG/M2)	13.9	37.9	22.1	3.8

Figure 32: Distribution of Cases according to BMI:



Among 115 patients, 67% patients belonged to normal BMI range. Only 5 patients (4.3%) were obese, 14 patients (12.2%) were overweight and 19 patients (16.5%) were underweight.

Table 5: Distribution of Abdominal Girth:

Descriptive Statistics	Min	Max	Mean	SD
ABDOMINAL GIRTH (CM)	51	171	87.6	19.9

Out of 115 patients, the mean abdominal girth was 87.6.

Table 6: Distribution of Cases according to Symptomatology:

The below table shows distribution of cases according to the various symptomatology. Out of 115 patients, most of the patients presented with pain abdomen (52.2%), followed by heart burn (26.1%), vomiting (26.1%) and dysphagia (18.3%).

Other presentations were hematemesis, malena, nausea, abdominal distension, loose stools, mass per abdomen, fever, generalised weakness, cough, foreign body in the throat, sore throat, swelling on the side of neck, throat pain.

SYMPTOMATOLOGY	N	Percent
PAIN ABDOMEN	60	52.2
HEART BURN	30	26.1
VOMITING	30	26.1
DYSPHAGIA	21	18.3
HEMATEMESIS	10	8.7
FEVER	5	4.3
MALENA	3	2.6
NAUSEA	3	2.6
GENERALISED WEAKNESS	2	1.7
ABDOMINAL DISTENSION	1	0.9
COUGH	1	0.9
FOREIGN BODY IN THROAT	1	0.9
LOOSE STOOLS	1	0.9
MASS PER ABDOMEN	1	0.9
SORE THROAT	1	0.9
SWELLING ON SIDE OF NECK	1	0.9
THROAT PAIN	1	0.9

Figure 33: Distribution of Cases according to Symptomatology

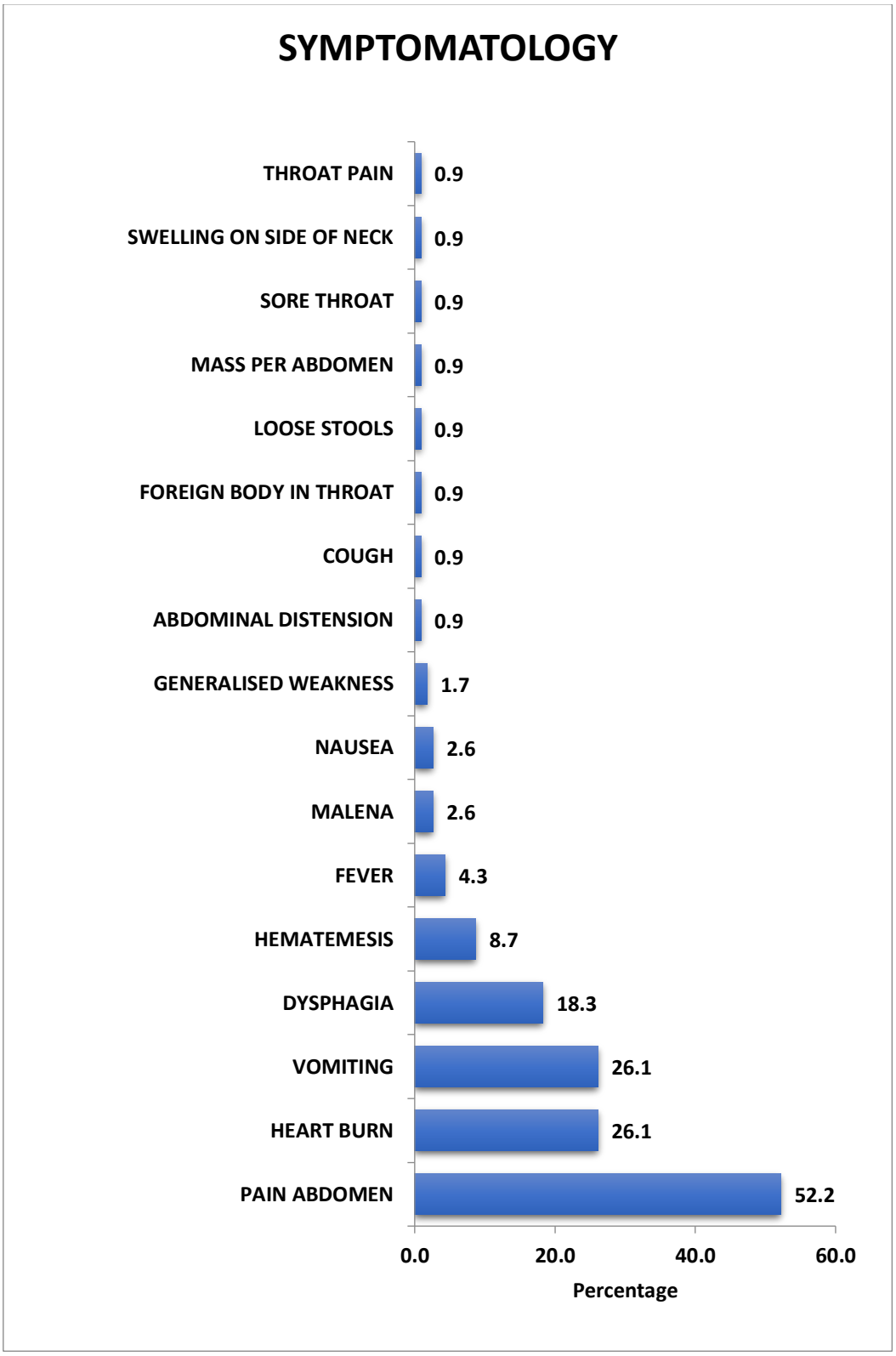
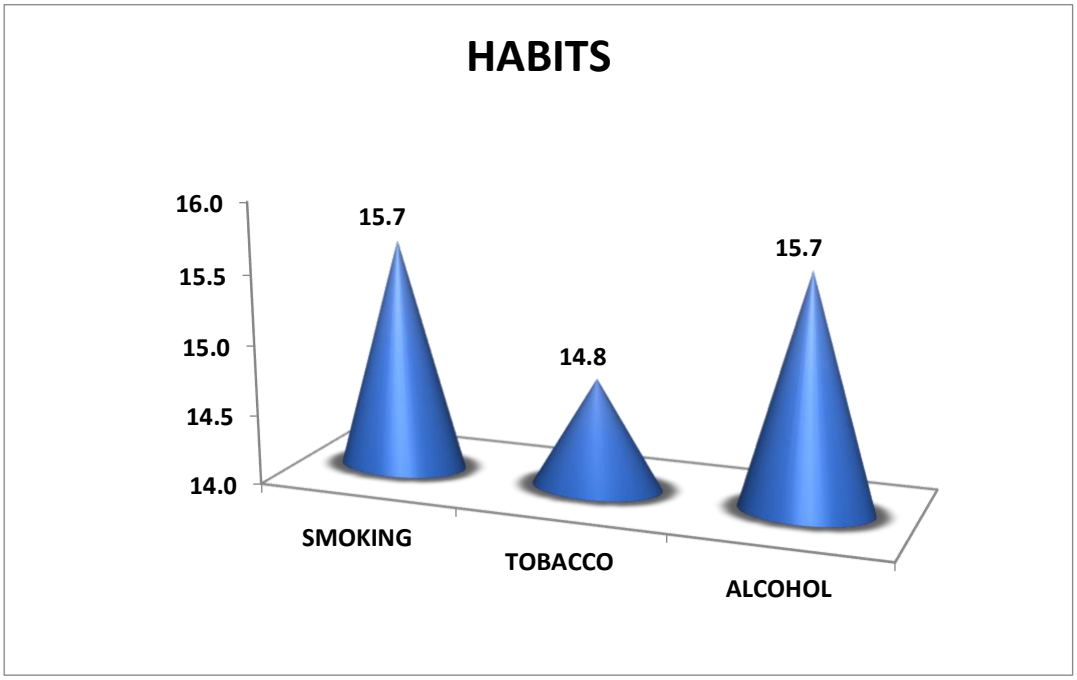


Table 7: Distribution of Cases according to Habits

HABITS	N	Percent
SMOKING	18	15.7
TOBACCO	17	14.8
ALCOHOL	18	15.7

Figure 34: Distribution of Cases according to Habits



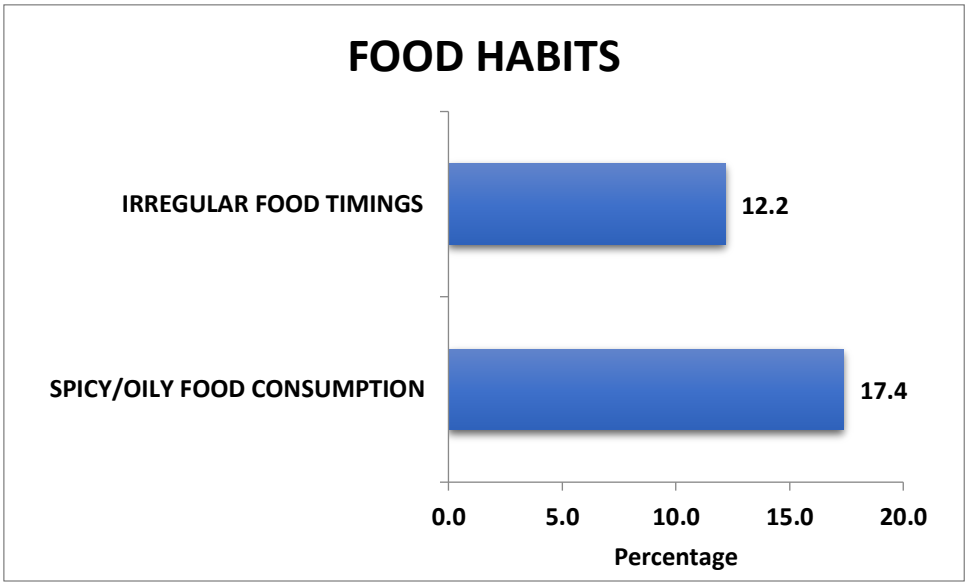
Out of 115 patients, only 53 (46%) patients had addictions. In the above table as shown, the distribution of these 53 patients as smokers was 15.7%, tobacco chewing habits was 14.8% and consumption of alcohol was 15.7%.

Remaining 62 patients out of 115 (54%) had no known habits.

Table 8: Distribution of Cases according to Food Habits

FOOD HABITS	N	Percent
SPICY/OILY FOOD CONSUMPTION	20	17.4
IRREGULAR FOOD TIMINGS	14	12.2

Figure 35: Distribution of Cases according to Food Habits



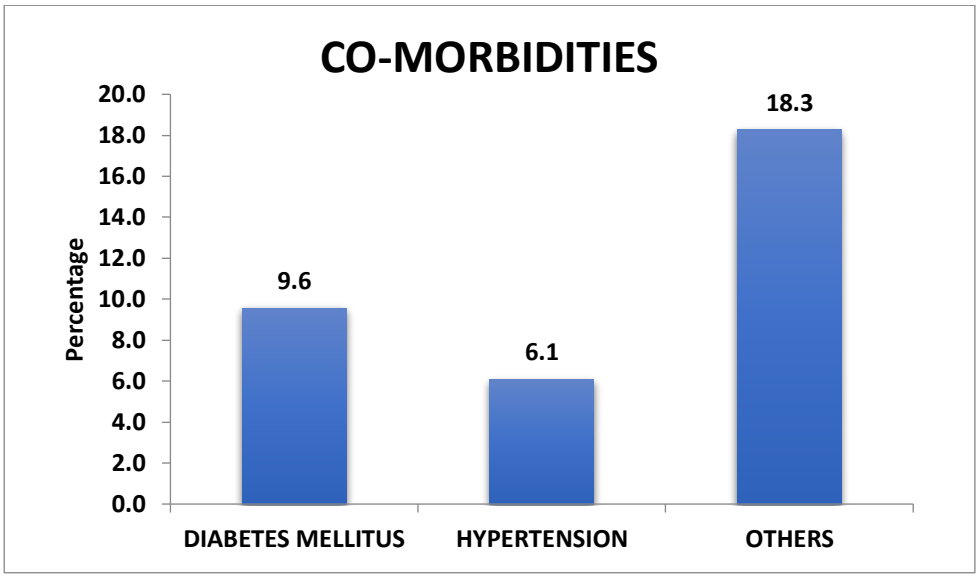
Out of 115 patients, only 34 patients (29.6%) had irregular food habits. 20 patients had a history of consumption of spicy and oily food (17.4%), and 14 patients (12.2%) had irregular food timings.

Majority of the patients, i.e., 81 out of 115 patients (70.4%) had regular and normal food habits.

Table 9: Distribution of Cases according to Co-Morbidities

CO-MORBIDITIES	N	Percent
DIABETES MELLITUS	11	9.6
HYPERTENSION	7	6.1
OTHERS	21	18.3

Figure 36: Distribution of Cases according to Co-Morbidities

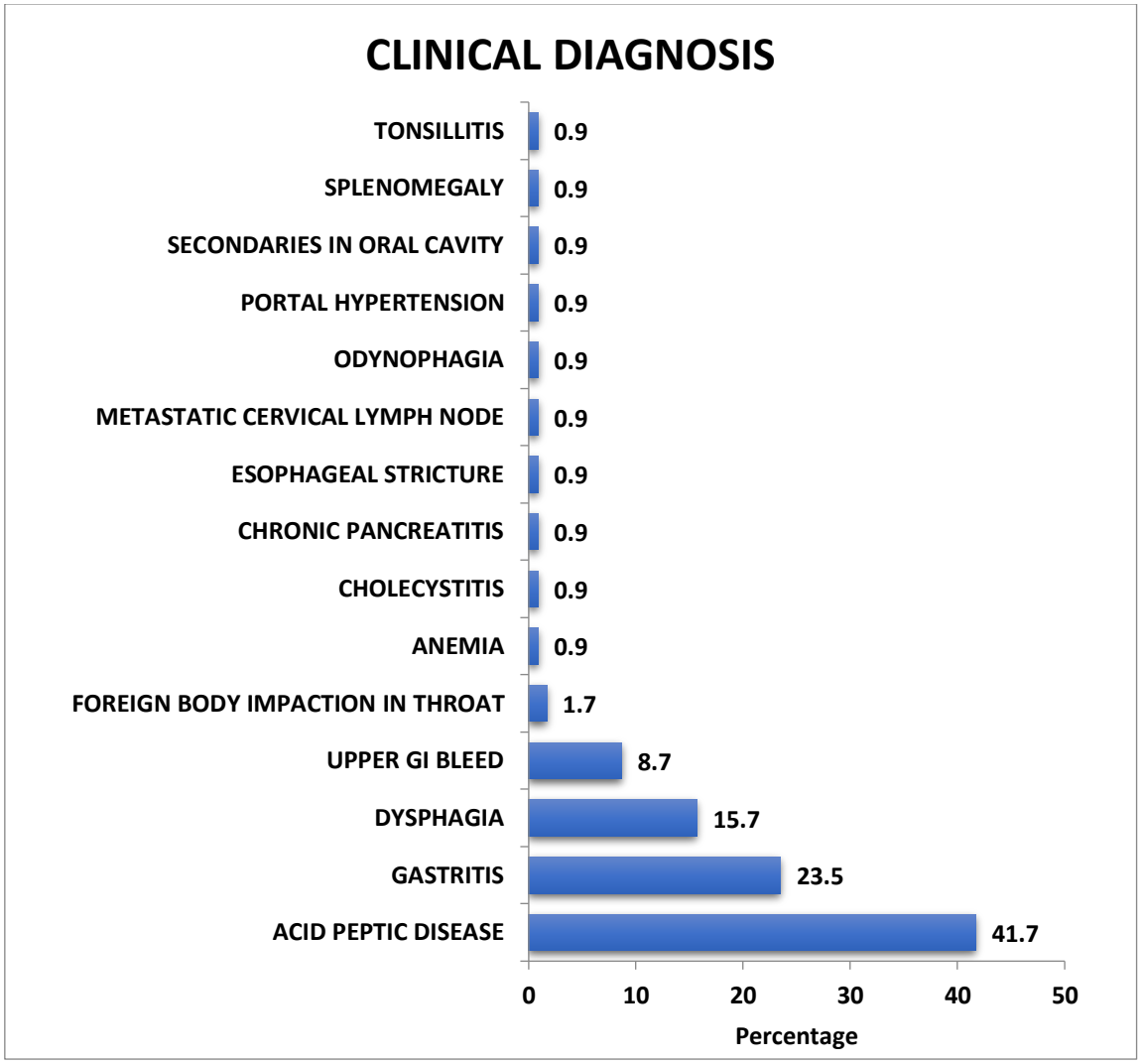


The above table and figure show distribution of cases according to comorbidities. Out of 115 patients, only 39 patients (34%) had various comorbidities including diabetes mellitus (9.6%), hypertension (6.1%), and other factors (18.3%) like history of heart surgeries, renal failure, hypothyroidism etc on medications. Rest of the 76 patients out of 115 (66%) did not have any comorbidities.

Table 10: Distribution of Cases according to Clinical Diagnosis

CLINICAL DIAGNOSIS	N	Percent
ACID PEPTIC DISEASE (GERD)	48	41.7
GASTRITIS	27	23.5
DYSPHAGIA	18	15.7
UPPER GI BLEED	10	8.7
FOREIGN BODY IMPACTION IN THROAT	2	1.7
ANEMIA	1	0.9
CHOLECYSTITIS	1	0.9
CHRONIC PANCREATITIS	1	0.9
ESOPHAGEAL STRICTURE	1	0.9
METASTATIC CERVICAL LYMPH NODE	1	0.9
ODYNOPHAGIA	1	0.9
PORTAL HYPERTENSION	1	0.9
SECONDARIES IN ORAL CAVITY	1	0.9
SPLENOMEGALY	1	0.9
TONSILLITIS	1	0.9
Total	115	100

Figure 37: Distribution of Cases according to Clinical Diagnosis



The above table and figure show the distribution of cases according to clinical diagnosis made before upper GI endoscopy was performed.

Out of 115 patients, maximum number of patients, i.e., 48 (41.7%) were diagnosed with GERD (acid peptic disease) on clinical examination, followed by gastritis (23.5%), dysphagia (15.7%) and upper gastro-intestinal bleed (8.7%). Other clinical diagnoses made were foreign body impaction in throat, pancreatitis, cholecystitis, portal hypertension, oral cavity malignancies etc.

Table 11: Distribution of Cases according to Other Findings on Endoscopy

OTHER FINDINGS	N	Percent
REFLUX ESOPHAGITIS	86	74.8
ANTRAL GASTRITIS	3	2.6
GRADE 1 ESOPHAGEAL VARICES	3	2.6
EROSIVE GASTRITIS	2	1.7
PAN GASTRITIS	2	1.7
ESOPHAGEAL VARICES WITH DUODENAL EROSIONS	1	0.9
GRADE 2 ESOPHAGEAL VARICES WITH GASTRITIS	1	0.9
LAX OG JUNCTION	1	0.9
MALLORY WEISS TEAR AT GE JUNCTION WITH REFLUX ESOPHAGITIS	1	0.9
REFLUX GASTRITIS	1	0.9
Total	115	100

The above table shows distribution of cases according to other findings found on upper GI endoscopy, in addition to hiatus hernia.

Out of 115 patients, majority of them i.e., 86 patients (74.8%) had hiatus hernia with reflux esophagitis and the rest of the patients had various other findings like gastritis (6.9%), esophageal varices, duodenal erosions, etc.

Figure 38: Distribution of Cases according to Other Findings on Endoscopy

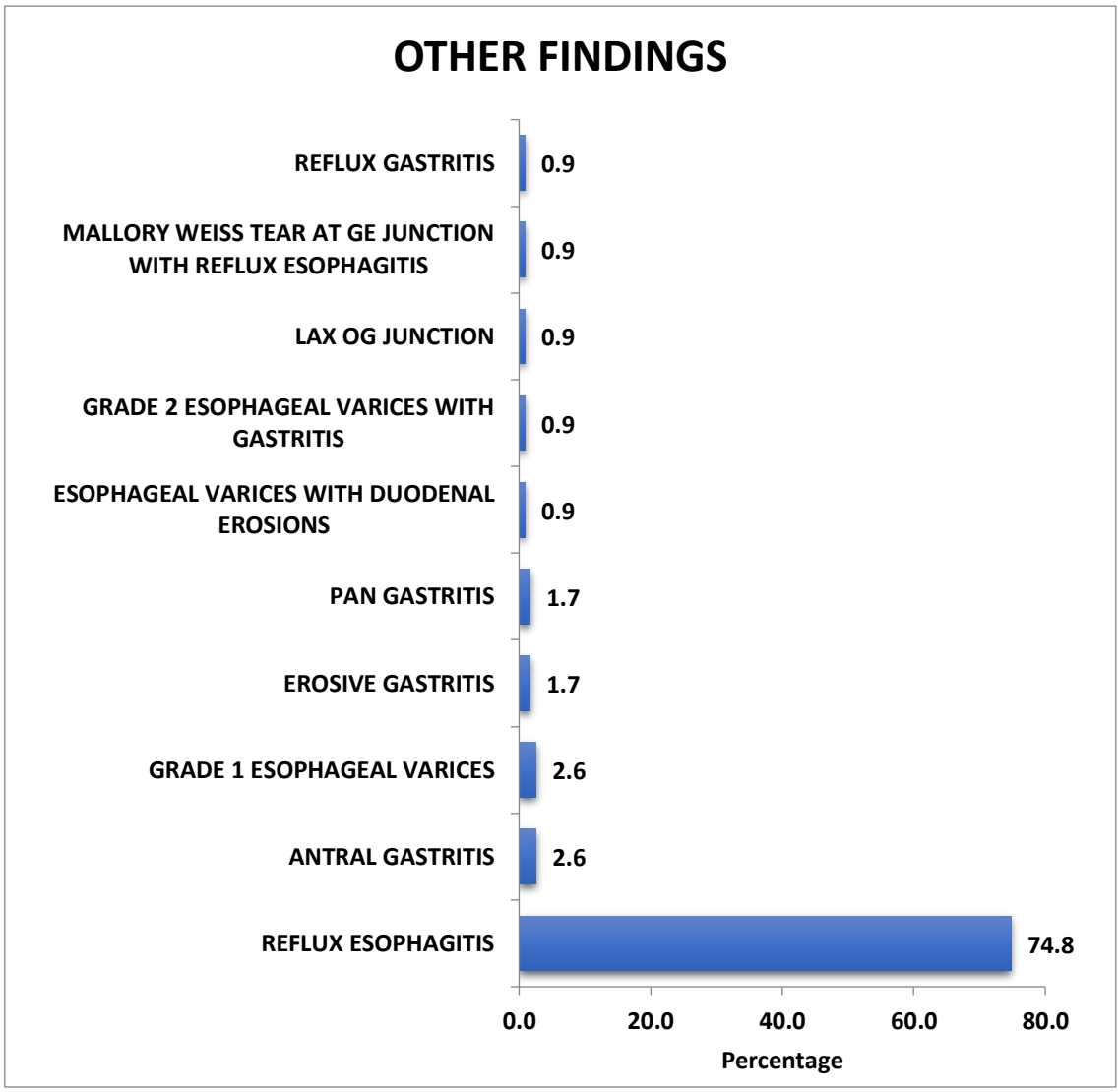
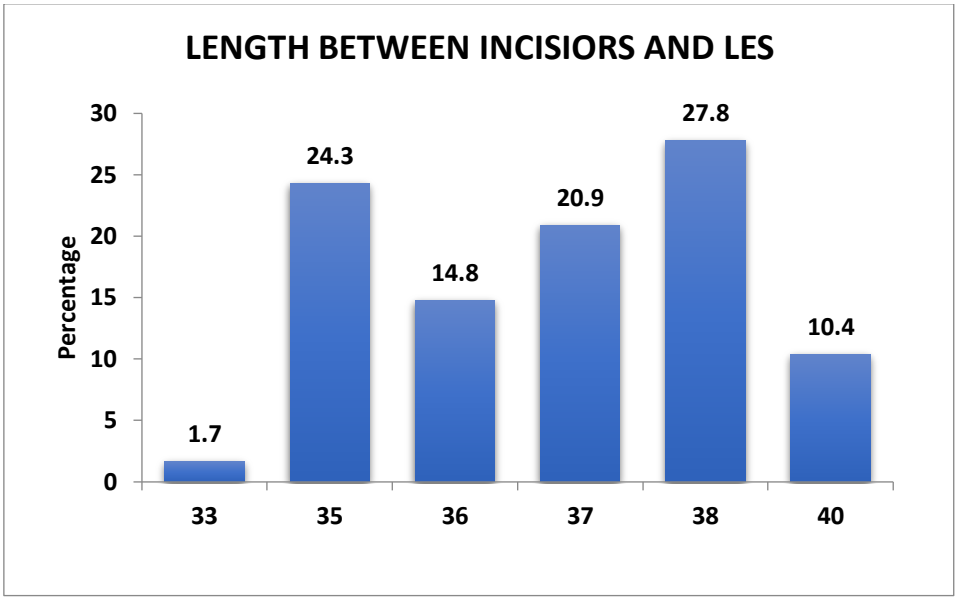


Table 12: Distribution of Length between Incisors and LES

LENGTH BETWEEN INCISIORS AND LES (cm)	N	Percent
33	2	1.7
35	28	24.3
36	17	14.8
37	24	20.9
38	32	27.8
40	12	10.4
Total	115	100

Descriptive Statistics	Min	Max	Mean	SD
LENGTH BETWEEN INCISIORS AND LES (CM)	33	40	36.9	1.6

Figure 39: Distribution of Length between Incisors And LES



The above table and figure show distribution of length between incisor and lower esophageal sphincter on upper GI endoscopy, with the mean length being 36.9.

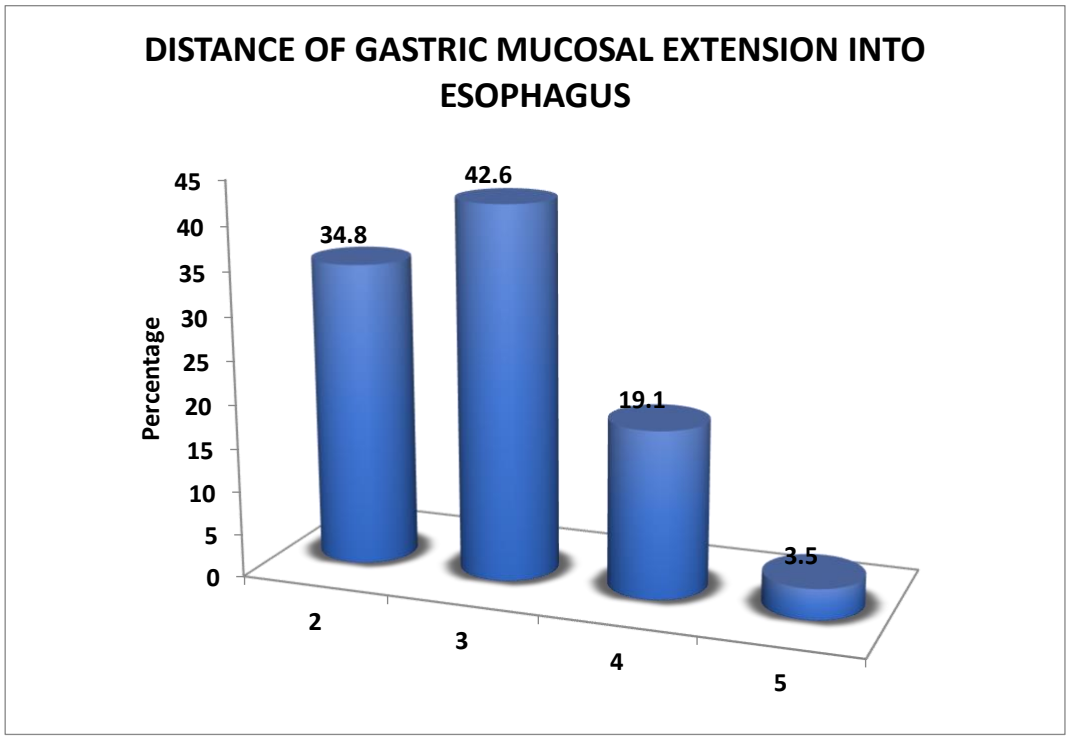
Table 13: Distribution of Distance of Gastric Mucosal Extension Into Esophagus

DISTANCE OF GASTRIC MUCOSAL EXTENSION INTO ESOPHAGUS (CM)	N	Percent
2	40	34.8
3	49	42.6
4	22	19.1
5	4	3.5
Total	115	100

Descriptive Statistics	Min	Max	Mean	SD
DISTANCE OF GASTRIC MUCOSAL EXTENSION INTO ESOPHAGUS (CM)	2	5	2.9	0.8

The above table shows the distribution of cases according to distance of gastric mucosal extension into the esophagus as seen on upper GI endoscopy, with the mean distance being 2.9.

Figure 40: Distribution of Distance of Gastric Mucosal Extension Into Esophagus



DISCUSSION

Hiatus hernia has become one of the most common findings on upper GI endoscopy. But not much is known about its incidence.

Simplistically, the hiatus hernia can be caused by one or more of three mechanisms[5]:

(i) widening of the diaphragmatic hiatus[5],

(ii) pulling up of the stomach by oesophageal shortening[5], and

(iii) pushing up of the stomach by increased intra-abdominal pressure[5].

In our study, a total of 115 patients presenting with various symptomatology who have been diagnosed with hiatus hernia on upper GI endoscopy have been studied. Various factors like age, sex, symptomatology, habits, comorbidities, BMI, abdominal girth, clinical diagnosis on presentation and other endoscopic findings were studied.

In our study, hiatus hernia was found to be most prevalent in young adults between the age group of 21-30 years (29.6%). Only 9.6% of the patients were more than 60 years old. This is significant in contrast to the available literature which observes that hiatus hernia is a finding in older age groups. In a study by Loffeld et al. in 2002, on incidence of hiatus hernia, they concluded that hiatus hernia developed in significantly older patients[6].

According to our present study, out of 115 patients, a significant male predominance (69.6%) was observed for the incidence of hiatus hernia. In a

study by Loffeld et al. in 2002, they concluded that the number of women developing hiatus hernia was significantly higher than the number of men developing hiatus hernia ($p<0.0001$)[6]. In another 20 year retrospective study on hiatus hernia by P.R. Allison, in 1973, out of 898 patients, 538 were females and 361 were males, giving a ratio of 3:2 in favour of females[7].

Lax phreno-esophageal ligament is the main pathogenesis behind development of hiatus hernia. A high BMI indicates obesity which predisposes to the laxity of the phreno-esophageal ligament and thus development of hiatus hernia. In this study, a significant number of the patients diagnosed with hiatus hernia had a normal BMI range i.e., 67% of the patients had BMI ranging from 18.5 to 24.9. Only 4.3% patients were obese and 12.2% were overweight. This is in contrast to other studies which conclude that hiatus hernia is more prevalent in the obese individuals. In a study conducted by Fredrick Che et al. on the prevalence of hiatus hernia in the morbidly obese, they identified the presence of hiatus hernia in nearly 40% of morbidly obese patients[8].

In another study by Louis. J. Wilson et al. in 1999, on the association of obesity with hiatal hernia and esophagitis, they concluded that excessive body weight is a significant independent risk factor for hiatus hernia[9]. This retrospective case-control study suggested that obesity, as defined by BMI, is significantly associated with both hiatal hernia and esophagitis, through various mechanisms like increased intra-abdominal pressure, increased intra-gastric pressure, greater

lower esophageal sphincter (LES) relaxation, an abnormal diaphragmatic pinchcock and a delayed acid clearance[9].

In our study, abdominal girth of all the 115 patients were measured at the umbilicus level, to assess the variations and prevalence of waist belt obesity in patients with hiatus hernia. An abdominal girth less than 90cm was considered to be in the normal range. Most of the patients (68.6%) had an abdominal girth measuring less than 90cm irrespective of their age and sex.

Hiatus hernia patients are significantly more likely to have regurgitation symptoms with heart burn and dyspepsia, with or without the presence of reflux esophagitis on upper GI endoscopy (i.e., non-erosive disease). In this study, we studied the different symptomatology with which hiatal hernia patients presented. Majority of the symptoms with which the patients presented included pain abdomen (52.2%), heart burn and dyspepsia (26.1%), vomiting (26.1%) and dysphagia (18.3%). Other presenting complaints included hematemesis (8.7%), fever (4.3%), melena (2.6%), nausea (2.6%), generalized weakness (1.7%), abdominal distension (0.9%), cough (0.9%), foreign body in throat (0.9%), loose stools (0.9%), mass per abdomen (0.9%) and sore throat (0.9%).

In a 2004 review article by Gordon et al., on the role of hiatus hernia in gastro-esophageal reflux disease, out of 57 healthy subjects, 62% with gastro-esophageal reflux symptoms had hiatus hernia on upper GI endoscopy compared with only 14% of asymptomatic subjects[5]. And out of 930 patients, with exclusion of patients with reflux esophagitis, patients with hiatus hernia

were significantly more likely to have heartburn and regurgitation compared to those without[5]. In another 20 year retrospective study, done in 1973, on hiatus hernia, by P.R. Allison, it was concluded that in hiatal hernia patients, the commonest symptoms were pain, flatulence, regurgitation and vomiting and dysphagia[7]. These findings of other studies are in alignment with the findings in our present study with respect to the presenting symptoms of the patients with hiatus hernia.

Smoking a cigarette reduces the gastro-esophageal barrier pressure and may allow reflux, with accompanying symptoms to occur[10]. The probable mechanism is that inhaled nicotine blocks the cholinergic control mechanism. In-vitro nicotine causes relaxation of circular muscle fibres at the lower esophagus[10]. In a 1972 study by Stanciu et al., on gastroesophageal reflux and smoking, they studied the gastroesophageal sphincter pressure in 25 chronic smokers with complaints of heartburn and concluded that cigarette smoking is a common reversible cause of gastro-esophageal reflux[10]. In our present study, out of 115 patients, only 15.7% were smokers, 14.8% consumed tobacco and 15.7% consumed alcohol. These findings are not suggestive of a strong association of smoking, alcohol or tobacco consumption with the incidence of hiatus hernia.

In our study, we studied the association of dietary habits with incidence of hiatus hernia. Out of 115 subjects, only 17.4% reported consumption of spicy and oily foods on a daily basis, and only 12.2% had irregular meal timings

which might lead to underlying reflux symptoms. These findings don't appear to be significantly associated with the incidence of hiatus hernia.

Out of 115 subjects in the present study, it was observed that 9.6% had diabetes mellitus, 6.1% were hypertensive and 18.3% had other co-morbidities like alcoholic liver disease, renal failure, pancreatitis, oral cavity malignancies etc.

Gastro-esophageal reflux disease (GERD) is a condition that is defined as mild symptoms that occur atleast 2 times per week or moderate to severe symptoms that occur atleast once per week, that result from the reflux of gastric contents into the esophagus or beyond into the oral cavity or lung[2]. Heartburn, regurgitation, dysphagia and epigastric pain are considered as typical symptoms of GERD. In our study, most of the cases were initially diagnosed clinically as GERD (acid peptic disease) (41.7%) and gastritis (23.5%) based on the symptomatology of presentation. Other clinical diagnosis included upper GI bleed, dysphagia, cholecystitis, chronic pancreatitis, oral cavity malignancies and secondaries, foreign body impaction etc.

Hiatus hernia is considered one of the most important factor in reflux mechanisms. Many studies have shown that reflux esophagitis and hiatus hernia coexist. In our study, a significant number of patients (74.8%) had associated reflux esophagitis. In another supporting study on relationship of hiatus hernia to reflux esophagitis, by Berstad et al., in 1986, they concluded that hiatus hernia is significantly associated with reflux esophagitis[11]. In another study on the importance of hiatal hernia in reflux esophagitis compared with lower

esophageal sphincter pressure or smoking, by Stephen et al., in 1991, they concluded that the correlations between hiatal hernia and esophagitis, hiatal hernia and frequency, and hiatal hernia and acid contact time suggest that hiatal hernia exerts its deleterious effect on the esophageal mucosa by allowing more frequent episodes of acid reflux, which in turn increase the time that the esophageal mucosa is exposed to the acid[12].

The normal endoscopic length of esophagus from the incisors to lower esophageal sphincter ranges from 38 to 40cm[2]. During a normal swallow, the oesophagus shortens by up to 2 cm[13]. The elasticity of supporting structures, especially the phreno-esophageal ligament, returns the anatomy to its normal position[13]. In hiatus hernia, the phreno-esophageal ligament becomes lax. In our study, the mean length between incisors and lower esophageal sphincter was 36.9 with a standard deviation of 1.6. In a study by Peter J Kahrilas et al., on attenuation of esophageal shortening during peristalsis with hiatus hernia, in 1995, they concluded that esophageal shortening with hiatus hernia maybe the secondary consequence of the distal esophageal longitudinal muscle remaining partially shortened because of reduced opposing forces[13].

SUMMARY

This study on 'clinical profile of patients with hiatus hernia' is conducted in BLDE (DU) Shri B.M. Patil Medical College Hospital and Research Center, Vijayapur, between November 2018 to June 2020. All the patients with upper gastrointestinal symptoms were subjected to a routine upper GI endoscopy and the patients diagnosed with hiatus hernia were noted and their clinical profiles were studied. In our study of 115 patients with hiatus hernia -

- It was most commonly seen in young adults between 21-30 years.
- Males were more commonly noted to have hiatus hernia i.e., 69.6%.
- Hiatus hernia was often diagnosed on upper GI endoscopy in individuals with normal BMI.
- 78.4% of total patients presented with complaints of epigastric pain and heart burn and were diagnosed as GERD clinically.
- Hiatus hernia does not seem to be associated significantly with habits like smoking, alcohol or tobacco consumption.
- Hiatus hernia does not seem to be associated with spicy/oily food consumption.
- Associated co-morbidities in individuals like diabetes, hypertension, etc does not seem to play much role in incidence of hiatus hernia.
- Reflux esophagitis remains the most common (74.8%) associated finding with hiatus hernia on upper GI endoscopy, followed by gastritis.

CONCLUSION

Hiatus hernia is becoming one of the most common findings on upper GI endoscopy. But not much data is available regarding its incidence. It was previously believed that hiatus hernia was more commonly a part of gastro-esophageal reflux disease. Older studies have concluded that hiatus hernia was mainly a presentation in old age, with female preponderance, and obese individuals. Not much recent studies have been done on this subject.

In our study, we found that hiatus hernia is one of the common findings on upper GI endoscopy. We concluded that it is more common in young adults, having a male preponderance. Hiatus hernia does not seem to be specifically associated with obese individuals or lifestyle changes like smoking, alcohol consumption or spicy food consumption, as presumed.

This change in trends of incidence of hiatus hernia is in need of dire attention and further investigation. There is an obvious need for more studies to recognise hiatus hernia as its own entity, but not just as a part of gastro-esophageal reflux disease. Our study is an effort to note these changing trends of incidence of hiatus hernia.

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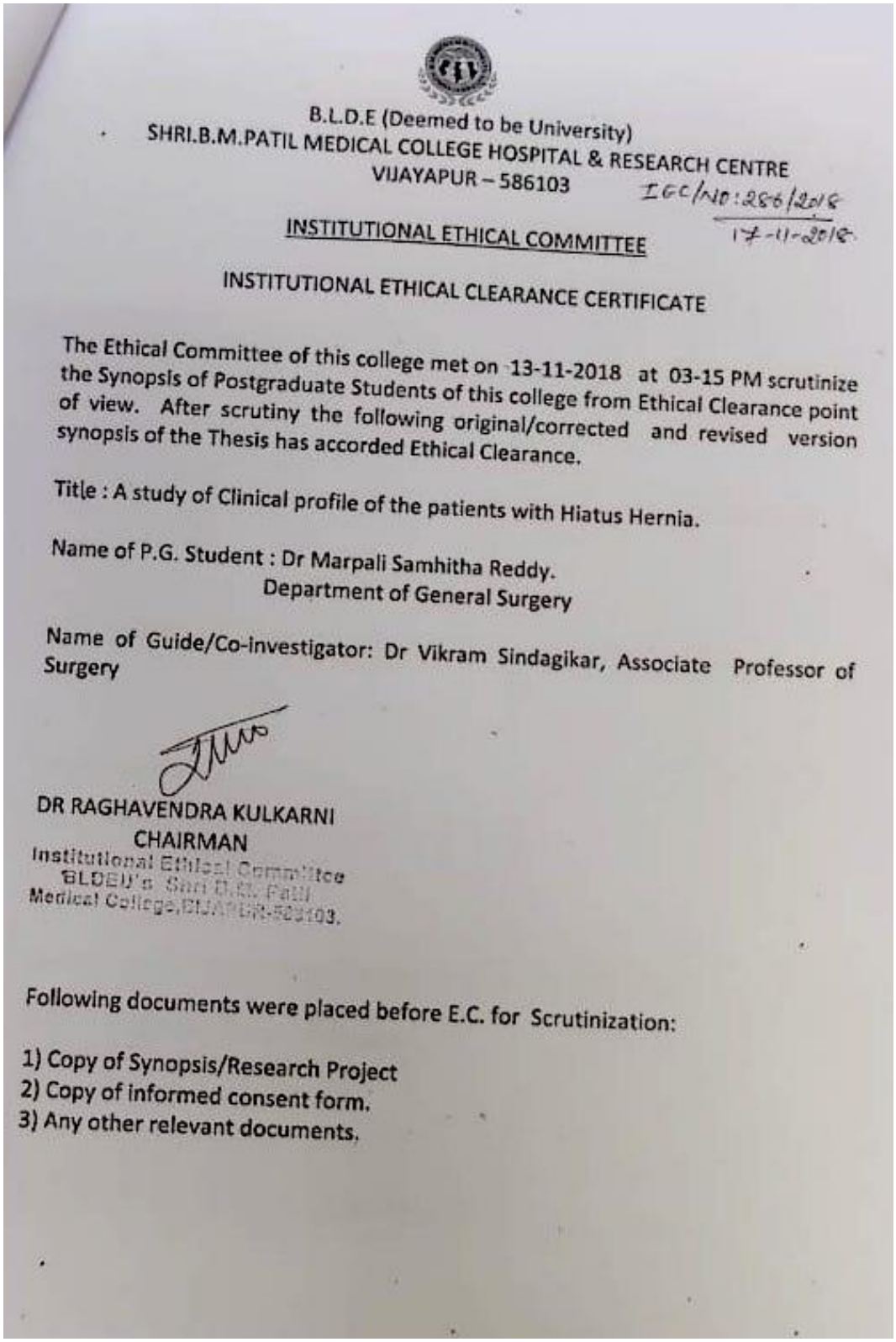
ANNEXURE I: CERTIFICATE OF ETHICAL CLEARANCE



B.L.D.E (Deemed to be University)
SHRI.B.M.PATIL MEDICAL COLLEGE HOSPITAL & RESEARCH CENTRE
VIJAYAPUR – 586103

IEC/NO:286/2018
17-11-2018

INSTITUTIONAL ETHICAL COMMITTEE

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Ethical Committee of this college met on 13-11-2018 at 03-15 PM scrutinize the Synopsis of Postgraduate Students of this college from Ethical Clearance point of view. After scrutiny the following original/corrected and revised version synopsis of the Thesis has accorded Ethical Clearance.

Title : A study of Clinical profile of the patients with Hiatus Hernia.

Name of P.G. Student : Dr Marpali Samhitha Reddy.
Department of General Surgery

Name of Guide/Co-investigator: Dr Vikram Sindagikar, Associate Professor of Surgery

DR RAGHAVENDRA KULKARNI
CHAIRMAN
Institutional Ethical Committee
BLDEU's Shri B.M. Patil
Medical College,BIJAPUR-586103.

Following documents were placed before E.C. for Scrutinization:

1) Copy of Synopsis/Research Project
2) Copy of informed consent form.
3) Any other relevant documents.

ANNEXURE II: PARTICIPANT CONSENT FORM

TITLE OF THE PROJECT:

"STUDY OF CLINICAL PROFILE OF PATIENTS WITH HIATUS HERNIA"

NAME OF THE INVESTIGATOR: **Dr. MARPALI SAMHITHA REDDY**

NAME OF THE GUIDE: **Dr. VIKRAM SINDGIKAR**

INTERVENTION: **Upper GI Endoscopy**

CONFIDENTIALITY OF RECORDS:

I understand that medical information produced by this study will become a part of this hospital records and will be subjected to the confidentiality and privacy regulation of this hospital. Information of a sensitive, personal nature will not be a part of the medical records, but will be stored in the investigator's research file only by a code number. The code key connecting name to numbers will be kept in the medical records. If the data are used for publication in the medical literature or for teaching purpose, no names will be used and other identifiers such as photographs and audio or video tapes will be used only with my special written permission. I understand that I may see the photograph and videotapes and hear audiotapes before giving this permission.

REQUEST FOR MORE INFORMATION:

I understand that I may ask more questions about the study at any time, Dr. MARPALI SAMHITHA REDDY is available to answer my questions or concerns. I understand that I will be informed of any significant new findings discovered during the course of this study, which might influence my continued participation.

If during this study, or later, I wish to discuss my participation in or concerns regarding this study with a person not directly involved, I am aware that the social worker of the hospital is available to talk with me, and that a copy of this consent form will be given to me to keep it and for careful reading.

REFUSAL OR WITHDRAWAL OF PARTICIPATION:

I understand that my participation is voluntary and I may refuse to participate or may withdraw consent and discontinue participation in the study at any time without prejudice to my present or future care at this hospital.

I also understand that Dr. Marpali Samhitha Reddy, will terminate my participation in this study at any time after she has explained the reasons for doing so and has helped arrange for my continued care by my own physician or therapist, if this is appropriate.

INJURY STATEMENT:

I understand that in the unlikely event of injury to me/my ward, resulting directly in my participation in this study, if such injury were reported promptly, then medical treatment would be available to me, but no further compensation will be provided.

I understand that by my agreement to participate in this study, I am not waiving any of my legal rights.

I have explained to _____ the purpose of this research, the procedures required and the possible risks and benefits, to the best of my ability in patient's own language.

Date:

Dr. Vikram Sindgikar
(Guide)

Dr. Marpali Samhitha Reddy
(Investigator)

Participant's name:

Address:

TITLE OF THE PROJECT:

"STUDY OF CLINICAL PROFILE OF PATIENTS WITH HIATUS HERNIA"

The details of the study have been provided to me in writing and explained to me in my own language. I confirm that I have understood the above study and had the opportunity to ask questions. I understand that my participation in the study is voluntary and that I am free to withdraw at any time, without giving any reason, without the medical care that will normally be provided by the hospital being affected. I agree not to restrict the use of any data or results that arise from this study provided such a use is only for scientific purpose(s). I have been given an information sheet giving details of the study. I fully consent to participate in the above study.

_____

_____

(Participant) (Date)

_____

_____

(Witness to signature) (Date)

_____

_____

(Investigator to signature) (Date)

ANNEXURE III: PROFORMA

CASE NUMBER: IP NUMBER:

NAME: OPD NUMBER:

AGE/SEX: WARD/UNIT:

OCCUPATION: DATE OF ADMISSION:

ADDRESS: DATE OF ENDOSCOPY:

DATE OF DISCHARGE:

CHIEF COMPLAINTS:

HISTORY OF PRESENTING ILLNESS:

PAST HISTORY:

TREATMENT HISTORY:

SURGICAL HISTORY:

PERSONAL HISTORY: Dietary Habits-

Appetite- Sleep-

Bowel and bladder habits-

Habits-

GENERAL PHYSICAL EXAMINATION:

BUILT: Well / Moderate / Poor

NOURISHMENT: Well / Moderate / Poor

WEIGHT: kg , HEIGHT: cm , BMI: kg/m^2

PALLOR- ICTERUS- CYANOSIS- CLUBBING-

PEDAL EDEMA- GENERALISED LYMPHADENOPATHY-

VITALS: Temperature-

Pulse- bpm , Blood Pressure- mmHg

Respiratory Rate- cpm

SYSTEMIC EXAMINATION:

PER ABDOMEN: Abdominal Girth- cm

Abdominal Tone-

RESPIRATORY SYSTEM:

CARDIOVASCULAR SYSTEM:

CENTRAL NERVOUS SYSTEM:

CLINICAL DIAGNOSIS:

LABORATORY TESTS:

Complete Blood Count: Total Count- Hb- gm%

RBC- mil/cumm , Platelet count-

Chest X-Ray:

INDICATIONS FOR ENDOSCOPY:

ENDOSCOPY FINDINGS:

Length between incisors and lower oesophageal sphincter-

Distance of gastric mucosal extension into the oesophagus-

Other associated findings-

KEY TO MASTER CHART

SL NO - Serial Number

IP NO - In patient Number

M - Male

F - Female

BMI - Body Mass Index

LES - Lower Esophageal Sphincter

S.NO.	NAME	AGE	SEX	IP/OP NO.	DATE OF ENDOSCOPY	SYMPTOMATOLOGY					HABITS			FOOD		CO-MORBIDITIES			BMI (KG/M2)	ABDOMINAL GIRTH (CM)	CLINICAL DIAGNOSIS	ENDOSCOPY FINDINGS		
						PAIN ABDOMEN	HEART BURN	DYSPHAGIA	VOMITING	OTHERS	SMOKING	TOBACCO	ALCOHOL	SPICY/OILY FOOD CONSUMPTION	IRREGULAR FOOD TIMINGS	DIABETES MELLITUS	HYPERTENSION	OTHERS				LENGTH BETWEEN INCISIORS AND LES (CM)	DISTANCE OF GASTRIC MUCOSAL EXTENSION INTO ESOPHAGUS (CM)	OTHER FINDINGS
1	BHIMARAYYA	50	M	30809	11/09/18	YES	NO	NO	NO	NO	YES	NO	YES	YES	YES	NO	NO	H/O HEART SURGERY ON MEDICATION	23	83	ACID PEPTIC DISEASE	40	3	REFLUX ESOPHAGITIS
2	BASAVESHWAR	33	M	3E+05	10/09/18	YES	YES	NO	NO	NO	NO	NO	NO	YES	NO	NO	NO	NO	25.2	91	ACID PEPTIC DISEASE	40	3	REFLUX ESOPHAGITIS
3	ANASABAI	30	F	34478	09/10/18	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	24	83	ACID PEPTIC DISEASE	40	3	
4	SOMAYYA	9	M	4E+05	17/11/18	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	13.9	51	ESOPHAGEAL STRICTURE	35	2	REFLUX ESOPHAGITIS
5	VISHAL	33	M	4E+05	17/11/18	NO	NO	YES	YES	COUGH	YES	NO	NO	NO	NO	YES	NO	H/O RTA	26.2	94	DYSPHAGIA	38	2	REFLUX ESOPHAGITIS WITH FUNDAL EROSIONS
6	SHIVAPPA	17	M	39175	17/11/18	YES	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	16.6	68	ACID PEPTIC DISEASE	40	3	REFLUX ESOPHAGITIS
7	NAGAPPA	46	M	4E+05	19/11/18	NO	NO	YES	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	24.8	89	DYSPHAGIA	40	3	GRADE 1 ESOPHAGEAL VARICES
8	KALLAPPA	27	M	4E+05	19/11/18	YES	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	23.4	85	ACID PEPTIC DISEASE	40	3	
9	SURYAKANTH	67	M	4E+05	25/12/18	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	YES	NO	H/O STENTING +, K/C/O ARTHRITIS	24.2	95	DYSPHAGIA	40	3	REFLUX ESOPHAGITIS
10	ANAND	28	M	4E+05	28/12/18	NO	YES	NO	NO	NO	NO	NO	NO	YES	YES	NO	NO	NO	23	68	GASTRITIS	40	3	REFLUX ESOPHAGITIS
11	MALLAPPA	45	M	1124	12/01/19	NO	YES	NO	YES	HEMATEMESIS	NO	YES	YES	NO	NO	NO	NO	NO	20.4	81	UPPER GI BLEED	38	4	REFLUX ESOPHAGITIS WITH FUNDAL EROSIONS
12	BASAVARAJ	28	M	15045	12/01/19	NO	YES	NO	NO	NO	NO	YES	NO	NO	YES	NO	NO	NO	22.5	82	ACID PEPTIC DISEASE	35	3	REFLUX ESOPHAGITIS
13	ANISA	45	F	19905	17/01/19	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	YES	H/O TUBECTOMY 20 YEARS BACK	32.5	105	GASTRITIS	38	5	LAX OG JUNCTION
14	AMBIKA	14	F	53098	11/02/19	YES	NO	NO	NO	NAUSEA	NO	NO	NO	NO	NO	NO	NO	NO	20.8	84	GASTRITIS	35	4	REFLUX ESOPHAGITIS
15	VINOD	28	M	53193	11/02/19	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	19.5	82	GASTRITIS	38	4	REFLUX ESOPHAGITIS
16	MANENDRA	33	M	65263	20/02/19	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	22	90	ACID PEPTIC DISEASE	36	3	REFLUX ESOPHAGITIS
17	SUSHILA	30	F	73149	27/02/19	NO	NO	NO	NO	THROAT PAIN	NO	NO	NO	NO	YES	NO	NO	NO	22.2	84	TONSILLITIS	38	5	
18	NINGAPPA	42	M	7030	08/03/19	NO	NO	NO	NO	NAUSEA	NO	NO	NO	NO	NO	NO	NO	NO	23.5	90	ACID PEPTIC DISEASE	40	3	REFLUX ESOPHAGITIS
19	VIJAYALAXMI	29	F	7033	12/03/19	NO	YES	NO	YES	[illegible]	NO	NO	NO	NO	NO	NO	NO	NO	23.4	80	ACID PEPTIC DISEASE	35	4	REFLUX ESOPHAGITIS
20	KAMALABAI	58	F	97248	18/03/19	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	YES	NO	28.5	88	DYSPHAGIA	35	4	REFLUX ESOPHAGITIS WITH SCHATZKI RING
21	BHIMAPPA	50	M	8458	19/03/19	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	H/O SURGERY FOR DU PERFORATION 2YRS BACK	22	84	GASTRITIS	40	3	REFLUX ESOPHAGITIS WITH DUODENAL EROSIONS
22	MURARILAL	43	M	1E+05	25/03/19	NO	NO	NO	NO	SORE THROAT	NO	NO	NO	YES	NO	NO	NO	H/O SURGERY AND RADIOTHERAPHY FOR GLOTTIS CA	26.5	103	SECONDARIES IN ORAL CAVITY	38	4	SEVERE ESOPHAGITIS
23	DATTATREYA	27	M	1E+05	25/03/19	YES	NO	NO	NO	NAUSEA	NO	NO	YES	NO	NO	NO	NO	NO	17.9	166	GASTRITIS	38	3	GRADE 1 ESOPHAGITIS
24	CHANDRASHEKAR	34	M	14020	04/04/19	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	21.3	68	ACID PEPTIC DISEASE	38	4	REFLUX ESOPHAGITIS
25	PARVATI	46	F	1E+05	16/04/19	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	YES	NO	NO	26	52	ACID PEPTIC DISEASE	40	3	GRADE 1 ESOPHAGITIS
26	KAMALABAI	45	F	11588	17/04/19	YES	NO	NO	YES	NO	NO	NO	NO	NO	NO	YES	NO	H/O RENAL CALCULUS	23.4	80	GASTRITIS	40	3	GRADE 1 ESOPHAGITIS
27	SIDDALINGAPPA	45	M	2E+05	01/05/19	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	H/O RIGHT MESH HERNIOPLASTY 2 YRS BACK	20.9	87	ACID PEPTIC DISEASE	35	3	REFLUX ESOPHAGITIS
28	ABDUL	45	M	2E+05	01/05/19	YES	NO	NO	NO	NO	NO	NO	NO	YES	NO	NO	NO	NO	20.3	81.5	GASTRITIS	38	3	REFLUX ESOPHAGITIS
29	SHASHIKANTH	24	M	2E+05	04/05/19	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	22	85	ACID PEPTIC DISEASE	37	3	REFLUX ESOPHAGITIS
30	SIDRAMAPPA	38	M	16700	28/05/19	YES	YES	NO	YES	HEMATEMESIS	NO	NO	NO	NO	NO	NO	NO	NO	22.3	82	UPPER GI BLEED	35	4	REFLUX ESOPHAGITIS WITH EROSIVE GASTRITIS
31	BABU	55	M	2E+05	12/06/19	YES	YES	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	18.8	171	ACID PEPTIC DISEASE	35	3	REFLUX ESOPHAGITIS WITH ANTRAL GASTRITIS
32	DIVYA	21	F	2E+05	17/06/19	NO	NO	NO	YES	NO	NO	NO	NO	YES	NO	NO	NO	NO	20.3	71	ACID PEPTIC DISEASE	38	2	GRADE 2 REFLUX ESOPHAGITIS
33	NAGAPPA	45	M	2E+05	24/06/19	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	H/O EXCISION OF RIGHT VOCAL CORD POLYP	24	92	DYSPHAGIA	38	3	REFLUX ESOPHAGITIS
34	SHRISHAIL	20	M	2E+05	22/06/19	NO	NO	NO	YES	NO	NO	NO	NO	NO	YES	NO	NO	NO	23.4	67	ACID PEPTIC DISEASE	35	4	REFLUX ESOPHAGITIS
35	KASHIBAI	35	F	19829	22/06/19	NO	NO	NO	YES	FEVER	NO	NO	NO	NO	NO	NO	NO	H/O SNAKE BITE WITH CELLULITIS AND K/C/O AKI	20.8	79	ACID PEPTIC DISEASE	37	4	
36	GANAPATI	30	M	2E+05	21/06/19	NO	NO	YES	NO	[illegible]	NO	NO	NO	NO	NO	NO	NO	NO	25.8	96	FOREIGN BODY IMPACTION IN THROAT	35	3	
37	AMBRESH	35	M	19583	20/06/19	NO	NO	NO	YES	HEMATEMESIS	NO	NO	YES	NO	NO	NO	NO	NO	17.2	70	UPPER GI BLEED	35	5	MALLORY WEISS TEAR AT GE JUNCTION WITH REFLUX ESOPHAG
38	SHARANAPPA	34	M	18436	11/06/19	YES	NO	NO	NO	NO	NO	NO	YES	NO	NO	NO	NO	NO	25	154	ACID PEPTIC DISEASE	38	3	REFLUX ESOPHAGITIS WITH GASTRIC EROSIONS
39	CHANDIBAI	50	F	20277	29/06/19	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	YES	NO	NO	32	100	ACID PEPTIC DISEASE	38	4	REFLUX ESOPHAGITIS
40	DATTU	49	M	20919	06/07/19	YES	NO	NO	NO	[illegible]	NO	NO	YES	NO	NO	NO	NO	H/O CHRONIC ALCOHOLIC LIVER DISEASE	15.2	75	GASTRITIS	36	3	REFLUX ESOPHAGITIS
41	MAHANTESH	40	M	21572	09/07/19	YES	NO	YES	NO	NO	YES	NO	YES	YES	YES	NO	NO	NO	15.9	168	ODYNOPHAGIA	35	4	REFLUX ESOPHAGITIS
42	PODU	25	M	21540	09/07/19	NO	NO	NO	YES	[illegible]	NO	YES	YES	NO	NO	NO	NO	NO	20.7	160	UPPER GI BLEED	38	2	REFLUX ESOPHAGITIS WITH ANTRAL GASTRITIS
43	DARYAPPA	25	M	2E+05	09/07/19	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	24.2	83	DYSPHAGIA	35	3	REFLUX ESOPHAGITIS WITH ANTRAL GASTRITIS
44	SHRIDEVI	38	F	2E+05	09/07/19	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	31.8	95	ACID PEPTIC DISEASE	35	4	REFLUX ESOPHAGITIS
45	SHUBHAM	45	M	2E+05	15/07/19	NO	YES	NO	NO	NO	NO	NO	NO	YES	YES	NO	NO	NO	25.2	92	ACID PEPTIC DISEASE	36	4	REFLUX ESOPHAGITIS
46	VEERESH	20	M	23741	23/07/19	YES	NO	NO	NO	FEVER	NO	NO	NO	NO	NO	NO	NO	NO	26.2	87	GASTRITIS	35	3	REFLUX ESOPHAGITIS
47	LINGARAJ	29	M	24150	25/07/19	NO	YES	NO	YES	HEMATEMESIS	NO	NO	NO	YES	YES	NO	NO	NO	18.8	74	ACID PEPTIC DISEASE	38	2	REFLUX ESOPHAGITIS WITH LINEAR EROSIONS AT GE JUNCTION
48	RAVI	18	M	3E+05	27/07/19	YES	NO	NO	NO	NO	NO	NO	NO	YES	YES	NO	NO	NO	16.3	67	ACID PEPTIC DISEASE	35	4	REFLUX ESOPHAGITIS
49	MALASIDDA	35	M	3E+05	30/07/19	NO	YES	NO	YES	NO	NO	NO	NO	YES	YES	YES	NO	NO	22.5	87	ACID PEPTIC DISEASE	35	3	REFLUX ESOPHAGITIS WITH BARRETT'S MUCOSA
50	LAKKAPPA	30	M	25530	03/08/19	YES	NO	NO	NO	NO	NO	YES	NO	YES	YES	NO	NO	NO	22.3	82	ACID PEPTIC DISEASE	33	4	EROSIVE GASTRITIS
51	MALAKAPPA	40	M	3E+05	03/08/19	YES	NO	NO	YES	NO	NO	NO	YES	NO	NO	NO	NO	H/O PANCREATITIS 1 YEAR BACK	22.8	88	CHRONIC PANCREATITIS	36	3	REFLUX ESOPHAGITIS
52	SHRISHAIL	47	M	3E+05	09/08/19	NO	NO	NO	YES	NO	NO	YES	NO	NO	NO	NO	NO	NO	29	99	GASTRITIS	37	3	REFLUX ESOPHAGITIS
53	DEVAMMA	45	F	3E+05	09/08/19	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	26.2	83	ACID PEPTIC DISEASE	35	3	REFLUX ESOPHAGITIS
54	ANASUYA	85	F	30043	10/09/19	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	24.7	98	ACID PEPTIC DISEASE	35	4	REFLUX ESOPHAGITIS
55	UMA	53	F	27711	03/09/19	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	K/C/O STAGE 5 RENAL FAILURE	26.2	92	DYSPHAGIA	33	5	
56	HUSEENBASHA	37	M	4E+05	19/11/19	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	20.3	72	GASTRITIS	38	2	REFLUX ESOPHAGITIS
57	GANGABAI	65	F	39407	26/11/19	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	22.2	82	ACID PEPTIC DISEASE	37	3	REFLUX ESOPHAGITIS
58	PRAHALAD	51	M	4E+05	02/12/19	NO	YES	YES	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	18.2	78	DYSPHAGIA	35	4	
59	SONAVVA	58	F	39827	03/12/19	YES	NO	NO	NO	[illegible]	NO	NO	NO	NO	NO	NO	NO	NO	20	75	SPLENOMEGALY	38	2	REFLUX ESOPHAGITIS
60	AKSHAY	24	M	4E+05	05/12/19	YES	NO	NO	NO	NO	NO	NO	NO	YES	NO	NO	NO	NO	20.3	75	ACID PEPTIC DISEASE	35	2	REFLUX ESOPHAGITIS
61	ANITA	18	F	40845	06/12/19	YES	NO	NO	NO	NO	NO	NO	NO	YES	NO	NO	NO	NO	22.9	77	ACID PEPTIC DISEASE	38	2	REFLUX ESOPHAGITIS
62	BASANAGOUDA	65	M	41667	12/12/19	NO	NO	NO	NO	[illegible]	YES	YES	NO	NO	NO	NO	NO	NO	21.3	76	METASTATIC CERVICAL LYMPH NODE	37	3	
63	KASHAPPA	64	M	42010	01/08/19	NO	NO	YES	YES	LOOSE STOOLS	NO	YES	YES	NO	NO	NO	NO	NO	19.6	81	DYSPHAGIA	35	3	REFLUX ESOPHAGITIS
64	GANGAWWA	30	F	43030	24/12/19	YES	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	20.4	60	ACID PEPTIC DISEASE	38	2	REFLUX ESOPHAGITIS
65	BASAVARAJ	32	M	43800	30/12/19	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	24.5	92	ACID PEPTIC DISEASE	35	4	GRADE 1 ESOPHAGEAL VARICES
66	SIDDAPPA	45	M	2076	18/01/20	NO	NO	NO	YES	HEMATEMESIS	NO	NO	YES	NO	NO	NO	NO	NO	18.7	71	UPPER GI BLEED	36	3	REFLUX ESOPHAGITIS
67	SATYAMMA	30	F	2E+05	27/06/20	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	K/C/O HYPOTHYROIDISM ON TREATMENT	37.9	110	GASTRITIS	37	3	REFLUX ESOPHAGITIS
68	MANJUNATH	85	M	15147	18/06/20	NO	NO	NO	NO	MALENA, FEVER	NO	NO	NO	NO	NO	YES	YES	H/O CVA 2 YEARS BACK	18.4	74	UPPER GI BLEED	38	2	REFLUX ESOPHAGITIS
69	MUTTAPPA	60	M	1E+05	16/06/20	NO	NO	YES	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	17.9	74	DYSPHAGIA	38	2	
70	SHANTAGOUDA	60	M	14842	16/06/20	YES	NO	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	22.8	94	CHOLECYSTITIS	36	4	REFLUX GASTRITIS
71	VEERESH	28	M	1E+05	03/06/20	NO	YES	NO	NO	NO	NO	YES	NO	NO	NO	NO	NO	K/C/O ORAL SUBMUCOUS FIBROSIS SINCE 1 YEAR	22.8	84	ACID PEPTIC DISEASE	38	2	REFLUX ESOPHAGITIS
72	SANDYA	22	F	1E+05	03/06/20	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	22.2	74	ACID PEPTIC DISEASE	36	4	
73	SHANTANU	27	M	13076	18/05/20	NO	YES	NO	NO	NO	YES	NO	NO	YES	YES	NO	NO	NO	22	70	GASTRITIS	37	3	REFLUX ESOPHAGITIS
74	RENUKA	43	F	1E+05	20/03/20	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	18.5	62	GASTRITIS	36	3	REFLUX ESOPHAGITIS
75	RAMESH	20	M	99135	09/03/20	YES	NO	NO	NO	NO	NO	NO	NO	YES	NO	NO	NO	NO	17.8	64	ACID PEPTIC DISEASE	37	2	REFLUX ESOPHAGITIS
76	PADMAJA	36	F	80537	25/02/20	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	22.2	74	ACID PEPTIC DISEASE	38	2	REFLUX ESOPHAGITIS WITH LOWER ESOPHAGEAL MUCOSAL ER
77	SHIVU	23	M	70550	18/02/20	YES	NO	NO	NO	NO	NO	NO	NO	YES	NO	NO	NO	NO	25.8	91	GASTRITIS	36	2	REFLUX ESOPHAGITIS
78	ASHOK	30	M	5396	15/02/20	NO	NO	NO	NO	[illegible]	NO	NO	NO	NO	NO	NO	NO	NO	18.4	74	ANEMIA	37	3	REFLUX ESOPHAGITIS
79	BAGANNA	21	M	65805	14/02/20	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	H/O DUODENAL PERFORATION	17.8	67	ACID PEPTIC DISEASE	38	2	REFLUX ESOPHAGITIS WITH GASTRIC EROSIONS

80	BASAVARAJ	47	M	57248	06/02/20	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	YES	NO	NO	23.4	90	ACID PEPTIC DISEASE	37	3	
81	SUREKHA	32	F	4504	07/02/20	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	K/C/O POLIOMYELITIS, H/O HEMARROIDS 3 YEARS BACK	22.4	82	GASTRITIS	36	3	REFLUX ESOPHAGITIS
82	SHRISHAIL	24	M	51607	05/02/20	NO	YES	NO	YES	[illegible]	NO	YES	NO	NO	NO	NO	NO	NO	23.5	83	UPPER GI BLEED	35	2	REFLUX ESOPHAGITIS
83	MALLIKARJUN	22	M	44996	31/01/20	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	20.4	68	ACID PEPTIC DISEASE	38	2	REFLUX ESOPHAGITIS
84	MEHEBOOB	28	M	40490	28/01/20	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	23.6	81	GASTRITIS	38	2	REFLUX ESOPHAGITIS
85	GIRIJA	40	F	24530	18/01/20	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	35.1	114	DYSPHAGIA	35	3	REFLUX ESOPHAGITIS
86	GURUSIDDAYYA	56	M	2176	21/01/20	NO	NO	NO	NO	[illegible]	NO	NO	YES	NO	NO	NO	NO	K/C/O CHRONIC LIVER DISEASE WITH PORTAL HYPERTENSION AND HEPATITIS	22.2	91	PORTAL HYPERTENSION	38	2	GRADE 2 ESOPHAGEAL VARICES WITH GASTRITIS
87	MD SHAFEEQ	40	M	15901	29/06/20	NO	NO	NO	YES	NO	NO	NO	YES	NO	NO	NO	NO	K/C/O ALCOHOLIC LIVER DISEASE	26.9	97	GASTRITIS	37	3	REFLUX ESOPHAGITIS WITH GASTRIC EROSIONS WITH PAN GAS
88	NITESH	21	M	2E+05	02/07/20	NO	NO	NO	YES	HEMATEMESIS	NO	YES	YES	NO	NO	NO	NO	NO	21	80	UPPER GI BLEED	37	3	REFLUX ESOPHAGITIS
89	SUSALABAI	20	F	1E+05	01/05/20	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	20	93	FOREIGN BODY IMPACTION IN THROAT	35	4	REFLUX ESOPHAGITIS
90	RAVI	29	M	1E+05	04/05/20	NO	NO	YES	NO	NO	YES	NO	NO	YES	NO	NO	NO	NO	19.8	83	DYSPHAGIA	38	2	
91	BASAPPA	64	M	1E+05	05/05/20	YES	YES	NO	NO	NO	NO	NO	YES	NO	NO	NO	YES	NO	22	98	ACID PEPTIC DISEASE	36	3	REFLUX ESOPHAGITIS
92	ANITA	21	F	1E+05	06/05/20	YES	NO	NO	NO	NO	NO	NO	NO	NO	YES	NO	NO	NO	21	88	GASTRITIS	37	3	PAN GASTRITIS
93	HUSSENBASHA	38	M	1E+05	07/05/20	YES	NO	NO	YES	NO	YES	YES	NO	NO	NO	NO	NO	NO	20.4	101	ACID PEPTIC DISEASE	37	3	REFLUX ESOPHAGITIS
94	ASHOK	58	M	12574	09/05/20	YES	NO	NO	YES	HEMATEMESIS	YES	NO	YES	NO	NO	YES	NO	NO	21.2	90	UPPER GI BLEED	36	2	GRADE 1 ESOPHAGEAL VARICES
95	MOHAN	76	M	12663	09/05/20	NO	YES	NO	NO	NO	YES	YES	NO	NO	NO	NO	YES	NO	18.2	78	ACID PEPTIC DISEASE	37	3	REFLUX ESOPHAGITIS
96	SAVITA	21	F	1E+05	09/05/20	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	20.8	94	GASTRITIS	35	2	PAN GASTRITIS
97	BHIMU	55	M	1E+05	12/05/20	NO	NO	NO	YES	HEMATEMESIS	YES	NO	YES	NO	NO	NO	NO	NO	22.4	97	UPPER GI BLEED	38	2	
98	HARINI	21	F	1E+05	14/05/20	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	17.9	81	GASTRITIS	36	2	REFLUX ESOPHAGITIS
99	PARASAPPA	70	M	1E+05	14/05/20	NO	NO	YES	NO	NO	YES	YES	NO	YES	YES	YES	NO	NO	22.2	108	DYSPHAGIA	37	3	EROSIVE GASTRITIS
100	DANAMMA	63	F	1E+05	18/05/20	YES	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	17.8	81	GASTRITIS	36	2	REFLUX ESOPHAGITIS
101	MALLIKARJUN	35	M	12996	18/05/20	YES	NO	NO	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	21.4	93	ACID PEPTIC DISEASE	37	3	ANTRAL GASTRITIS
102	HANAMANTH	32	M	1E+05	19/05/20	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	19.3	89	ACID PEPTIC DISEASE	37	2	REFLUX ESOPHAGITIS
103	JAYASHREE	49	F	1E+05	20/05/20	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	YES	NO	NO	23	98	DYSPHAGIA	36	2	REFLUX ESOPHAGITIS
104	MANSUR KHAN	29	M	1E+05	22/05/20	YES	NO	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	19.9	84	ACID PEPTIC DISEASE	37	2	ANTRAL GASTRITIS
105	MAHADEVI	35	F	13271	21/05/20	YES	YES	NO	NO	NO	NO	NO	NO	YES	NO	NO	YES	NO	21.3	95	GASTRITIS	36	3	REFLUX ESOPHAGITIS
106	HUSSENKHAN	65	M	1E+05	21/05/20	NO	NO	YES	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	19.8	84	DYSPHAGIA	37	2	REFLUX ESOPHAGITIS
107	RIYAZ	50	M	1E+05	26/05/20	NO	NO	YES	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	20.8	81	DYSPHAGIA	38	2	
108	JANABAI	38	F	1E+05	27/05/20	YES	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	17.5	80	GASTRITIS	35	2	ANTRAL GASTRITIS
109	GURULINGAPPA	45	M	45328	28/05/20	YES	NO	NO	NO	NO	NO	NO	YES	NO	NO	NO	YES	K/C/O ALCOHOLIC LIVER DISEASE	23.7	104	ACID PEPTIC DISEASE	37	2	ESOPHAGEAL VARICES WITH DUODENAL EROSIONS
110	SHASHIKALA	42	F	1E+05	28/05/20	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	18.3	90	GASTRITIS	38	2	REFLUX ESOPHAGITIS
111	RAVINDRA	57	M	1E+05	28/05/20	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	20	87	DYSPHAGIA	37	2	REFLUX ESOPHAGITIS WITH ANTRAL GASTRITIS
112	APPASAB	42	M	1E+05	29/05/20	YES	NO	NO	YES	NO	YES	NO	NO	NO	NO	NO	NO	NO	19.7	93	GASTRITIS	37	2	REFLUX ESOPHAGITIS
113	SURESH	45	M	17235	21/07/20	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	21.4	98	DYSPHAGIA	38	2	REFLUX ESOPHAGITIS
114	SANGAWWA	24	F	2E+05	25/07/20	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	18.2	79	ACID PEPTIC DISEASE	36	2	REFLUX ESOPHAGITIS
115	SHARANAPPA	34	M	2E+05	29/07/20	YES	YES	NO	NO	NO	YES	YES	NO	NO	NO	NO	NO	NO	22.2	96	ACID PEPTIC DISEASE	37	2	REFLUX ESOPHAGITIS