

**THE SPECTRUM OF HISTOPATHOLOGICAL CHANGES IN  
ENDOSCOPIC GASTRODUODENAL BIOPSY IN PATIENTS  
WITH FUNCTIONAL DYSPEPSIA**

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**IN**

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**THE SPECTRUM OF HISTOPATHOLOGICAL  
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BIOPSY IN PATIENTS WITH FUNCTIONAL  
DYSPEPSIA**



**MASTER OF SURGERY  
In GENERAL SURGERY**

**LIST OF ABBREVIATIONS**

<b>ABBREVIATIONS</b>	<b>FULL FORMS</b>
FD	Functional dyspepsia
GERD	Gastro esophageal reflux disease
DM	Diabetes Mellitus
UGI	Upper Gastro intestinal
EGD	Esophagogastroduodenoscopy
H. Pylori	Helicobacter pylori
ACG	American College of Gastroenterology
PPI	Proton Pump Inhibitor
RCT	Randomized Control Trails
PDS	Post-Prandial Distress Syndrome
EPS	Epigastric Pain Syndrome
NSAID	Non-steroidal anti-inflammatory drug
TCA	Tri Cyclic antidepressants
Sl. No	Serial number
Fig	Figure

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## **INTRODUCTION**

The term dyspepsia is used for a spectrum of symptoms localized by the patient to the epigastric region (between the navel and the xiphoid process) and the flanks<sup>(1)</sup>.

The Greek terms “dys” and “pepsin,” which translate to “poor” and “digestive system,” respectively, are the source of the phrase dyspepsia <sup>(1)</sup>. Since ancient times, people have complained about stomach pain and discomfort. Since its initial use in the middle of the 18th century, it has become very popular. Along with hypochondria and hysteria, dyspepsia was considered one of the “nervous illnesses” in the 18th century <sup>(2)</sup>.

It has been used somewhat loosely, and symptoms have included belching, early satiety, abdominal bloating, nausea with or without vomiting, and unexplained upper abdomen or periumbilical discomfort or pain.

In surgical practice, dyspepsia is a common symptom that affects approximately a fourth of the population in industrialized countries. The prevalence of dyspepsia is high and consumes considerable medical and economic resources.

According to several global population studies, the prevalence of dyspepsia is approximately 25% (range 10-40%) for a 3–12month period.

The evaluation and management of dyspepsia constitute a significant clinical and economic burden<sup>(3)</sup>.



There are many criteria and updates on defining functional dyspepsia. The Rome Committee is a multinational group of experts, first convened in 1990 and meets regularly to revise the diagnostic criteria for all functional disorders. The Rome IV criteria being the latest one was proposed in 2016.

**AIM AND OBJECTIVE OF THE STUDY:**

Evaluation of the histopathological changes of endoscopic gastroduodenal biopsies in patients with Functional dyspepsia.

## **NEED FOR STUDY:**

Most dyspeptic patients have FD, as organic causes are uncommon. A study from India reported a prevalence of dyspepsia to be 30.4%. However, none of these investigations used the Rome criteria or other generally accepted diagnostic criteria for FD<sup>(3)</sup>.

Upper GI endoscopy has a vital role in the evaluation of dyspepsia. In FD, the mucosa of the upper GI tract appears normal, but the patients continue to manifest with upper GI symptoms. Identifying the definite explainable etiology is still a grey area. There can be a possibility that subtle changes in the mucosa at a microscopic level which can be missed on gross examination, could explain the symptomatology of the dyspepsia.

Thus, we are taking up this study of FD to evaluate the possible histopathological changes at the upper GI mucosa that can be missed on gross examination by subjecting the patients to multiple biopsies from the stomach and the initial part of the duodenum.

## **REVIEW OF LITERATURE**

### **EPIDEMIOLOGY**

According to several global population studies, the prevalence of dyspepsia is approximately 25% (range 10-40%) for a 3–12month period <sup>(4)</sup>. However, the prevalence estimate will only be about 3–15% if heartburn or regurgitation are not included <sup>(5)</sup>. According to longitudinal research, symptoms improve over time in less than 50% of patients <sup>(6)</sup>.

Compared to males, women are more likely than men to have dyspepsia. With age, this difference becomes smaller. About 5% of the population will get new onset dyspepsia yearly <sup>(7)</sup>.

A study from India reported a prevalence of dyspepsia to be 30.4%. In another multi-centric study from India, the frequency of dyspeptic symptoms was as high as 49% in the community. Therefore, it may be inferred from the scant available information that 7.6 to 49% of the Indian population report dyspeptic symptoms <sup>(3)</sup>. However, none of these investigations used the Rome criteria or other generally accepted diagnostic criteria for FD.

Depending on the symptoms, the prevalence <sup>(8)</sup> is

- Reflux symptoms 25%
- Dyspepsia without reflux symptoms 15%
- Irritable bowel symptoms 15%
- GERD 10%

Dyspepsia-related illnesses is divided into two categories: organic and functional.

**1. Organic dyspepsia:**

Organic dyspepsia means that there is a clear anatomic or pathophysiologic reason for the dyspeptic complaints, such as an ulcer disease or mass <sup>(2)</sup>. Peptic ulcer disease, gastroesophageal reflux disease, biliary tract disease, and stomach cancer are the major organic causes of dyspepsia <sup>(9)</sup>.

**2. Functional dyspepsia:**

Functional dyspepsia is the presence of symptoms thought to originate in the gastroduodenal region in the absence of any organic, systemic, or metabolic disease that is likely to explain the symptoms <sup>(10)</sup>.

Overall, most dyspepsia patients do not have a well-defined underlying illness process.

Patients with either new or potentially recurrent dyspeptic symptoms in whom no previous investigations have been made are said to have uninvestigated dyspepsia. Dyspeptic problems may therefore be considered as investigated dyspepsia and separated into organic dyspepsia and FD <sup>(2)</sup>.

Alarming features, often known as alert signs, red flags, or warning signs, are linked to dyspepsia. These unique characteristics are believed to be associated with serious gastrointestinal disorders such as underlying cancer or significant pathologies like stricture or ulcers.

The warning signs when serious pathology should be taken into consideration include

- any persistent dyspepsia in patients over the age of 55
- weight loss
- gastrointestinal bleeding
- persistent vomiting
- dysphagia
- epigastric mass
- jaundice
- gastric surgery
- history of gastric ulcer
- NSAID's usage
- iron deficiency anemia
- suspicious barium meal
- hospitalization for epigastric pain

## **CAUSES OF ORGANIC DYSPEPSIA**

### **1) LUMINAL GI TRACT**

• Peptic ulcer disease
• Gastroesophageal disease
• Gastric or esophageal neoplasia
• Gastroparesis (eg. DM, post-vagotomy, scleroderma, chronic intestinal Pseudo-obstruction, post-viral, idiopathic)
• Infiltrative and inflammatory gastric disorders (eg. Crohn's disease, eosinophilic gastroenteritis, sarcoidosis, amyloidosis)
• Gastric infections (cytomegalovirus, fungus, TB, syphilis)
• Parasites (Giardia lamblia, Strongyloides stercoralis)
• Chronic gastric volvulus
• Chronic gastric or intestinal ischemia
• Food intolerance
• Irritable bowel syndrome

### **2) MEDICATIONS**

• Ethanol
• Gemfibrozil
• Estrogens
• Glucocorticoids

<ul style="list-style-type: none"><li>• Colchicine</li></ul>
<ul style="list-style-type: none"><li>• Iron</li></ul>
<ul style="list-style-type: none"><li>• Aspirin (other NSAIDs, including COX-2 selective agents)</li></ul>
<ul style="list-style-type: none"><li>• Digitalis preparations</li></ul>
<ul style="list-style-type: none"><li>• Levodopa</li></ul>
<ul style="list-style-type: none"><li>• Narcotic</li></ul>
<ul style="list-style-type: none"><li>• Niacin</li></ul>
<ul style="list-style-type: none"><li>• Nitrates</li></ul>
<ul style="list-style-type: none"><li>• Orlistatin</li></ul>
<ul style="list-style-type: none"><li>• Potassium chloride</li></ul>
<ul style="list-style-type: none"><li>• Quinidine</li></ul>
<ul style="list-style-type: none"><li>• Sildenafil</li></ul>
<ul style="list-style-type: none"><li>• Theophylline</li></ul>

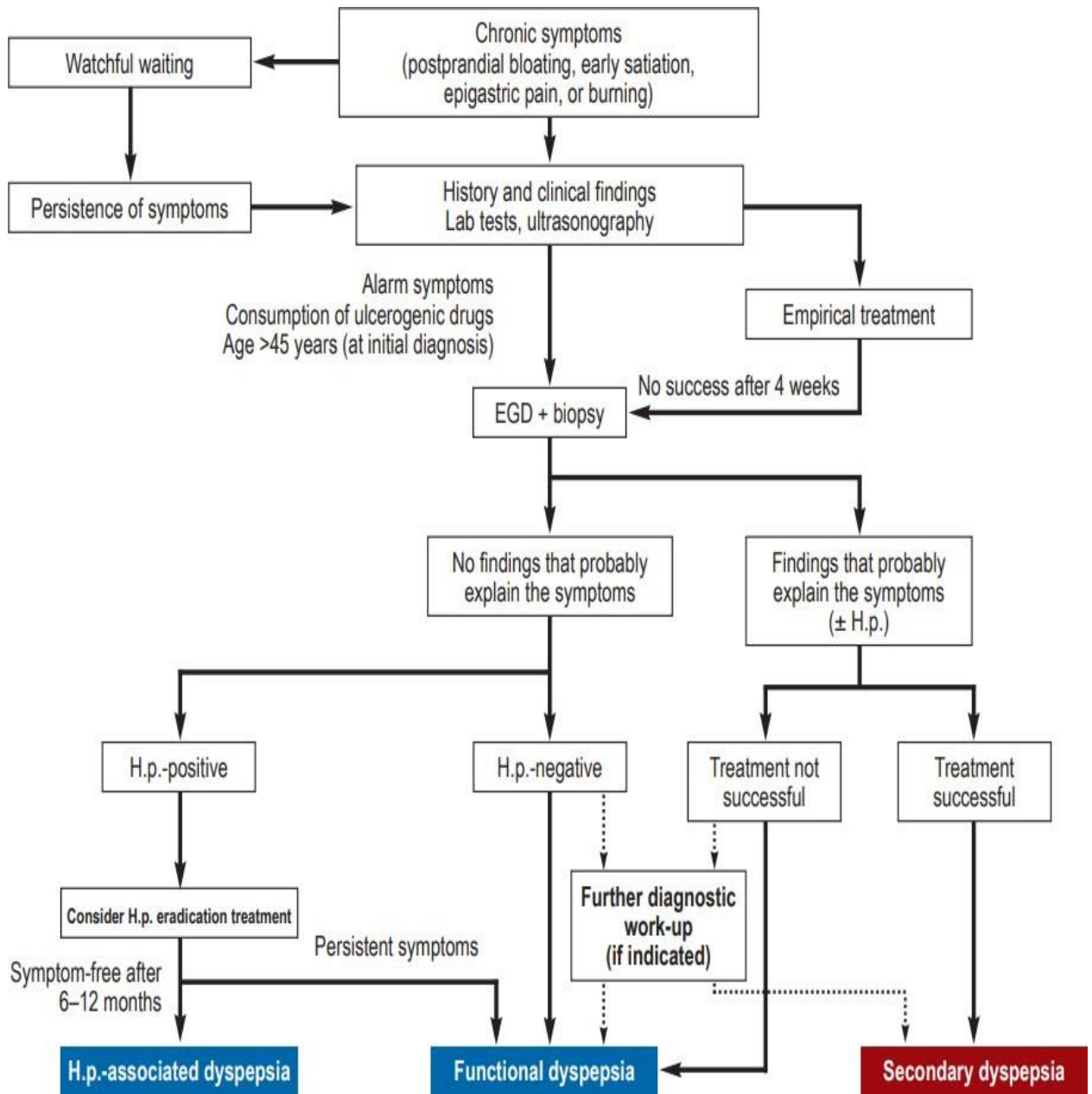
### **3) PANCREATIC AND BILIARY DISORDERS**

<ul style="list-style-type: none"><li>• Biliary pain (cholelithiasis, choledocholithiasis, sphincter of Oddi dysfunction)</li></ul>
<ul style="list-style-type: none"><li>• Chronic pancreatitis</li></ul>
<ul style="list-style-type: none"><li>• Pancreatic neoplasms</li></ul>

#### 4) SYSTEMIC DISORDERS

• Myocardial ischemia
• Congestive cardiac failure
• Diabetes mellitus
• Thyroid disease
• Hyperparathyroidism
• Intra-abdominal malignancy
• Pregnancy
• Renal insufficiency





**Fig 1: Diagnostic procedure in patients with dyspeptic symptoms**

## **FUNCTIONAL DYSPEPSIA**

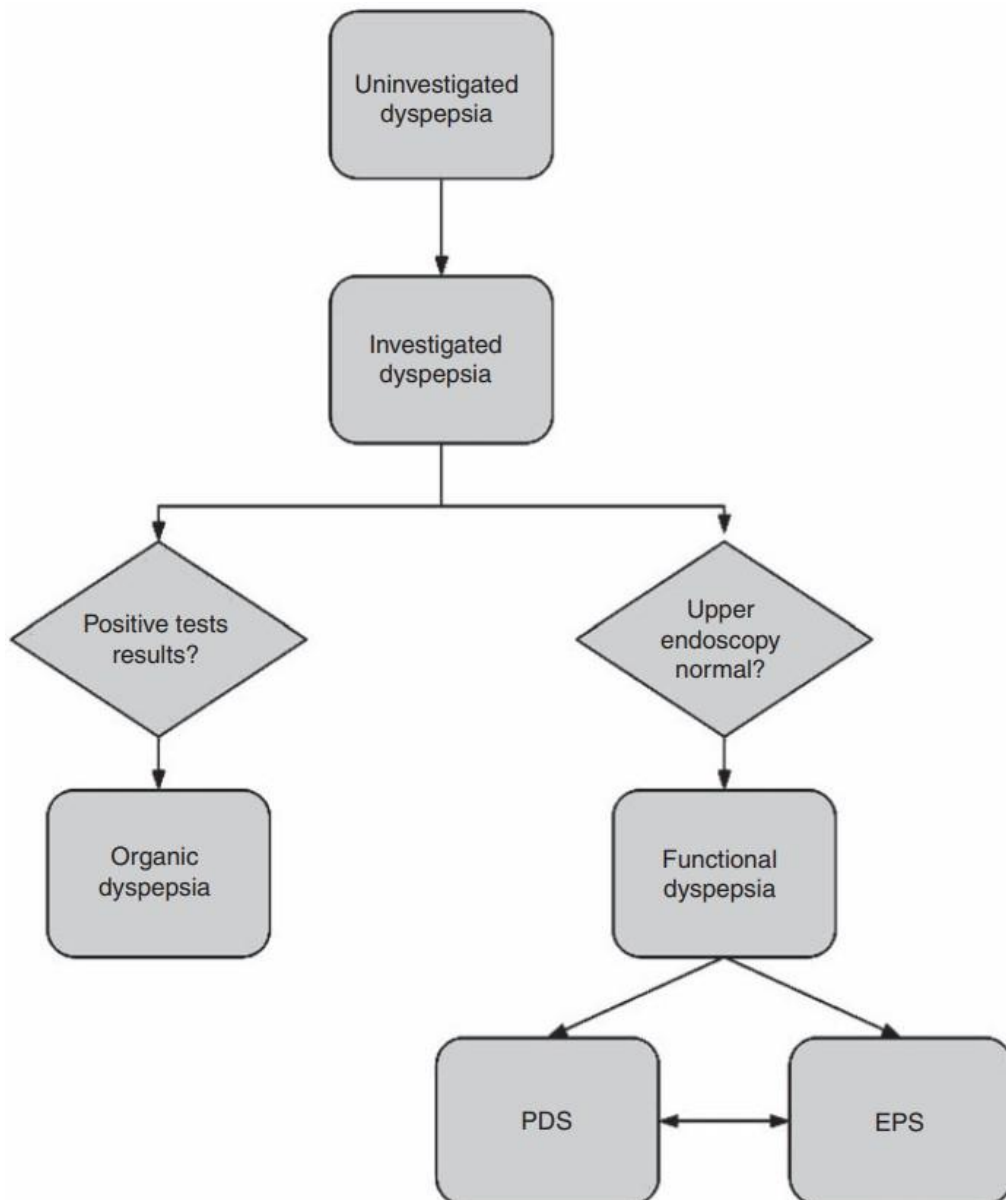
Functional dyspepsia is when structural or biochemical abnormalities cannot explain the chronic or recurrent gastrointestinal symptoms. From the oropharynx to the large intestine, including the biliary tract, malfunction may be the cause. This syndrome is diverse. There are different subtypes of FD, which could indicate an underlying pathology. It contains.

- 1) **Ulcer-like dyspepsia:** they typically have peptic ulcer symptoms
- 2) **Dysmotility-dyspepsia:** symptoms including nausea, fullness, and regurgitation that point to gastric stasis or intestinal dysmotility
- 3) **Reflux-like dyspepsia:** symptoms include heartburn or regurgitation along with epigastric pain

In epidemiological studies, H pylori have not been found to be significantly linked to non-ulcer dyspepsia. There is no clear or strong evidence linking FD to other causes, including increased acid secretion, stress, nutritional, or psychological issues.

There are many criteria and updates on defining functional dyspepsia. The Rome Committee is a multinational group of experts, first convened in 1990 and meets regularly to revise the diagnostic criteria for all functional disorders. The Rome criteria were amended as the Rome IV criteria, launched at the site of Digestive Disease Week (DDW2016) in San Diego, California, USA, on May 21-

25, 2016. Rome IV criteria, as opposed to Rome III criteria, mainly improve the specificity of the definition and significantly reduces the overlap between PDS and EPS groups. In order to stratify FD patients, this categorization is, therefore, probably more effective in practical practice <sup>(11)</sup>.



**Fig 2: Dyspepsia nomenclature. PDS, postprandial distress syndrome; EPS, epigastric pain syndrome.**

**ROME IV CRITERIA** <sup>(11)</sup> -

**A1. FUNCTIONAL DYSPEPSIA\***

*Diagnostic criteria\*\**

1. *One or more* of the following:
  1. Bothersome postprandial fullness
  2. Bothersome early satiation
  3. Bothersome epigastric pain
  4. Bothersome epigastric burning

AND

2. No evidence of structural disease (including at upper endoscopy) that is likely to explain the symptoms

\*Must fulfil criteria for A1a. PDS and/or A1b. EPS

\*\*Criteria fulfilled for the last 3 months with symptom onset at least 6 months prior to diagnosis

**A1a. Postprandial Distress Syndrome (PDS)**

*Diagnostic criteria\**

*Must include **one or both** of the following at least 3 days a week:*

1. Bothersome postprandial fullness (i.e., severe enough to impact on usual activities)

2. Bothersome early satiation (i.e., severe enough to prevent finishing a regular size meal)

No evidence of organic, systemic, or metabolic disease that is likely to explain the symptoms on routine investigations (including at upper endoscopy)

\*Criteria fulfilled for the last 3 months with symptom onset at least 6 months prior to diagnosis

*Supportive criteria*

1. Postprandial epigastric pain or burning, epigastric bloating, excessive belching, and nausea can also be present
2. Vomiting warrants consideration of another disorder
3. Heartburn is not a dyspeptic symptom but may often co-exist
4. Symptoms that are relieved by evacuation of feces or gas should generally not be considered as part of dyspepsia
5. Other individual digestive symptoms or groups of symptoms (e.g., from GERD and IBS) may co-exist with PDS

**A1b. Epigastric Pain Syndrome (EPS)**

*Diagnostic criteria\**

*Must include **one or both** of the following symptoms at least 1 day a week:*

1. Bothersome epigastric pain (i.e., severe enough to impact on usual activities)

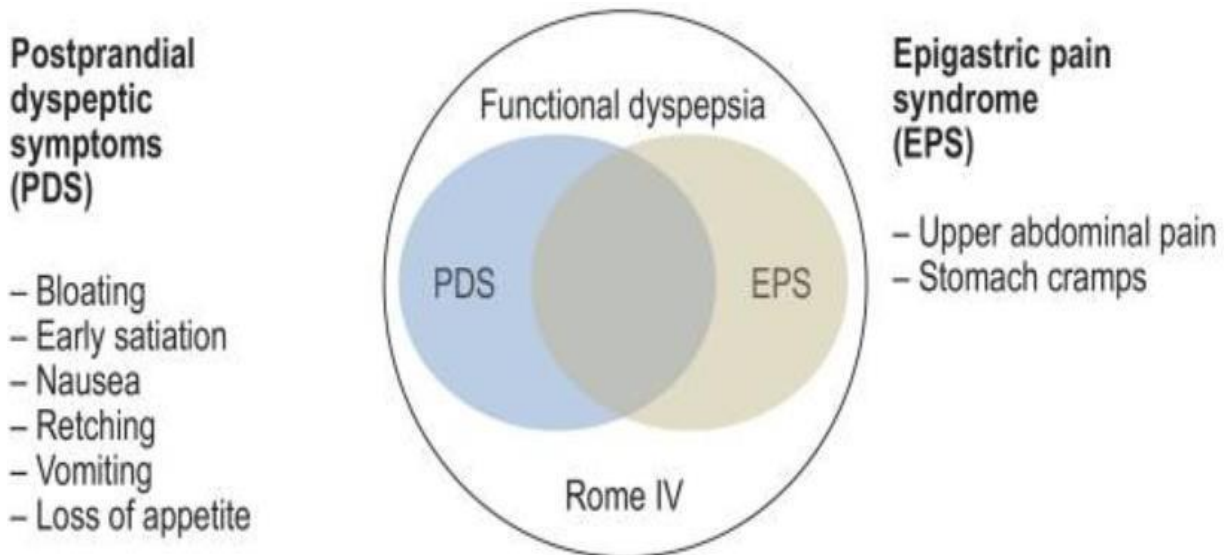
2. Bothersome epigastric burning (i.e., severe enough to impact on usual activities)

No evidence of organic, systemic, or metabolic disease that is likely to explain the symptoms on routine investigations (including at upper endoscopy).

\*Criteria fulfilled for the last 3 months with symptom onset at least 6 months prior to diagnosis

*Supportive criteria*

1. Pain may be induced by ingestion of a meal, relieved by ingestion of a meal, or may occur while fasting
2. Postprandial epigastric bloating, belching, and nausea can also be present
3. Persistent vomiting likely suggests another disorder
4. Heartburn is not a dyspeptic symptom but may often co-exist
5. The pain does not fulfil biliary pain criteria
6. Symptoms that are relieved by evacuation of faeces or gas generally should not be considered as part of dyspepsia
7. Other digestive symptoms (such as from GERD and IBS) may co-exist with EP



**Fig 3: Definition of FD according to Rome IV criteria**

### **Symptom Definition**<sup>(2)</sup>

#### **Epigastric pain:**

Epigastric refers to the region between the umbilicus and lower end of the sternum and is marked by the midclavicular lines. Pain refers to a subjective, unpleasant sensation; some patients may feel that tissue damage is occurring.

#### **Epigastric burning:**

Epigastric refers to the region between the umbilicus and the lower end of the sternum and is marked by the midclavicular lines. Burning refers to an unpleasant subjective sensation of heat.

#### **Postprandial fullness:**

An unpleasant sensation like the prolonged persistence of food in the stomach.

### **Early satiation:**

A feeling that the stomach is overfilled soon after starting to eat, out of proportion to the size of the meal being consumed so that the meal cannot be finished. Previously, the term ‘early satiety’ was used, but satiation is the correct term for the disappearance of the sensation of appetite during food ingestion.

### **PREVALENCE**

It is difficult to determine the true prevalence of FD in population studies. Various studies have been done to provide an accurate picture of the prevalence of FD. The prevalence of FD varies considerably between different populations.

This variation may be due to three factors:

- (1) actual variations in the condition’s frequency;
- (2) diagnostic criteria utilized and
- (3) level of care used to rule out organic causes.

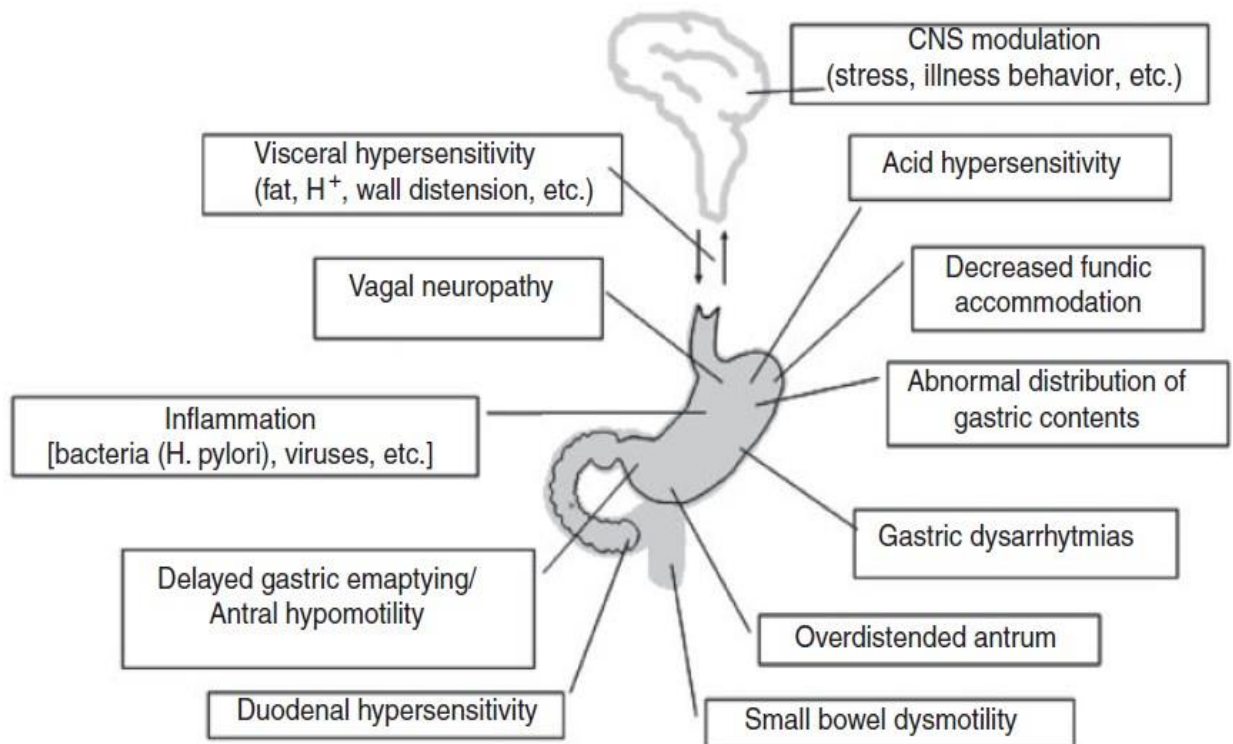
With the definition of “upper abdominal pain,” Singapore has the lowest frequency, at 7%–8%, among the South East Asian countries. The rates among Scandinavians (14.5%) are slightly higher. In the US, prevalence rates range from 23 to 25.8%; the greatest rates are found in New Zealand (34.2%) and India (30.4% of the population) <sup>(12)</sup>.



Thus, the overall prevalence of FD is roughly 16%, though this number may change depending on the nation and the criteria employed to establish its presence (13).

There are not many studies that use the Rome IV criteria. However, even when solely using the Rome III criteria, the prevalence of FDs varies greatly. According to the Rome IV criteria, three-nation research in the general populations of Canada, the USA, and the UK found a prevalence of 10%; however, this ranged from 8% in the UK and Canada to 12% in the USA (13).

## **PATHOPHYSIOLOGY**



**Fig 4: Pathophysiologic mechanisms in FD. H<sup>+</sup>, acid exposure.**

## **1. Delayed gastric emptying**

It occurs when the duodenal resistance increases or the antral peristalsis is impaired. To evaluate the effectiveness of gastric neuromuscular action, the measurement of gastric emptying during a meal is used. Typically, it lies in the 20–50% range. There are few patients who have delayed gastric emptying for solids. It is more common in women than men, according to studies. However, numerous investigations have found no association between dyspepsia and delayed gastric emptying.

Water consumption after meals reduces antral gastric motility and elevates cholecystokinin levels. This elevation is due to the flow of fatty chyme into the duodenum, which inhibits antral peristalsis, called a ‘duodenal break’. The rapid initial inflow into the duodenum caused by low-viscosity meals inhibits antral peristalsis, delaying gastric emptying more than high-viscosity meals. Therefore, especially in postprandial distress syndrome, these may be more critical factors in generating symptoms.

## **2. Hypersensitivity to gastric distension**

Gastric distension might cause hypersensitivity in some FD patients.

- chemoreceptor or mechanoreceptors sensitization
- increased excitability of spinal cord neurons
- dysfunction of spinal inhibitory symptoms

- altered CNS modulation and processing of visceral stimuli

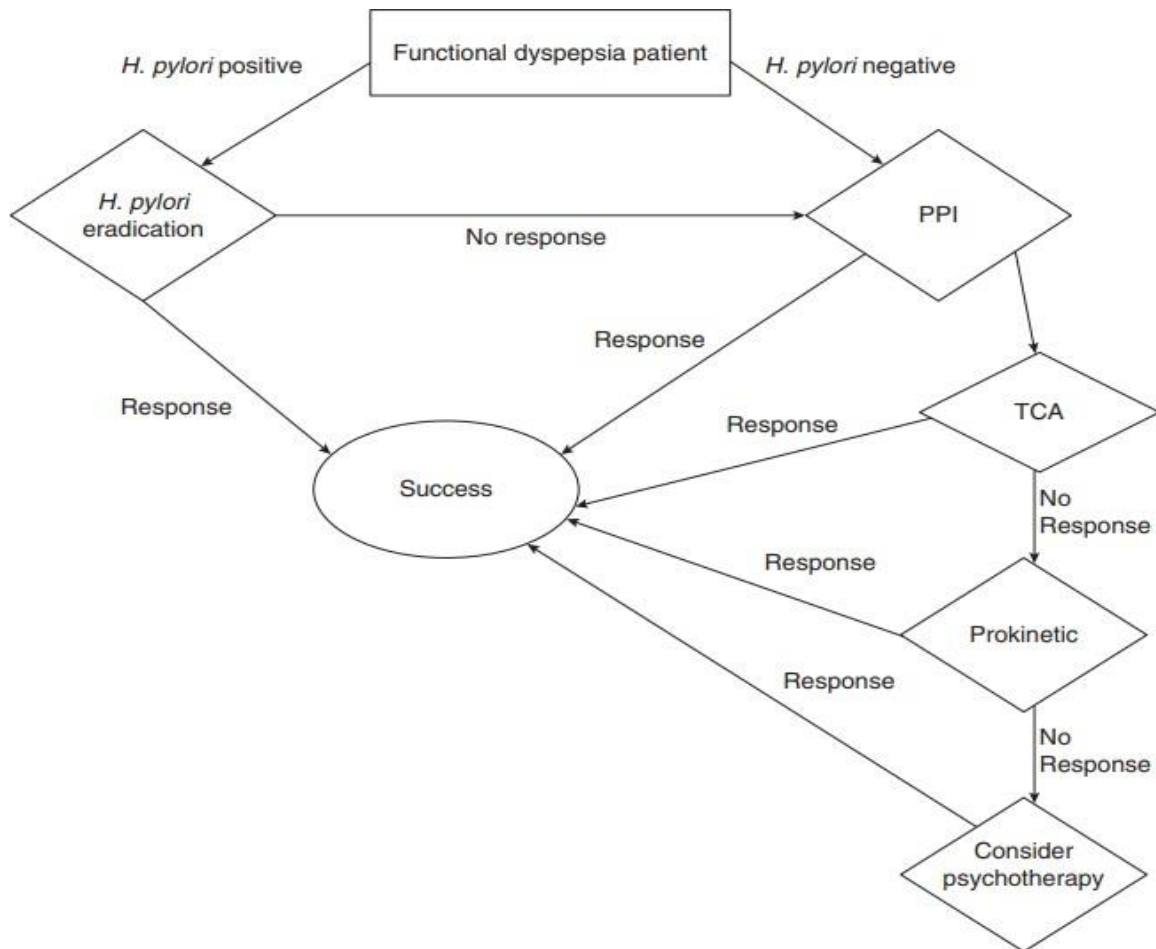
### **3. Altered duodenal sensitivity to lipids**

The release of cholecystokinin by nutrient lipids heightens the sensation of stomach distension. compared to healthy individuals, patients with FD showed higher levels of duodenal acid exposure during duodenal pH monitoring. Acid clearance is impaired, which causes this rise.

### **4. Impaired gastric accommodation**

Following a meal, the proximal stomach serves as a food reservoir, while the distal stomach breaks down food into small enough pieces to pass into the pylorus. The proximal stomach cannot accommodate the huge volume of food if vagal reflux does not allow for enough relaxation of the stomach. Around 40% of patients with FD exhibit reduced stomach accommodation, according to numerous research.

## TREATMENT (14)



**Fig 5: Algorithm for the management of FD**

### 1. DIETARY MODIFICATION

A low-risk solution for patients is to urge dietary changes to reduce items related to FD. Foods high in fat, wheat, FODMAPs (fermentable oligosaccharides, disaccharides, monosaccharides, and polyols), and naturally occurring food chemicals such as caffeine were associated with symptoms of FD. It's unclear whether drinking alcohol is associated with FD symptoms, and it could vary depending on the type of alcohol.

## **2. H. PYLORI ERADICATION**

H. pylori test and treatment methodologies, when compared with endoscopy, are safer, more effective and less expensive for managing patients with dyspepsia. A urea breath test and an H. pylori stool antigen test are two non-invasive H. pylori tests. In a meta-analysis of 23 studies, H. pylori eradication therapy showed improvement at one year but no difference at six months in the symptoms of FD. FD symptoms were reduced by 50% in an RCT that compared the removal of H. pylori with a placebo (number required to treat [NNT] = 8). Another recommendation was to eradicate H. pylori infection if it was the root cause of dyspepsia in some patients. Patients should begin taking a proton pump inhibitor (PPI) if their H. pylori test is negative or if they still experience symptoms following treatment.

## **3. ACID SUPPRESSION**

The two main kinds of drugs used to reduce acid secretion: PPIs and histamine H<sub>2</sub> receptor antagonists.

PPIs should be provided for eight weeks at the standard dosage of one pill per day. Doubling the dosage doesn't have any benefits.

H<sub>2</sub> antagonists are another option for treating FD. H<sub>2</sub> antagonists demonstrated a relative risk reduction of 23% in dyspepsia symptoms in twelve RCTs that compared them with placebo (NNT = 7).

PPIs were superior in four of the seven RCT studies, which led the ACG to endorse PPIs as first-line therapy. FD cannot be treated with antacids or sucralfate (Carafate). Despite a slight advantage, bismuth is not advised due to the possibility of long-term harm.

#### **4. PSYCHOTROPIC MEDICATIONS**

For FD that is not improving with treatment for *H. pylori* or PPI use, guidelines advise trying TCAs such as 25 mg of amitriptyline per day and 50 mg of imipramine per day before utilizing prokinetics. Buspirone (Buspar), according to small placebo-controlled research, lessens bloating and fullness.

#### **5. PROKINETICS**

Prokinetic medications help ease symptoms in people with FD with postprandial distress syndrome, which is defined by symptoms that are associated with meals (predominantly cisapride). The ACG's recommendations for treatment include metoclopramide.

#### **6. NONPHARMACOLOGIC THERAPY**

Psychological therapy was statistically significantly beneficial for patients with FD (i.e., cognitive behavioral therapy and other forms of psychotherapy). Due to the inadequate quality of the data, the high risk of bias brought on by the absence of blinded psychological interventions, and subjective symptom scoring, psychotherapy should not be used as a first-line treatment for

FD. If medication therapy has failed, it is reasonable to consider psychological therapy.

## **7. COMPLEMENTARY AND ALTERNATIVE MEDICINE**

Although the ACG states that individuals who are motivated can consider these alternatives, it does not suggest their habitual usage for the treatment of FD. The herbal remedy Iberogast, which contains extracts of bitter candytuft, Matricaria flower, peppermint leaves, caraway, licorice root, and lemon balm, was statistically better but only slightly more effective clinically.

## **ANATOMY**

### **STOMACH**

The most dilatable and widest part of the alimentary tract is the stomach.

It is 25 cm in length. The stomach is divided into

- a) cardiac part
- b) pyloric part

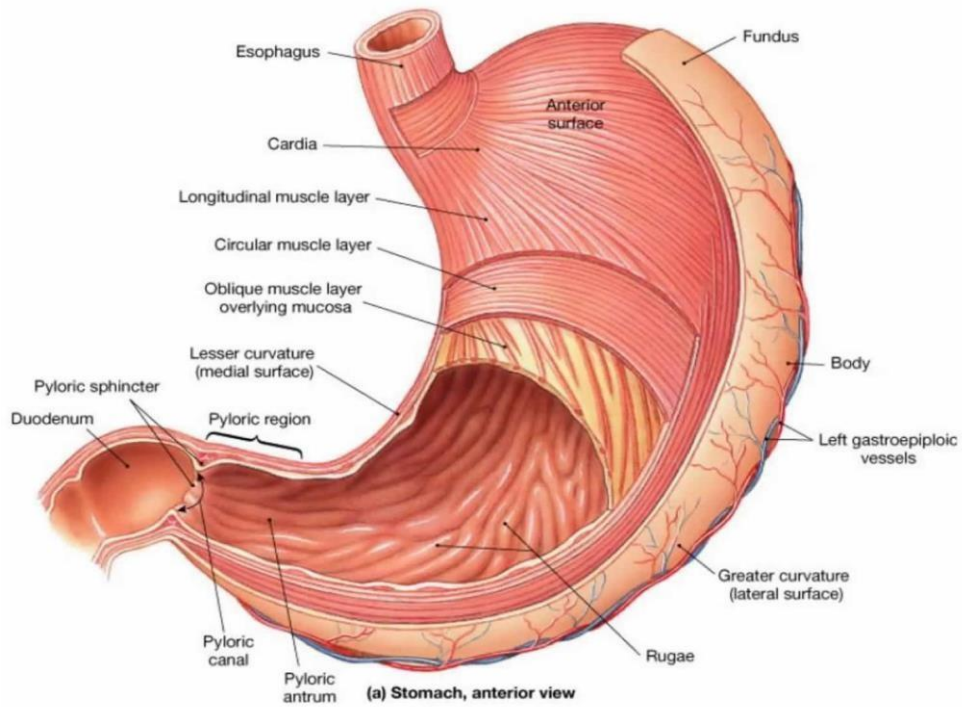
The cardiac part is further subdivided into

- a) fundus
- b) body

Pylorus is subdivided into

- a) antrum
- b) canal

The fundus of the stomach is the area that is located above its cardiac end. Along the lesser curvature, the stomach's body extends from the fundus to the incisura angularis. The pylorus is located below the body of the stomach, which is separated into the antrum and canal.

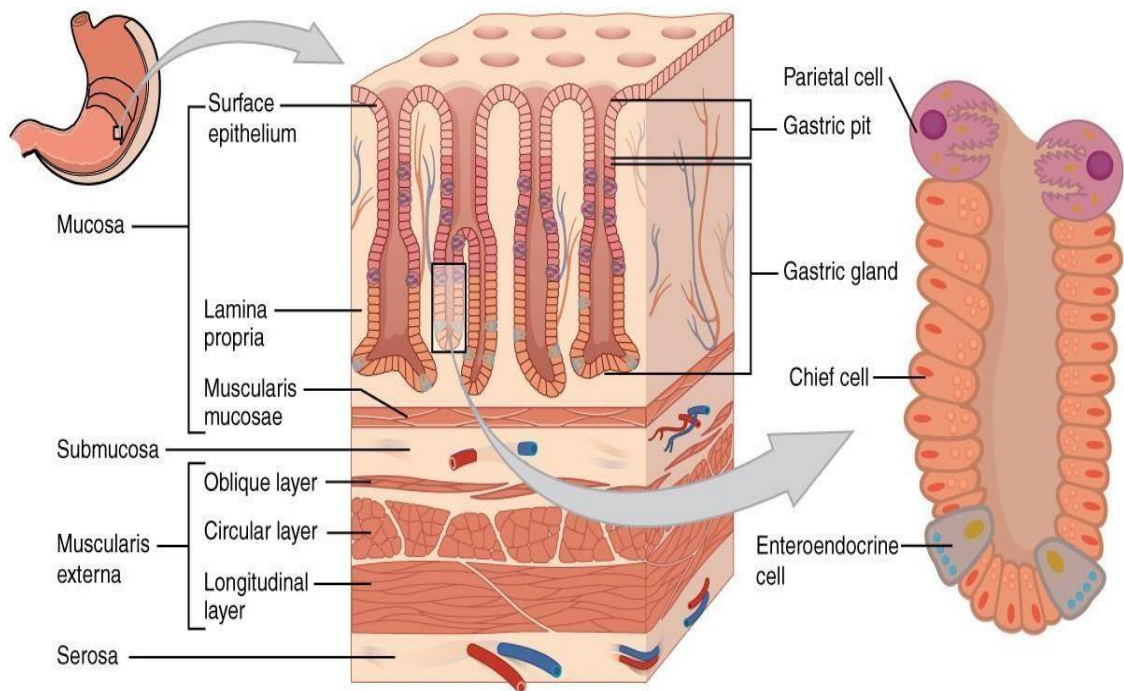


**Fig 6: Anatomy of stomach**

Layers of the stomach are

- a) mucosa
- b) submucosa
- c) muscle coat
- d) serosal coat





**Fig 7: Layers of stomach**

The mucosa has rugae when the stomach is empty. Except for areas of lesser curvature, where they are longitudinal, these rugae are irregular. Rugae vanish in a bloated stomach. The single layer of lining epithelium is called ‘surface mucosal cells’ as they secrete mucus. The mucus shields the stomach from the effects of acids and enzymes. The most frequent location of a peptic ulcer is a smaller curvature, which liquids typically reach. Many gastric pits, also known as depressions in the mucosal layer, exist.

### **Gastric Glands**

It is subdivided into

1. Cardiac glands
2. Main gastric glands
3. Pyloric glands

## **1. Cardiac glands**

They are located in the cardia of the stomach, which is the area closest to the heart and covers the gap where the esophagus connects to the stomach.

Here, the only glands are cardiac glands, which mostly release mucus. They are smaller and located more shallowly in the mucosa than the other gastric glands.

There are two different types: compound racemose, which resembles the duodenal Brunner's glands, and simple tubular with short ducts.

## **2. Main gastric glands**

They are found in the stomach's fundus and body. Peptic cells, oxyntic cells, neck cells, and endocrine cells are among the many cells that they contain.

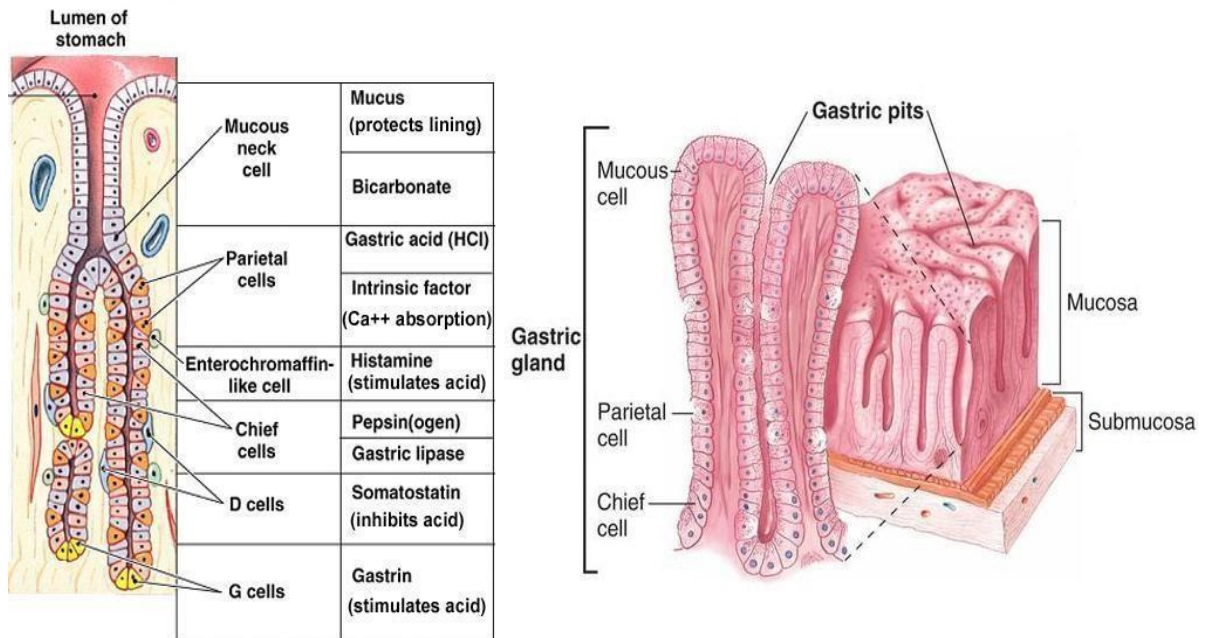
Peptic cells are found in the basal region and secrete pepsin.

Both hydrochloric acid and an intrinsic factor are secreted by oxyntic cells. Oxyntic and mucous cells are both found in the upper region. D-cells and enterochromaffin-like cells, which secrete the hormones somatostatin and histamine, respectively, are abundant throughout the gland. G-cells, which secrete gastrin, are found in the bottom portion of the stomach glands.

Numerous cells lack differentiation, and it is unknown what their roles are.

### 3. Pyloric glands

They are found in the antrum and pylorus. They release parietal, endocrine and mucous cells.



**Fig 8: Gastric glands and their secretions**

### PHYSIOLOGY

Daily, around 2.5 liters of gastric juice is produced. The juice's hydrochloric acid serves as a medium for pepsin's action. The mucus shields the gastric mucosa from acid damage by producing a gel-like layer and secreting bicarbonate. As a result, even if the pH is lower on the luminal side, it becomes basic on the surface of the epithelial cells, preventing epithelial damage. Since the mucus layer contains a high concentration of *H. pylori*, injury to this layer triggers the pathogenesis of *H. pylori*.

Gastric secretion and motility are regulated by

1. neural mechanism involving autonomic cholinergic neurons
2. hormonal mechanism involving gastrointestinal hormone

The three phases of gastric secretions are

1. cephalic phase
2. gastric phase
3. intestinal phase

#### **1. Cephalic phase**

Oxyntic and parietal cells are triggered directly by cholinergic stimulation through activation of the vagal centre, resulting in the production of digestive enzymes and acid.

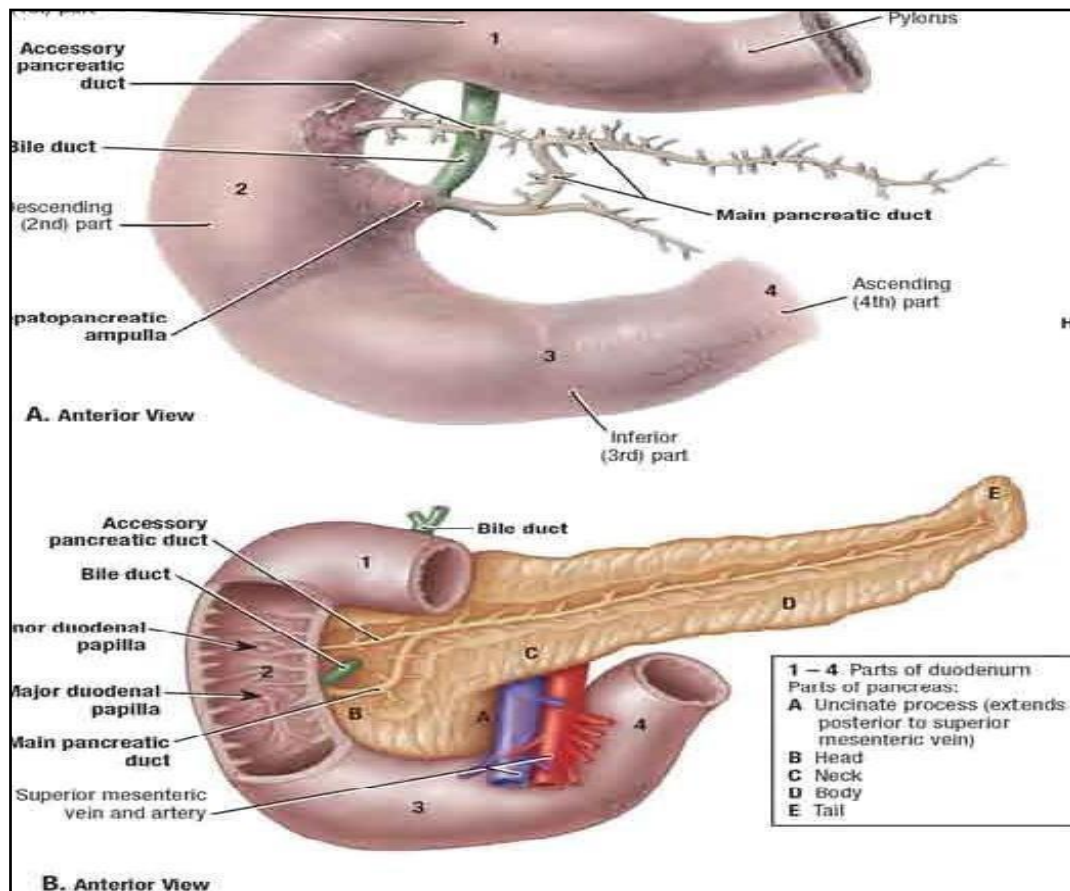
#### **2. Gastric phase**

Food causes the stomach to distend, which triggers the vagal reflex and causes the G-cells to secrete gastrin.

#### **3. Intestinal phase**

Similar to the gastric phase, when food enters the intestine, neural receptors become active, causing the production of intestinal gastrin. As soon as the pH in the antrum and duodenum drops, vagal inhibition prevents the release of additional acids.

## DUODENUM :



**Fig 9: Anatomy of duodenum**

The duodenum is 25cm long. It bends in a “C” form around the pancreatic head. It consists of three parts.

1st part:

Extends upwards, backward, and to the right to the level of the upper border of the first lumbar vertebrae. It is about 5cm long.

2nd part:

Extends downwards to the level of the lower border of the 3rd lumbar vertebrae. It is about 7.5cm long.

3rd part:

Extends to the left, then upwards to the level of the left side of the 2nd lumbar vertebrae. It is 12.5cm long. The duodenum derives its blood supply from the gastroduodenal, superior and inferior pancreatic duodenal arteries. The venous drainage corresponds to arterial supply.

## **Histology**

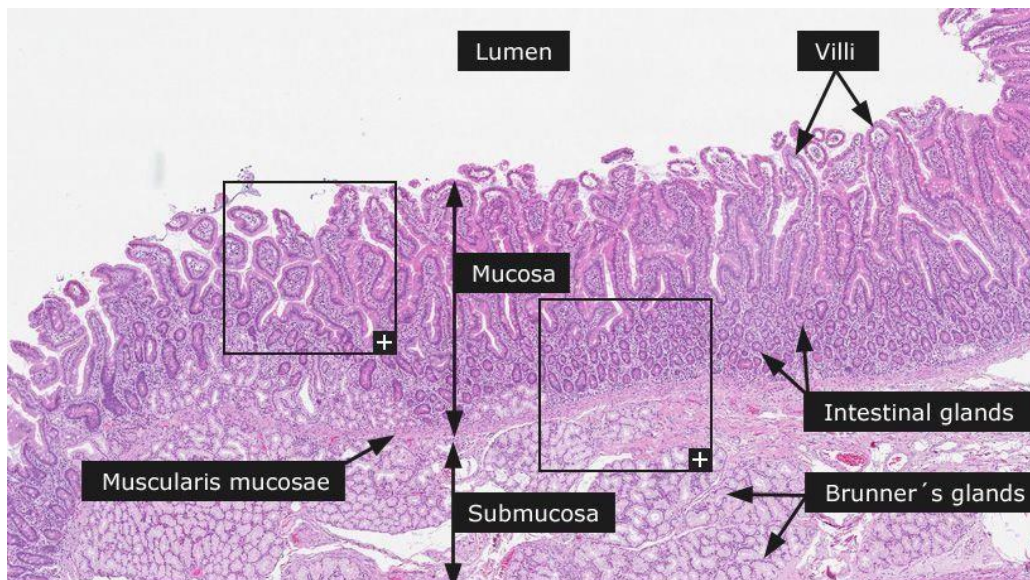
In terms of histology, the duodenum is similar to other hollow organs of gastrointestinal organs with mucosa, submucosa, and muscularis.

- The mucosa is made up of a layer of smooth muscle, a layer of connective tissue, and a simple columnar epithelium (lamina epithelial) (lamina muscularis). Enterocytes, the intestinal epithelial cells, are covered in a coating of mucin and glycoproteins.
- The Meissner's plexus, numerous blood arteries, and loose connective tissue make up the submucosa.
- The inner circular and outer longitudinal muscles of the muscularis meet at the Auerbach's plexus.

The circular folds of the mucosa and submucosa, finger-shaped villi, and microvilli (hairlike structures protruding from the surface) are common in all portions of the small intestine (valves of Kerckring). The duodenum's absorption area is increased by these structures by up to 1500 times.

The duodenum is abundant in absorbing enterocytes, goblet cells, which produce mucus, and endocrine cells, which produce peptide hormones.

The Brunner's glands implanted in the submucosa are a distinguishing feature of the duodenum. These products, among other things, include mucus secretions that include bicarbonate to balance the stomach acid. The Lieberkuhn crypts are also located between the villi. These crypts' lumen contains paneth cells. Although their roles in antimicrobial defense are known to exist today, Paneth cells still have unknown functions.



**Fig 10: Histology of Duodenum**

## Physiology

The duodenum's main function is to initiate the body's digestive process of dissolving and absorbing nutrients. It blends the chyme with digestive enzymes to break down the food, adds bicarbonate to balance acids, and gets the chyme

ready for the jejunum, where the majority of the body's nutrient absorption takes place, to break down fats and proteins.

The duodenum's specific functions include:

- Receiving the small, mixed-up pieces of food from the stomach
  - Reducing the pH level of the chyme's acidity
  - Helping the digestive process along with bile from the liver, pancreatic digestive enzymes, and intestinal secretions secreted by the walls of the duodenum and other digestive organs.
  - Setting up the chyme for subsequent digestion by including bile to aid in the breakdown of lipids
- 
- Absorption of certain nutrients such as folate, iron, and vitamin D3

### **Other Functions**

The duodenum releases two important hormones:

- **Secretin**, which is released when the duodenum's pH has to be adjusted (specific pH levels are needed for proper digestion of fats and proteins)
- **Cholecystinin**, which is released to facilitate the digestion and uptake of nutrients like fats and proteins



Immune support is one of the duodenum's key functions. In order to stop hazardous microorganisms from entering the body, the duodenum serves as a barrier.

## **UPPER GI ENDOSCOPY**

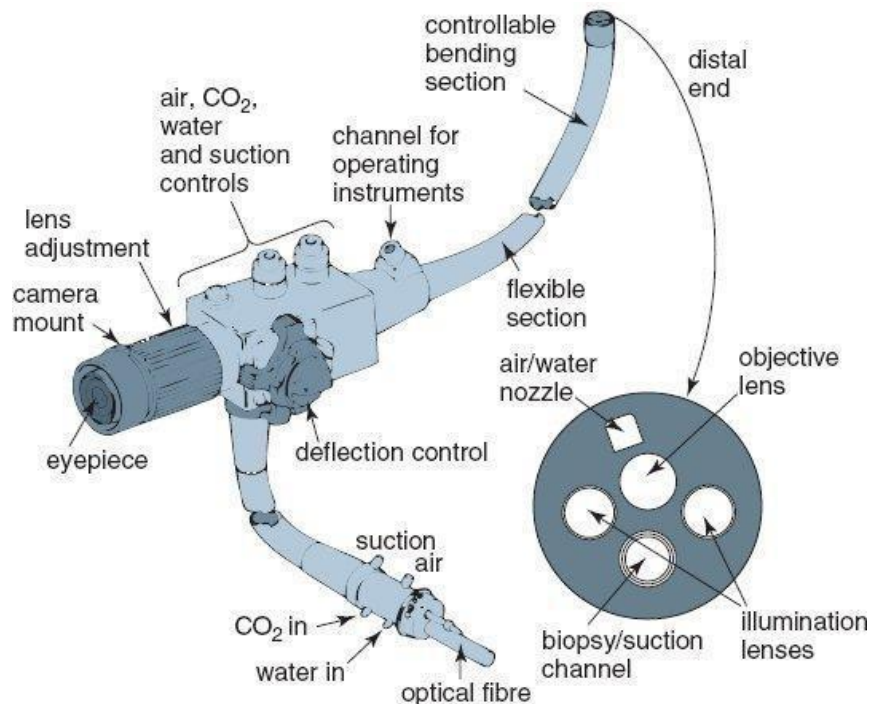
UGI endoscopy is an established mode of investigation, particularly where radiology has been negative and treatment of a wide range of upper gastrointestinal conditions <sup>(15)</sup>. The advantage of negative endoscopy is that it reduces patient anxiety & increases patient satisfaction.

The word "endoscopy" is derived from the Greek word by combining the prefix "endo," meaning "within," and the verb "skopein," "to view or observe"<sup>(16)</sup>. The term "upper endoscopy," sometimes known as "endoscopy," "EGD," or "esophagogastroduodenoscopy," is a procedure that allows a physician to visually inspect the esophagus, stomach, and duodenum in the upper part of the gastrointestinal tract<sup>(17-22)</sup>.

## **HISTORY AND DEVELOPMENT**

A rigid endoscope with a smaller lamp was utilized in the past. However, it has the drawback of overheating, and because of its rigidity, it is unable to pass through intestinal curvatures. Johann Von Mikulicz Radecki, a Polish

surgeon, designed it in the 19<sup>th</sup> century.



**Fig 11: Parts of an endoscope**

Flexible endoscopy, which is semi-rigid, was first developed by R. Schindler and German physician Georg Wolf in 1936. In 1928, Baird made the initial discovery of the flexible fiberoscope

C. Wilbur Peters and Lawrence Curtis, two physics students, developed the first flexible endoscope that could be used in clinical settings. The discovery of fibers coated in glass for insulation solved the issue of fiber cross-talk that rendered interpretation difficult. As a result, the fiberscope was created.

Hirschowitz made the discovery of the gastroscope with a controlled tip in 1962. Inc Norwalk CT produced the first endoscope for commercial use in 1961. Numerous research been conducted to determine the effectiveness against

endoscope complications, and currently, it has advanced to the point where endoscopic procedures are performed



**Fig 12: Flexible endoscope**

### **Endoscopic Equipment:**

The equipment comprises an endoscope with a chip camera at the tip, an irrigation channel to clean the lens, suction/insufflation/working channels, and a control handle. The endoscope also has a non-coaxial optic fiber system to carry light to the tip. Along with the other components mentioned, it also has a stack, a light source, an electrosurgical unit, a video recorder/photo printer, and tools, including biopsy forceps, snares, and injection needles. Chemicals utilized include indigo carmine, dimethicone, and acetate <sup>(23-25)</sup>.

The endoscope needs to be correctly positioned and manipulated for an adequate evaluation. The control head is typically held in place with the left hand, the thumb holds the knob, and the air insufflation and suction buttons are held by

the middle and index fingers. During the inspection, the flexible shaft is adjusted using the right hand.



**Fig 13: Position of Endoscope**

## **PATIENT PREPARATION**

The patient is first given a thorough explanation of the procedure once their consent is taken. The history of prior treatments is assessed. Antibiotics, for example, may be prescribed only when needed. The patient should be instructed to fast overnight. Since the majority of instances involve outpatients, an attendant must accompany them.

Sedation may not be necessary for calm patients, although it may be necessary for anxious patients to be sedated. Before sedation, consider a topical lignocaine spray. Diazepam or midazolam are typically used for sedation.

## **TECHNIQUE**

### **Introduction of endoscope**

Following the administration of the necessary topical anesthesia or sedation, the patient is placed in the left lateral decubitus posture. The teeth are spaced apart by a mouthpiece.

The midline of the endoscope is advanced to allow visualization of the structures within the oral cavity. The patient is instructed to swallow when the endoscope has reached the cricopharyngeus, and light pressure is then used to help the instrument enter the esophagus.

### **Examination of Esophagus**

The endoscope tip should be in the center of the lumen for optimal vision and optimal insufflation. It is advisable to withdraw the scope if the vision is obscured.

There will be external compression at the four sites when the endoscope travels along the esophageal lumen. The esophagogastric junction is typically visible about 38–40 cm from the incisors, and Z lines, or differences in mucosal color, are also present. The patient is instructed to breathe deeply so that the diaphragmatic hiatus leaves a mark on the esophageal and stomach walls, which can be used to determine the position of the esophageal hiatus.

## **Passage into the stomach**

The lower esophageal junction should be examined before entering the stomach to determine whether it is normal, lax, or constricted. The endoscope can penetrate the stomach without any resistance because of its broad lumen. As the stomach fills with air, the patient could experience discomfort. The posterior wall and a greater curvature are seen when the tip is bent just a little to the left and downward. For a clear view of the stomach and to avoid aspiration, all stomach liquids are suctioned out. The entire stomach body can be seen if the endoscope is rotated. Using the J maneuver, the proximal portion of the curvature is visualized. When the stomach is swollen, the J maneuver is performed by a 180-degree upward rotation of the scope.

The pyloric ring is immediately viewed through the angulated endoscope tip in order to view the antrum. Under clear visibility, the endoscope is advanced via the pylorus. Once past the pylorus, the first portion of the duodenum can be seen all the way up to the superior duodenal angle.

Once the duodenum has been viewed, the endoscope is removed while the stomach is inflated in order to examine the proximal portion of the stomach along the lesser curvature. The endoscope's tip should be straightened as it is brought back through the esophagus.

After the procedure, the patient is monitored and instructed to avoid eating or drinking for the following 30 minutes.

**Biopsy Techniques:**

EGD in the evaluation of upper GI symptoms and disease allows definitive histological assessment. Biopsy or cytologic sampling should be anticipated whenever this endoscopic procedure is undertaken, and proper informed consent for a biopsy should be obtained from the patient. The surgeon–endoscopist must supervise that proper instrumentation is available to allow adequate and efficient biopsy, and he or she must supervise the handling of all tissue for histologic or cytologic interpretation <sup>(26)</sup>.

The biopsy tool is known as a forceps. A biopsy tool is attached at one end of the lengthy wire. Many regularly used biopsy tools contain two cups on either side of a short metal spike to spike, grip, and pull off tissue. The doctor inserts the forceps through the endoscope’s biopsy channel, opens them, pinches a piece of tissue, locks them around the tissue, and then removes the forceps with the biopsy inside of them from the endoscope. The majority of patients do not feel the biopsy, and there is little bleeding. The biopsy is a tiny piece of tissue about the size of a cooked rice grain. It's put in the fixative and delivered to the pathologist for histopathology analysis <sup>(27)</sup>.



**Fig 14: Endoscopic biopsy instrument**

A biopsy may be important even though gross mucosal lesions are not evident. In these circumstances, histologic assessment of “normal” mucosa may identify microscopic evidence of malignant or dysplastic tissue and determine therapy.

#### **Biopsy handling – Esophagus, Stomach and Duodenum:**

The biopsy specimen, which typically has a diameter of 1 to 5 mm, consists of epithelium, lamina propria, and rarely a slip of muscularis mucosae. In some circumstances, an aspiration technique may be employed instead, producing specimens that are deeper and larger, usually containing some submucosa. In order to facilitate appropriate orientation for embedding and sectioning, the biopsy specimens should ideally be mounted at the time of procurement by placing them submucosal side down on a supporting medium such as filter paper, nylon mesh, or gel foam and then placing them immediately in fixative. The



specimen can be immediately dropped into fixative without mounting if mounting methods are unavailable, although this orientation might not be ideal. While some pathologists prefer picric acid fixatives or fixatives based on mercury, standard formalin fixation usually suffices <sup>(28)</sup>.

20 to 30 serial slices at a thickness of 3 to 5 micrometers should be prepared for each biopsy specimen. Depending on the size of the biopsies, these may be put on one to three slides. For routine purposes, hematoxylin and eosin (H&E) staining is satisfactory, and special stains are generally not required in the first instance. However, some laboratories find it more convenient to routinely prepare special stains for *Helicobacter* organisms in case of gastric and duodenal biopsies. Depending on the disease present, different numbers of biopsies should be taken<sup>(28)</sup>.

Light and electron microscopy, special stains, immunohistochemical techniques, fluorescent in situ hybridization (FISH), and polymerase chain reaction (PCR) are some of the pathologic methods used to evaluate endoscopic biopsy specimens. Often, light microscopy and H&E staining are enough to make a diagnosis or confirm it. Protozoan infections of the small intestine can be detected using electron microscopy because tiny organisms like cryptosporidia and microsporidia are easier to see under this microscope. To emphasize specific disease traits, special stains might be used: Grocott's Methenamine Silver (GMS) stains show fungi like candida, histoplasmosis, and cryptococcus; Periodic Acid Schiff with Diastase (PASD) stains highlight acid mucopolysaccharides,

glycogen, and pseudohyphae in candidiasis; Mycobacterial Bacilli are visible in Acid-Fast Bacilli (AFB) stains; Spirochetes are detected using Warthin-Starry stains, while the diagnosis of microsporidia is made using Brown-Brenn stains<sup>(29)</sup>.

## **INDICATIONS**

### **Diagnostic indications<sup>(30)</sup>**

1. To identify the site and lesion in upper gastrointestinal bleeding (acute or chronic).
2. Accurate follow-up during the treatment of upper gastrointestinal lesions.
3. Primary evaluation of patient symptoms related to the upper gastrointestinal tract.
4. Part of the workup for occult malignancy.
5. To evaluate further, those lesions seen on X-ray (plain /contrast studies)
6. Intraoperative assessment of gastroduodenal lesions.
7. To evaluate early and late postoperative complications.

### **Therapeutic indications<sup>(30)</sup>**

1. Control of bleeding points with electrocautery or laser
2. Gastric or esophageal polypectomy.
3. Removal of foreign bodies
4. Disintegration of bezoars

5. Placement of guide wire for esophageal dilatation.
6. Sclerosis of esophageal varices.
7. Placement of nasogastric feeding tube.
8. Placement of percutaneous gastrostomy

## **CONTRAINDICATIONS**

### **Absolute Contraindication <sup>(30)</sup>**

1. An unstable patient (hypotension, respiratory distress, seizures, or mechanical instability of the neck)
2. Acute myocardial infarction.

### **Relative Contraindications <sup>(30)</sup>**

1. An un-cooperative patient.
2. Coagulopathy -- particularly in therapeutic endoscopic procedures.
3. Myocardial ischemia
4. Thoracic aortic aneurysm
5. Early in the postoperative period after upper gastrointestinal surgery

## **COMPLICATIONS OF ENDOSCOPY <sup>(30, 31)</sup>**

1. Drug reactions related to premedication, like anaphylactic reaction. Aspiration of secretion, respiratory depressions in elderly patients, cirrhotic patients and

patients with chronic obstructive pulmonary disease

2. Bleeding
3. Perforation of esophagus
4. Cardiopulmonary complications like sinus tachycardia, ventricular und atrial premature beats.
5. Infection

## **METHODOLOGY**

A prospective cross-sectional observational study “The spectrum of histopathological changes in endoscopic gastroduodenal biopsy in patients with FD” was undertaken at General surgery department, B.L.D.E.(D.U) Shri B.M.Patil Medical College Hospital,. The period of study was from January 2021 to November 2022. The patient selection was by convenience sampling.

A detailed clinical history was elucidated, followed by careful clinical examination and relevant investigation. All the patients meeting the Rome IV criteria were included in the study. UGI endoscopy was performed on each patient in the study group, and the biopsies were taken after obtaining written informed consent from all the patients with a detailed explanation of the procedure which was performed on them, the risk factors and complications involved and the advantages and disadvantages of the same as per the proforma.

## **INCLUSION CRITERIA**

- Patients diagnosed having functional dyspepsia as per ROME IV criteria.
- Age > 18years.

## **EXCLUSION CRITERIA**

- Patients with detectable organic causes on endoscopy or USG abdomen
- Refusal for endoscopy.

## **PROCEDURE:**

UGI endoscopy was performed on each patient in the study group under topical anesthesia. The patients were asked to fast overnight for the procedure.

For the purpose of a local anesthetic, lignocaine sprays were administered to the patient ten minutes prior to the treatment. Patients were placed in left lateral positions when the UGI endoscopy was performed using a flexible, fiberoptic endoscope.

All the patients included in the study underwent UGI endoscopy as described before. Five endoscopic biopsies were randomly taken from the fundus, body, antrum and pylorus of the stomach and the duodenum in cases where normal endoscopic results were found. Once the procedure was over, the patient was observed for the next 30 min. The UGI endoscopy was performed by the same team of surgeons.

**Fig 15: ENDOSCOPIC PICTURES OF NORMAL UGI MUCOSA**



**GEJ**



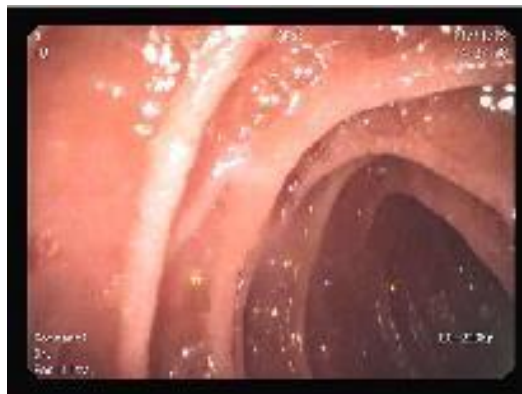
**FUNDUS**



**ANTRUM**



**PYLORUS**



**DUODENUM**

## RESULTS AND ANALYSIS OF OBSERVED DATA

### EPIDEMIOLOGY

A total of 72 patients in the study underwent UGI endoscopy, and five endoscopic biopsies were randomly taken from the fundus, body, antrum and pylorus of the stomach and the duodenum.

The majority of the patients were from Vijayapur and the surrounding area.

The age distribution of patients is shown in the table.

**Table 1: A ge and sex distribution of study subjects**

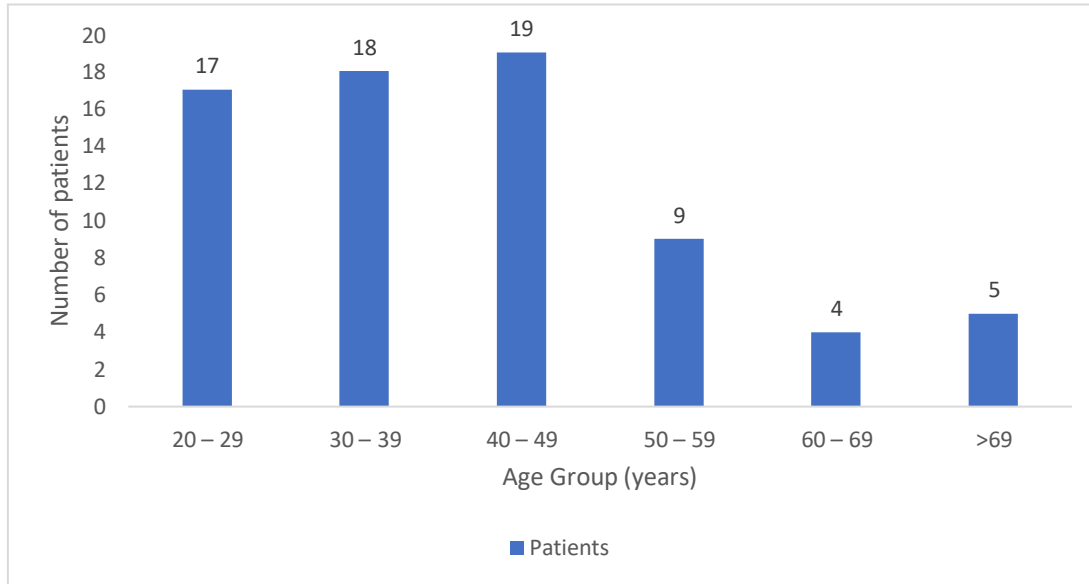
Age in Years	Sex		Total	Percentage
	Male	Female		
20 – 29	13	4	17	23.6
30 – 39	10	8	18	25
40 – 49	9	10	19	26.4
50 – 59	6	3	9	12.5
60 – 69	1	3	4	5.6
>69	1	4	5	6.9
Total	40	32	72	100
Mean		41.11 years		
Standard deviation		14.724 years		



The patients' ages varied from 20 to 87 years.

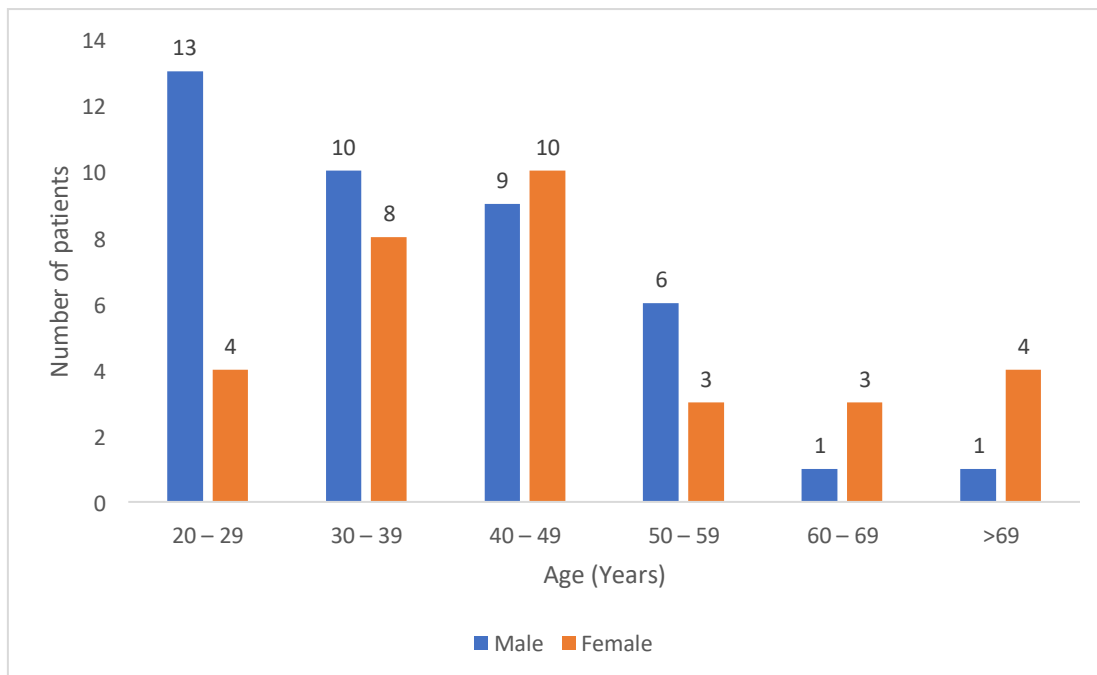
The study population's mean age was 41.11 years.

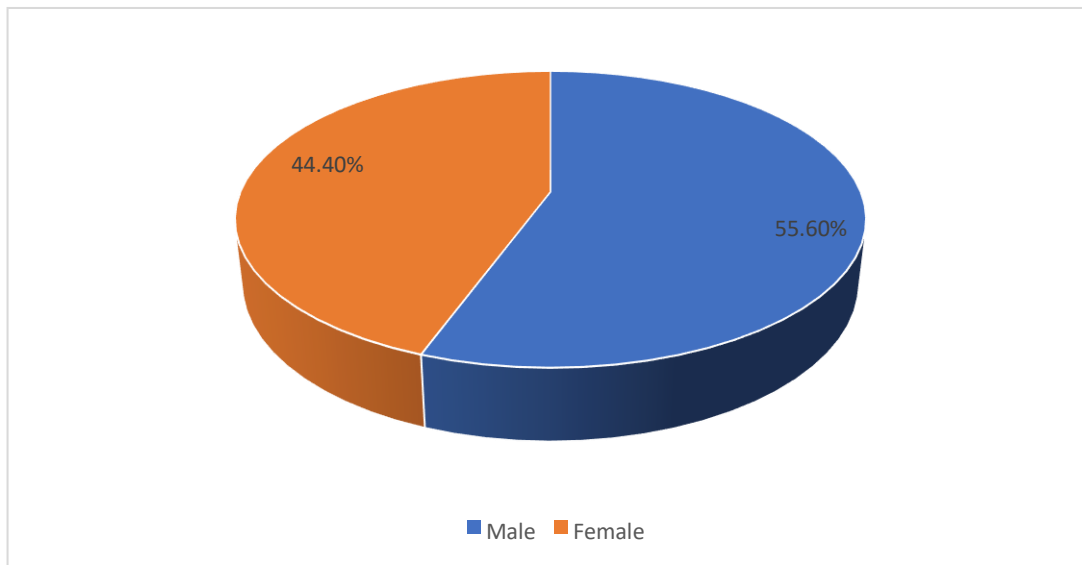
**Graph 1: Showing age distribution of study subjects**



The majority of FD patients were in the age group of 20 – 49 years with the highest in 40-49 age group.

**Graph 2: Showing age and sex-wise distribution of study subject**



**Graph 3: Graph Showing sex-wise distribution of study subject**

Of the 72 patients, 40 (55.6%) were male, and 32 (44.4%) were female.

### **SYMPTOMATOLOGY- DURATION**

The length of symptoms ranged from six months to ten years.

The duration of the presenting symptoms is shown in table 2.

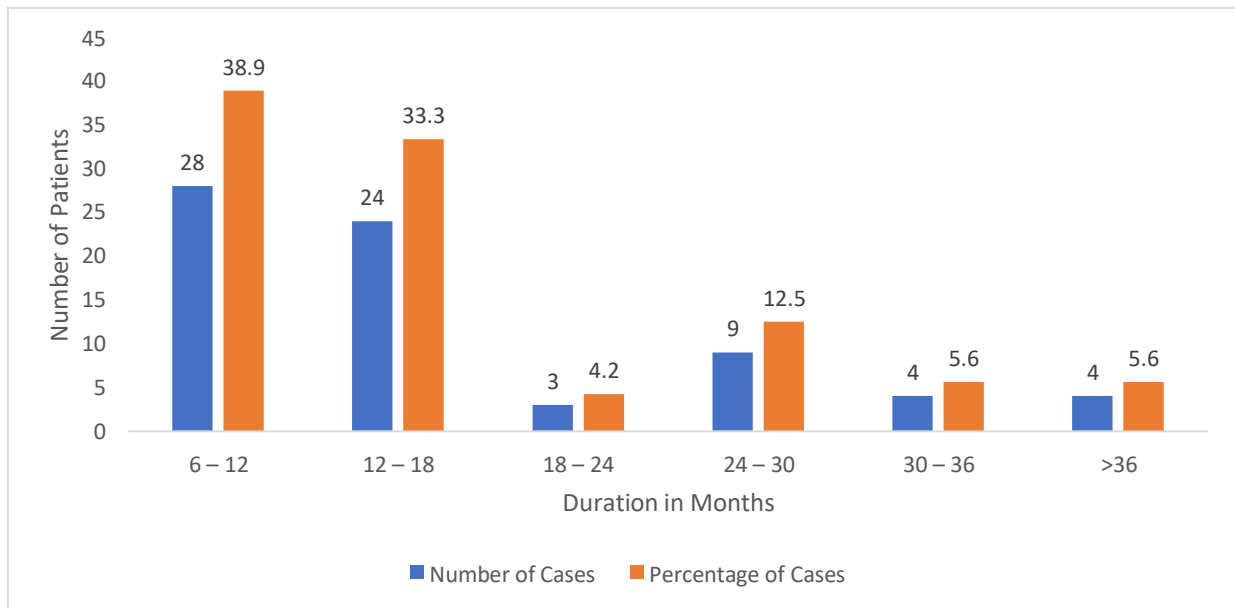
**TABLE 2: DURATION OF SYMPTOMS**

<b>Duration of symptoms (in months)</b>	<b>Cases</b>	
	<b>No.</b>	<b>%</b>
<b>6 – 12</b>	28	38.9
<b>12 – 18</b>	24	33.3
<b>18 – 24</b>	3	4.2
<b>24 – 30</b>	9	12.5
<b>30 – 36</b>	4	5.6
<b>&gt;36</b>	4	5.6
<b>MEAN</b>	16.58	
<b>S.D</b>	18.50	

Out of the 72 patients, 52 (72.2%) presented within 6 to 18 months of the onset of symptoms, with majority in 6 to 12 months (38.9%). The mean duration of symptoms was 16.58 months.

The duration of symptoms is depicted in graph 4.

**Graph 4: Showing the duration of symptoms**

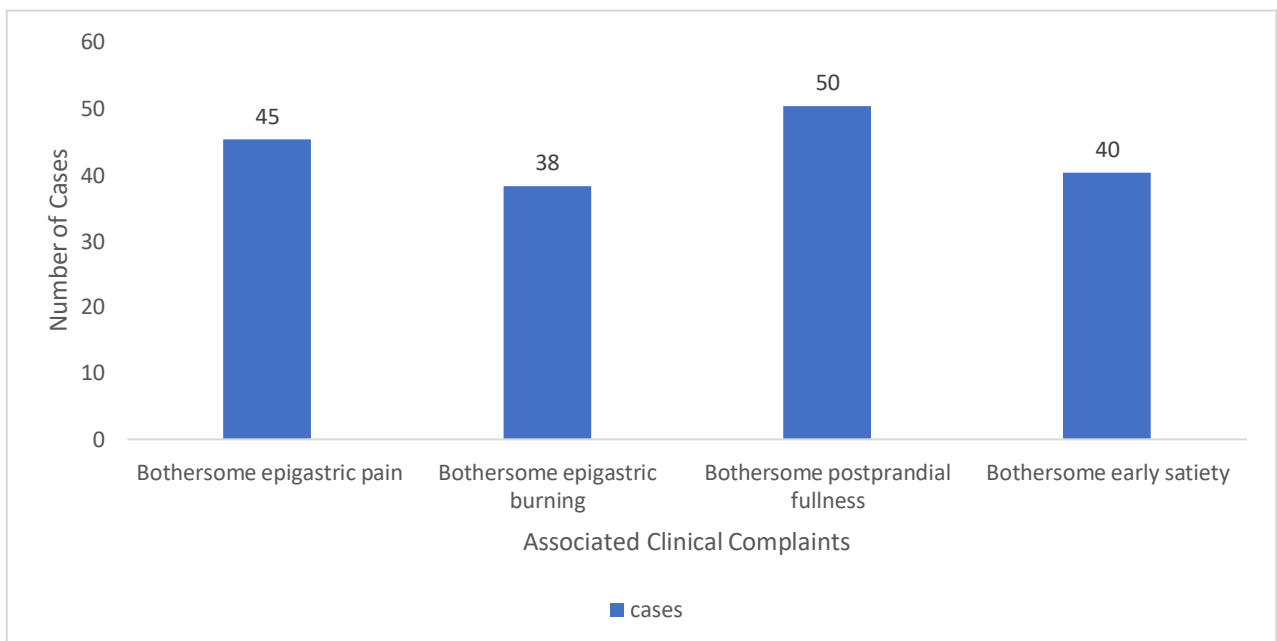


## ASSOCIATED CLINICAL COMPLAINTS

Symptoms of FD include bothersome epigastric pain, epigastric burning, postprandial fullness and early satiety. The occurrence of varied FD symptoms is shown in Table 3 and Graph 5.

**Table 3: Associated clinical complaints**

<b>Associated Clinicalcomplaints</b>	<b>Total Number of Cases</b>	<b>Percentage</b>
Bothersome epigastric pain	45	62.5 %
Bothersome epigastric burning	38	52.8 %
Bothersome postprandial fullness	50	69.4 %
Bothersome early satiety	40	55.6 %

**Graph 5: Showing associated clinical complaints**

69.4% of the patients presented with bothersome postprandial fullness, 62.5% with bothersome epigastric pain, 55.6% with bothersome early satiety and 52.8% with bothersome epigastric burning. Bothersome postprandial fullness was the most typical presentation of all.

## ASSOCIATED CLINICAL SYNDROMES

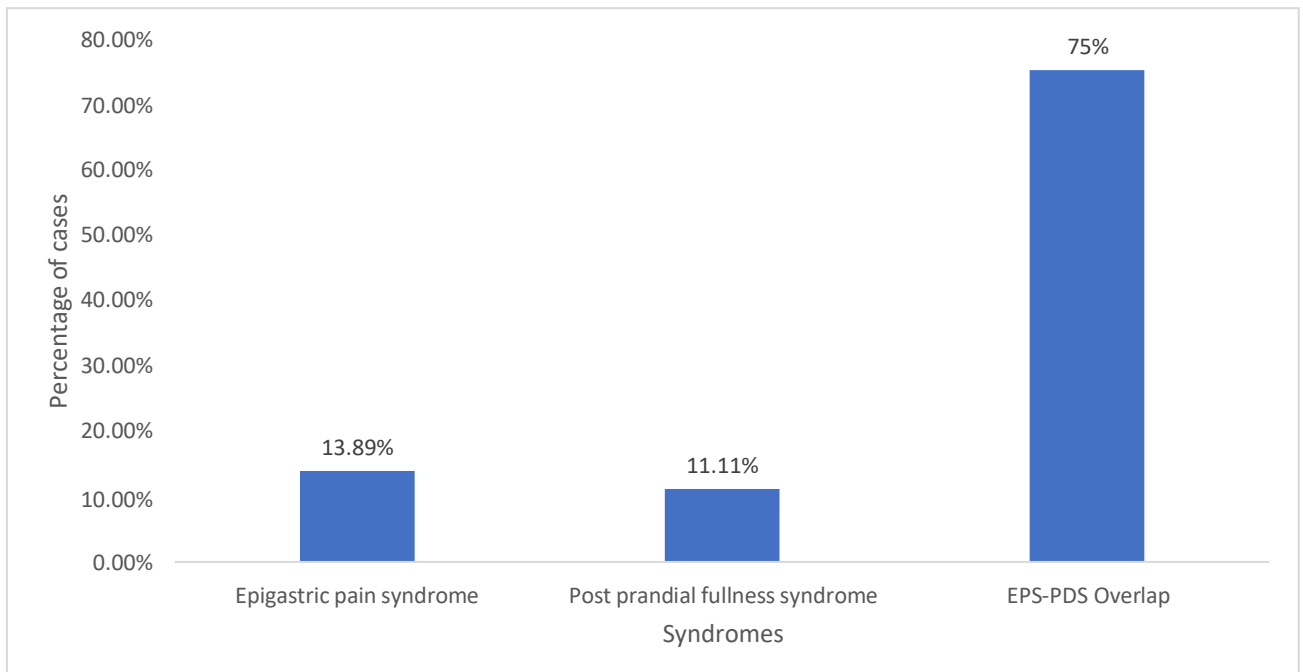
FD remains an umbrella term that includes both epigastric pain syndrome (EPS) and postprandial distress syndrome (PDS) with substantial overlap.

The frequency of clinical syndrome is shown in table 4 and graph 6.

**Table 4: Associated Syndrome Presentation**

Syndrome	Number of cases	Percentage
EPS	10	13.89%
PDS	08	11.11%
EPS-PDS Overlap	54	75%

**Graph 6: Showing associated Syndrome Presentation**



Out of the study group, 10 (13.89%) patients had epigastric pain syndrome, 8

(11.11%) patients had postprandial distress syndromes, and 54 (75%) patients had EPS-PDS overlap.

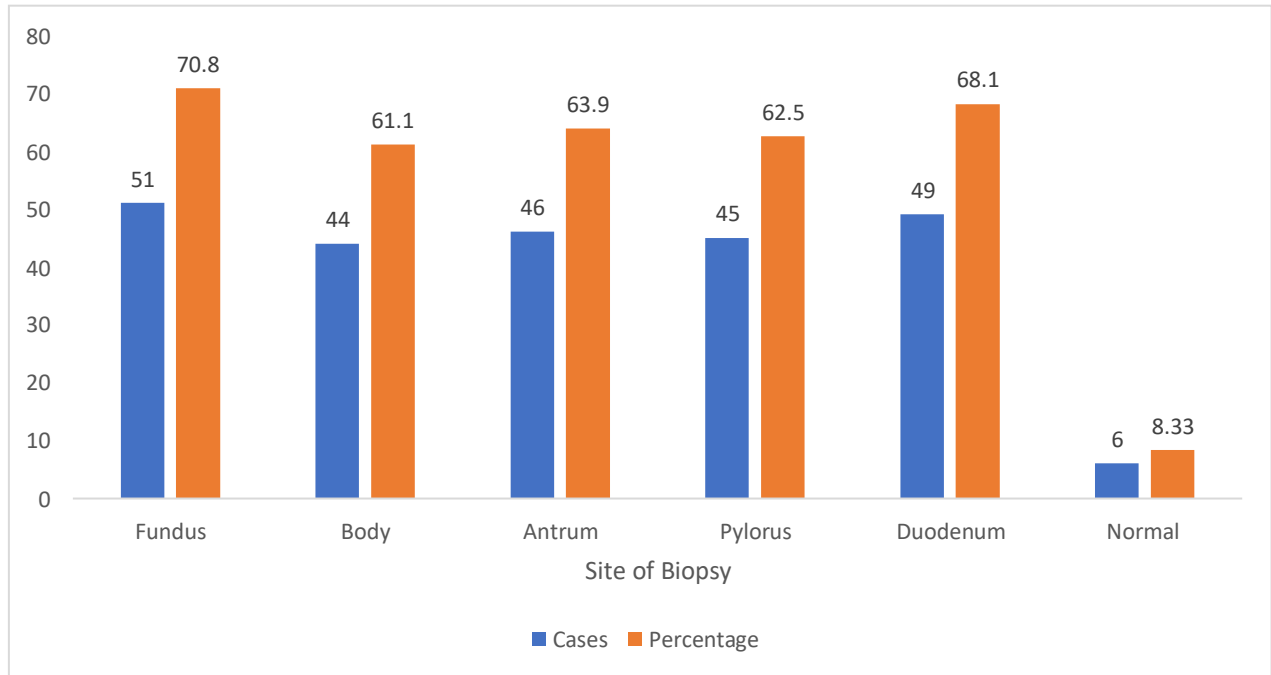
### **PREVALENCE OF HISTOPATHOLOGIC LESIONS ACCORDING TO SITE**

Five endoscopic biopsies were randomly taken from the fundus, body, antrum and pylorus of the stomach and the duodenum. The Distribution of histopathologic lesions in different sites is shown in table 5 and Graph 7.

**Table 5: Site-wise distribution of histopathologic lesions**

<b>Site</b>	<b>No of Cases</b>	<b>Percentage</b>
Fundus	51	70.8 %
Body	44	61.1 %
Antrum	46	63.9 %
Pylorus	45	62.5 %
Duodenum	49	68.1 %
Normal	06	8.33%

**Graph 7: Showing site-wise distribution of histopathologic lesions**



Out of 72 patients, 66 (91.67%) had histological lesions in either stomach or the duodenum and the rest 6 were normal. (8.33%).

Among the study group, 15 (20.83 %) patients had normal findings in the stomach with histopathological changes in duodenum. 26 (36.11 %) patients had normal findings in the duodenum with histopathological changes in stomach.

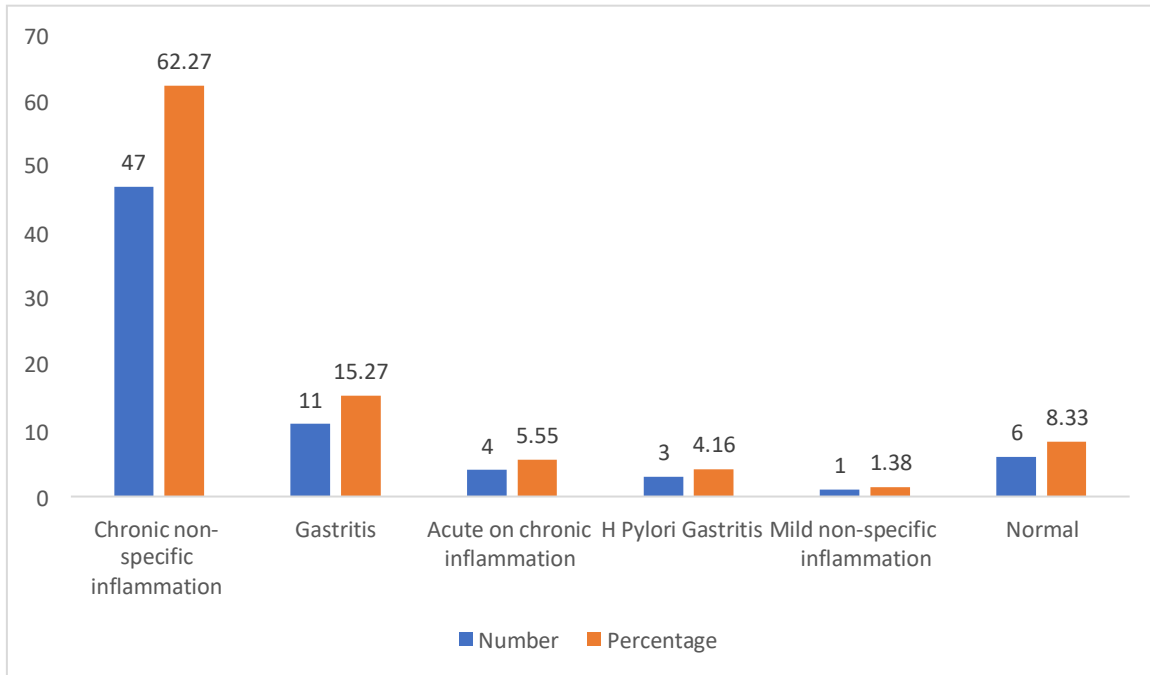
The fundus (70.8 %) was the most commonly involved region in the stomach and overall, followed by the duodenum (68.1%).

## HISTOPATHOLOGICAL FINDINGS SEEN IN BIOPSIES

The various histopathological findings were noted.

Graph 8 shows the frequency of various histopathological findings seen in biopsies.

### **Graph 8: Frequency of various histopathological findings seen in biopsies**



The histological lesions were found in 91.67% (66 out of 72 patients).

Chronic non-specific inflammation was the commonest finding (62.27%). Features of Gastritis (15.27%), acute on chronic inflammation (5.55%), H Pylori Gastritis (4.16%) and mild non-specific inflammation (1.38%) were other findings.

## **PRESENCE OF H. PYLORI**

Features of H Pylori Gastritis was present in only 3 patients.



## DISCUSSION

Upper endoscopy, also known as endoscopy, EGD, or esophago-gastroduodenoscopy, is a procedure that allows a specialist to examine the upper part of the gastrointestinal (G.I.) tract, which includes the oesophagus, stomach, and duodenum. Endoscopic biopsy is currently an important diagnostic method for gastrointestinal diseases <sup>(17-22)</sup>.

Endoscopic biopsy sampling of the upper gastrointestinal tract provides useful information that aids in the diagnosis of various lesions <sup>(32,33)</sup>. Good clinical and endoscopic information is a critical component of adequacy, and it has a significant impact on how a biopsy should be interpreted. In the present study, endoscopy along with biopsy was done on patients presenting with symptoms of FD.

A study entitled “The spectrum of histopathological changes in endoscopic gastroduodenal biopsy in patients with functional dyspepsia” was undertaken at the general surgery department, B.L.D.E. (D.U.) Shri B.M.Patil Medical College Hospital. A total of 72 patients included in the study underwent UGI endoscopy, and five endoscopic biopsies were randomly taken from the fundus, body, antrum, pylorus of the stomach and the duodenum and various findings were noted. The study’s findings were comparable to those of numerous previous studies.

### **COMPARISON OF AGE DISTRIBUTION:**

The majority of patients with FD were in the age group of 20 – 49 years. The mean age of our study subjects was 41.11 years. In the studies conducted by various authors, the mean age was as shown in table no 6.

**Table no 6: Comparison of age distribution:**

<b>Sl. No</b>	<b>Name of study</b>	<b>Mean age in years</b>
1	Hosman M Dawod et al <sup>(34)</sup>	45.54
2	Anouar Teriaky et al <sup>(35)</sup>	44
3	S Nwokediuko et al <sup>(36)</sup>	44.03
4	Present study	41.11

The above studies also had similar observations regarding the mean age in patients with dyspepsia.

### **COMPARISON OF GENDER DISTRIBUTION:**

The study population was 55.6% male and 44.4% female. Males had a slightly higher prevalence of the various FD presentations than females.

The gender distribution in various similar studies is shown in table no 7.

**Table no 7: Comparison of gender distribution:**

Sl. No	Name of study	Percentage of male	Percentage of female
1	Dawod and Emara <sup>(34)</sup>	48.6%	51.4%
2	S Nwokediuko et al <sup>(36)</sup>	50.7%	49.3%
3	K. Van den Houte et al <sup>(37)</sup>	23%	77%
4	Ali Jafari Heidarloo et al <sup>(38)</sup>	40%	60%
5	Tariq Sarfaraz et al <sup>(39)</sup>	65%	35%
6	Present study	55.6 %	44.4%

Studies done by Dawod, Nwokediuko and Tariq Sarfaraz had similar results, with presentations of FD slightly higher in males than females.

Contrary to our study, a study conducted by K. Van den Houte et al. and Ali Jafari Heidarloo had dyspeptic symptoms more common in females with male to female ratio of 1:3 and 2:3, respectively.

## COMPARISION OF CLINICAL PRESENTATION

In our study, the chief complaint of the majority of the patients was bothersome postprandial fullness. It was present in 50 (69.4%) patients. It was followed by bothersome epigastric pain in 45 (62.5%) patients. Bothersome early

satiety and Bothersome epigastric burning were present in 40 (55.6%) and 38 (52.8%), respectively.

**Table no 8: Comparison of clinical symptoms**

Symptoms	K Van den Houte et al <sup>(40)</sup>	Present study
Bothersome epigastric pain	72%	62.5%
Bothersome epigastric burning	38%	52.8%
Bothersome postprandial fullness	91%	69.4%
Bothersome early satiety	58%	55.6%

In a similar study done by K. Van den Houte et al. in 2020, 91% of all patients reported postprandial fullness. In addition, 72% of all patients reported epigastric pain. Early satiation and epigastric burning were present in respectively 58% and 38% of all patients. The results matched our study.

**Table no 9: Comparison of the clinical syndrome**

Study	EPS	PDS	OVERLAP
Imran Aziz et al <sup>(41)</sup>	18%	61%	21%
Ami D. Sperber et al <sup>(42)</sup>	19.2%	62.8%	17.8%
Ghosal and Singh <sup>(3)</sup>	9%	27%	64%
Present study	13.9%	11.1%	75%

Two studies that used the Rome IV criteria reported the prevalence of the different subtypes of dyspepsia.

In a three-nation study by Imran Aziz et al., conducted in Canada, the UK, and the USA, the subtype distribution was 61% postprandial distress syndrome, 18% epigastric pain syndrome, and 21% overlapping variant with both syndromes; this pattern was similar across the countries.

In a multinational study done by Ami D. Sperber et al., again, the pooled prevalence of PDS was higher (62.8%) than the pooled prevalence of either EPS (19.2%) or overlap (17.8%).

In a study conducted by Ghosal and Singh in India with 528 patients, 9% had epigastric pain, 27% had postprandial distress syndromes and 64% EPS-PDS overlap.

Our study had similar results to the study by Ghosal and Singh, with more prevalence in EPS-PDS overlap, different from studies of other countries.

## **COMPARISON OF HISTOPATHOLOGIC LESIONS**

In a similar study by Nwokediuko and Okafor<sup>(43)</sup>, 53 out of 75 (70.7%) patients with normal UGI endoscopy exhibited one or more indices of gastritis in varying degrees of severity. The remaining 22 (29.5%) had histologically normal gastric mucosa. Furthermore, female patients had mucosal inflammation more frequently than their male counterparts did.

A study by Dawod and Emara <sup>(34)</sup> reported similar findings where histological lesions were found in 65.7% (69 out of 105). The commonest finding was chronic inflammation. Neutrophilic activity, glandular atrophy, and mild intestinal metaplasia were found in 27, 45, and 6 individuals, respectively (22.8, 42.8, and 5.7%). A significant difference ( $p = 0.045$ ) was found between the presence of *H. pylori* in 54 patients with histopathological lesions and 6 patients without.

Another study by Tariq Sarfaraz et al. <sup>(34)</sup> on 100 patients had similar results, with 30% of patients having no notable histological findings, while 70% of patients had gastritis-related histological characteristics. 70 cases (70%) of chronic inflammation, 15 cases (15%) of activity, 2 cases (2%) of glandular atrophy, and 2 cases (2%) of intestinal metaplasia, were observed. Based on hematoxylin and eosin (H and E) staining and modified Giemsa staining, *H. Pylori* was discovered in 25 instances (or 25% of the cases).

Contrary to our results, according to a recent study by Anouar Teriaky et al. <sup>(35)</sup> with 1054 patients, 58 % of patients with normal histology, yet 23% of patients having histological features of chronic gastritis, which was also the most typical finding in our study, followed by 7% with reactive gastropathy in stomach. 88% of the biopsies from the duodenum were normal followed by features of duodenitis in 6% of the patients.

## SUMMARY

Considering the current study on — The spectrum of histopathological changes in endoscopic gastroduodenal biopsy in patients with FD, we conclude that-

- Males were more likely than females to have FD, and it was more prevalent in the 40- to 49-year-old age group.
- Bothersome postprandial fullness is the most common presenting symptom in patients with FD.
- Majority of the patients presented with EPS-PDS overlap.
- The histopathological examination allowed the detection and grading of gastric and duodenal pathology in patients with FD. It showed that Chronic non-specific inflammation was the commonest histopathological finding
- Significantly low frequency of H. pylori in patients with FD.
- UGI endoscopy is incomplete without biopsy, which is the gold standard to determine the underlying pathology.

## CONCLUSION

The prospective study of “The spectrum of histopathological changes in endoscopic gastroduodenal biopsy in patients with functional dyspepsia” is aimed to identify the possible histopathological changes of the mucosa in an otherwise normal appearing upper GI tract in patients with FD, defined by Rome IV criteria.

Based on the study results, the following observations are made.

- Prevalence of FD is more in the 40-49 years age group and more common in males than females.
- Bothersome postprandial fullness is the most common presenting symptom in patients with FD.
- Majority of the patients presented with EPS-PDS overlap.
- Histopathological examination showed changes in 66 patients out of 72. Chronic non-specific inflammation was the most common finding, followed by gastritis. Fundus was the most common site.
- Significantly low frequency of *H. pylori* in patients with FD.

Based on the above findings, we conclude that the histopathological changes are noted in patients with FD, having grossly appearing normal mucosa in UGI endoscopy. However, more studies are required to identify the cause of inflammation. We also recommend endoscopic biopsy for patients with FD, even though the gross endoscopic findings may be normal.



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**ANNEXURE - I**

**SAMPLE INFORMED CONSENT FORM**

**B.L.D.E.(D.U.) SHRI B.M. PATIL MEDICAL COLLEGE HOSPITAL AND  
RESEARCH CENTRE, VIJAYAPUR – 586103, KARNATAKA**

**TITLE OF THE PROJECT :** THE SPECTRUM OF  
HISTOPATHOLOGICAL CHANGES IN ENDOSCOPIC  
GASTRODUODENAL BIOPSY IN PATIENTS WITH FUNCTIONAL  
DYSPEPSIA

**PRINCIPAL INVESTIGATOR:** DR. PRAJWAL P S

DEPARTMENT OF GENERAL SURGERY

**PG GUIDE**

:DR. VIKRAM SINDGIKAR

M.S. GENERAL SURGERY

ASSOCIATE PROFESSOR

DEPARTMENT OF GENERAL SURGERY

**CO GUIDE**

:DR. VIJAYALAXMI PATIL

M.D. PATHOLOGY

ASSOCIATE PROFESSOR

DEPARTMENT OF PATHOLOGY

**PURPOSE OF RESEARCH:**

I have been informed that this study will analyse the endoscopic biopsy findings for functional dyspepsia. I have been explained about the reason for doing this study and selecting me/my ward as a subject for this study. I have also been given free choice for either being included or not in the study.

**PROCEDURE:**

I understand that relevant history will be taken and I will undergo detailed clinical examination and will also be explained about the required investigations as per standard protocol.

**RISKS AND DISCOMFORTS:**

I understand that I/my ward may experience some pain and discomfort during the examination or during any intervention. This is mainly the result of my condition and the procedure of this study is not expected to exaggerate these feelings which are associated with the usual course of diagnosis and treatment.

**ALTERNATIVES:**

Even if you decline in participation, you will get the routine line of management.

**BENEFITS:**

I understand that I/my ward's participation in this study will help to analyse the endoscopic biopsy findings in functional dyspepsia.



**CONFIDENTIALITY:**

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 and identified only by a code number. The code key connecting name to numbers will be kept in a separate secure location. 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.PRAJWAL P S 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. PRAJWAL P S 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 to 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    Dr VIJAYALAXMI PATIL    Dr PRAJWAL P S

(Guide)

(Co guide)

(Investigator)

**STUDY SUBJECT CONSENT STATEMENT:**

I confirm that Dr.PRAJWAL P S has explained to me the purpose of this research, the study procedure that I will undergo and the possible discomforts and benefits that I may experience, in my own language.

I have been explained all the above in detail in my own language and I understand the same.

Therefore,I agree to give my consent to participate as a subject in this research project.

\_\_\_\_\_  
(Participant)

\_\_\_\_\_  
Date

\_\_\_\_\_  
(Witness to above signature)

\_\_\_\_\_  
Date

**ANNEXURE - II**

**PROFORMA**

NAME:

OPD NUMBER:

AGE/SEX:

WARD/UNIT:

OCCUPATION:

DATE OF ADMISSION:

DATE OF ENDOSCOPY:

DATE OF DISCHARGE:

CHIEF COMPLAINTS:

HISTORY OF PRESENTING ILLNESS:

PAST HISTORY:

PERSONAL HISTORY:

Diet-

Appetite-

Sleep-

Bowel and bladder-

Habits-

**GENERAL PHYSICAL EXAMINATION:**

**BUILT:** Well / Moderate / Poor

**NOURISHMENT:** Well / Moderate / Poor

**PALLOR-**      **ICTERUS-**      **CYANOSIS-**      **CLUBBING-**      **PEDAL**  
**EDEMA-**      **GENERALISED LYMPHADENOPATHY-**

**VITALS:**

**Pulse-**      bpm, **Blood Pressure-**      mmHg, **Respiratory Rate-**      cpm

**SYSTEMIC EXAMINATION:**

**PER ABDOMEN:**

**RESPIRATORY SYSTEM:**

**CARDIOVASCULAR SYSTEM:**

**CENTRAL NERVOUS SYSTEM:**

**CLINICAL DIAGNOSIS:**

**INDICATIONS FOR ENDOSCOPY:**

**UPPER GI ENDOSCOPY FINDINGS:**

**HISTOPATHOLOGICAL EXAMINATION OF BIOPSY:**

- 1)Fundus
- 2)Body
- 3)Antrum
- 4)Pylorus
- 5)Duodenum

## ANNEXURE - III

### ETHICAL COMMITTEE CLEARANCE CERTIFICATE



B.L.D.E. (DEEMED TO BE UNIVERSITY)

IEC/09/2021  
Date-22/01/2021

(Declared vide notification No. F.9-37/2007-U.3 (A) Dated. 29-2-2008 of the MHRD, Government of India under Section 3 of the UGC Act, 1956)

The Constituent College

SHRI. B. M. PATIL MEDICAL COLLEGE, HOSPITAL AND RESEARCH CENTRE

#### INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Institutional ethical committee of this college met on 11-01-2021 at 11-00 am to 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 been accorded Ethical Clearance

**Title:** Evaluation of upper gastrointestinal biopsy in functional dyspepsia.

**Name of PG student:** Dr Prajwal P S, Department of Surgery

**Name of Guide/Co-investigator:** Dr Vikram Sindgikar, Associate Professor  
Department of Surgery

  
DR .S.V.PATIL  
CHAIRMAN, IEC

**Institutional Ethical Committee**  
**B.L.D.E (Deemed to be University)**  
**Shri B.M. Patil Medical College,**  
**VIJAYAPUR-586103 (Karnataka)**

**Following documents were placed before Ethical Committee for Scrutinization:**

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

## ANNEXURE - IV

### MASTER CHART

Sl. No	Name	Age	Sex	OP No	EP	EB	PPF	ES	Duration	Habits	CO Mob	Fundus	Body	Antrum	Pylorus	Duodenum	H P
1	RAMMANNA	35	M	74327	A	P	P	P	12	A/S	Y	MNSI	MNSI	MNSI	MNSI	MNSI	A
2	SUDHABAI	35	F	136143	A	P	A	P	8	NIL	N	GAST	GAST	GAST	GAST	CNSI	A
3	PRABHU	45	M	156245	P	P	P	A	3	A/T	N	N	N	N	N	N	A
4	SHANKREMMMA	48	F	158508	P	P	A	A	18	NIL	N	GAST	GAST	GAST	CNSI	N	A
5	ADAVAYYA	75	M	159564	P	A	A	P	6	S	Y	N	N	N	N	N	A
6	YELLAPPA	32	M	1540	A	P	A	P	8	NIL	N	CNSI	CNSI	CNSI	CNSI	CNSI	A
7	SHARADA	40	F	18702	P	P	A	P	4	NIL	N	N	CNSI	N	N	CNSI	A
8	RAJSHEKAR	58	M	110815	P	A	P	A	6	NIL	N	N	N	N	N	CNSI	A
9	MALLANAGOUDA	55	M	117885	P	P	P	P	3	S	N	N	N	N	N	N	A
10	NINGANNA	24	M	1664	P	A	P	P	12	NIL	N	CNSI	CNSI	CNSI	CNSI	CNSI	A
11	LAXMAN	55	M	225432	P	P	A	A	12	NIL	N	N	N	N	N	N	A
12	UMA	40	F	228687	P	A	P	P	36	NIL	N	CNSI	N	N	N	N	A
13	SIVALEELA	40	F	196125	P	A	P	A	10	NIL	N	N	N	N	N	CNSI	A
14	BHAGIRATHI	61	F	190571	P	P	P	A	12	NIL	N	CNSI	N	N	N	CNSI	A
15	SAVANTARAWWA	75	F	115948	A	P	A	P	6	NIL	Y	CNSI	N	N	N	N	A
16	CHANDRAKANTH	45	M	62445	A	A	P	P	12	NIL	Y	CNSI	N	N	N	N	A
17	KAMALAKSHI	47	M	70154	A	P	A	P	3	NIL	N	CNSI	N	N	N	CNSI	A
18	MAYAWWA	48	M	77478	A	P	P	A	12	NIL	N	CNSI	CNSI	CNSI	CNSI	N	A
19	ANNAPURNA	55	F	78540	A	A	P	P	7	NIL	N	GAST	GAST	GAST	GAST	N	A
20	DEVRAJ	34	M	86873	A	P	P	A	3	NIL	N	GAST	GAST	GAST	GAST	N	A
21	BABURAY	23	M	98027	P	A	P	P	10	NIL	N	N	N	CNSI	N	CNSI	A
22	RENUKA	28	F	102629	P	A	A	P	12	NIL	N	N	N	N	N	CNSI	A
23	SAVITRI	31	F	159661	P	P	A	A	6	NIL	N	ACI	ACI	ACI	ACI	ACI	A
24	GURU	26	M	159513	P	A	P	A	3	NIL	N	ACI	ACI	ACI	ACI	ACI	A
25	AMBIKA	32	F	169414	A	A	P	P	12	NIL	N	CNSI	CNSI	CNSI	CNSI	CNSI	A
26	DYAVAPPA	55	M	271918	P	A	P	A	8	S	Y	CNSI	CNSI	CNSI	CNSI	CNSI	A
27	RAVIKUMAR	20	M	181367	P	A	A	A	12	NIL	N	CNSI	CNSI	CNSI	CNSI	CNSI	A
28	LACHUBAI	73	F	171864	P	A	P	A	6	NIL	Y	ACI	ACI	ACI	ACI	ACI	A
29	SHIVANAND	23	M	187530	P	A	P	P	12	NIL	N	N	N	CNSI	CNSI	CNSI	A
30	HUSSAINBI	45	F	195633	A	P	P	P	24	NIL	N	CNSI	N	N	N	N	A
31	LAXMI	45	F	200808	P	P	P	A	12	NIL	N	CNSI	CNSI	CNSI	N	CNSI	A
32	MODINSAB	27	M	205839	P	A	P	A	60	NIL	Y	CNSI	CNSI	CNSI	CNSI	CNSI	A
33	MAHADEVI	38	F	205931	A	A	P	P	6	NIL	N	N	N	N	CNSI	CNSI	A
34	PARAMESH	21	M	213485	P	P	P	P	4	NIL	N	CNSI	N	CNSI	CNSI	N	A
35	MAITRABAI	52	F	213481	P	A	P	P	8	NIL	Y	CNSI	CNSI	CNSI	CNSI	CNSI	A
36	SHEKHAR	44	M	34233	A	P	P	A	8	NIL	N	N	N	N	N	CNSI	A
37	BABUGOUDA	50	M	205286	A	A	P	P	12	A	N	GAST	GAST	CNSI	GAST	N	A
38	PINKUDEVI	33	F	173963	A	P	P	A	36	NIL	N	ACI	ACI	ACI	ACI	ACI	A
39	MARUTI	61	M	173967	A	P	P	P	12	NIL	N	GAST	GAST	GAST	GAST	N	A
40	AISHWARYA	21	F	208711	A	A	P	P	12	NIL	N	CNSI	CNSI	CNSI	CNSI	CNSI	A
41	ASHA	29	F	198306	A	P	P	P	24	NIL	N	N	N	N	N	CNSI	A
42	SHIVANAND	39	M	207022	P	A	P	P	4	NIL	N	CNSI	CNSI	CNSI	CNSI	CNSI	A
43	BHIMARAYA	36	M	229998	A	P	A	P	12	NIL	N	CNSI	CNSI	CNSI	N	CNSI	A
44	SAVITRI	60	F	227107	P	A	A	P	8	NIL	N	CNSI	CNSI	CNSI	CNSI	CNSI	A
45	SUSHMA	20	F	224295	A	P	P	P	12	NIL	N	GAST	GAST	GAST	GAST	N	A
46	MALLAPPA	25	M	233677	P	A	A	P	24	NIL	N	GAST	GAST	GAST	CNSI	N	A
47	BASAVARAJ	40	M	272658	P	A	P	P	24	NIL	N	CNSI	N	CNSI	CNSI	CNSI	A
48	SAROJA	60	F	226744	A	A	P	P	36	NIL	N	GAST	GAST	GAST	GAST	N	A
49	PARAMAWWA	32	F	239613	A	A	P	P	18	NIL	N	N	CNSI	N	N	N	A
50	SUSHILA	45	F	271359	A	P	P	P	18	NIL	N	GAST	GAST	GAST	GAST	CNSI	P
51	PRAVEEN	28	M	271982	A	A	P	P	8	NIL	N	CNSI	CNSI	CNSI	CNSI	CNSI	A
52	BABUGOUDA	20	M	181991	P	A	A	P	24	NIL	N	CNSI	N	N	N	N	A
53	KENCHAPPA	37	M	80038	P	P	P	A	12	NIL	N	N	N	N	N	N	A
54	MALLIKARJUN	35	M	283036	A	P	P	A	12	NIL	N	GAST	GAST	GAST	GAST	CNSI	P
55	SHIVAKUMAR	28	M	233673	P	A	P	P	10	NIL	N	GAST	GAST	GAST	GAST	N	A
56	SAVITA	34	F	283488	P	A	A	P	8	NIL	N	CNSI	CNSI	CNSI	CNSI	CNSI	A
57	AMOGI	42	M	283533	P	P	A	A	12	NIL	N	GAST	GAST	GAST	GAST	CNSI	P
58	HANAMANTH	34	M	89947	P	A	P	A	36	NIL	N	N	N	N	N	N	A



59	SAVITRI	60	F	305401	P	P	P	A	60	NIL	N	CNSI	CNSI	CNSI	CNSI	CNSI	A
60	BABU	28	M	286997	P	P	A	A	10	NIL	N	GAST	GAST	GAST	GAST	N	A
61	RESHMA	41	F	339970	P	P	P	A	24	NIL	N	CNSI	CNSI	CNSI	CNSI	CNSI	A
62	SHAHJAHAN	52	M	333474	P	P	P	A	24	NIL	N	CNSI	CNSI	CNSI	CNSI	CNSI	A
63	RAVI	42	M	340596	P	P	A	A	12	NIL	N	CNSI	N	N	N	N	A
64	SHETTAVVA	40	F	340582	P	A	P	A	24	NIL	N	N	CNSI	CNSI	N	N	A
65	HAZARTBEE	87	F	313053	P	A	P	A	8	NIL	N	CNSI	CNSI	CNSI	CNSI	CNSI	A
66	SANJEEVKUMAR	34	M	360396	A	P	P	P	24	NIL	N	CNSI	CNSI	CNSI	CNSI	CNSI	A
67	SIDDU PARVATI	25	M	362048	P	A	P	A	12	NIL	N	CNSI	CNSI	CNSI	CNSI	CNSI	A
68	DURGAMMA	50	F	181719	P	P	A	A	12	NIL	Y	N	N	N	N	CNSI	A
69	UDAY	40	M	181087	P	P	A	P	8	NIL	N	CNSI	CNSI	CNSI	CNSI	CNSI	A
70	LALITHA PATIL	35	F	180552	P	A	P	P	12	NIL	N	N	N	N	N	CNSI	A
71	PAVADIBASAWESHWAR	30	M	180536	P	P	A	A	6	NIL	N	N	N	N	N	CNSI	A
72	RENUKA	45	F	180560	A	P	P	A	4	NIL	N	CNSI	CNSI	CNSI	CNSI	CNSI	A

## KEY TO MASTER CHART

SI No – Serial Number

Age – Age in Years

OP No – Out Patient Number

EP – Epigastric Pain

EB – Epigastric Burning

PPF – Post Prandial Fullness

ES – Early Satiety

Duration – Duration in Months

Habits – A: Alcoholic

S: Smoking

T: Tobacco Chewing

CO Mob – Co Morbidities

MNSI – Mild Non-specific Inflammation

CNSI – Chronic Non-Specific Inflammation

GAST – Gastritis

ACI – Acute on Chronic Inflammation

N - Normal

H P – Helicobacter Pylori

A – Absent

P – Present

## ANNEXURE – V









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TO STUDY THE SPECTRUM OF HISTOPATHOLOGICAL CHANGES IN ENDOSCOPIC GASTRODUODENAL BIOPSY IN PATIENTS WITH FUNCTIONAL DYSPEPSIA By Dr Prajwal P S  
INTRODUCTION

The term dyspepsia is used for a spectrum of symptoms localized by the patient to the epigastric region (between the navel and the xiphoid process) and the flanks (1).