"HISTOPATHOLOGICAL SPECTRUM OF UPPER GASTROINTESTINAL TRACT LESIONS IN PATIENTS UNDERGOING UPPER GASTROINTESTINAL ENDOSCOPIC BIOPSIES"

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LIST OF ABBREVIATIONS USED

Abbreviation	-	**Expansion**
AIDS	-	Acquired immunodeficiency syndrome
CMV	-	Cytomegalovirus
DPAS	-	Diastase resistant Periodic acid-Schiff
EBV	-	Epstein Barr virus
GEJ	-	Gastroesophageal junction
GERD	-	Gastroesophageal reflux disease
GIST	-	Gastrointestinal stromal tumor
H & E	-	Hematoxylin and eosin
H. pylori	-	Helicobacter pylori
IHC	-	Immunohistochemistry
MALT	-	Mucosa associate lymphoid tissue
PAS	-	Periodic acid-Schiff
PCR DNA	-	Polymerase chain reaction deoxyribonucleic acid
SCC	-	Squamous cell carcinoma
WHO	-	World health organization

ABSTRACT

Title: “Histopathological spectrum of upper gastrointestinal tract lesions in patients undergoing upper gastrointestinal endoscopic biopsies”

Background & objectives:

Tissue sampling of the gastrointestinal tract during endoscopy is the mainstay of many gastrointestinal diagnoses. Histopathological examination of the endoscopic biopsies obtained from the upper gastrointestinal tract helps in providing an accurate diagnosis. The main objective is to study the histopathological spectrum of upper gastrointestinal endoscopic biopsies and correlate it with endoscopic findings of the same.

Methods:

An observational study was conducted on 135 upper gastrointestinal endoscopic biopsies received in the histopathology section, KLE’S Dr. Prabhakar Kore Hospital from 1st January 2023 to 31st December 2023 for histopathological evaluation and their correlation with endoscopic findings.

Results:

The age of patients varied from 10-85 years with a male to female ratio of 1.81:1. The most common site for biopsy was oesophagus (36.29%) followed by duodenum (28.9%) and stomach (23.70%). Out of 135 upper gastrointestinal endoscopic biopsies on histopathology 54 cases (40.75%) were neoplastic and 80 cases (59.25%) were non-neoplastic lesions. The most common lesion as well as malignancy present in oesophageal biopsies was squamous cell carcinoma of oesophagus (55%). The most common lesion present in gastric biopsies was chronic

gastritis (65.62%) followed by gastric adenocarcinoma (18.76%) which was the most common malignancy. The most common lesion from the gastroesophageal junction biopsies was oesophageal adenocarcinoma (30%). The most common lesion in duodenal biopsies was chronic duodenitis (79.4%). Endoscopy findings correlated with histopathology findings in 96.5% of oesophageal malignancies, 55.5% of stomach malignancies, 75% of gastroesophageal malignancies, and 33.3% of duodenal malignancies. Overall endoscopy and histopathology findings correlation were present in 85.7% of oesophageal biopsies, 78.1% of gastric biopsies, 70% of gastroesophageal junction biopsies, and 84% of duodenal biopsies. Endoscopy correlated with histopathology in 83.7% of overall cases. ($p < 0.0001$).

Interpretation and conclusion:

Endoscopy findings should be complemented with histopathological examination of biopsy to provide definitive diagnosis and appropriate management in patient care.

Keywords: Upper gastrointestinal tract; Endoscopy; Biopsy; Histopathology; Malignancy

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

LIST OF GRAPHS

INTRODUCTION

Tissue sampling of the gastrointestinal tract during endoscopy is the mainstay of many gastrointestinal diagnoses.[1] The American Society for Gastrointestinal Endoscopy has prepared a protocol for using upper gastrointestinal endoscopy. Upper gastrointestinal endoscopy is performed in patients with complaints of dysphagia, dyspepsia, significant pain in the upper abdomen, odynophagia, recurrent vomiting, and palpable epigastric mass. Upper gastrointestinal endoscopy is used for detecting lesions and obtaining a biopsy from the lesional site; apart from that, it is also used as a screening tool where gastric cancer cases are higher and for therapeutic purposes.[2] Endoscopy findings, along with a histopathological examination of the endoscopic biopsies obtained from the upper gastrointestinal lesions from the oesophagus, stomach, and up to the second part of the duodenum, help in providing an accurate diagnosis of the lesions which will aid the clinician in providing appropriate patient care.[3]

Upper gastrointestinal tract disorders are classified into benign, pre-malignant, and malignant lesions. A wide range of these disorders are the main contributors to long-term illness and death.[2,3] According to GLOBOCAN 2020, gastric cancer is the fifth most common cancer and oesophageal cancer is the eighth most common cancer globally. In terms of global mortality, gastric cancer is the fourth most common cancer whereas oesophageal cancer is the sixth most common cancer.[4,5] Benign and malignant lesions do show similar clinical complaints; so, most of the time, malignancies are detected in late stages where it is difficult to treat patients. Hence, endoscopic observations along with histopathological diagnosis of the lesion have become a necessity.[3]

WHO has classified oesophageal tumors into benign epithelial tumors and precursors such as squamous papilloma, squamous dysplasia and malignant epithelial tumors such as squamous cell carcinoma, adenocarcinoma and adenosquamous carcinoma.[6] Oesophageal SCC is common worldwide and is associated with alcohol drinking and tobacco smoking, whereas adenocarcinoma is on the rise in Western countries. Gastroesophageal reflux disease and Barrett's oesophagus are precursors for adenocarcinoma. Gastritis can be caused by various etiological factors, like infection by H. pylori organisms, autoimmunity, granulomatous lesions, and radiation.[7] WHO has classified gastric tumors into benign epithelial tumors and precursors such as glandular dysplasia, intestinal-type adenoma and malignant epithelial types such as tubular adenocarcinoma, papillary adenocarcinoma, signet ring cell carcinoma, squamous cell carcinoma, and adenosquamous carcinoma.[6] Most of the gastric malignancies are adenocarcinomas on histopathology and are classified into intestinal and diffuse types by Lauren.[7] In duodenal lesions celiac disease is common among causes of malabsorption syndrome which shows shortening of villi and increased lymphocytes in lamina propria.[8] Chronic non-specific duodenitis is a commonly observed lesion in adults. There are various infections like giardia, shigella, and microorganisms affecting immunocompromised patients which can be detected on duodenal biopsy.[7]

AIMS AND OBJECTIVES

Primary objective:

To study the histopathological spectrum of upper gastrointestinal endoscopic biopsies and correlate with endoscopic findings of the same.

Secondary objective:

To know the distribution of various neoplastic and non-neoplastic lesions according to age, sex, and site with brief clinical data.

REVIEW OF LITERATURE

History of endoscopy:

Desormeaux suggested in 1853 that the required parts for making an endoscope are a light source and an optical lens. Kussmaul passed a rigid tube in 1868 as an endoscope to see the upper gastrointestinal tract. Johann Mikulicz in 1881 made better instruments which had 30 degree field of mirrors and did the first upper gastrointestinal biopsy. Henry Elsner made an instrument with both bendable and stiff parts which was known as the first flexible endoscope. Then, in 1930, the Wolf Schindler gastroscope with a flexible angle of 30 to 34 degrees and a suction channel of the lens was made. Basil Hirschowitz made a flexible gastroscope in 1957, depending on Hopkin's fiberoptic principles which was used in his endoscope in 1954.[2] Recent endoscopes are equipped with components including a light source, optical lenses with a video recording device, image processing, and most importantly an accessory channel that has 2 to 6 mm diameter and through which instruments for tissue collection can pass.[1] Revolutionary video endoscopy came into the picture in 1983.[2]The upper gastrointestinal flexible fiberoptic endoscope came into patient care in 1968.[3] Also, wireless capsule endoscopy has become a great innovation.[9]

Tissue processing:

In routine practice, biopsy material is fixed in formalin for tissue sampling, and Hematoxylin and eosin (H & E) staining is required for histopathological analysis. However, specific preparations need to be made when tissue collection is intended for molecular testing, cytology, or culture.[1]

Embryology of upper gastrointestinal tract:

The digestive system starts developing in the fourth week, then arises the foregut, midgut, and hindgut.[10] The foregut gives origin to the oesophagus. Initially, it is short but eventually becomes longer as the diaphragm goes down during the neck formation. There is one tracheoesophageal septum ventral part that gives rise to respiratory lineage while the dorsal part gives rise to the oesophagus. Usually by the seventh week oesophagus comes to the actual destination.[11]

The foregut also gives origin to the stomach. During the formation of the stomach by the seventh week there are two initial borders: ventral and dorsal, which later transform to become lesser curvature (right) and greater curvature (left), respectively, when 90-degree stomach rotation occurs. The second time, transverse (anterioposterior) rotation occurs. In the end, cardia/fundus goes upwards, and the pylorus is placed below.[11]

The foregut gives rise to first (superior) part and second part of duodenum. The midgut gives rise to the most proximal duodenum part. Meso-duodenum attaches to the posterior abdominal wall. After this, most of the duodenum becomes retroperitoneal.[11]

Anatomy of upper gastrointestinal tract:

The oesophagus is a fibromuscular tube structure measuring around 18–26 cm in length.[12] It connects the sixth cervical vertebrae to the eleventh thoracic vertebrae. The whole length of the oesophagus is divided into cervical, thoracic, and abdominal oesophagus. Oesophageal constrictions are present at the cricoid cartilage, the left major bronchus, and the aortic arch, and the diaphragm. The upper oesophageal

sphincter is made of the cricopharyngeus muscle and pharyngeal inferior constrictors. The lower oesophageal sphincter is not a real anatomical structure.[13]

The stomach is J-shaped which can expand to store food. The length of the stomach is approximately 25 cm and it has a storage capacity of 1500-2000 ml. The cardia, fundus, body, and pyloric antrum and canal are the four major parts of the stomach. There are some linear rugae in the mucosa of the stomach.[14]

The duodenum has four sections: the duodenal bulb or cap, the second vertical or descending portion, the third horizontal or transverse portion, and the fourth oblique or ascending portion. The duodenum starts at the end of the stomach pylorus, in the plane of the first lumbar vertebra.[15]

Histology of upper gastrointestinal tract:

Stratified squamous epithelium usually lines the oesophagus. The lamina propria consists of lymphoid follicles and connective tissue.[16] The submucosa of the oesophagus has very few submucosal glands compared to the other gastrointestinal tissues.[12] It does contain blood vessels, lymphatics, and a mesenchymal plexus. Muscularis propria has inside circular, and outside longitudinal muscle layers. The upper third of the oesophagus is made up of voluntary skeletal muscle, while the lower third is made up of involuntary smooth muscles. The shift from striated to the smooth muscle is slow, and there is a part of the middle third of the oesophagus that can have both muscles. Between two layers of muscularis propria, there is a myentric plexus.[16] Because the oesophagus lacks a serosal coating, infections, and cancers can spread through it.[17]

In the stomach, the fundus and body mucosa do have foveolae, or gastric pits, that occupy only 25% of the mucosal thickness; they usually have straight types of glands. In the glands, there are various cell types, including chief (zymogenic) cells having a basal nucleus, highly basophilic cytoplasm, and separated granules, which are basophilic. Parietal (acid-secreting) cells, which have a round nucleus in the middle, have highly acidophilic cytoplasm. Other types of cells are mucous neck cells, neuroendocrine cells, and stem cells. Pyloric mucosa does have gastric pits that occupy 50% of the mucosal thickness. Coiled glands are present which are mostly lined by mucus-secreting cells. Other layers are the submucosa, muscularis propria, and serosa.[16]

The duodenum has glandular mucosa which corresponds with the columnar mucosa of the small intestine which shows elongated villi and crypts which are shorter. Brunner gland occupies the submucosa and muscularis mucosa. Cell types in intestinal epithelium are enterocytes, goblet cells, paneath cells, neuroendocrine, stem cells, and intraepithelial lymphocytes.[16]

Figure 1: Oesophagus histology (H & E stain) (4X)

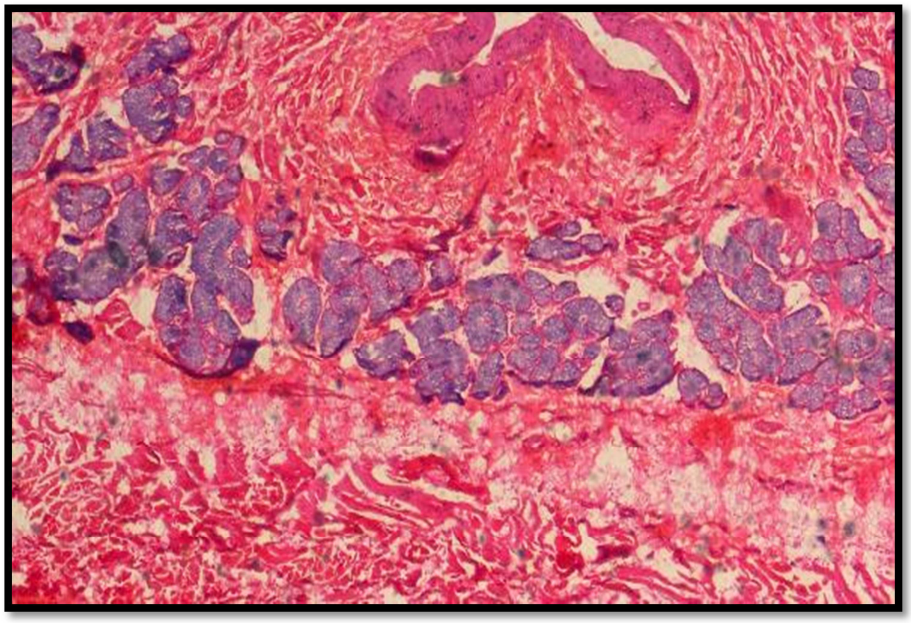


Figure 2: Gastric fundus and body histology (H & E stain) (4X)

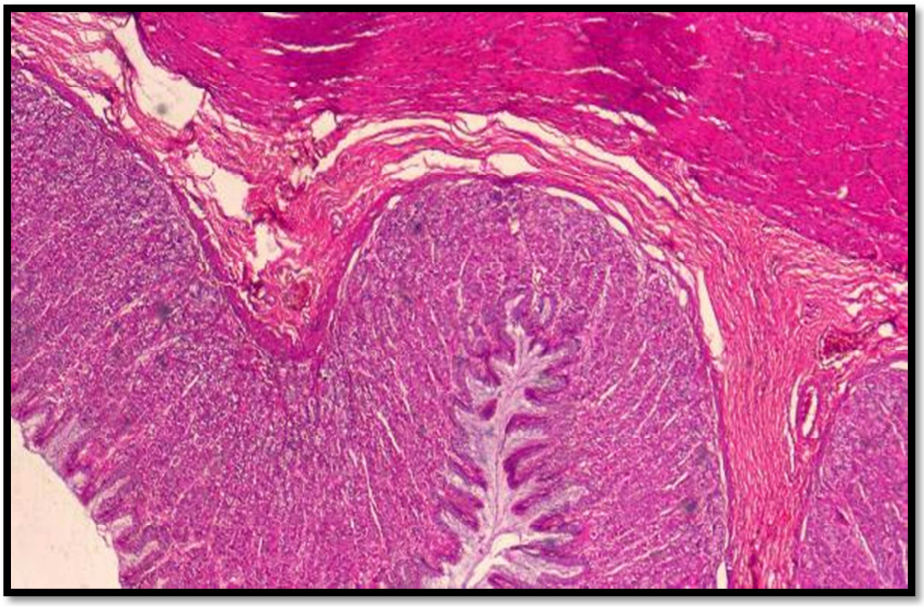


Figure 3: Gastric fundus and body histology (H & E stain) (10X)

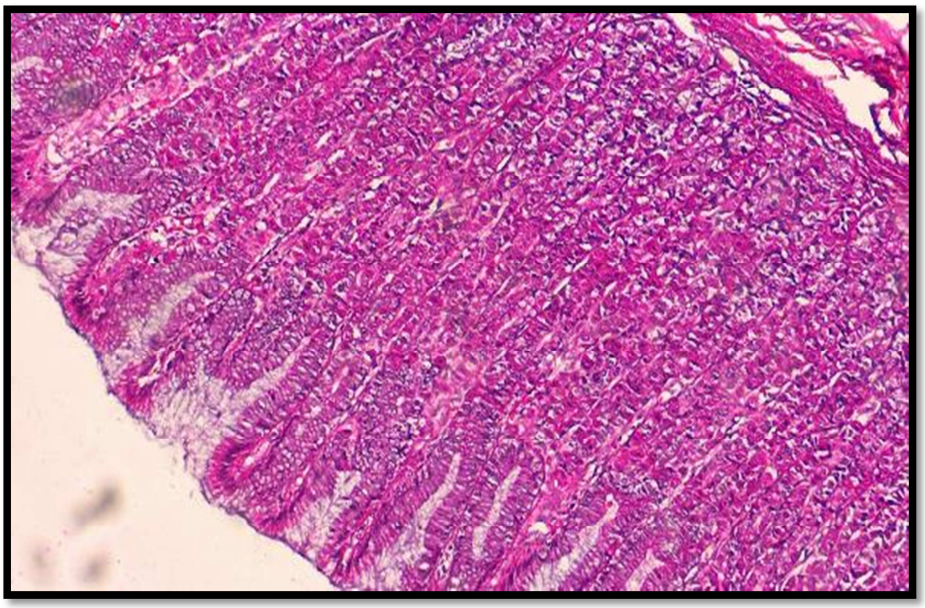


Figure 4: Gastric pyloric antrum histology (H & E stain) (10X)

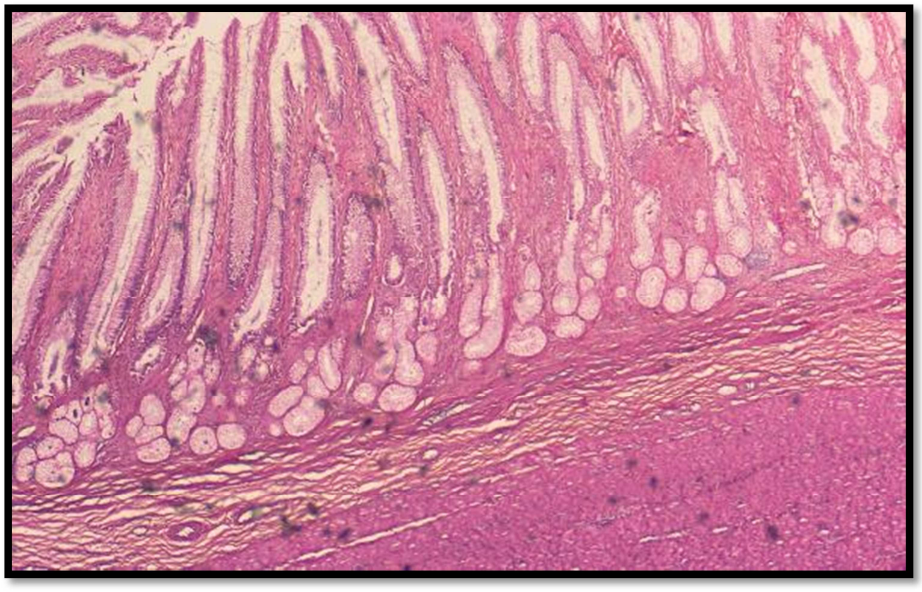
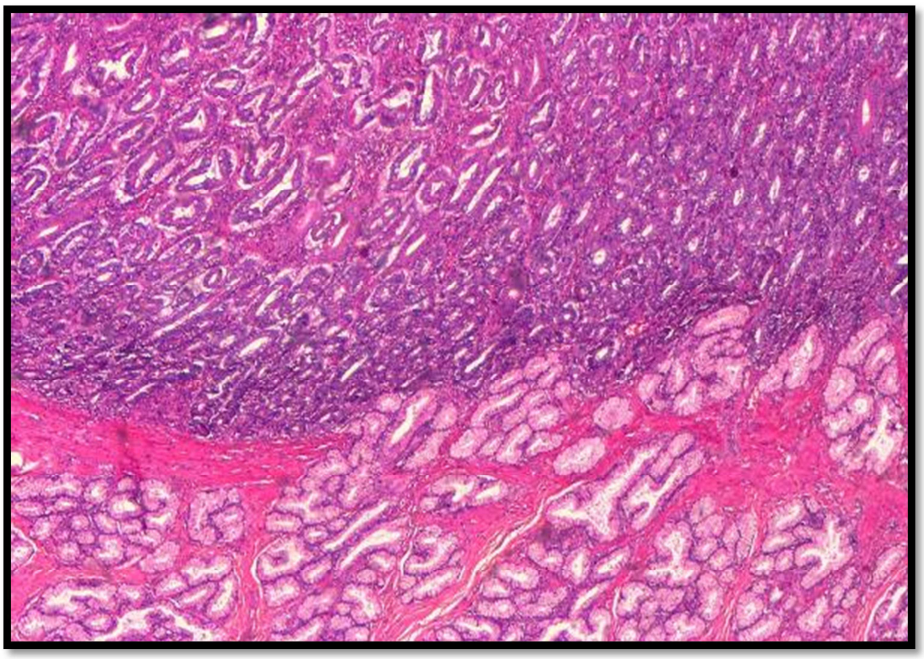


Figure 5: Duodenum histology (H & E stain) (10X)



Oesophageal lesions:

Infectious oesophagitis:

Various bacteria, fungi, viruses, and parasites can lead to oesophageal infections. Certain immunocompromised states and cancers, and treatments used for cancers, also predispose to infectious esophagitis. Candidal esophagitis presents as whitish plaques on the oesophageal mucosa on endoscopy. Yeast or pseudo-hyphae with invasion into the mucosa can be seen in histopathology. Herpes simplex esophagitis is presented as volcano-type or punched out ulcers in the lower oesophagus on endoscopy. Peripheral margin of ulcer is used for taking biopsy. Large epithelial cells with multiple nuclei and ground-glass cytoplasm and eosinophilic intranuclear inclusions are histopathological findings. The majority of cytomegalovirus (CMV) related ulcers are deep, straight, or elongated. Large, isolated

ulcers or erosions are noted in endoscopy. The biopsy reveals tissue injury and intracytoplasmic or intranuclear inclusions usually seen in the endothelium of capillaries or mesenchymal cells.[18]

Eosinophilic oesophagitis:

Eosinophilic oesophagitis is multifactorial inflammatory disease with genetic predisposition. It presents as a combination of deficient oesophageal mucosal barrier with an abnormal immune reaction mediated by Th2 interleukins. Up to four oesophageal biopsies from the upper, mid, and lower oesophagus are mandatory for diagnosis.

It can show oesophageal feline (ring-like appearance) or longitudinal furrows on endoscopy. For histopathology, there are major and minor criteria. One of the major criteria comprised more than 15 eosinophils per high power field in the lamina propria. Minor criteria show a basal segment with papillary hyperplasia, eosinophilic small-abscesses, eosinophil degranulation, desquamation, and fibrosis in the lamina propria.[19]

Gastroesophageal reflux disease (GERD):

Gastroesophageal reflux disease is a chronic mucosal irritation caused by gastric acid coming up into the lower oesophagus. The most important cause is short-term lower oesophageal sphincter relaxation. There is also raised abdominal pressure by coughing. Other etiological factors include pregnancy, alcohol, cigar use, and obesity.[7]

Of the 24 hours in a day, the oesophageal mucosa is normally exposed to a pH <4 for less than an hour which can be measured by 24 hr pH studies. If the length of the cardiac mucosa increases, the amount of reflux also increases. Clinically, patients can have no symptoms; typical symptoms are heartburn and regurgitation. Endoscopic changes in general are erosions or ulcerations in the squamous mucosa which are not specific. Cardiac mucosa, oxyntic cardiac mucosa, and intestinal metaplasia constitute flattened glandular mucosa that is placed in the gastroesophageal junction on endoscopy.[20]

Table 1: Endoscopy and histopathology findings of GERD

Reflux	Endoscopy	Histopathology
Mild	Normal	Cardiac mucosa , oxyntic cardiac mucosa
Moderate	Abnormal	Cardiac mucosa + oxyntic cardiac mucosa length < 2 cm
Severe	Abnormal	Cardiac mucosa + oxyntic cardiac mucosa length > 2 cm

Table 2: Scoring system of GERD based on histopathology findings

Histopathological findings	**Criteria**	**Score**
Basal layer of cells hyperplasia	<15%	0
	15-25%	1
	>25%	2
Stromal papillae elongation	<33%	0
	33-66%	1
	>66%	2
Intraepithelial T lymphocytes	5	0
	6-10	1
	>10	2
Intraepithelial Eosinophils/high power field	5	0
	6-10	1
	>1	2
Metaplasia	Gastric	2
	Intestinal	4
Maximum score	-	12

The above table shows scoring system of GERD and helps in better diagnosis as it suggests the score more than 3 do signify GERD. [21]

Barret's oesophagus:

American College of Gastroenterology (2016) defines Barret's oesophagus as extension of salmon or red-coloured mucosa into the lower oesophagus nearest to the gastroesophageal junction for more than 1 cm, and a biopsy proved intestinal metaplasia having with goblet cells.[22]

British Society of Gastroenterology (2014) defines Barret's oesophagus as goblet cells may or may not be present in the columnar epithelium, but the columnar epithelium can extend more than 1 cm nearest to the gastroesophageal junction.[23]

If metaplastic columnar epithelium length is more than 3 cm it is considered a long segment and if the length is less than 3 cm it is considered a short segment, and if less than 1 cm it is considered an ultrashort segment. Some of the predisposing factors for the condition are old age, obesity, and cigar usage. [22]

Endoscopy of Barret's oesophagus:

There is an extension of the red velvety or salmon-coloured mucosa near the gastroesophageal junction. Approximately 16 endoscopic biopsies are taken from the suspicious red or salmon coloured mucosa to detect true positive Barret's oesophagus cases.[7,24]**Histopathology of Barret's oesophagus:**

Columnar epithelium having intestinal metaplasia mostly along with goblet cells can present and replace the squamous epithelium of the oesophagus. Goblet cells have a wineglass appearance, are randomly distributed, have cytoplasmic mucin vacuoles that are pale blue and can indent the nucleus of cells. This can be confused with pseudo goblet cells which are columnar, barrel-shaped, and have surface mucin

vacuoles that give the cytoplasm a blueish tinge on H & E stain. Acidic mucin (sialomucin) in true goblet cells stains purplish blue with alcian blue at pH 2.5; it is sometimes used for differentiating pseudo-goblet cells, which generally do not stain because they have neutral mucin.[24,25]

In low-grade dysplasia, only the lower half of the epithelium is affected, the crypts are bending, and there is minimal to mild hyperchromasia of the cells. In high-grade dysplasia, there is complex cribriform patterning of crypts. Cells show a high nuclear-to-cytoplasmic ratio and hyperchromatism. If dysplastic changes with no inflammation are there and if it is challenging to differentiate from regeneration, a diagnosis of indefinite dysplasia is given.[26]

Oesophageal tumors

WHO classification of oesophageal tumors.[6]

Epithelial tumors of oesophagus:

- **Benign tumors and precursors**

- Squamous cell papilloma
- Squamous papillomatosis
- Oesophageal glandular dysplasia, (intraepithelial neoplasia) low grade and high grade
- Oesophageal squamous intraepithelial neoplasia (dysplasia) low grade and high grade

- **Malignant tumors**

- Squamous cell carcinoma
    - Verrucous squamous cell carcinoma
    - Spindle cell squamous cell carcinoma
    - Basaloid squamous cell carcinoma
- Adenocarcinoma
- Adenosquamous carcinoma
- Adenoid cystic carcinoma
- Mucoepidermoid carcinoma
- Undifferentiated carcinoma
- Lymphoepithelioma like carcinoma
- Neuroendocrine tumor grade I,II,III
- Neuroendocrine carcinoma large or small cell
- Mixed neuroendocrine-non neuroendocrine neoplasm

- Combined small cell adenocarcinoma
- Combined small cell squamous cell carcinoma

Squamous papilloma:

Squamous papilloma finding is incidental to upper gastrointestinal endoscopy. Human papilloma virus, alcohol drinking, smoking tobacco, and injuries are risk factors for papilloma. This tumor is usually small but can show a verrucous appearance on endoscopy, which is confused with verrucous carcinoma. Typically, it is observed in the lowest part of the oesophagus. Histopathological features of squamous papilloma show a papillary-type arrangement of cells with a real fibrovascular core is there. Squamous cells with perinuclear halo known as koilocytes are seen in multiple layers of squamous epithelium.[27]

Squamous dysplasia:

According to the WHO, squamous dysplasia is classified as low-grade dysplasia when the lower half of the epithelium has mild or moderate atypia and high dysplasia when the upper half of the epithelium has severe atypia.[27]

Squamous cell carcinoma (SCC):

According to GLOBOCAN 2020, oesophageal cancer is the eighth most common cancer worldwide. In terms of global mortality, it ranks sixth. Most of the cases are encountered in East Asia, the south and east parts of Africa, and south-central Asia. Some of the less-income countries like Iran, China, and Brazil show quite high cases of SCC.[5]

The majority of cases of SCC occur in the middle third of the oesophagus. Known liable factors for this cancer are alcohol drinking, smoking, bad food, plummer-Vinson syndrome, and oesophageal achalasia. SCC is linked to alcohol drinking and cigarette smoking in developed nations. In underdeveloped countries, malnutrition, heat damage, opium, hydrocarbons, and tobacco use are linked with cancer.[7,28] In terms of genetics, TP53 alterations, along with NOTCH1, NOTCH3, EGFR, CDH1 mutations, and SOX2 amplification are linked to oesophageal dysplasia and SCC.[7,29]

Dysphagia is the chief complaint, mostly present in the majority of SCC cases; weight loss is usually associated with SCC. Endoscopy usually shows cauliflower mass, strictures, growth, polypoid lesions, or ulcerated lesions.7 Histopathology shows three grades. Malignant tumor cells cross the basement membrane in SCC.[27]

Table 3: Histopathology grades of SCC

Histopathology grades of SCC		
GRADE I	Well-differentiated	Tumor cells with mild nuclear pleomorphism, a large amount of eosinophilic cytoplasm, a low number of mitotic figures, and keratinization seen frequently
GRADE II	Moderately differentiated	Mixed features of grade I and III minimal keratinization
GRADE III	Poorly differentiated	Tumor cells with high-grade nuclear pleomorphism, arranged in nests or sheets. High numbers of mitotic figures, desmoplasia, and lympho-vascular invasion are also seen.

Verrucous carcinoma is a rare type that is seen in the lower oesophagus. Endoscopy reveals verrucous growth. Histopathology shows a well-differentiated SCC-type appearance with expanding margins.

Spindle SCC can appear as a polyp on endoscopy. On histopathology, tumor cells are squamous and spindle-type, with a cartilaginous or osseous stroma. Immunohistochemistry (IHC) is positive for vimentin in the spindle component, and cytokeratin and p40 are positive in the squamous component.

Basaloid SCC on histopathology shows tumor cells have hyperchromatic nuclei with nuclear palisading. Tumor cells are arranged in nests, cribriform, microcystic, or solid patterns. Comedo necrosis inside the nests can be seen.

Both adeno-squamous and mucoepidermoid oesophageal carcinomas have a bad prognosis as poorly differentiated SCC.[27]

Oesophageal adenocarcinoma:

Globally oesophageal adenocarcinoma comprises approximately 15% of oesophageal carcinoma. It is most commonly seen in high-income countries such as Australia, Canada, a few countries in Northern and Western Europe, and the United States and, the United Kingdom. GERD and Barret's oesophagus are precursor lesions for adenocarcinoma.[5]

TP53, EGFR, and CyclinD1 gene alterations are linked to adenocarcinoma of oesophagus.[7,28] For starting anti-HER2 drug treatment in this cancer, it is good to know the status of ERBR2 (HER2) which often undergoes amplification used as a prognostic marker.[28] Clinical features and endoscopy have similarities with SCC. On

histopathology, oesophageal adenocarcinoma can be seen as an intestinal type, diffuse type, or mixed type according to Lauren's classification.[7,29]

Fibrovascular polyp:

Fibrovascular polyps are like growths attached to the mucous membrane of the oesophagus by a stalk. They are most commonly present in the upper oesophagus. Histopathology shows a polypoidal structure lined by non-keratinized squamous epithelium, with blood vessels and adipocytes admixed with myxoid stroma.[30]

Non epithelial tumors:

Leiomyomas, granular cell tumors, and gastrointestinal stromal tumors are mesenchymal polyps, that can be seen situated in the sub-epithelium.[30,33]

Oesophageal leiomyoma:

Oesophageal leiomyomas are benign, non-epithelial tumors. It comprises more than 60% of all benign oesophageal tumors. Histopathology of oesophageal leiomyoma shows cross bundles of spindle cells with a large amount of eosinophilic cytoplasm. Desmin and alpha-smooth muscle actin are positive IHC markers for oesophageal leiomyomas

Granular cell tumor:

Oesophageal granular cell tumors arise from Schwann cells of neural origin. They are generally seen in the lower oesophagus. Tumor cells are large, polygonal, with eosinophilic to basophilic cytoplasm and round nuclei, arranged in sheets or nests. IHC is positive for S-100, Vimentin, and CD68 and negative for smooth muscle actin, Desmin, CK, CD117, and CD34 markers.[32]

Gastrointestinal stromal tumor (GIST):

Interstitial cells of Cajal give rise to Gastrointestinal stromal tumors which is mesenchymal tumor. The C-kit gene is usually altered. GISTs are mainly seen in the stomach; oesophageal GISTs are quite rare. In the lower oesophagus, they are most prevalent. Microscopically, spindle type with little granular cytoplasm, arranged in crossing bundles or epitheloid type can be seen. Mitotic activity is uncommon. IHC is positive for KIT (CD117), DOG1, CD34, smooth muscle actin (SMA), Desmin, and S100 protein markers.[33]

Lymphoma of oesophagus:

Primary oesophageal lymphoma is very rare, but it can be present as metastases from mediastinal lymph nodes or gastric lymphoma invasion. The gastrointestinal tract is commonly affected by extra-nodal sites in non-Hodgkin's lymphoma and is comprised mostly of mature B cells. IHC Markers like CD19, 20, 79a, and BCL 2 are positive, while CD5 and CD10 are negative.[34]

Gastric lesions:

Updated Sydney system of gastritis:

In this system, five variables are checked they are as: 1) Grade of gastritis 2) activity of gastritis 3) gastric atrophy 4) intestinal metaplasia 5) Helicobacter pylori (H. pylori) thickness. It also suggests techniques for getting an appropriate biopsy sample from not only the pyloric antrum and corpus but also from the incisura angularis.[35]

Gastritis is classified based on etiology into acute gastritis, chronic gastritis, special types (infectious, lymphocytic, fungal), granulomatous gastritis and reactive gastropathy.[36]

Acute gastritis:

Acute gastritis is usually caused by stress, pain relief drugs, shock, or an H. pylori infection. Histopathology shows neutrophilic inflammatory infiltrates along with a few mixed inflammatory infiltrates along with edematous mucosa and congested blood vessels.[37]

H. pylori gastritis:

Helicobacter pylori is a gram-negative curve shaped microorganism that can lead to chronic active or non-specific gastritis, dysplasia of the gastric epithelium, gastric and duodenal ulcers, atrophic gastritis, MALT lymphoma and gastric cancer, chronic duodenitis.[38]

Table 4: Diagnostic tests for H. pylori

Diagnostic tests for H. pylori	
Invasive tests	**Non-invasive tests**
Endoscopy/ biopsy	Urea breath test
Rapid urease test	Stool antigen test
Culture and PCR-DNA	Serological and PCR-DNA

Familial transmissions are quite common in H. pylori infections. H. pylori infections are transmitted via fecal-oral route. Developing nations do have high

chances of H. pylori infection mostly in backward classes. Most of the H. pylori infection cases are encountered in Asia, Africa, Mexico, and Latin America.[7,39]

H. pylori organisms usually tend to involve the antrum first by attaching to gastric mucins (TFF1) at an appropriate pH. There are four main host-damaging elements: flagella, urease, adhesions, Cag A (cytotoxin-associated gene A) and Vac A (vacuolating cytotoxin A). This organism is seen attaching to the mucous neck cells of the surface gastric mucosa.[40]

When searching for a location, an antrum biopsy is advised, as the organisms are generally present there. If atrophy or intestinal metaplasia are suspected, a corpus biopsy is also done so organisms do not miss out. Endoscopy can show erosions, easily breaking gastric mucosa, and mucosal erythema, nodularity but they are not specific findings for diagnosing H. pylori, as any type of gastritis can have these findings.[38]

On histopathology, chronic gastritis shows infiltration of the lamina propria by mixed inflammatory cells like lymphocytes, plasma cells, macrophages and a few eosinophils.[7]

A study done by Hirachand et al.[41] and Ganga H et al.[42] revealed that in chronic active gastritis with H. pylori infection, there was the presence of mostly neutrophils and a few mixed inflammatory infiltrates in the lamina propria.

H. pylori organisms can be stained by routine H & E. Special stains are not in routine use, but for good visualization, they can be used. There are a few silver-based stains like Warthin-Starry, Steiner, El-Zimaity dual stain, and Genta, and non-silver-based stains like Diff-Quik, Giemsa, and Leung Alcian Yellow.[43]

Granulomatous gastritis:

Granulomas are seen commonly in the antrum. Tuberculosis, syphilis, Crohn's disease, and sarcoidosis cause immune granulomas. There are granulomas caused by foreign bodies or malignancies. Microscopically, granulomas show central necrosis or can be without necrosis; epitheloid cells, multinucleated giant cells surrounded by lymphocytic infiltration, can have plasma cells also.[44]

Lymphocytic gastritis:

The lesion most likely matches a varioliform gastritis case that was originally documented in 1945. It shows thick gastric folds on endoscopic examination. The key characteristic here, on histopathology as defined by recent terminology, is the presence of a minimum of 25 intraepithelial lymphocytes (IEL) per 100 gastric pits and outer epithelial cells. Microscopically, it can show halo surrounds hyperchromatic nuclei in the cells.[45] It is associated with celiac disease, which shows antral predominance or diffuse involvement, and H. pylori infection, which shows corpus predominance.[46]

Reactive (chemical) gastropathy:

It is commonly induced by bile and pancreatic secretions, but it can also be caused by nonsteroidal anti-inflammatory drugs, chemotherapeutics, and alcohol.[47] Endoscopy shows erythema or friable mucosa, corkscrew appearance of glands, and a minimum of two times the elongation of gastric pits. Dixon's criteria for histopathology variables that are checked are hyperplasia of the foveola, lamina propria fibrosis, vasodilatation, and congestion of capillaries for the diagnosis of reactive gastropathy.[48]

Gastric tumors:

Who classification of stomach tumors.[6]

Epithelial tumors of stomach

- **Benign tumors**

- Glandular intraepithelial neoplasia low grade
- Glandular intraepithelial neoplasia high grade
- Serrated dysplasia, low grade
- Serrated dysplasia, high grade
    - Intestinal type dysplasia
    - Foveolar type dysplasia
    - Gastric pit dysplasia
- Intestinal-type adenoma low grade
- Intestinal-type adenoma high grade
    - Sporadic intestinal-type adenoma
    - Syndromic intestinal-type adenoma
- Adenomatous polyp, low grade dysplasia
- Adenomatous polyp, high grade dysplasia

- **Malignant tumors**

- Adenocarcinoma
    - Tubular adenocarcinoma
    - Parietal cell carcinoma
    - Papillary adenocarcinoma
    - Mixed subtypes adenocarcinoma
    - Micropapillary carcinoma
    - Mucinous adenocarcinoma

  - Signet ring cell carcinoma
  - Poorly cohesive carcinoma
  - Medullary carcinoma with lymphoid stroma
  - Hepatoid adenocarcinoma
  - Paneath cell carcinoma
- Squamous cell carcinoma
- Adenosquamous carcinoma
- Undifferentiated carcinoma
  - Large cell carcinoma with rhabdoid phenotype
  - Pleomorphic carcinoma
  - Sarcomatoid carcinoma
- Gastroblastoma
- Neuroendocrine tumor grade
- Neuroendocrine tumor grade I,II,III
- Somatostatinoma
- Enterochromaffin cell carcinoid
- Enterochromaffin cell malignancy
- Neuroendocrine carcinoma Large and small cell
- Mixed neuroendocrine-non neuroendocrine neoplasm

Gastric polyps:

There are three known epithelial polyps named as fundic gland polyps, hyperplastic polyps, and adenomatous polyps. fundic gland polyps are the most commonly noticed gastric polyps. Histopathology shows dilatation of the fundic glands. On endoscopy, these polyps have a flat surface and a wide base. They are

usually linked with familial adenosis polyposis. When it comes to adenomatous polyps, most of the population affected is from Japan or China. Adenomatous polyps with genetic alterations can lead to dysplasia, which eventually can be transformed into gastric malignancy.[49]

In fundic gland polyps, the adenomatous polyposis coli/beta-catenin pathway alteration can be seen. In occasional fundic gland polyps, CTNNB1 gene mutations are seen; they are also related to wild-type APC. Hyperplastic polyps are mostly present flat or with stalk lesions. On histopathology, corkscrew appearance of glands are situated into the depth of the sub-epithelium. These polyps show TP53 gene alteration or overexpression of TP53.[28]

Gastric adenoma:

APC, KRAS, ERBB2, ARID2, and microsatellite instability gene alterations are linked with intestinal types of adenomas.[50,51] Foveolar-type adenomas are linked to familial adenosis polyposis; occasionally, APC and KRAS gene alterations are seen.[50] Pyloric gland adenomas are linked to H. pylori infection. Pyloric gland adenomas can cause alterations of the GNAS, APC, and/or KRAS genes.[52]

Sequential events leading to gastric malignancy:

Gastritis tends to undergo metaplasia. It is divided into two types: Pseudopyloric type and intestinal type, which further divides into a complete type where a small intestinal mucosa is present, and an incomplete type where a colon mucosa is present. It is also known that it undergoes dysplastic change which is linked to TP53 gene deletion or APC gene alteration. Ultimately malignant transformation can occur.[28]

Two types of molecular classifications of gastric cancer.[53,54,55]			
Asian cancer research group			
Microsatellite instability	Microsatellite stable Epithelial-mesenchymal transition	Microsatellite stable TP53 active	Microsatellite stable TP53 inactive
The cancer genome atlas research network			
EBV-positive	Microsatellite stable	Microsatellite instable	Chromosomally instable

GIST, leiomyoma, and granular cell tumors are mesenchymal polyps that can be seen situated in the sub-epithelium.[49]

Gastric neuroendocrine tumors arises from enterochromaffin cells. The common location for this tumor is the corpus and fundus, where more than 90% of the tumors are seen. According to Recent Updates in 5th Edition of the World Health Organization "Blue Book" Neuroendocrine tumors are classified as only 2 types well and poorly differentiated.[28]

Gastric cancer:

According to GLOBOCAN 2020, gastric cancer is the fifth most common cancer observed and fourth most common cancer in terms of global mortality. Most of the cases of gastric cancer are seen in Eastern Asia, central and Eastern Europe, and South America. Japan is a country where cases are on par with the Western countries.[4]

Incisura angularis is the prevalent site for adenocarcinoma of the stomach. Usually, cardia-gastric cancer is seen in the gastroesophageal junction and non-cardia gastric cancer is seen in the distal stomach. Non cardia gastric cancer is more common overall. There are certain causes like GERD and obesity which are linked with the occurrence of gastric cardia cancer, while H. pylori infection, backward class, preserved food, and salty food which are linked with the occurrence of non-cardia gastric cancer. H. pylori gastritis ulcers can lead to metaplasia which can lead to dysplasia which lead to gastric malignancies. Other causes like intrinsic factor deficiency, and blood group A are also linked with the occurrence of gastric cancer.[56]

On endoscopy, adenocarcinoma presents as an ulcero-proliferative growth type lesion; sometimes carcinoma can present as longstanding ulcers or diffuse infiltration causing thickening of mucosa called as linitis plastica. For a better diagnosis in histopathology, if possibly seven biopsies are taken from the margin or base of the ulcerated lesion or growth.[57]

Intestinal type adenocarcinoma shows APC gene loss and beta-catenin gene gain whereas for diffuse gastric carcinomas there is CDH1 gene loss (E Cadherin loss) can be there.[7]

Lauren classification:

1. The intestinal type is composed of neoplastic glands with cells showing surface mucin vacuoles.
2. The diffuse type shows malignant cells are dispersed. In signet ring-type malignancy, cells show abundant mucin, which displaces the nucleus to the

periphery. On H & E, these cells have a clear cytoplasm with a peripheral nucleus.[7]

Mucosa associated lymphoid tissue (MALT) lymphoma:

MALT lymphoma on endoscopy presents as nodules or polypoidal mass, thick gastric mucosa, oedema, erosions, and ulcers. Due to the focal presence of MALT lymphoma on endoscopy, it is suggested to take a biopsy from different sites of lesion where focal area is there. Histopathology reveals B-cell-derived lymphocytes and centrocytes.[7,58]

Duodenal lesions:

Celiac disease:

Celiac disease is an immune-mediated disease that produces auto-antibodies against tissue transglutaminase and affects genetically predisposed individuals with HLA DQ8 and DQ2 phenotypes. Gluten diets like wheat, barley, or oats cause this disease. Western and Northern European Europe countries show more celiac disease cases. For celiac disease, the most common age groups affected in children are those around the age of 10 years. Adults are usually affected between 30 to 60 years of age group.[7,59]

Scalloped folds of the duodenum or a mosaic pattern of vessels leading to a fissured appearance of the duodenum can be found on endoscopy, which can suggest celiac disease, but they are not specific findings.[60]

There are Marsh-modified systems that are useful for celiac disease evaluation. Endoscopic biopsies are taken from the second and third parts of the

duodenum. A minimum of 4 biopsies are taken, 2 from the second and 2 from the third part of the duodenum. On histopathology, intraepithelial T lymphocytes (IEL) of more than 30/100 enterocytes suggest pathogenic lymphocytosis, along with eosinophils, plasma cells, or mast cells, hyperplasia of crypts, and villous atrophy. Celiac disease patients are prone for malignancy of T-cell lymphoma [61]

Duodenitis:

Endoscopic findings can show erythematous, nodular, or ulcerated mucosa.[1] On histopathology, chronic inflammatory infiltrates are usually lymphoplasmacytic in the subepithelial lamina propria for chronic non-specific duodenitis or neutrophils for chronic active duodenitis, more than which is usually present, along with hyperplasia of Brunner's glands and distortion of villi. In eosinophilic duodenitis, eosinophil influx into the lamina propria is seen, along with a few lymphoplasmacytic infiltrates.[62]

Infections of duodenum :

Villous surface giardia lamblia can be found. Trophozoites of giardia lamblia can penetrate the mucosa. Histological characteristics of AIDS enteropathy can include normal villi or villous atrophy; CD4 cells decrease while CD8 cells increase.

Histological characteristics of Whipple's illness there can be hyperplastic villi of duodenum and dense foamy macrophages in the lamina propria. These foamy macrophage granules are PAS-positive.[62]

Duodenal biopsy findings of infections in immunocompromised patients:

Some findings that are not specific but are usually seen in the histopathology of these infections. They are villous shortening, hyperplastic crypts, mostly eosinophilic inflammatory cell infiltrates, along with plasma cells in the lamina propria.

Microsporidia are usually present in the enterocytes, whereas cryptosporidiosis is present on the surface, with the cryptal epithelium having varying sizes.

Isosporiasis shows dysplastic epithelium with nuclear polarity loss. These organisms have banana-shaped elongation present in the below-nucleus cytoplasm, which looks like a T-shaped structure organized horizontally in the enterocytes. It usually does not stain. Cryptosporidiosis is present on the surface, and the cryptal epithelium has varying sizes.

Mycobacterium shows curved, acid-fast diasterase PAS-positive macrophages stuffed with DPAS-positive, acid-fast, curved bacilli in the foamy macrophages.

CMV has basophilic or eosinophilic intranuclear inclusion bodies, giving the appearance of an owl eye seen in the capillary endothelial or mesenchymal cells. Immunoperoxidase stain is useful to detect CMV-infected cells.

Cryptococcosis has a short neck; apart from H & E, DPAS or methamine silver stains are used for detecting cryptococcosis neoformans.

Leishmaniasis shows involvement of reticuloendothelial system cells; a 100x round or oval amastigote is there. This amastigote has a bar kinetoplast and nucleus.[62,63]

Previous studies on histopathological spectrum of upper gastrointestinal tract lesions:

A study done by Krishnappa Rasmi et al.[64] (2013) in Karnataka showed that in terms of the distribution of upper GI endoscopic biopsies there were total 100 endoscopic biopsies, of which 68 gastric biopsies, 25 oesophageal biopsies, and 7 duodenal biopsies with males affected 2 times more than females. Most of the cases were from 5th decade. In gastric biopsies majority were from pylorus showed non-neoplastic lesion chronic gastritis was most common followed by stomach adenocarcinoma malignancy. In oesophageal biopsies majority were from middle oesophagus showed oesophagitis was most common non-neoplastic lesion followed by squamous cell carcinoma. There was 91% and 74% correlation was established between endoscopy and histopathology diagnosis for oesophageal and gastric malignancies respectively. Overall 78% cases matched endoscopy and histopathology diagnosis.

A study done by Begum et al.[65](2024) in Telangana showed that in terms of distribution of upper gastrointestinal endoscopic biopsies there were total 177 endoscopic biopsies of which 90 gastric biopsies, 21 oesophageal biopsies and 66 duodenal biopsies with male: female ratio of 1.39:1. Most of the cases were from 6th decade. Here in gastric biopsies gastritis and gastric polyps are commonly observed lesions. In oesophagus, oesophagitis and Barret's oesophagus commonly observed. Almost all inflammatory lesions of upper gastrointestinal endoscopic biopsies showed

correlation between endoscopy and histopathology diagnosis. For adenocarcinoma of stomach 91% of cases matched endoscopy and histopathology diagnosis. 60% of SCC showed correlation between endoscopy and histopathology diagnosis.

Anjana M.L. et al.[66], Somani NS et al.[67], and Ganga H et al.[42] showed that in the oesophagus, SCC was the most common lesion and most common malignancy, comprising 62%, 49%, and 63.3%, respectively, mostly seen in the middle oesophagus with male preponderance.

Hirachand et al.[41], and Mishra R et al.[68] showed the most common lesion was gastritis comprising 79%, and 58%, respectively, and the most common malignancy was adenocarcinoma comprising 12.3%, and 21.06%, respectively in gastric biopsies.

Anjana M.L. et al.[66] and Begum et al.[65]showed chronic duodenitis was the most common lesion comprising 61.8%. and 66%, respectively in duodenal biopsies.

Krishnappa Rasmi et al.[64], P Uma Rani et al.[65], and Mishra R et al.[68] showed that out of all cases, endoscopic and histopathology correlation was seen in 78%, 79%, and 80% of cases, respectively.

METHODOLOGY

Source of data: KLE's Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi's Hi-Tech Histopathology Laboratory

Ethical consideration: Ethical clearance was obtained and approved by Ethical Committee of Jawaharlal Nehru Medical College and Research Centre. All the personal information obtained from the study participant were kept confidential.

Study design: A hospital-based observational study

Collection of data period: 1st January 2023 to 31st December 2023

Sampling technique: Universal

Study population: After obtaining informed consent from all the participants, 135 upper gastrointestinal biopsies were collected from patients who underwent upper gastrointestinal endoscopy from outpatient departments and wards for gastrointestinal symptoms and suspected upper gastrointestinal tract disorders in KLE's Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi. Endoscopic biopsies were sent to Hi-Tech Histopathology Laboratory.

Inclusion criteria: Patients who underwent upper gastrointestinal endoscopy for gastrointestinal symptoms, biopsies from the oesophagus, stomach, and duodenum were taken from the lesional site at KLE's Dr. Prabhakar Kore Hospital and Medical Research Centre in Belagavi.

Exclusion criteria: Inadequate biopsies

Method of collection of data:

A gastroenterologist, with the help of a fiberoptic endoscope, selects the proper lesional site, and then a biopsy from that site was taken. The biopsy was put in 10% formalin, and with the proper details of the patient, it was labeled and sent to the histopathology section.

Biopsies were fixed overnight, and then they were taken for processing. Biopsies were put with the epithelial sides up. After embedding in paraffin, 4 to 7-micron-thick tissue sections are cut at the right angle to the biopsy surface. Routine H & E staining was done for histopathological examination.

Mucosal lining and foveola were searched for H. pylori in H & E. H. pylori detection is necessary for cases of gastritis with dysplasia or intestinal metaplasia, and suspected malignancy cases and Giemsa stain was done in these cases to give a better picture of H. pylori microorganisms.

The patient's clinical data and clinical diagnosis were obtained from the histopathology requisition form. Endoscopy reports were taken from the endoscopy room of the gastroenterology outpatient department. Histopathology findings of biopsies were correlated with endoscopic findings and brief clinical data.

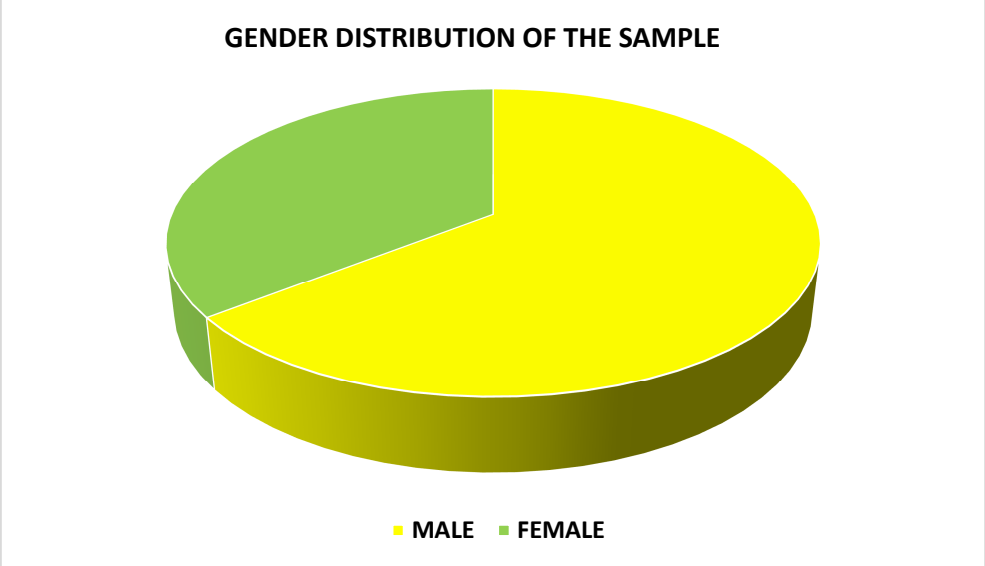
Statistical analysis used: p values were calculated using Chi square test to know statistical significance between clinical, endoscopy and histopathological findings of upper gastrointestinal biopsies. SPSS software was used for the chi-square test.

RESULTS

Table 5: Gender distribution of lesions

Gender	Number	Percentage%
Male	87	64.44
Female	48	35.56
Total	135	100.00

Graph 1: Gender distribution of lesions

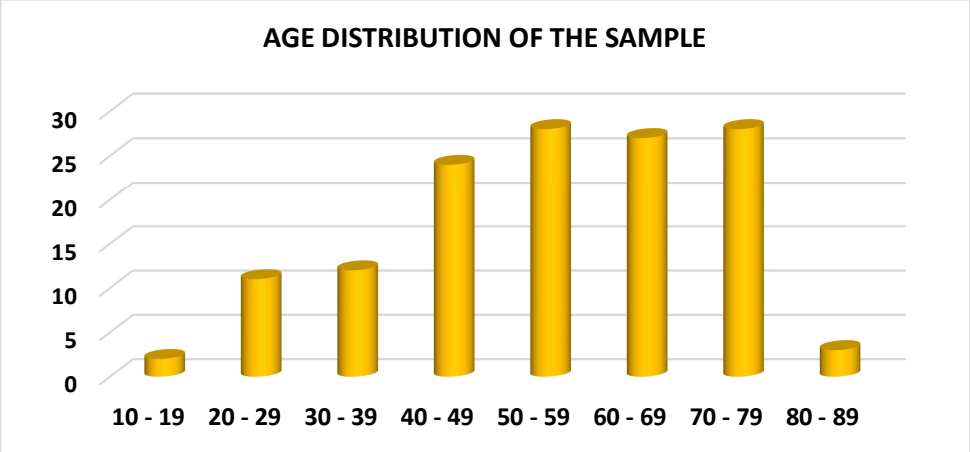


In this study a total of 135 total upper gastrointestinal endoscopic biopsies were studied of which 87(64.44%) were from males and 48 (35.66%) were from females. Overall cases showed male preponderance with a male-to-female ratio of 1.81:1.

Table 6: Age-wise distribution of lesions

Age	Number	Percentage%
10 - 19	2	1.48
20 - 29	11	8.15
30 - 39	12	8.89
40 - 49	24	17.78
50 - 59	28	20.74
60 - 69	27	20.00
70 - 79	28	20.74
80 - 89	3	2.22
Total	135	100.00

Graph 2: Age-wise distribution of lesions



	MEAN	S.D.	MIN	MAX
AGE	54.25	16.68	10	85

This study included cases between the age group of 10 to 85 years. The majority of the patients (28 cases each) were in the age group of 50-59 years and 70-79 years, followed by 60-69 years (27 cases) and 40-49 years (24 cases) of age group respectively.

Table 7.1: Biopsy site-wise distribution of lesions

Biopsy site	Number	Percentage%
Oesophagus	49	36.29%
Gastroesophageal junction (GEJ)	10	7.41%
Stomach	32	23.7%
Duodenum	39	28.88%
Stomach and duodenum	05	3.70%
Total biopsies	135	100%

In this study of the 135 endoscopic biopsies, 49 (36.29%) were from the oesophagus, 10 (7.40%) from the gastroesophageal junction, 32 (23.7%) from the stomach, 39 (28.88%)from the duodenum and 5(3.70%) biopsies were from both the stomach and duodenum.

Table 7.2: Biopsy site-wise distribution of oesophageal lesions

Biopsy site	Number	Percentage%
Upper oesophagus	10	20.40%
Middle oesophagus	25	51.02%
Lower oesophagus	14	28.57%
Total oesophageal biopsies	49	100%

Of the 49 oesophageal biopsies, the majority of the cases (25) were from the middle oesophagus, 14 were from the lower oesophagus and 10 were from the upper oesophagus.

Table 7.3: Biopsy site-wise distribution of gastric lesions

Biopsy site	Number	Percentage%
Greater curvature	1	3.12%
Fundus	2	6.25%
Gastric body	5	15.62%
Pyloric antrum	24	75%
Total gastric biopsies	32	100%

Of the 32 gastric biopsies, the majority of the cases (24) were from the pyloric antrum, 5 from the body, 2 from the fundus and 1 from greater curvature respectively.

Table 8.1: Clinical complaints of patients in overall lesions

Clinical complaints	Total number	Percentage%
Abdominal pain	55	30.89%
Nausea, vomiting	46	25.84%
Dysphagia	39	21.91%
Dyspepsia	10	5.61%
Diarrhoea	10	5.61%
Loss of weight	8	4.49%
Loss of appetite	5	2.80%
Hematemesis/melena	2	1,12%
Others	3	1.68%
Total	178	100

In this study, abdominal pain was the most common complaint in 55 cases (30.89%) followed by nausea and vomiting in 46 cases (26%) in chronic gastritis and duodenitis patients and for overall cases. Dysphagia was present in all cases (100%) of oesophageal and gastroesophageal junction malignancies. Dyspepsia as the complaint is seen in almost equal numbers in chronic gastritis and gastric malignancy patients. Loss of weight and appetite is usually seen with chronic duodenitis.

Table 8.2: Clinical complaints of patients in oesophageal, GEJ, gastric and duodenal lesions

Clinical complaints	Oesophageal lesions	GEJ lesions	Gastric lesions	Duodenal lesions
Abdominal pain	8	3	22	22
Nausea, vomiting	6	3	18	19
Dysphagia	30	4	4	1
Dyspepsia	-	1	9	-
Diarrhoea	1	-	9	-
Loss of weight	2	-	-	6
Loss of appetite	2	-	-	3
Hematemesis/melena	-	-	1	1

The most common complaint in patients undergoing oesophageal biopsy was dysphagia (30 cases), in patients undergoing gastric biopsy was abdominal pain (22 cases) and nausea with vomiting (18 cases), in patients undergoing duodenal biopsy abdominal pain (22 cases) and nausea with vomiting (19 cases) was the most common complaint.

Table 9: Oesophageal lesions distribution

Neoplastic lesions		
Neoplastic lesions	**Number**	**Percentage%**
Moderately differentiated SCC	20	40.81%
Poorly differentiated SCC	7	14.28%
Poorly differentiated adenocarcinoma	1	2.04%
Adeno squamous carcinoma	1	2.04%
Mild dysplasia	1	2.04%
Moderate dysplasia	2	4.08%
Total oesophageal Neoplastic lesions	32	65.31%
Non-neoplastic lesions		
Non-neoplastic lesions	**Number**	**Percentage%**
Oesophagitis with ulcer	8	16.32%
Eosinophilic oesophagitis	1	2.04%
Barret's oesophagus	1	2.04%
Squamous epithelial hyperplasia	6	12.24%
Fibrovascular polyp	1	2.04%
Total oesophageal non-neoplastic lesions	17	34.69%
Total oesophageal lesions	49	100

Of the 135 upper GI biopsies, 49 were from the oesophagus, 32(65.31%) were neoplastic and 17(34.69%) were non-neoplastic lesions. The commonest lesion of all the oesophageal biopsies was SCC in 27 cases (55%). There was one case each of poorly differentiated adenocarcinoma and adenosquamous carcinoma. Among non-neoplastic lesions oesophagitis with ulcer was the commonest 8 cases (16.32%) followed by squamous epithelial hyperplasia 6 cases (12.24%). Of the 49 oesophageal biopsies, Barret's oesophagus and eosinophilic oesophagitis was seen only in one case (2.04%) respectively.

Table 10: Gastric lesions distribution

Non-neoplastic lesions		
Non-neoplastic lesions	**Number**	**Percentage%**
Chronic gastritis	13	40.62%
Chronic gastritis with intestinal metaplasia	6	18.75%
H. pylori gastritis with intestinal metaplasia	1	3.12%
Total gastric non-neoplastic lesions	20	62.5%
Unremarkable	1	3.12%
Neoplastic lesions		
Neoplastic lesions	**Number**	**Percentage%**
H. pylori with dysplasia	1	3.12%
Moderately differentiated adenocarcinoma	3	9.38%
Poorly differentiated adenocarcinoma stomach	3	9.38%
Signet ring cell carcinoma	2	6.25%
Neuroendocrine tumor	1	3.12%
Moderately differentiated squamous cell carcinoma	1	3.12%
Total gastric neoplastic lesions	11	34.3%
Total gastric lesions	32	100

Of the 32 gastric biopsies, 11(34.3%) were neoplastic and 20(62.5%) were non-neoplastic lesions and one was unremarkable on histopathology. The most common lesion present in gastric biopsies was chronic gastritis in 21 cases (65.62%) followed by gastric adenocarcinoma in 6 cases (18.76%).

This study had 2 cases of H. pylori gastritis of which one was associated with intestinal metaplasia and the other was associated with dysplasia. This study also included two cases of signet ring cell carcinoma (6.25%), one case of SCC (3.12%), and one case of neuroendocrine tumor (3.12%).

Table 11: Gastroesophageal (GEJ) lesions distribution

Non neoplastic lesions		
Non neoplastic lesions	**Number**	**Percentage%**
Barret's oesophagus	2	20%
Chronic gastritis	2	20%
Squamous epithelial hyperplasia with ulcer	1	10%
Fibrovascular polyp	1	10%
Total non-neoplastic GEJ lesions	6	60%
Neoplastic lesions		
Neoplastic lesions	**Number**	**Percentage%**
Moderately differentiated adenocarcinoma	2	20%
Poorly differentiated adenocarcinoma	1	10%
Poorly differentiated squamous cell carcinoma	1	10%
Total neoplastic GEJ lesions	4	40%
Total GEJ lesions	10	100

Of 10 gastroesophageal junction biopsies, 6(60%) were non-neoplastic and 4 (40%) were neoplastic lesions. The most common lesion from the gastroesophageal junction was oesophageal adenocarcinoma in 3 cases (30%). Barret's oesophagus was seen in 2 cases (20%) and chronic gastritis was seen in 2 cases (20%).

Table 12: Duodenal lesions distribution

Non-neoplastic lesions		
Non-neoplastic lesions	**Number**	**Percentage%**
Chronic duodenitis	26	66.6%
Eosinophilic duodenitis (chronic)	2	5.12%
Chronic duodenitis with focal erosion	3	7.69%
Lymphangiectasia	1	2.56%
Leukocytoclastic vasculitis	1	2.56%
Total non-neoplastic duodenal lesions	33	87.62%
Neoplastic lesions		
Neoplastic lesions	**Number**	**Percentage%**
Spindle tumor	1	2.56%
Neuroendocrine tumor	1	2.56%
Moderately differentiated adeno carcinoma	3	7.69%
Villous adenoma/dysplasia	1	2.59%
Total neoplastic duodenal lesions	6	15.38%
Total duodenal lesions	39	100

There were a total of 39 duodenal biopsies of which 6(15.38%) were neoplastic and 33(87.62%) were non-neoplastic lesions. The most common lesion in duodenal biopsies was chronic duodenitis with 31 cases (79.4%). Of the 6 neoplastic lesions, 3 cases (7.69%) were of adenocarcinoma (7.69%) and one each was a spindle cell tumor, neuroendocrine tumor, and villous adenoma/dysplasia respectively.

Table 13: Gastric and duodenal lesions (two site biopsies)

Non-neoplastic lesions	Number	Percentage%
Chronic gastritis and chronic duodenitis	3	60%
Neuroendocrine tumor of duodenum and chronic gastritis	1	20%
Gastric intestinal metaplasia with Brunner's gland hyperplasia	1	20%
Total	5	100

In this study 2 site biopsies were also included in that most common lesion was chronic duodenitis with chronic gastritis (60%).

Distribution of total lesions:

In this study, on histopathology 54 cases (40.75%) were neoplastic and 80 cases (59.25 %) were non- neoplastic lesions.

Table 14: Age interval of non-neoplastic lesions

Age interval	Oesophagitis with ulcer	Dysplasia	Barret's oesophagus	Gastritis	Duodenitis
<20	-	-	-	-	3
20-29	1	1	-	2	5
30-39	2	-	-	2	6
40-49	-	-	3	3	11
50-59	1	-	-	7	3
60-69	3	-	-	6	5
70-79	2	2	-	4	-
80-89	-	-	-	1	1
Total	9	3	3	25	34

The commonest non-neoplastic lesions in upper gastrointestinal biopsies in oesophagus, stomach, and duodenum were oesophagitis, gastritis, and duodenitis. The majority of the cases of oesophagitis were observed in the seventh decade 3 cases (33.3%), gastritis cases were observed in the sixth decade 7 cases (28%) and in the seventh decade 6 cases (24%), and duodenitis cases were observed in fifth decade 11 cases (33.3%).

Table 15: Sex-wise distribution of non-neoplastic lesions

Lesions	Male	Female	M:F ratio
Oesophagitis with ulcer	7	2	3.5:1
Barret's oesophagus	3	-	-
Oesophageal dysplasia	1	2	1:2
Gastritis	14	11	1.27:1
Duodenitis	20	14	1.42:1

In this study non-neoplastic lesions showed male preponderance in all the cases except oesophageal dysplasia where female preponderance was there.

Table 16: Sex-wise distribution of malignancies

Gender	Oesophageal squamous cell carcinoma	Gastric adenocarcinoma	Duodenal adeno carcinoma
Male	13	04	3
Female	14	02	-
Total	27	06	3

Oesophageal SCC showed a female: male ratio was 1.071:1 with slight female preponderance whereas gastric and duodenal adenocarcinoma showed a male preponderance.

Table 17: Age distribution of malignancies

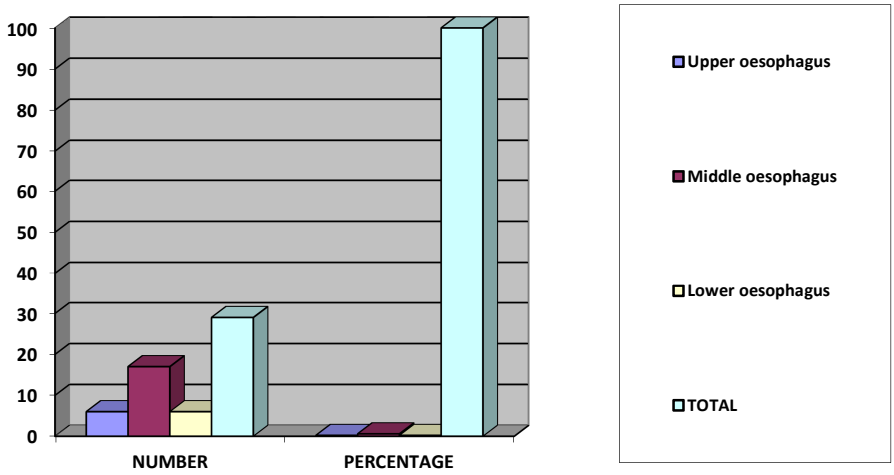
Age interval	Oesophageal carcinoma	Gastric/GEJ Carcinoma	Duodenal carcinoma
30-39	1	-	-
40-49	3	1	-
50-59	9	4	1
60-69	6	4	-
70-79	10	2	2
80-89	-	2	-
Total	29	13	3

Most of the cases of upper gastrointestinal malignancies were seen between the sixth to eighth decade of life.

Table 18: Site-wise distribution of oesophageal malignancies

Biopsy site	Number	Percentage%
Upper oesophagus	6	20.68%
Middle oesophagus	17	58.62%
Lower oesophagus	6	20.68%
Total	29	100

Graph 3: Site-wise distribution of oesophageal malignancies

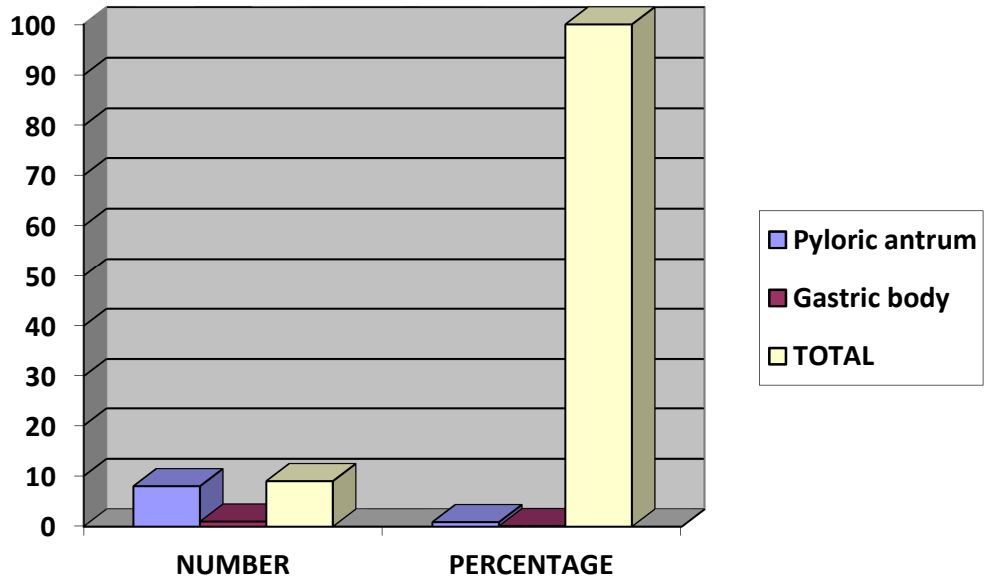


Of the 29 oesophageal malignancies,17(58.62%) were seen in the middle oesophagus, of which 16(94.11%) were squamous cell carcinoma.

Table 19: Site-wise Distribution of gastric Malignancies

Biopsy site	Number	Percentage%
Pyloric antrum	8	88.88%
Gastric body	1	11.11%
Total	9	100

Graph 4: Site-wise distribution of gastric malignancies

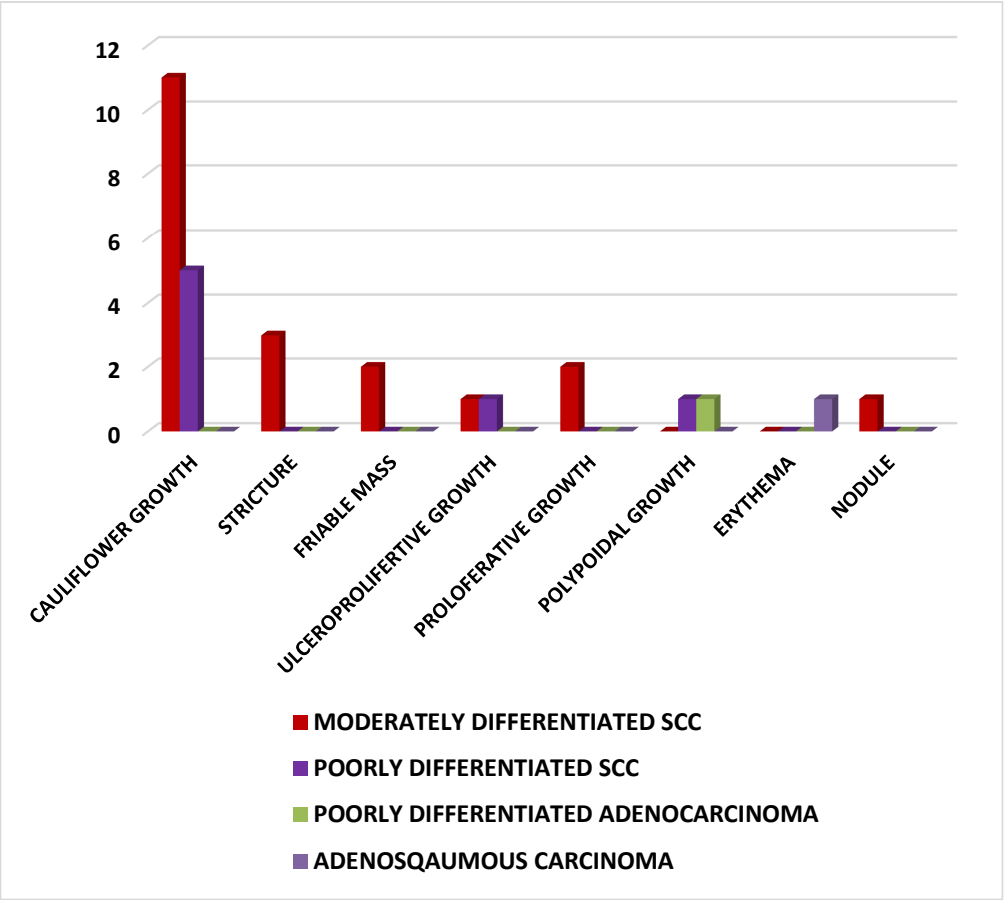


Most of the gastric malignancies, 8 (88.8%) were from Pyloric antrum and one (11.1%) was from the gastric body.

Table 20: Endoscopy and histopathology findings of oesophageal malignancies

Oesophagus	**Histopathology findings**			
Endoscopic findings	**Moderately differentiated SCC**	**Poorly differentiated SCC**	**Poorly differentiated adenocarcinoma**	**Adenosquamous carcinoma**
Cauliflower growth	11	5	-	-
Stricture	3	-	-	-
Friable mass	2	-	-	-
Ulceroproliferative growth	1	1	-	-
Proliferative growth/luminal narrowing	2	-	-	-
Polypoidal growth	-	1	1	-
Erythema	-	-	-	1
Nodule	1	-	-	-
Total	20	7	1	1

Graph 5: Endoscopy and histopathology findings of oesophageal malignancies



Clinically of the 42 suspected cases of oesophageal carcinoma, 29 were proven to be malignancies on histopathology where all the patients presented with dysphagia.

On endoscopy, oesophageal malignancy commonly presented as cauliflower growth in 16 cases. The remaining cases presented with stricture, ulceroproliferative growth, proliferative growth, polypoidal growth, nodular lesion, and mucosal erythema. One case of mucosal erythema on endoscopy which was clinically diagnosed as oesophagitis on histopathological examination turned out to be

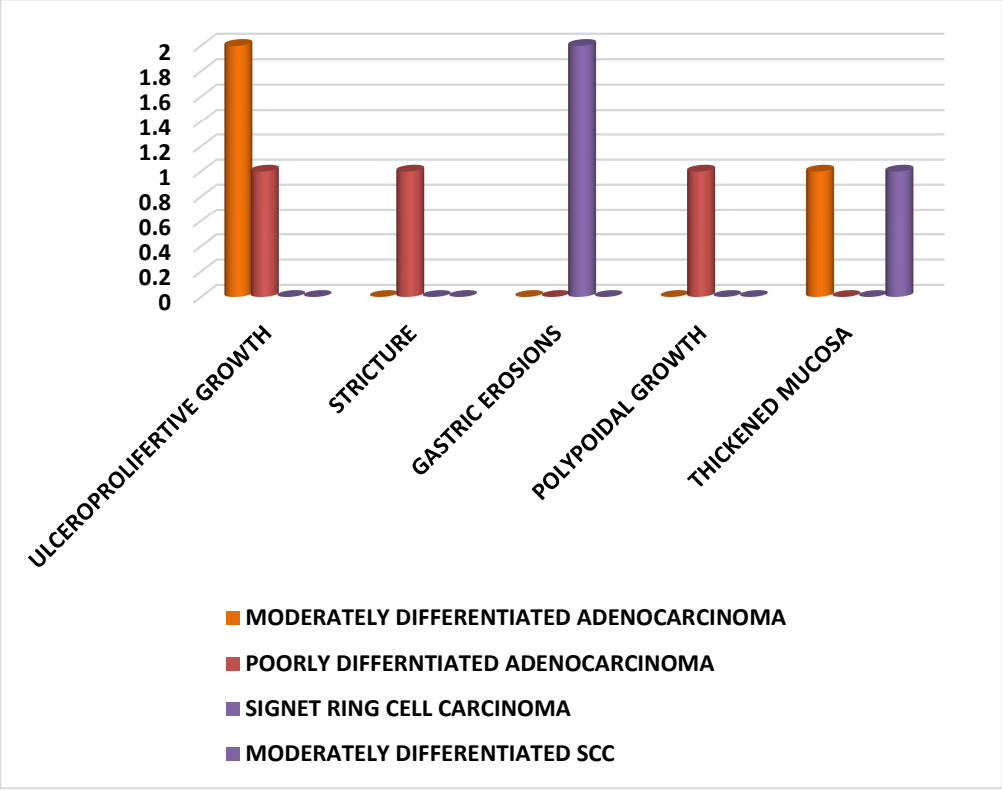
adenosquamous carcinoma. Thus, endoscopy findings were correlated with histopathology findings in 28 out of 29 cases (96.5%) of oesophageal malignancies.

Also, two cases with clinical suspicion of carcinoma oesophagus which on endoscopy showed strictures turned out to be epithelial hyperplasia on histopathological examination. One case of post-cricoid anatomical stricture on endoscopy which was treated for oesophageal carcinoma turned out to be moderate dysplasia on histopathology. One case of the nodule with luminal narrowing on endoscopy clinically suspected carcinoma oesophagus, which turned out to be moderate dysplasia on histopathology.

Table 21: Endoscopy and histopathology findings of gastric malignancies

Stomach	Histopathology findings			
Endoscopic findings	**Moderately differentiated adenocarcinoma**	**Poorly differentiated adenocarcinoma**	**Signet ring cell carcinoma**	**Moderately differentiated SCC**
Ulceroproliferative growth	2	1	-	-
Stricture	-	1	-	-
Gastric erosions	-	-	2	-
Polypoidal growth	-	1	-	-
Thickened mucosa	1	-	-	1
Total	3	3	2	1

Graph 6: Endoscopy and histopathology findings of gastric malignancies



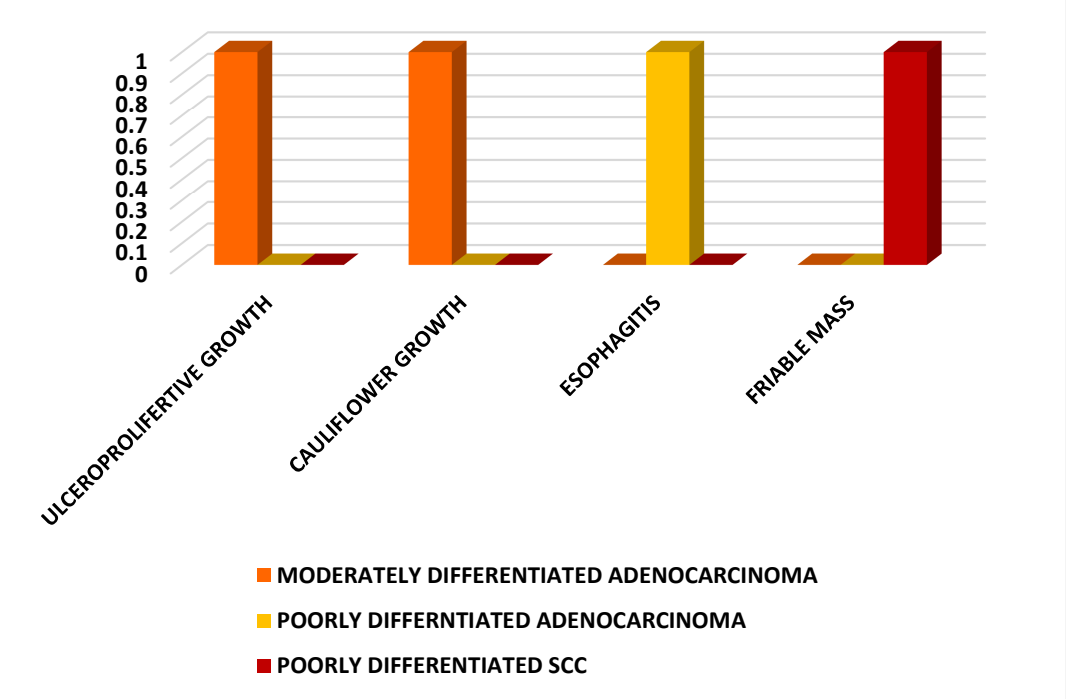
Clinically of the 10 suspected cases of gastric carcinoma, 6 turned out to be positive on histopathology. Suspected cases presented with vague symptoms of dyspepsia associated with nausea and vomiting, and abdominal pain.

Gastric erosion was the most common finding in chronic gastritis and adenocarcinoma presented mostly as ulceroproliferative growth on endoscopy. Here, of the 9 histopathological diagnoses of gastric carcinoma, endoscopic findings in 5 cases showed ulceroproliferative growths, two cases showed gastric erosions and the remaining two cases showed thickened gastric mucosa. Two cases of gastric erosions on endoscopy revealed signet ring cell carcinoma on histopathology and another two cases of thickened mucosa on endoscopy revealed adenocarcinoma and squamous cell carcinoma. Thus, endoscopy findings correlated with histopathology in 5 out of 9 cases (55.5%) of gastric malignancies

Table 22: Endoscopy and histopathology findings of GEJ malignancies

GEJ	Histopathology findings		
Endoscopic findings	**Moderately differentiated adenocarcinoma**	**Poorly differentiated adenocarcinoma**	**Poorly differentiated SCC**
Ulceroproliferative growth	1	-	-
Cauliflower growth	1	-	-
Esophagitis	-	1	-
Friable mass	-	-	1
Total	2	1	1

Graph 7: Endoscopy and histopathology findings of GEJ malignancies

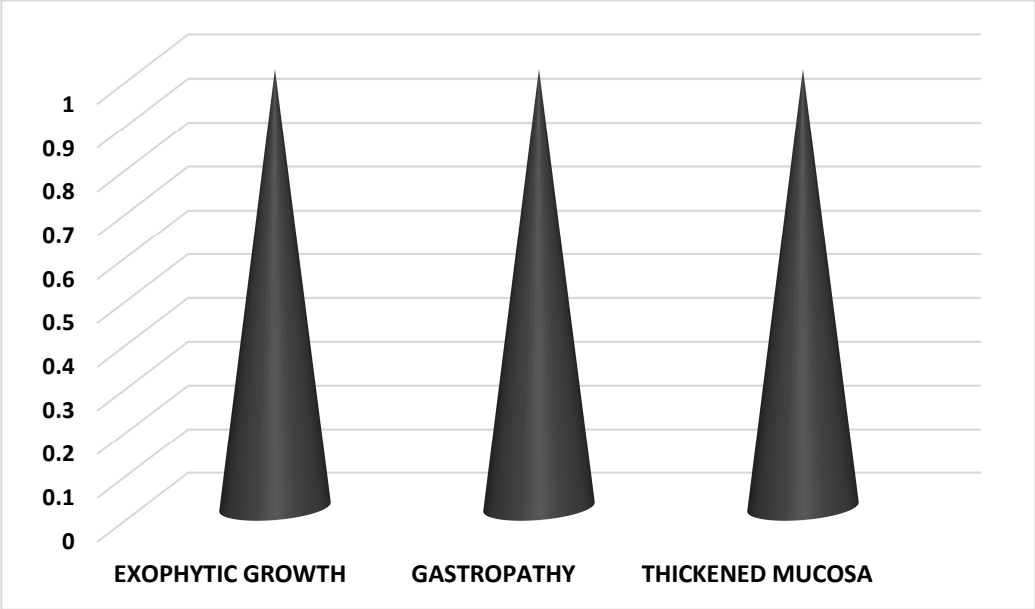


In gastroesophageal junction malignancies, all the cases suspected of carcinoma oesophagus turned out to be malignancies on histopathology. One of the cases endoscopically diagnosed as oesophagitis was diagnosed as a malignancy of the oesophagus. Thus, endoscopy findings correlated with histopathology findings in 3 out of 4 cases (75%) of GEJ malignancies.

Table 23: Endoscopy and histopathological findings of duodenal malignancies

Duodenum	Histopathological findings
Endoscopic findings	**Moderately differentiated adenocarcinoma**
Exophytic growth	1
Gastropathy	1
Thickened mucosa	1

Graph 8: Endoscopy and histopathological findings of duodenal malignancies



Of the 3 duodenal biopsies diagnosed as a malignancy on histopathology, on endoscopy presented as exophytic growth, gastropathy, and thickened mucosa respectively. Thus, endoscopy findings correlated with histopathology findings in 1 out of 3 cases (33.3%) of duodenal malignancies.

Endoscopy and histopathology correlation:

Endoscopy findings correlated with histopathology findings in 96.5% of oesophageal malignancies, 55.5% of stomach malignancies, 75% of GEJ malignancies, and 33.3% of duodenal malignancies. Lesions like Barret's oesophagus, Neuroendocrine tumors, and dysplasia do not have specific findings on endoscopy, mostly they were diagnosed on histopathology in the present study. Endoscopy and histopathological correlation were observed in 42 of 49 oesophageal biopsies (85.7%), 25 of 32 cases in gastric biopsies (78.1%), 7 of 10 (70%) GEJ biopsies and 37 out of 44 (84%) duodenal biopsies. Overall, 113 out of 135 cases showed endoscopy and histopathology findings correlation. Thus, endoscopy with histopathology correlation was 83.7% of overall cases. ($p < 0.0001$)

Statistical analysis:

p values were calculated using a chi-square test to find the overall association between (1) clinical and endoscopy findings showed $p < 0.0001$, (2) clinical and histopathology findings showed $p < 0.0001$, (3) endoscopy and histopathology findings showed $p < 0.0001$ which proved statistically significant association. SPSS software was used for the chi-square test. (p value<0.05 is generally significant). Master chart with clinical, endoscopic and histopathology diagnosis of all patients given in annexures.

Endoscopy images of lesions:

Figure 6: Endoscopy image of oesophagitis with circumferential ulceration

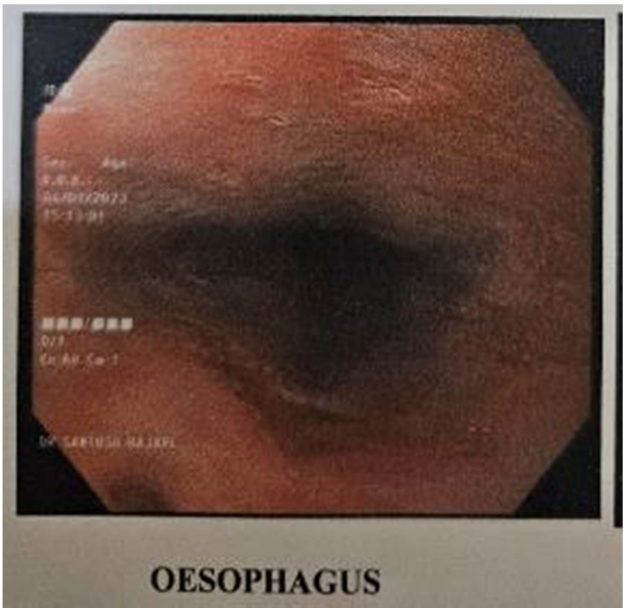


Figure 7: Endoscopy image of diffuse erosive gastritis

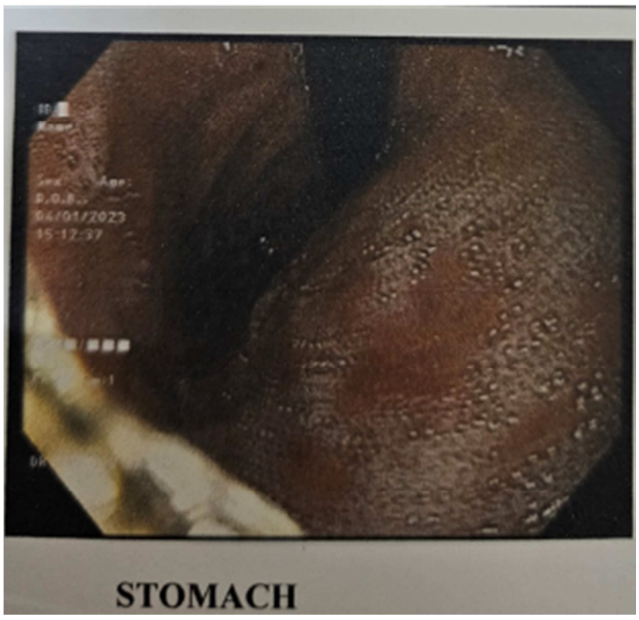


Figure 8: Endoscopy image of duodenitis

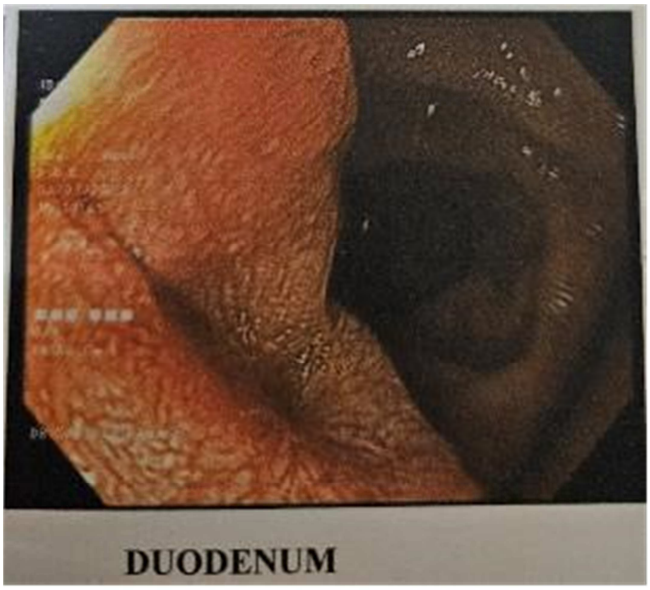


Figure 9: Endoscopy image of cauliflower mass in oesophagus

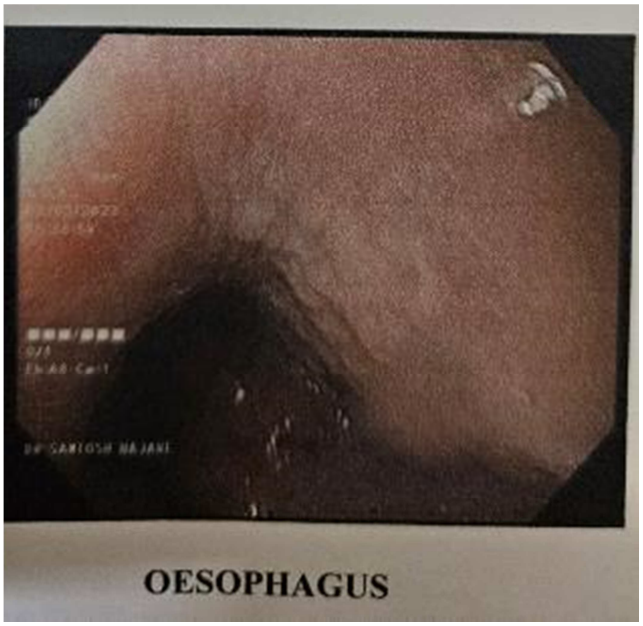


Figure 10: Endoscopy image of ulceroproliferative growth of oesophagus

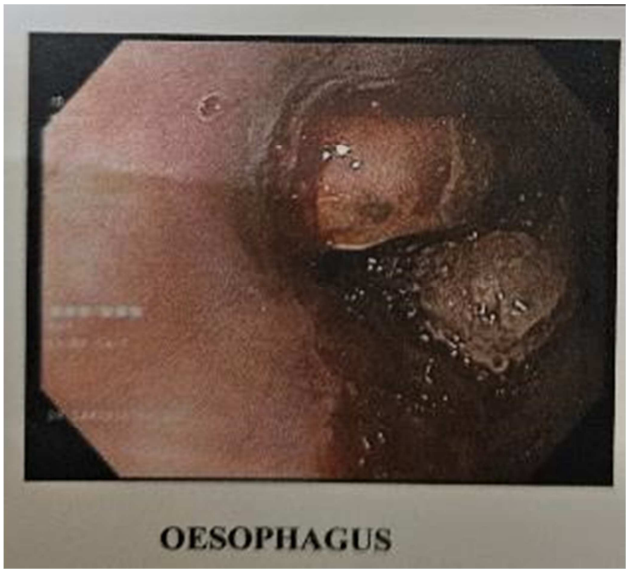
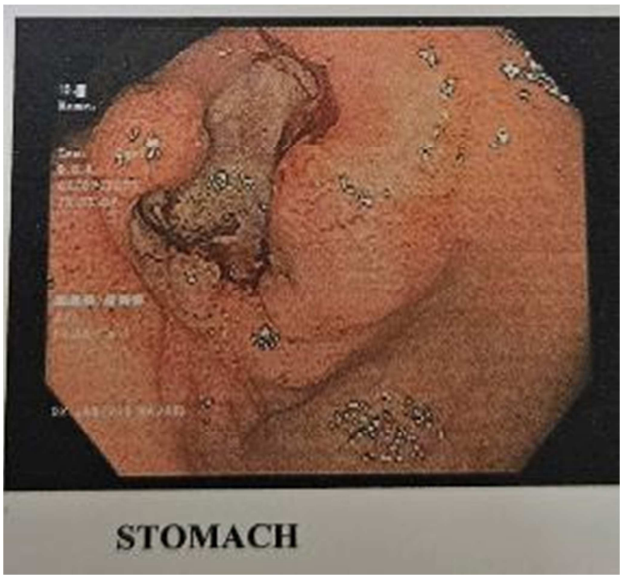


Figure 11: Endoscopy image of ulceroproliferative growth of stomach



Histopathology images of lesions:

Figure 12: Acute on chronic oesophagitis (H & E stain) (10X)

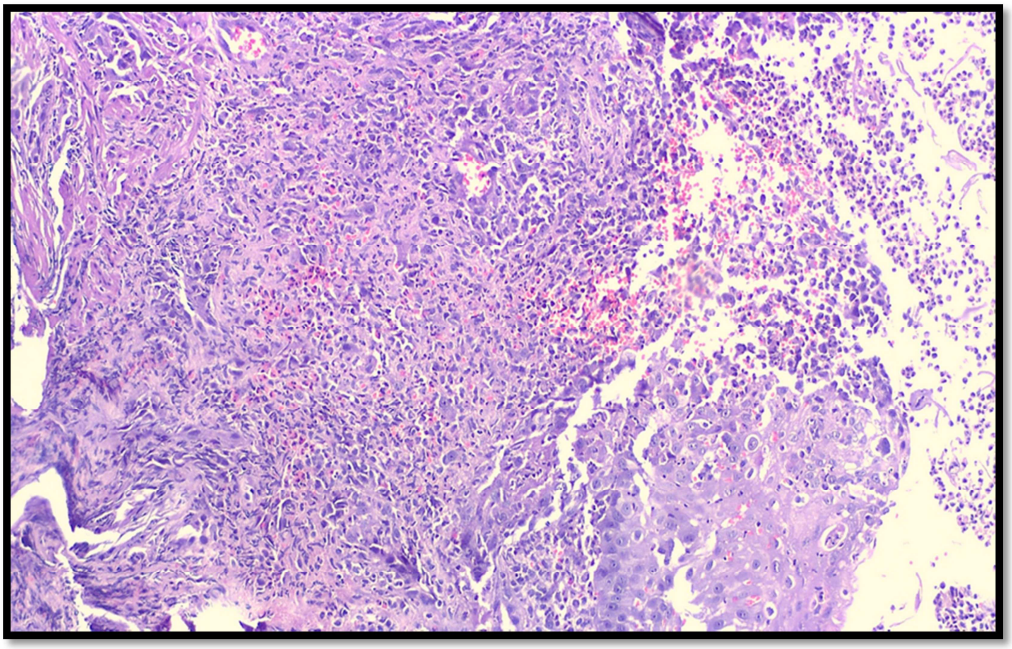


Figure 13: Barret's oesophagus (H & E stain) (20X)

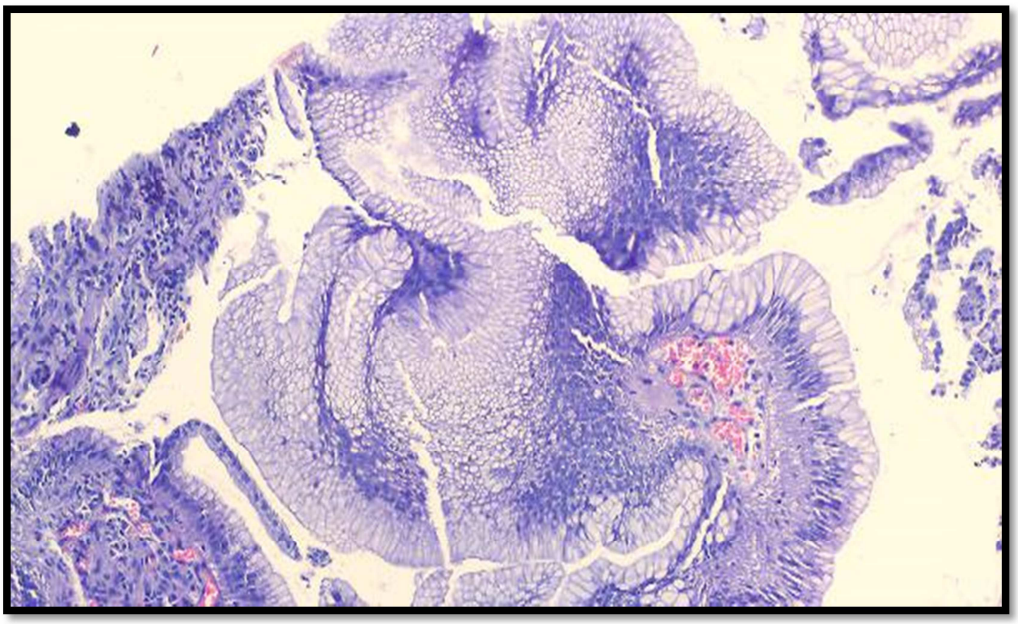


Figure 14: Mild dysplasia of oesophagus (H & E stain) (10X)

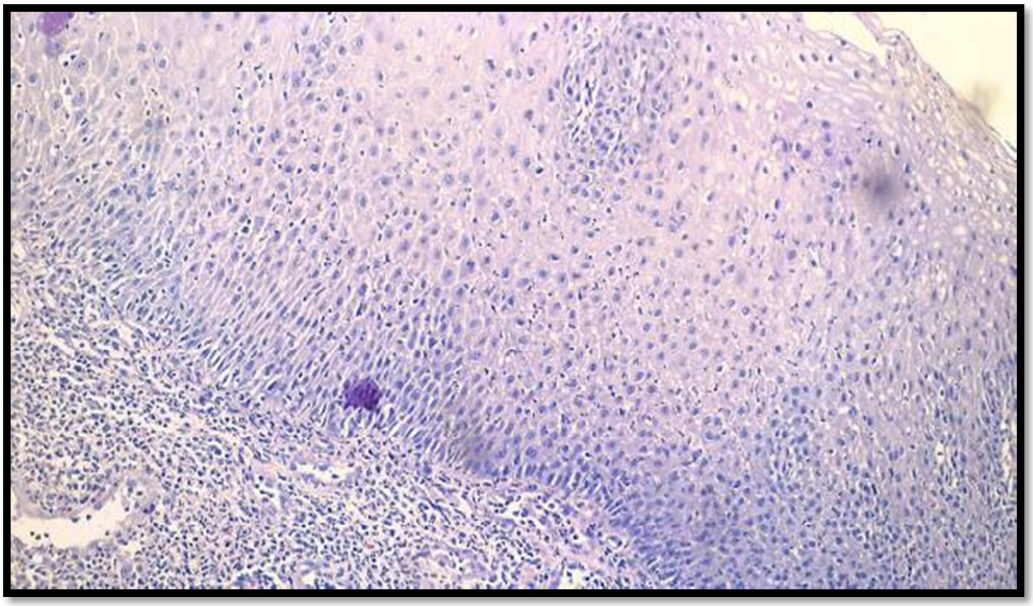


Figure 15: Moderate dysplasia of oesophagus (H & E stain) (20X)

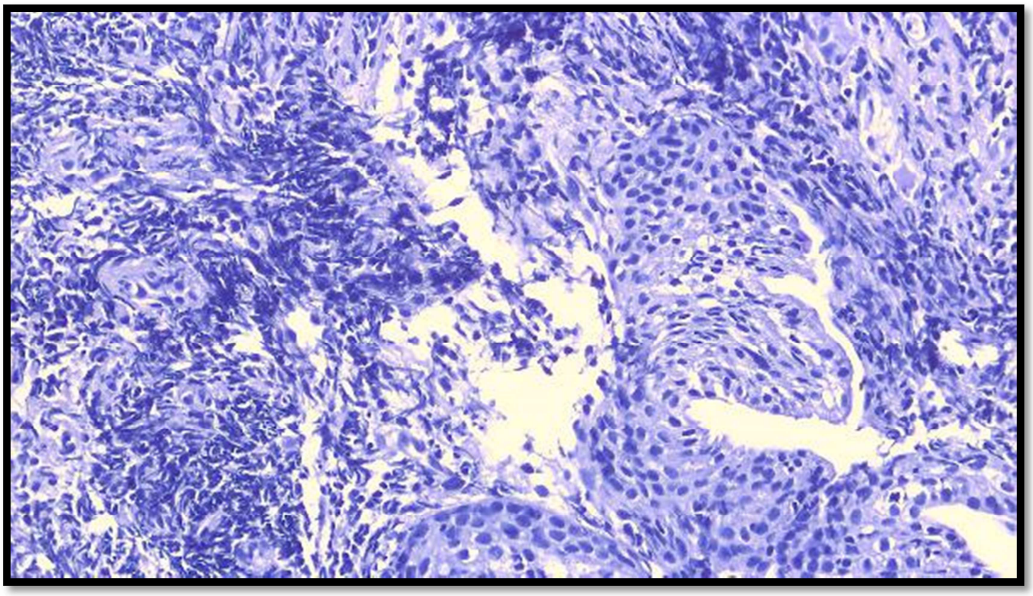


Figure 16: Moderately differentiated SCC (H & E stain) (20X)

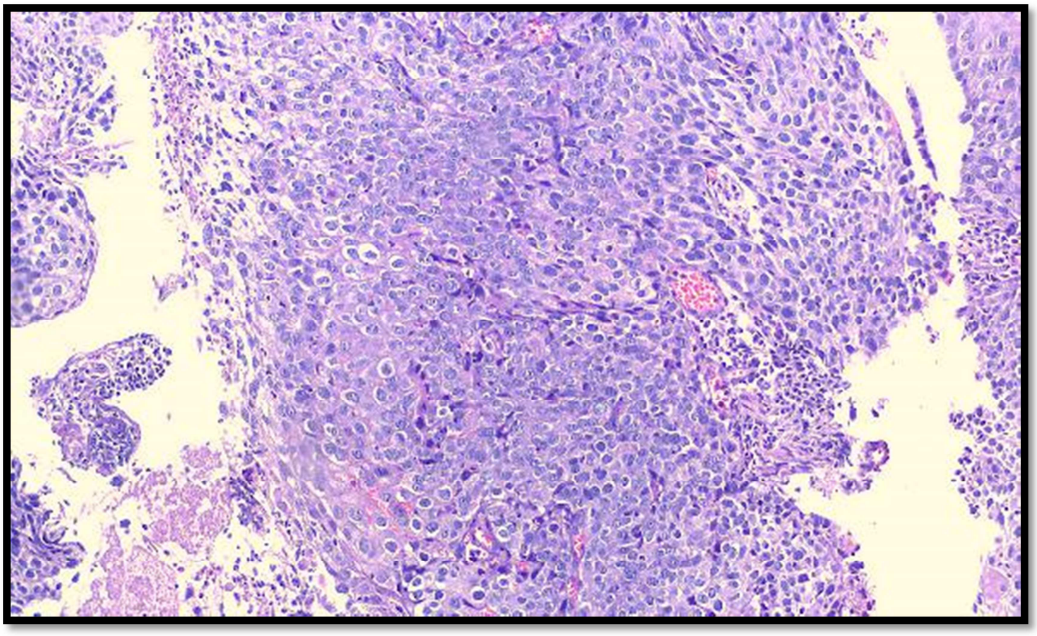


Figure 17: Poorly differentiated SCC (H & E stain) (20X)

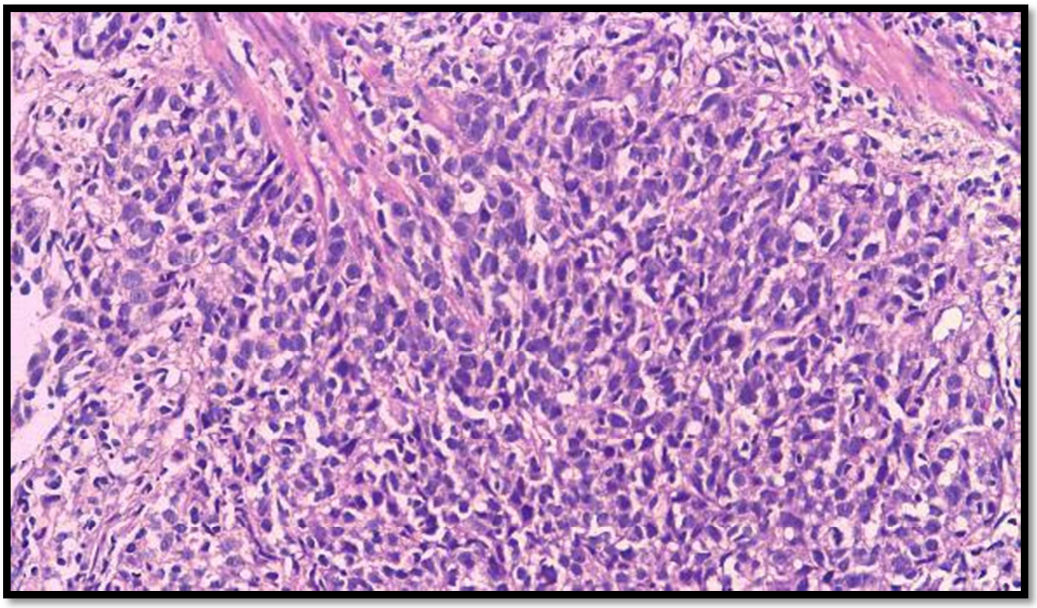


Figure 18: Poorly differentiated adenocarcinoma of oesophagus (H & E stain) (20X)

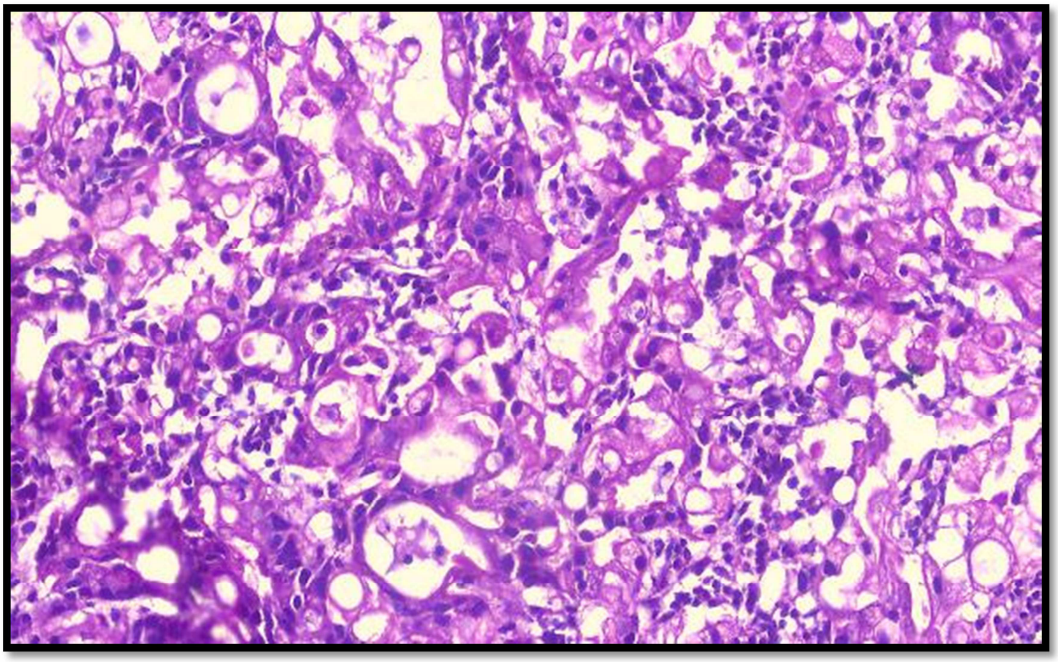


Figure 19: Chronic gastritis (H & E stain) (20X)

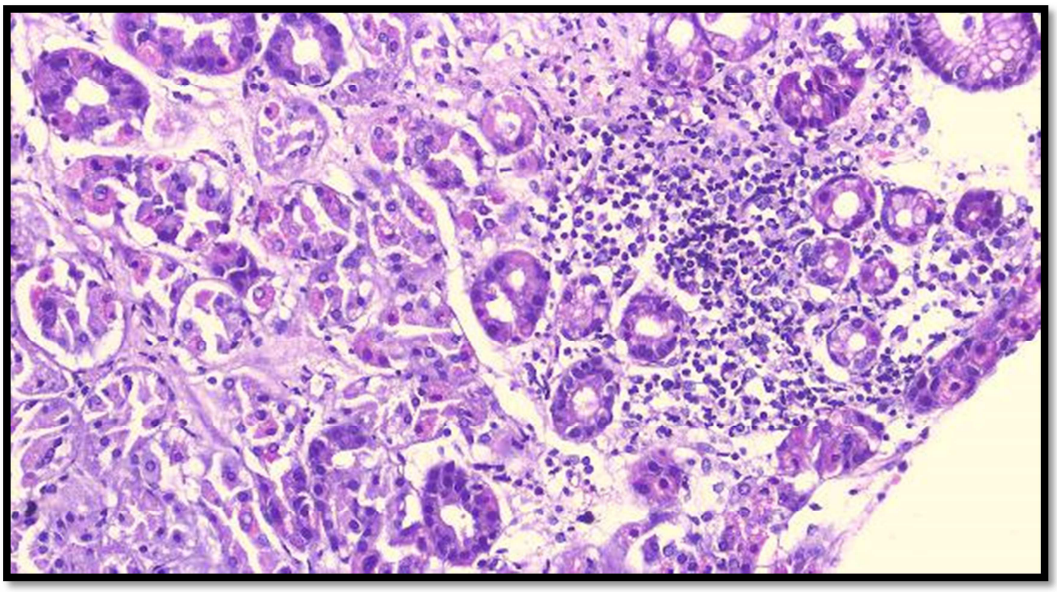


Figure 20: Chronic gastritis with intestinal metaplasia (H & E stain) (20X)

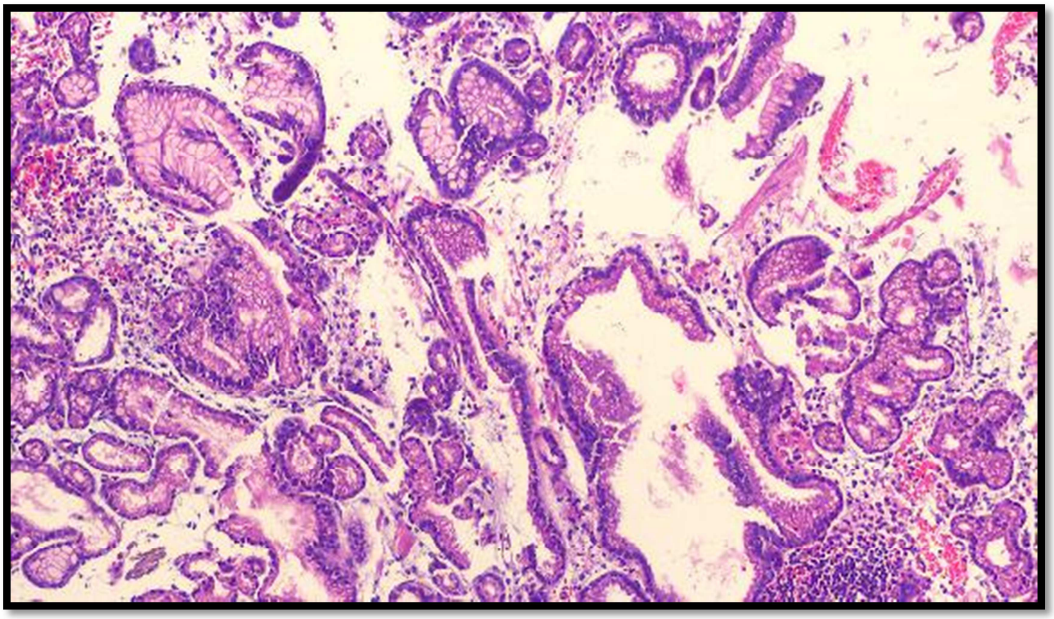


Figure 21: Chronic gastritis with H. pylori (H & E stain) (40X)

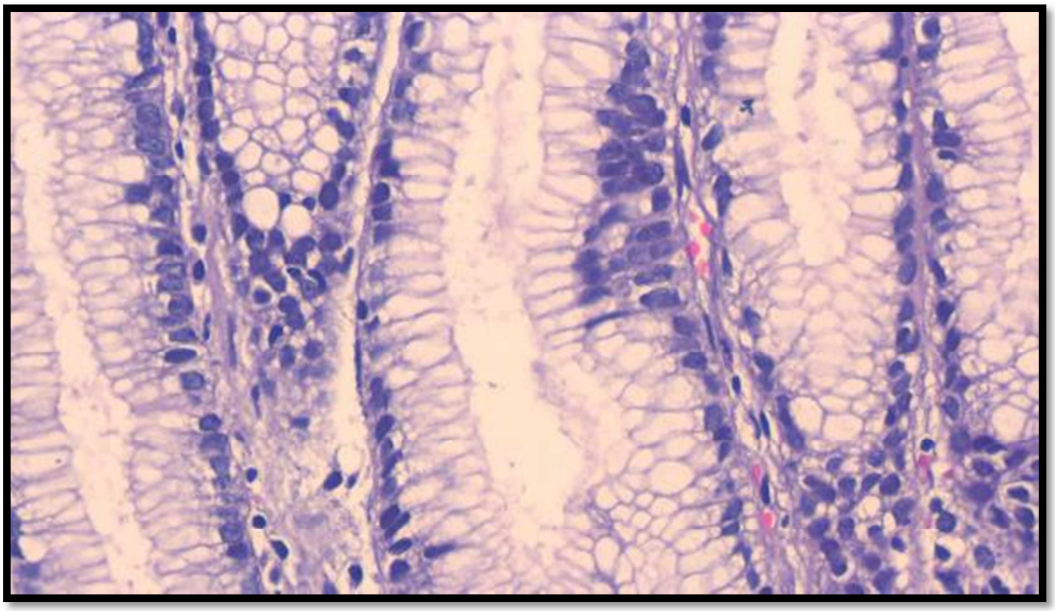


Figure 22: Chronic gastritis with H. pylori Giemsa stain (20X)

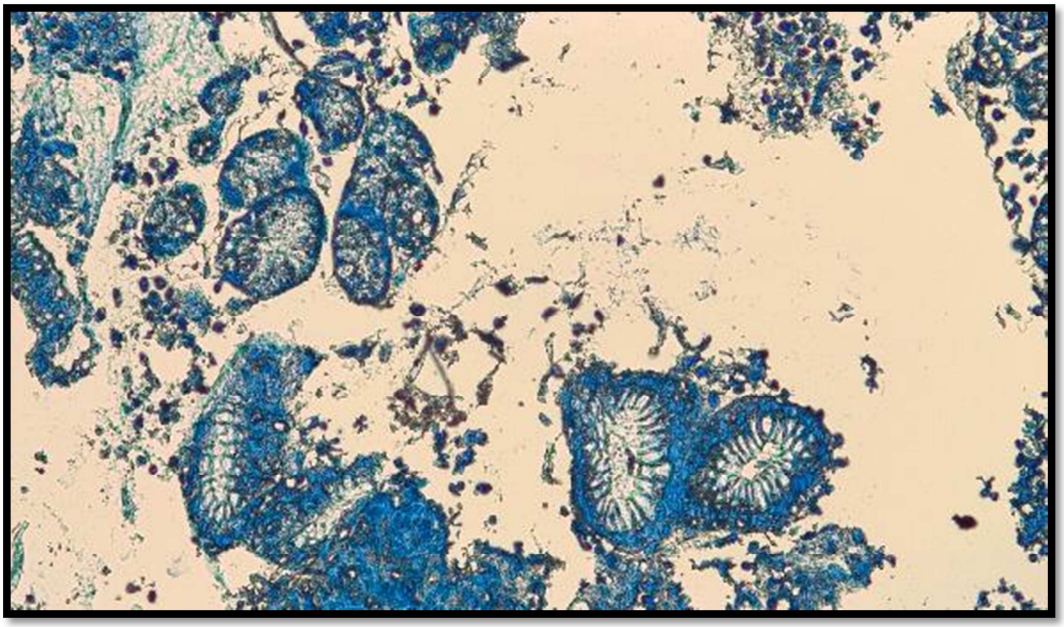


Figure 23: Gastric neuroendocrine tumor (H & E stain) (10X)

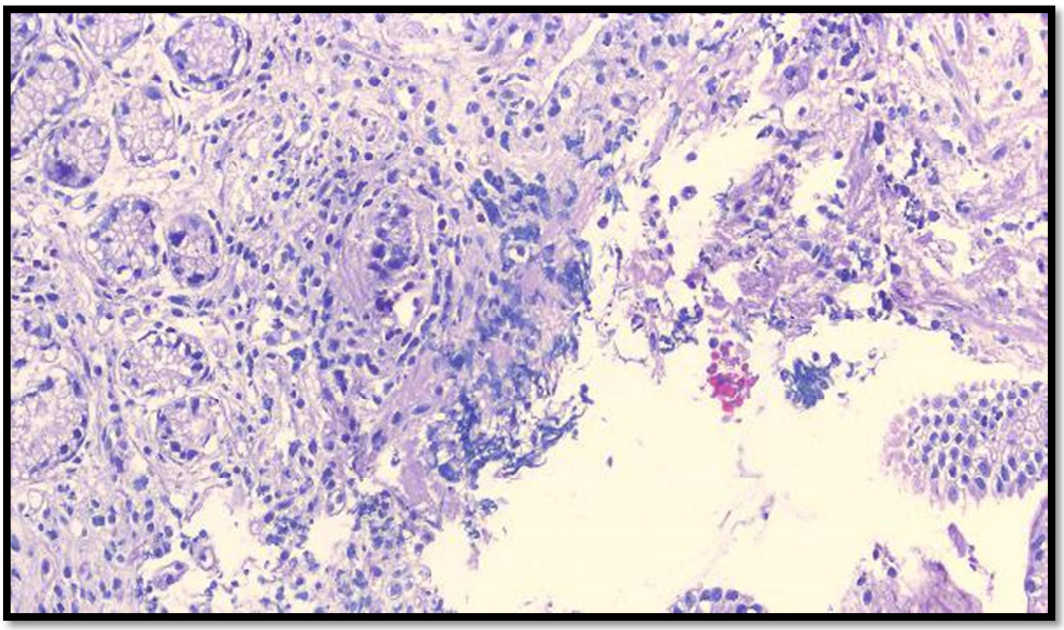


Figure 24: Moderately differentiated adenocarcinoma stomach (H & E stain) (20X)

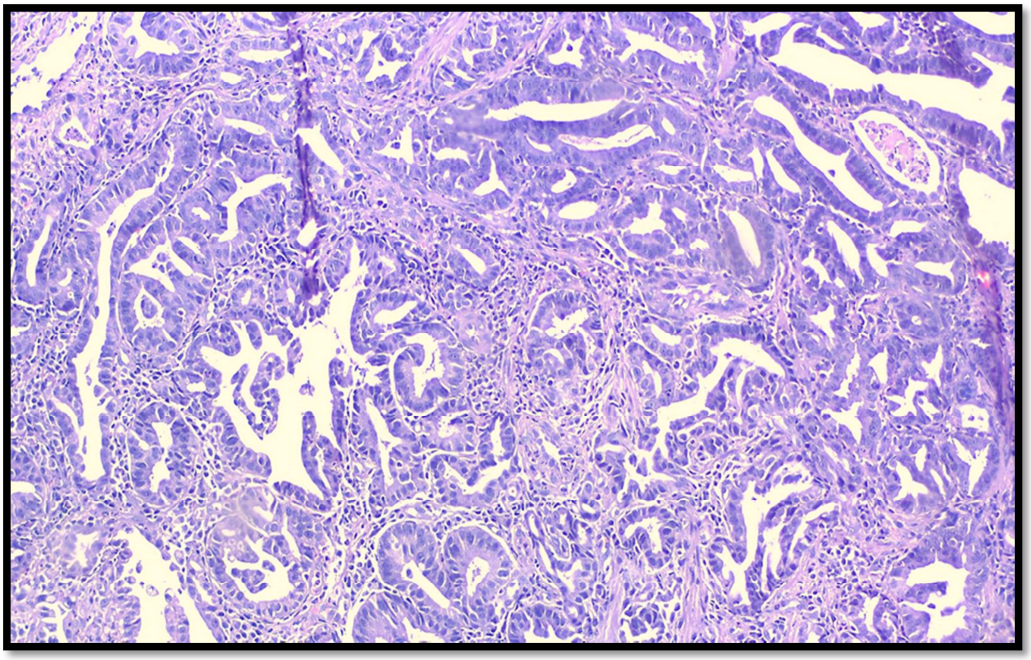


Figure 25: Signet ring cell carcinoma of stomach (H & E stain) (20X)

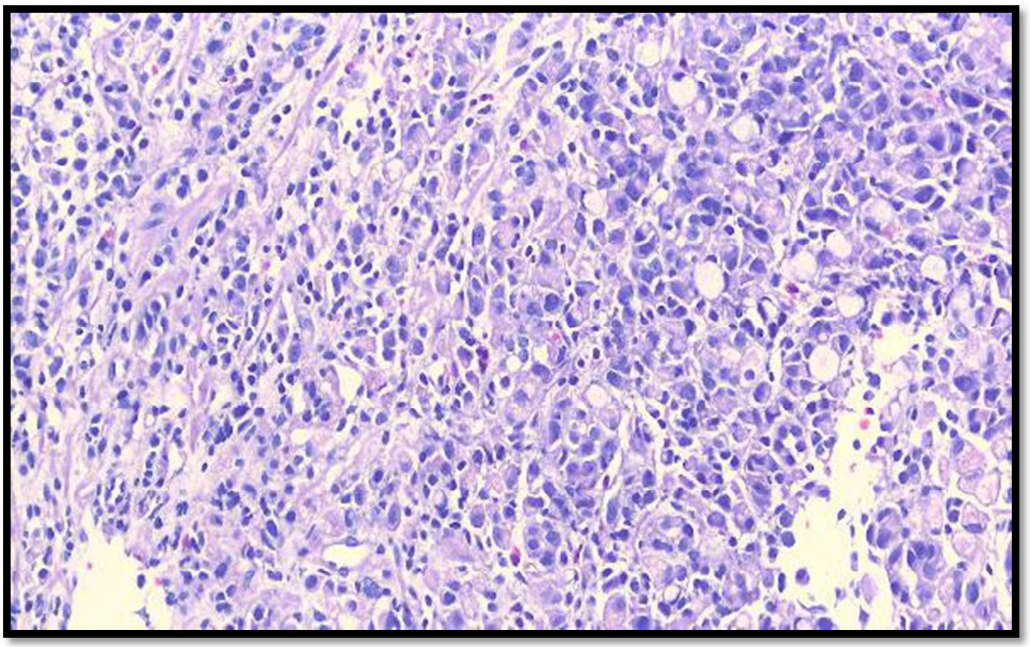


Figure 26: Chronic duodenitis (H & E stain) (10X)

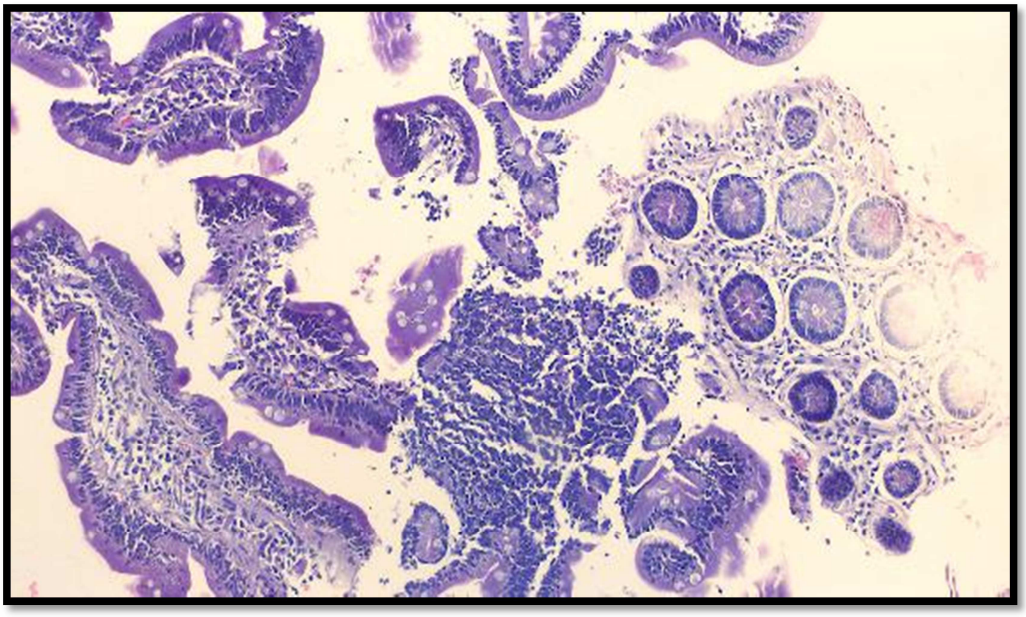


Figure 27: Neuroendocrine tumor duodenum (H & E stain) (10X)

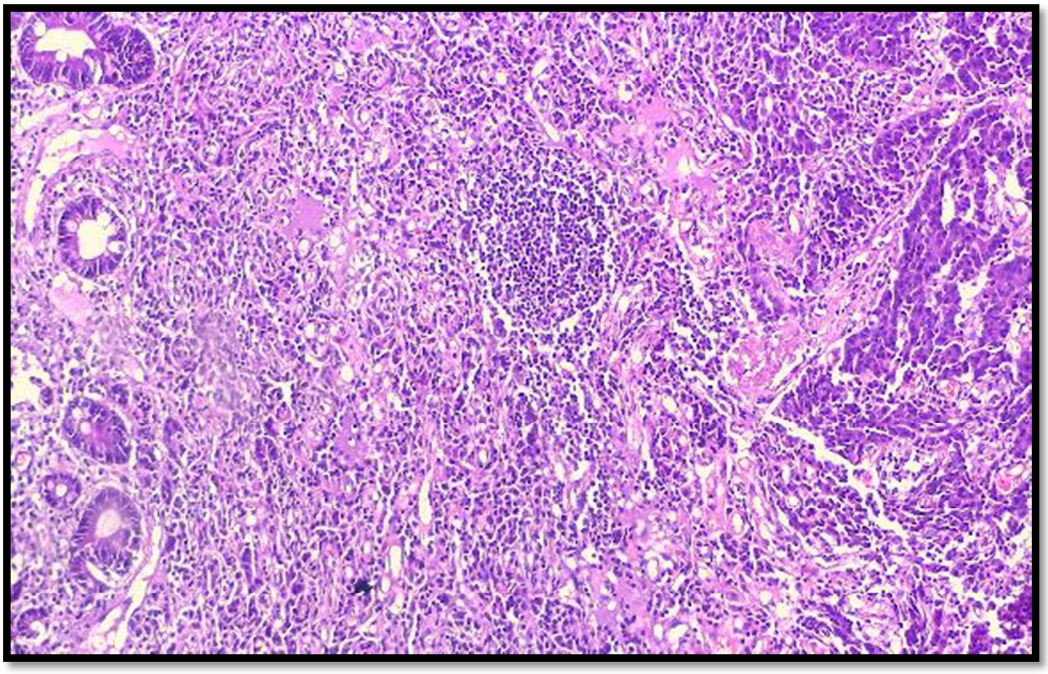
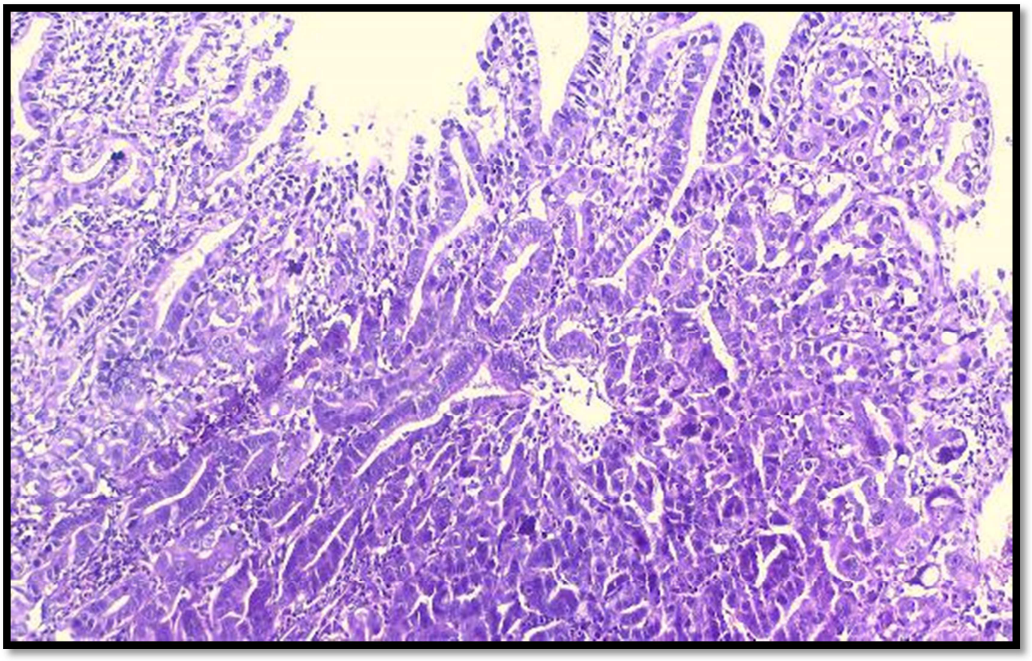


Figure 28: Moderately differentiated duodenal carcinoma (H & E stain) (20X)



DISCUSSION

Upper gastrointestinal tract disorders especially malignancies are the main contributors to long-term illness and death. Upper gastrointestinal endoscopy is used for detecting lesions and obtaining a biopsy from the lesional site. Endoscopy findings along with a histopathological examination of the endoscopic biopsies obtained from the upper gastrointestinal tract helps in providing an accurate diagnosis and appropriate patient care. An observational study was conducted on 135 upper gastrointestinal endoscopic biopsies received in the histopathology section for histopathological evaluation and their correlation with endoscopic findings.

Gender distribution:

Table 24: Comparison of gender distribution of lesions

Study	Male: female ratio	Total endoscopic biopsies
Jaynul Islam et al.[70](2014)	1.44:1	110
Parikh et al.[71] (2024)	1.03:1	150
Deepa Rani et al.[72](2019)	1.5:1	100
Present study	1.81:1	135

In our study male-to-female ratio of 1.81:1 was seen with overall cases showing male preponderance. Similar findings were shown in other studies done by Jaynul Islam. et al.[70], Deepa Rani et al.[72], and Parikh et al.[71] where male preponderance was observed.

Age-wise distribution:

In our study, most of the lesions in patients occurred in the age groups of 50–59 years and 70–79 years. Similar to our findings a study done by Parikh et al.[71] (2024) comprising 150 endoscopic biopsies showed that the majority of patients were from 41–50 years, followed by 51–70 years. A study done by Rashid Sarker et al.[73] (2018) comprising 344 endoscopic biopsies showed maximum patients were from 41-60 years followed by 61-80 years.

Site of biopsy:

Table 25: Comparison of biopsy site-wise distribution of lesions

Site of biopsy	Sunita et al.[74](2019)	Rashid Sarker et al[73](2018)	Anjana M.L. et al.[66](2021)	Present study
Oesophagus	54 (49.54%)	72 (20.9%)	250 (63.13%)	49(36.29%)
GEJ	03 (2.75%)	-	-	10(7.40%)
Stomach	35 (52.1%)	235(68.3%)	104 (26.26%)	32 (23.70%)
Duodenum	17 (15.59%)	37 (10.68%)	42 (10.6%)	39 (28.88%)
Stomach and duodenum (2 site biopsies)	-	-	-	05 (3.70%)
Total endoscopic biopsies	109 (100%)	344 (100%)	396 (100%)	135 (100%)

In our study, oesophagus was the most common site of biopsy. This is similar to the studies done by Sunita et al.[74](2019) and Anjana M.L.et al.[66(]2021) where oesophagus was the most common site for biopsy whereas the study done by Rashid Sarker et al[73](2018) showed stomach was the most common site of biopsy.

Clinical complaints of patients:

Table 26: Comparison of clinical complaints with other studies

Clinical complaints	Hussain et al.[75](2015) (total endoscopic biopsies=132)	Mohan et al.[76](2019) (total endoscopic biopsies=106)	Present study (total endoscopic biopsies=135)
Abdominal pain	18.2%	56%	30.89%
Nausea, vomiting	4.5%	-	25.84%
Dysphagia	30.3%	51%	21.91%
Dyspepsia	37.9%	47%	5.61%
Diarrhoea	-	-	5.61%
Loss of weight	-	-	4.49%
Loss of appetite	-	19%	2.80%
Hematemesis/ melena	6.1%	-	1.12%

In our study, abdominal pain (30.89%) was the most common complaint followed by nausea and vomiting (26%) in chronic gastritis and duodenitis patients and for overall cases. Dysphagia was present in all cases (100%) of oesophageal and gastroesophageal junction malignancies. Dyspepsia as the complaint is seen in almost equal numbers in chronic gastritis and gastric malignancy patients. Loss of weight and appetite is usually seen with chronic duodenitis. Similar findings were seen where abdominal pain was the most common clinical complaint in a study done by Mohan et al.[75]

Whereas a study done by Hussain et al.[75](2015) showed in terms of gastric lesions, dyspepsia was the chief complaint in more than 70% of patients with gastritis, and abdominal pain was the chief complaint in 43% of patients with gastric malignancy.

A study done by Anjana M.L. et al.[66](2021) and Hussain et al.[75](2015), it was revealed that all cases of SCC of the oesophagus showed a dysphagia as clinical complaint similar to our study.

Distribution of lesions:

Table 27: Comparison of non-neoplastic and neoplastic lesions

Lesions	Tarun et al.[77] (2020) (total endoscopic biopsies= (1800)	Rohit Mathew et al.[78] (2020) (total endoscopic biopsies=259)	Veerendrasagar et al.[79](2020) (total endoscopic biopsies=59)	Present study (total endoscopic biopsies=135)
Non-neoplastic	73%	66.8%	30.50%	59.25%
Neoplastic	27%	33.2%	69.5%	40.75%

In our study, non-neoplastic lesions were more (59.25%) as compared to neoplastic lesions on histopathology. similar findings were seen in studies done by Tarun et al.[77] and Rohit Mathew et al.[78] Whereas study done by Veerendrasagar et al.[79]et al.[79] showed neoplastic lesions were more than non-neoplastic lesions.

Oesophageal lesions distribution

Table 28: Comparison of non-neoplastic and neoplastic lesions of oesophagus

Oesophageal biopsies	**Abhilash SC et al.[80] (2016)(ns=38)**	**Hirachand et al.[41] (2018)(ns=15)**	**Anjana M.L.et al.[66](2021)(ns=250)**	**Present study(ns= 49)**
Non-neoplastic lesions	52.26%	80%	32.8%	34.69%
Neoplastic lesions	47.74%	20%	67.2%	65.31%

In our study, 65.31% were neoplastic lesions of oesophagus which was in concordance with study done by Anjana M.L.et al.[66](2021) whereas Abhilash SC et al.[80] sand Hirachand et al.[41] showed non- neoplastic lesions predominance.

In the present study, most common lesion present in oesophageal biopsies was SCC of oesophagus 55% followed by esophagitis 18.36%, which was in concordance with a study done by Anjana M.L.et al.[66](2021), Somani NS et al.[67](2018), and Ganga H et al.[42](2018)

Studies done by Qureshi et al.[81](2007) and Rashid Sarker et al.[73](2018) showed 50% and 70% of cases of oesophageal adenocarcinoma which were more than SCC cases of oesophageal malignancy.

Gastric lesion distribution:

Table 29: Comparison of non-neoplastic and neoplastic lesions of stomach

Gastric biopsies	Hirachand et al.[41] (2018) (ns=219)	Keerthana et al.[82](2019) (ns=35)	Present study (ns=32)
Non-neoplastic lesions	87.67%	71%	62.5%
Neoplastic lesions	12.33%	29%	34.3%

In our study, 62.5% were non-neoplastic lesions of the stomach. Similarly, studies done by Hirachand et al.[41] (2018) and Keerthana et al.[82](2019) suggested non-neoplastic lesions predominance in gastric biopsies. In the present study, the most common lesion present in gastric biopsies was chronic gastritis (65.62%) of cases followed by gastric adenocarcinoma (18.76%) which was in concordance with studies done by Hirachand et al.[41] and Mishra R et al.[68]

Similarly, studies done by Parikh et al.[71] (2024), Anjana M.L.et al.[66] (2021), and Ganga H et al.[68](2018) also suggested chronic gastritis, comprising 93.6%, 52.8%, and 65.59% of cases, respectively, was the most common lesion in gastric biopsies.

A study done by Anjana M.L.et al.[66](2021) and Ujwala et al.[83](2021) suggested that in gastric biopsy cases, adenocarcinoma was the most common malignancy in the stomach, comprising 22.1% and 20% of gastric lesions, respectively similar to our study. Whereas a study done by Parikh et al.[71] and Eslavath Aruna et al.[84] showed that signet ring cell carcinoma was the most common gastric malignancy.

Compared with another study done by Rashid Sarker et al.[73](2018) suggested that out of 235 gastric biopsy cases, 63% were adenocarcinoma. Instead of gastritis, adenocarcinoma was the most common lesion overall.

There were 2 cases (6.24%) with H. pylori gastritis in the present study. Similar to this study, Parikh et al.[71] (2024) and Sandhya et al.[85] (2012) revealed 5.6% and 3.5% of cases with H. pylori gastritis. whereas 32.5% of cases with H. pylori gastritis in a study done by Elsawaf ZM et al.[86] (2017) in western Saudi Arabia .

Helicobacter pylori infection was usually seen in Asian countries like Bangladesh, India, Thailand, and Vietnam, where approximately 92%, 81%, 74%, and 75% of H. pylori infection cases were noted.[87-90]

In India, approximately more than 80% of cases showed H. pylori positive cases. Out of that, up to 3% of patients developed gastric cancer. [91,92,93] In general, studies from India do not show any proven association between H. pylori infection and gastric cancer.[91]

GEJ lesions distribution

In our study, the most common lesion from GEJ was oesophageal adenocarcinoma. Similar findings were seen in a study done by Ganga H et al.[42](2018), Sunita et al.[74](2019), and Rumana et al.[94](2005) showed that oesophageal adenocarcinoma was the most common lesion of GEJ.

Duodenal lesions distribution

In our study, the most common lesion was chronic duodenitis (76.9%) followed by adenocarcinoma (7.69%) which was in concordance with studies done by Sheik B A et al.[95](2015)

Age-wise distribution of non-neoplastic lesions

In our study, the majority of the cases of oesophagitis were observed in the seventh decade, gastritis cases were observed in the sixth decade and seventh decade, and duodenitis cases were observed in the fifth decade.

Whereas a study done by Manan et al.[96](2024) showed most of the cases of oesophagitis were seen in the third decade of life, gastritis was seen in the third and fourth decade of life; duodenitis cases were seen in the third and seventh decade of life.

Sex-wise distribution of malignancies:

Table 30: Comparison of sex-wise distribution of oesophageal squamous cell carcinoma

Study	Ratio
Ganga H et al.[42](2018)	1.37:1 (male: female)
Ujwala et al.[83](2021)	2.2:1 (male: female)
Present study	1.07:1 (female: male)

In our study for oesophageal squamous cell carcinoma, the female-to-male ratio was 1.071:1, with a slight female preponderance. Similarly, a study done by Begum et al.[65] (2024) suggested female preponderance. It was thought that tobacco consumption was higher in rural females than males.

Comparing the study done by Ganga H et al.[42] (2018) and Ujwala et al.[83](2021) also showed male preponderance for SCC of oesophagus. A study done by Anjana M.L.et al.[66](2021) and Ahamed et al.[3](2022) suggested males were more habitual for alcohol and smoking thus male preponderance was seen in most of the studies.

Table 31: Comparison of sex-wise distribution of gastric adenocarcinoma

Study	Male: female ratio
Durani et al.[97] (2009)	1.4:1
Krishnappa Rashmi et al.[64](2013)	2.2:1
Ujwala et al.[83](2021)	2.66:1
Somani NS et al.[67](2018)	3.2:1

In all-above mentioned studies male preponderance was seen which was similar to our study where the male: female ratio for gastric adenocarcinoma was 2:1.

Age-wise distribution of malignancies:

In our study, most of the cases of oesophageal carcinoma were seen in the sixth to eighth decade of life, and gastric/gastroesophageal junction malignancies were most commonly seen in the sixth and seventh decade of life.

Oesophageal carcinoma was most commonly seen in the sixth and seventh decade of life in studies done by Panjeta et al.[98](2012), Somani NS et al.[67](2018), and Keerthana et al.[82](2019) which was in concordance with our study. A study done by Somani NS et al.[67](2018) and Mabula et al.[99](2012) showed gastric carcinoma was most commonly seen in the fifth and sixth decade of life.

Site-wise distribution of malignancies:

Table 32: Comparison of site-wise distribution of oesophageal malignancies

Malignancy distribution	Upper oesophagus	Middle oesophagus	Lower oesophagus
Mishra R et al.[68](2022)	7.14%	21.43%	71.43%
Somani NS et al.[67](2018)	10.5%	57.9%	31.6%
Present study	20.68%	58.62%	20.68%

In our study, most of the oesophageal malignancies were seen in the middle oesophagus comprising 58.62%. Similar findings were seen in a study done by Somani NS et al.[67](2018). Studies done by Parikh et al.[71] and Tushar Kamble et al.[100](2022) also suggested concordance with the present study. Whereas a study done by Mishra R et al.[68](2022) suggested SCC was the most common malignancy in the oesophagus, but most of the cases were from the lower oesophagus.

Table 33: Comparison of site-wise distribution of stomach malignancies

Malignancy distribution	Pyloric antrum	Gastric body	Fundus/cardia
Ganga H et al.[42](2018)	71.7%	28.3%	13.3%
Present study	88.88%	11.11%	-

In our study, most of the stomach malignancies were from pyloric antrum comprising 88.88%. Similar findings were seen in studies done by Ganga H et al.[42](2018) where maximum stomach malignancies were found in pyloric antrum comprised 71.7%.

Endoscopy and histopathology correlation of oesophageal malignancy

Oesophageal malignancy was most commonly presented as cauliflower growth in our study. oesophageal malignancies were also presented as ulceroproliferative growth and strictures, which were commonly seen in studies done by Mishra R et al.[68](2022) and Tushar Kamble et al.[100](2022) in oesophageal carcinoma.

In our study endoscopy findings were correlated with histopathology findings in 28 out of 29 cases (96.5%) of oesophageal malignancies. Similarly to our study, a study done by Krishnappa Rashmi et al.[64](2013) suggested that 91% correlation between endoscopy and histopathology, 10 out of 11 cases were detected on endoscopy. Because oesophageal malignancies are present in old age, diagnosis becomes easy on endoscopy. A study by P Uma Rani et al.[69](2017) revealed that endoscopy detected 17 out of 18 cases of oesophageal carcinoma. So, the endoscopy and histopathology correlation in oesophageal carcinoma was 94.44%.

Endoscopy and histopathology correlation of gastric malignancy

Gastric erosions was the most common finding in chronic gastritis patients, and adenocarcinoma was presented as ulceroproliferative growth on endoscopy. Similarly, a study done by Mishra R et al.[68] (2022) revealed that on endoscopy, gastritis showed erosions. Adenocarcinoma was presented as ulceroproliferative growth on endoscopy.

In our study, endoscopy findings were correlated with histopathology findings in 5 out of 9 cases (55.5%) of gastric malignancies. A study done by Krishnappa Rashmi et al.[64](2013) suggested that 14 out of 19 gastric malignancies were detected on endoscopy suggesting a 74% correlation between endoscopy and histopathology because these malignancies are difficult to distinguish because they can present as ulcers in younger individuals. In our study, two gastric erosions turned out to be signet ring cell carcinoma on histopathology. A study done by P Uma Rani et al.[69](2017) showed that in gastric malignancy, endoscopic and histopathology findings were correlated in 19 out of 25 cases (76%) of gastric malignancies.

Endoscopy and histopathology correlation of duodenal malignancy

In our study, endoscopy findings correlated with histopathology findings in 1 out of 3 cases (33.3%) of duodenal malignancies. A study done by Keerthana et al.[82](2019) suggested that endoscopy and histopathology findings were correlated in 2 out of 4 duodenal malignancies suggesting a 50% correlation between endoscopy and histopathology.

Endoscopy and histopathology findings correlation in the present study was 83.7% of cases overall. ($p < 0.0001$) Similar to our results, Krishnappa Rashmi et

al.[64](2013), Mishra R et al.[68](2022) and Somani NS et al.[67](2018) showed there was a correlation between endoscopy and histopathology findings in 78%, 80% ($p < 0.000416$) and 91% of cases overall out of 100 endoscopic biopsies studied.

Limitations of the Study:

Inadequate biopsies were not taken into consideration.

CONCLUSION

This study is conducted to evaluate the histopathology of upper gastrointestinal endoscopic biopsies, correlate endoscopy and histopathology findings, determine the distribution of the lesions (neoplastic and non-neoplastic), identify the most common malignancies, detect H. pylori in gastric biopsies. Histopathology is more reliable as it confirms diagnosis when endoscopic findings vary. Thus, endoscopy findings should be complimented with histopathological examination of biopsy to provide definitive diagnosis and appropriate management in patient care.

SUMMARY

An observational study was conducted on 135 upper gastrointestinal endoscopic biopsies received in the histopathology section for histopathological evaluation and their correlation with endoscopic findings.

1. The most common site for biopsy was oesophagus (36.29%) followed by duodenum (28.9%) and stomach (23.70%).
2. Overall cases showed male preponderance male: female ratio 1.81:1.
3. The age of patients varied from 10-85 years Most of the lesions in patients occurred in age group of 50-59 years.
4. Pain in abdomen (30.89%) followed by nausea/vomiting (26%) was most common complaint overall. Dysphagia was present in all cases of oesophageal carcinoma.
5. Overall, 59.24 % of cases were non-neoplastic and 40.76% of cases were neoplastic lesions on histopathology.
6. The most common lesion as well as malignancy present in oesophageal biopsies was SCC of oesophagus (55%) followed by esophagitis (18.36%).
7. The most common lesion present in gastric biopsies was chronic gastritis (65.62%) followed by gastric adenocarcinoma (18.76%).
8. The most common lesion from gastroesophageal junction was adenocarcinoma (30%).
9. The most common lesion in duodenal biopsies was chronic duodenitis (79.4%).

10. majority of the cases of oesophagitis were observed in seventh decade, gastritis cases were observed in sixth decade and seventh decade, and duodenitis cases were observed in fifth decade.
11. Oesophageal SCC showed female: male ratio was 1.071:1 with slight female preponderance whereas gastric adenocarcinoma showed male: female ratio was 2:1.
12. Most of the cases of upper GI malignancies were seen between 6^{th} to 8^{th} decade of life.
13. Most of the oesophageal malignancies were seen in middle oesophagus (58.62%) (majority were squamous cell carcinoma)
14. Most of the stomach malignancies were from Pyloric antrum comprised total of 88.88%.
15. Endoscopy findings correlated with histopathology findings in 96.5% of oesophageal malignancies, 55.5% of stomach malignancies, 75% of GEJ malignancies and 33.3% of duodenal malignancies.
16. Overall endoscopy and histopathology findings correlation were present in 85.7% of oesophageal biopsies, 78.1% of gastric biopsies, 70% of gastroesophageal junction biopsies and 84% of duodenal biopsies
17. Overall, 113 out of 135 cases showed endoscopy and histopathology findings correlation. Thus, endoscopy with histopathology correlation was 83.7% of cases. ($p<0.0001$).

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ANNEXURE-I

PROCEDURES

Hematoxylin and Eosin (H&E) staining:

Harris hematoxylin solution:

Harris alum hematoxylin, 2.5 g

Absolute alcohol, 100%, 25 ml

Ammonium or potassium alum, 50 grams

Distilled water, 500 ml

Mercuric oxide 1.25 g or sodium iodate 0.5 g

Glacial acetic acid, 20 ml

Take a 2-liter jar with absolute alcohol and dissolve hematoxylin in the jar. In another 2-liter jar, dissolve potassium alum. Mix the two solutions, and then boil the mixture. Add sodium iodate or mercuric oxide. Then let it cool down with the help of cold water. After cooling down, glacial acetic acid is added to give accurate nuclear staining.

Eosin y:

Eosin, 10 grams

Distilled water, 50 ml

Glacial acetic acid, 10 ml

95% alcohol

Procedure:

1. The slides with tissue sections are incubated for 1 hour.
2. Remove paraffin wax by putting it into xylene I and II for 3 minutes.
3. Rehydrate with absolute alcohol (100%) and then put the smears in the descending grades of alcohol (90%, 80%, and 70%) for 30–60 seconds each.
4. Wash with water for 5–10 minutes.
5. Stain the tissue section with Harry's hematoxylin for 5–15 minutes.
6. Wash with running tap water till the tissue sections turn blue for 3 to 5 minutes.
7. Decolorize with 1% acid alcohol rapidly by 4 to 5 dips (1% HCL in 70% alcohol) for 5–10 seconds.
8. Wash in water or dip the section in diluted ammonia till it appears blue, then wash in water for 5 minutes.
9. Stain with eosin Y for 10 minutes.
10. Wash with water for 5–10 minutes.
11. Dip the slide in 95% alcohol for 30–60 seconds.
12. Dip into absolute alcohol. 2 changes, 30–60 seconds for each.
13. Put the slide in xylene I and II for 2 minutes.
14. Drain excess xylene and mount on DPX with a coverslip

Results:

Nuclei: blue/black

Cytoplasm: pink

Muscle fibers: deep pink or red

RBCs: orange/red

Fibrin: deep pink

Giemsa staining:

In this study for better visualization of H. pylori bacteria Giemsa stain was performed.

Working Giemsa solution:

Giemsa stock: 4 ml (Giemsa stain powder 4 grams, glycerol 250 ml, methanol 250 ml)

Acetate-buffered distilled water pH 6.8: 96 ml

Procedure:

1. Paraffin sections are fixed with Zenker's fixative.
2. Put it into different grades of alcohol.
3. Rinse in distilled water at pH 6.8.
4. Stain with working Giemsa and keep it overnight.
5. Wash in running tap water.
6. Put 0.2 % acetic acid rapidly, which acts as a differentiator, then put it in running tap water.
7. By putting different grades of alcohol into it, it quickly dehydrates.
8. Drain excess xylene and mount on DPX with a coverslip

Results:

Nuclei: blue

Cytoplasm: pink

H. pylori and other organisms: dark blue

ANNEXURE-II

KAHERs JNMC

BELAGAVI

INFORMED CONSENT FORM

"HISTOPATHOLOGICAL SPECTRUM OF UPPER GASTROINTESTINAL TRACT LESIONS IN PATIENTS UNDERGOING UPPER GASTROINTESTINAL ENDOSCOPIC BIOPSIES: A HOSPITAL BASED OBSERVATIONAL STUDY''

Name of Student/Principal Investigator:

Name of Guide/Co Investigators:

Name of Co guide:

Objectives:

1. To study histopathological spectrum of upper GI endoscopic biopsies and correlate it with endoscopic findings of the same.
2. To know the distribution of various neoplastic and non-neoplastic lesions according to age, sex, site and brief clinical data.

Introduction: Gastrointestinal conditions are associated with mortality and morbidity. Endoscopic biopsy findings and their histopathology play a role in diagnosing malignancies as early as possible, knowing the extent of particular malignancy grade, and effectiveness of treatment and not to be confused with benign lesions due to

similar clinical presentation by using both tools effectively. In the mismatch between endoscopic findings and histopathological diagnosis, histopathology is the definitive.

Explanation of procedure: Patients who will be undergoing upper GI endoscopic biopsies from OPDs and wards, for suspected upper GIT disorders, endoscopic findings will be noted, histopathology examination of these biopsies will be done for the same in the histopathology department, KLE'S Dr. Prabhakar Kore Hospital & Medical Research Centre and Charitable Hospital in Belagavi.

Withdrawal from participation in the study: Participation in this study is voluntary. You will be free to decide whether to participate in this study or continue participation once enrolled. In case you decide to withdraw your participation, you are free to do so. However, please convey the decision to the principal investigator.

Possible benefits from participating in the study: You will/will not have nor get any benefits by participating in this study. The data gathered will help the population at large.

Possible risks from participating in the study: There are no risks involved in participating in this study.

Privacy and confidentiality: The information collected from you will be coded, to prevent any person from identifying you. Your identity will never be revealed. The data collected from you will be kept confidential and only processed or aggregated data will be used for publication.

Financial incentives: You will not receive any payment for participating in this study.

Authorization for publication of aggregated data: Results obtained after processing of the aggregated data will be published for scientific purposes and or presented to scientific groups. However, your identity will never be revealed.

Questions: In case of any questions with regard to this study, you are free to contact: If you have any question or complaints with regard to your right as study participant you may contact Dr Harsha Hegde, Chairperson, Ethical committee of JNMC, 0831-2473777 Extension 4052.

Legal rights: By signing this consent form, we are not waving any of your legal rights.

CONSENT STATEMENT

I am making a voluntary decision to participate in the study **"Histopathological spectrum of upper gastrointestinal tract lesions in patients undergoing upper gastrointestinal endoscopic biopsies: A Hospital based observational study"** My signature below indicates that I have decided to participate and I have read the information provided above or the information provided above has been read to me in the language that I understand best. I was given the opportunity to ask questions and that they have been answered to my satisfaction.

Name of the participant:

Signature or left thumb impression of the participant:

Name of the witness:

Signature or left thumb impression of the witness:

Name of the investigator:

Signature of the investigator:

ANNEXURE-III

PROFORMA

"HISTOPATHOLOGICAL SPECTRUM OF UPPER GASTROINTESTINAL LESIONS IN PATIENTS UNDERGOING UPPER GASTROINTESTINAL ENDOSCOPIC BIOPSIES"

BIOPSY NO IP/OP.NO.

NAME:

AGE: SEX:

ADDRESS:

BRIEF CLINICAL HISTORY:

PRESENTING COMPLAINTS:

Pain in epigastrium Diarrhoea

Nausea vomiting Loss of appetite or weight

Dysphagia Hematemesis/melena

Dyspepsia

CLINICAL DIAGNOSIS:

NEED FOR ENDOSCOPIC BIOPSY:

ENDOSCOPIC FINDINGS:

HISTOPATHOLOGY DIAGNOSIS:

ANNEXURE-IV

KEY TO MASTERCHART

ANT	-	ANTRUM
B.NO.	-	BIOPSY NUMBER
B.SITE	-	BIOPSY SITE
BGHYP	-	BRUNNER GLAND HYPERPLASIA
CA DUO.	-	CARCINOMA DUODENUM
CA ESO.	-	CARCINOMA ESOPHAGUS
CA STO.	-	CARCINOMA STOMACH
CD	-	CHRONIC DUODENITIS
CDFI	-	CHRONIC DUODENITIS WITH FOCAL EROSION
CELIAC DS	-	CELIAC DISEASE
CG	-	CHRONIC GASTRITIS
CLD	-	CHRONIC LIVER DISEASE
CLI.DX	-	CLINICAL DIAGNOSIS
CU.GRTH	-	CAULIFLOWER GROWTH
DI	-	DUODENITIS
DIA	-	DIARROHEA
DU	-	DUODENUM
DUE	-	DUODENAL EROSIONS
DYS	-	DYSPLASIA
DYSP	-	DYSPEPSIA
DYSPHG	-	DYSPHAGIA
ED	-	EOSINOPHILIC DUODENITIS

EEI	-	EOSINOPHILIC ESOPHAGITS
EI	-	ESOPHAGITIS
EI/ULCER	-	ESOPHAGITIS WITH ULCER
ENDO.DX	-	ENDOSCOPY DIAGNOSIS
ERYM	-	ERYTHEMA
EXGR	-	EXOPHYTIC GROWTH
FRMASS	-	FRIABLE MASS
FU,B,DU	-	FUNDUS,BODY (STOMACH), DUODENUM
G.BODY	-	GASTRIC BODY
GAER	-	GASTRIC EROSIONS
GASTROP	-	GASTROPATHY
GEJ	-	GASTROESOPHAGEAL JUNCTION
GERD	-	GASTROESOPHAGEAL REFLUX DISEASE
GI	-	GASTRITIS
GLOBOMA	-	GLOBULAR MASS
GOO	-	GASTRIC OUTLET OBSTRUCTION
H.GI	-	H.PYLORI GASTRITIS
HEMT/MEL	-	HEMATEMESIS /MELENA
HISTO.DX	-	HISTOPATHOLOGY DIAGNOSIS
HYPS	-	HYPERPLASIA
IM	-	INTESTINAL METAPLASIA
IP/OP NO.	-	INPATIENT OUTPATIENT NUMBER
K.CA ESO	-	KNOWN CASE OF CA ESOPHAGUS
K.CA PYRF	-	KNOWN CASE OF CARCINOMA PYRIFORM FOSSA
L.YMPHCS	-	LYMPHANGECTASCIA

LA	-	LOSS OF APPETITE
LEUVAS	-	LEUCOCLASTIC VASCULITIS
LN	-	LUMINAL NARROWING
LOW ESO	-	LOWER OESOPHAGUS
LW	-	LOSS OF WEIGHT
M.ADECA	-	MODERATLY DIFFERENTIATED ADENOCARCINOMA
M.DYS	-	MODERATE DYSPLASIA
M.SCC	-	MODERATLY DIFFERENTIATED SQUAMOUS CELL CARCINOMA
MAS	-	MALABSORPTION SYNDROME
MI.AD,SCC	-	MIXED ADENO & SQUAMOUS CA
MI.DYS	-	MILD DYSPLASIA
MID ESO	-	MIDDLE OESOPHAGUS
NAU,VOM	-	NAUSEA,VOMITING
NET	-	NEUROENDOCRINE TUMOR
NOD	-	NODULE

ANNEXURE –V MASTER CHART

SL. NO.	B.NO.	IP/OP NO.	AGE	SEX	B.SITE	CLINICAL FEATURES									CLI.DX	ENDO.DX	HISTO.DX
						DYSPHG	PA	NAU,VOM	DYSP	LW	LA	DIA	HEMT/MEL	OTHERS			
1	28/23	5108951	70	F	MID ESO	1									CA ESO.	CU.GRTH	M.SCC
2	29/23	6878162	26	M	DU							1			MAS	DI	CD
3	45/23	1161969	39	M	MID ESO						1				EI	EI,GI,DI	EI/ULC
4	47/23	1161890	38	M	DU		1	1							DI	EI,DI	ED
5	68/23	1162451	60	F	DU							1			DI	EI,DI	CD
6	69/23	1162678	40	F	DU							1			DI	EI,DI	CD
7	147/23	6888954	52	M	ANT				1						CA STO.	ULPRGR	M.ADECA
8	159/23	6239443	26	M	DU		1			1					DI	EI,DI	CD
9	164/23	1163420	24	M	LOW.ESO						1				EI	EI,DI	EI/ULC
10	234/23	1164223	73	F	MID ESO			1							CA ESO.	STR	M.SCC
11	243/23	1164922	40	M	DU			1							DI	EI,DI	CD
12	246/23	6898429	75	M	GEJ	1									CA ESO.	ULPRGR,LN	M.ADECA
13	260/23	6899354	47	M	DU		1				1				DI	EI,DI	CD
14	261/23	6317023	78	M	LOW.ESO	1									CA ESO.	ULC	EI/ULC
15	262/23	5119817	40	F	DU			1				1			DI	EI,DI	CD
16	286/23	5869064	45	F	UP.ESO	1									CA ESO.	CU.GRTH	M.SCC
17	2808/23	7093742	55	F	GR.CUR			1							POLYP	POLYP	CGIM
18	451/23	6914951	45	M	DU			1							CELIAC DS	DUE	CD

19	450/23	6914952	27	M	DU			1							CELIAC DS	DI	CD
20	449/23	6913834	66	M	UP.ESO	1									CA ESO.	POLYPDG	P.SCC
21	415/23	6911159	61	M	UP.ESO	1									CA ESO.	PC.NR	M.SCC
22	400/23	1166867	80	M	PYLORUS			1							POLYP	POLYPDG	PDNS
23	312/23	1165868	68	M	DU			1							MAS	DI	CD
24	357/23	6906915	45	F	DU			1							MAS	EI,DI	ED
25	306/23	1167878	60	F	MID ESO	1									CA ESO.	CU.GRTH	M.SCC
26	1175/23	1176261	65	M	MID ESO	1									CA ESO.	EI	EI/ULC
27	1164/23	1176264	10	M	DU		1					1			MAS	EI,DI	CD
28	1150/23	6972226	22	M	DU		1	1							POLYP	EI,DI	CD
29	1233/23	6097723	56	M	DU	1									POLYP	NORMAL	NET
30	1235/23	1177382	48	M	GEJ		1								EI	TH.MU	BARRET
31	1254/23	6981963	48	F	MID ESO	1									CA ESO.	CU.GRTH	P.SCC
32	1255/23	6981151	60	F	LOW.ESO	1									CA ESO.	CU.GRTH	M.SCC
33	1285/23	6246040	63	M	DU		1								MAS	BARRET	CDFI
34	1286/23	5507683	65	F	ANT		1	1							POLYP	GAER	CGIM
35	1287/23	4976983	42	F	DU			1							MAS	EI,DI	CDFI
36	1329/23	6985279	28	M	UP.ESO	1									CA ESO.	ULC	MI.DY
37	1335/23	6310517	72	F	LOW.ESO	1									CA ESO.	ULPRGR	M.SCC
38	1149/23	5977799	52	M	MID ESO		1								EI	ULC	EI/ULC
39	1115/23	5977898	33	M	DU		1					1			DI	DI	CD
40	1114/23	5976892	25	M	DU		1	1							DI	DI	CD
41	1059/23	6962979	76	F	LOWESO	1									CA ESO.	POLYP	HYPS
42	1032/23	1174961	39	F	DU							1			MAS	DI	CD
43	994/23	6957906	78	F	MID ESO	1									CA ESO.	CU.GRTH	M.SCC
44	1004/23	6957906	17	F	DU		1	1							MAS	DI	CD

45	3068/23	3893675	72	F	UP.ESO	1				1					CA ESO.	PRGR,LN	M.SCC
46	3069/23	1199197	78	M	LOW.ESO								1		EI	EI	EI/ULC
47	3070/23	1199999	41	M	PYLORUS				1						CA STO.	ULPRGR,LN	PDNS
48	3067/23	1192278	42	F	DU		1								MAS	DI	CD
49	2131/23	1188988	50	M	DU		1	1							CA DUO	TH.MUD1	M.ADECA
50	2071/23	1188121	50	F	FU,B,DU		1	1							GI,DI	GAER,DI	CG,CD
51	2127/23	1188849	70	F	MID ESO		1								K.CA ESO.	PCAS	M.DYS
52	2065/23	1188219	66	M	DU			1							DI	DI	CD
53	2040/23	1187236	78	M	DU									YELSCL	PER CA	EXGR	M.ADECA
54	2013/23	5020340	50	F	GEJ			1							K.CA ESO.	GAER	CG
55	1929/23	1186281	70	M	ANT,DU					1	1				NET	GAER	NET,CG
56	1925/23	7032207	66	F	LOW.ESO	1									CA ESO.	CU.GRTH	M.SCC
57	1905/23	7030963	50	F	UP.ESO		1								CA ESO.	FRMASS	M.SCC
58	2262/23	1124532	55	F	LOW.ESO	1									CA ESO.	POLYPDG	P.ADECA
59	2417/23	7064439	31	F	MID ESO		1								EI	ULC	EI/ULC
60	1876/23	1185742	49	M	G.BODY		1								POLYP	POLYP	CG
61	1865/23	7025479	52	M	DU							1			MAS	DI	CD
62	1864/23	7027297	82	M	DU,G.BO		1				1				MAS	GAER,DI	CG,CD
63	1822/23	7025468	49	M	MID ESO										CA ESO.	STR	HYPLS
64	1821/23	7025445	55	F	GEJ	1		1							CA ESO.	CU.GRTH	M.ADECA
65	1804/23	1183926	33	M	DU					1		1			DI	DI	CD
66	1777/23	1184922	48	M	DU		1								MAS	GASTROP	CD
67	1781/23	1184651	50	F	DU		1			1					DI	DI	CD
68	1760/23	1188777	75	F	MID ESO	1									CA ESO.	CU.GRTH	P.SCC
69	3193/23	1201616	42	F	DU		1					1			DI	DI	CD
70	3194/23	1202087	52	M	MID ESO	1									CA ESO.	PRGE,LN	M.SCC

71	3288/23	1200276	78	M	LOW.ESO	1									CA ESO.	FRMASS	M.SCC
72	3306/23	1203352	70	M	G.BODY		1	1							CA STO.	VOLV	CG
73	3275/23	1202939	53	M	MID ESO	1									CA ESO.	STR	HYPS
74	3289/23	6795818	49	F	DU		1	1							DI	DI	CDFI
75	3326/23	6987058	20	M	DU		1			1					MAS	DI	CD
76	3309/23	6312849	51	M	ANT		1								COLONCA	GAER	SIGNET
77	3025/23	1198279	75	F	UP.ESO	1									CA ESO.	STR	M.SCC
78	3077/23	1200008	71	M	DU		1	1							GI,DI	GASTROP	M.ADECA
79	3070/23	1198881	45	M	DU		1	1							PER CA	NORMAL	SPI.TU
80	3235/23	1202457	66	M	UP.ESO	1									CA ESO.	CU.GRTH	M.SCC
81	3246/23	1203711	65	M	GEJ	1									CA ESO.	EI	P.ADECA
82	3168/23	7119885	38	F	MID ESO	1									CA ESO.	NOD	M.SCC
83	3245/23	6949066	28	M	GEJ		1								POLYP	POLYP	FVPOLYP
84	3169/23	7120492	50	M	MID ESO	1									CA ESO.	CU.GRTH	M.SCC
85	2459/23	1192586	52	M	MID ESO	1									CA ESO.	CU.GRTH	M.SCC
86	2464/23	7068686	58	M	MID ESO	1									CA ESO.	CU.GRTH	P.SCC
87	2448/23	1191713	59	M	ANT		1	1							GI	GAER	CG
88	2851/23	1197718	61	M	PYLORUS			1							GASTROP	GAED	CGIM
89	2914/23	1198279	75	F	UP.ESO	1									CA ESO.	NOD,LN	M.DYS
90	2909/23	1196605	40	M	MID ESO		1								EI	ERYM	MI.AD,SCC
91	2911/23	1196705	58	F	ANT									ANEMIA	GIST	GLOBMA	CG
92	2073/23	6105941	50	M	DU		1	1							L.YMPHCS	WHITSPO.	L.YMPHCS
93	3327/23	7131158	65	M	MID ESO		1	1							CA ESO.	EI	EI/ULC
94	3414/23	7131159	66	M	MID ESO	1									CA ESO.	EI	EEI
95	3430/23	1204065	77	F	DU		1	1							PER CA	POLYPDG	DYS
96	3444/23	1203355	50	M	PYLORUS		1	1							CA STO.	GAER	CGIM

97	1527/23	7004428	50	M	MID ESO	1									CA ESO.	CU.GRTH	P.SCC
98	1528/23	7004073	55	M	MID ESO	1									CA ESO.	CU.GRTH	P.SCC
99	1578/23	1182251	65	F	ANT		1	1							GI	GAER	NET
100	4609/23	5497427	77	M	ANT		1	1							GI	NOD,ULC	CGIM,H.GI
101	4608/23	1009476	45	F	G.BODY				1						CA STO.	TH.MU	M.SCC
102	4684/23	1011265	46	F	G.BODY					1					GI	GAER,ULC	H.GI,DYS
103	4685/23	1010458	56	F	LOW.ESO			1							POLYP	POLYP	FVPOLYP
104	4702/23	1011489	35	M	DU		1	1							VASC.	EI,DUE	LEUVAS
105	4703/23	7213058	53	M	LOW ESO	1									CA ESO.	CU.GRTH	M.SCC
106	4765/23	7218283	75	M	LOW ESO		1								LIPOMA	NOD,DUE	HYPLS
107	4836/23	10013621	42	M	ANT		1	1							GERD	GAER	CG
108	4832/23	7105138	63	M	MID ESO		1								K.CA ESO.	TH.MU	HYPLS
109	4903/23	7225984	24	M	ANT		1	1		1					GI	ULC	CG
110	4932/23	7009073	40	M	LOW ESO									RSBURN	BARRET	EI	BARRET
111	4931/23	7227812	63	F	ANT		1	1	1						GI	GAER,DI	CG
112	5039/23	7177837	61	F	GEJ				1						K.CA PYRF	TH.MU	CG
113	5085/23	10017179	32	M	PYLORUS		1	1							CA STO.	EI,TH.MU	UR
114	5101/23	10017222	61	F	DU		1	1							PER CA	DI	CD
115	5103/23	7237002	37	M	GEJ		1								EI	NOD	ULC,HYPLS
116	5157/23	10017959	77	M	DU		1	1					1		CLD	GASTROP	NET
117	5158/23	7218349	21	F	ANT		1								GI	GAER	CG
118	5190/23	6789679	62	F	FUNDUS				1						GI	GAER	CGIM
119	5256/23	7247352	85	M	GEJ	1									CA ESO.	FRMASS	P.SCC
120	5270/23	10018928	38	M	ANT,DU		1								GI,DI	GAER,DUE	CG,CD
121	5271/23	7248431	72	M	UP.ESO	1									CA ESO.	ULPRGR	P.SCC
122	5332/23	10020704	70	M	PYLORUS,DU		1	1							GOO	EI,DI	IM,BGHYP

123	5372/23	7257140	40	M	GEJ			1							POLYP	POLYP	BARRET
124	5401/23	10021893	65	M	ANT		1								POLYP	POLYP	CG
125	5420/23	10021668	65	M	PYLORUS		1								GI	GAER	SIGNET
126	5419/23	7260593	65	M	PYLORUS		1								CA STO.	POLYP	CGIM
127	3022/23	1197376	58	M	ANT				1						CA STO.	ULPRGR	PDNS
128	3625/23	1206953	55	M	G.BODY			1	1						GI	GAER	CG
129	3742/23	1208832	32	F	ANT				1						GI	GAER	CG
130	3980/23	7171407	64	M	ANT				1						CA STO.	ULPRGR	M.ADECA
131	3983/23	7171189	78	M	ANT			1							GI	GAER	CG
132	3021/23	1197815	79	M	LOW ESO	1									CA ESO.	CU.GRTH	M.SCC
133	3658/23	1207579	77	M	MID ESO	1									CA ESO.	TH.MU	HYPLS
134	2462/23	6911159	61	M	FUNDUS		1	1							GI	GAER	CG
135	2182/23	7045108	75	F	ANT			1							CA STO.	TH.MU	M.ADECA
						39	55	46	10	8	5	10	2	3			