SPECTRUM OF HISTOMORPHOLOGICAL PATTERNS OF UPPER GASTROINTESTINAL TRACT ENDOSCOPIC BIOPSIES

By

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Under the Guidance of

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When you come to the end of your rope, tie a knot and hang on.

~Franklin D. Roosevelt

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LIST OF ABBREVATIONS USED

GI	Gastrointestinal
UGIT	Upper gastrointestinal tract
UGE	Upper GI endoscopy
GERD	Gastroesophageal reflux disease
AIDS	Acquired immunodeficiency syndrome
I.V.	Intravenous
FNAC	Fine needle aspiration cytology
HPF	High power field
NSAIDS	Non steroidal antiinflamatory drugs
H. pylori	Helicobacter pylori
IHC	Immunohistochemistry
COPD	Chronic obstructive pulmonary disease
CRF	Chronic renal failure
NHL	Non-Hodgkin’s lymphoma

ABSTRACT

BACKGROUND

Upper gastrointestinal (GI) endoscopy is a safe and well tolerated procedure. However endoscopy alone is insufficient to diagnose mucosal lesions in about 15-30% of cases. In these cases histopathological examination can be useful for the diagnosis of the upper GI lesions. Thus endoscopy in combination with biopsy acts as a useful adjunct for diagnosis of upper GI lesions and plays an important role in management of patients.

OBJECTIVE

To study the histomorphological patterns and frequencies of lesions in upper GI endoscopic biopsies.

MATERIALS

Upper GI endoscopic biopsies from July 2010 to July 2014 were studied. Endoscopic biopsies done for lesions in esophagus, stomach, first and second part of duodenum up to the opening of common bile duct were taken. The biopsy specimens were stained with Hematoxylin and Eosin. Other special stains like PAS, Giemsa and immunohistochemistry were done wherever required.

RESULTS

Total 196 upper GI biopsies were studied. 83(42.34%) were from esophagus, 47(23.97%) cases were from stomach, 49(25%) were from duodenum and 15(7.65%) were from gastroesophageal junction. One case was from esophagogastric anastomosis site in a case of post trans-hiatal esophagectomy(0.5%) . Male to female

ratio was 2.26. Overall non-neoplastic lesions of upper GI biopsies were equal to neoplastic lesions. In esophagus most common lesion was squamous cell carcinoma. Among non neoplastic lesions, chronic esophagitis was more common. In stomach most commonly diagnosed lesion was adenocarcinoma followed by chronic gastritis.

In duodenum there were 41 cases of chronic duodenitis and there was one case of well differentiated adenocarcioma of periampullary region.

CONCLUSION

Endoscopic biopsy leads to an early diagnosis of various upper GI lesions. Hence the present study was done to determine the spectrum of upper GI lesions that help in early therapeutic decisions and management of the patients.

KEY WORDS: Endoscopic biopsy, histomorphology, upper gastrointestinal tract

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INTRODUCTION

Upper gastrointestinal (GI) endoscopy is a visual examination of the upper gastrointestinal tract (UGIT) using a lighted, flexible fibreoptic or video endoscope.[1] The use of flexible fibreoptic gastroscope is now a part of routine gastroenterological practice. Endoscopic examination is recommended with negative Barium X-rays, in patients with dyspeptic symptoms.[2] For most upper gastrointestinal lesions, the sensitivity and specificity of endoscopy is about 90% and nearly 100% respectively which is far higher than for barium radiography where sensitivity and specificity is about 50% & 90% respectively.[3]

The major advantages of endoscopy over contrast radiography in evaluation of diseases of the alimentary tract is direct visualization, resulting in a more accurate and sensitive evaluation of mucosal lesions. Other advantages are the ability to obtain biopsy specimens from superficial lesions and the ability to perform therapeutic interventions.[3] Endoscopic appearance may be valuable in diagnosis of mucosal lesions but more accurate and detailed information results from histological examination of mucosal biopsy specimens.[4]

The indications of upper GI endoscopic (UGE) biopsy includes – evaluation of dyspepsia, dysphagia, GERD, Barrett oesophagus, dysplasia, peptic ulcer disease and its complications, gastric and oesophageal carcinomas.[5]

UGIT is a common site for tumors, especially malignant tumors. In India, according to the National Cancer Registry, esophageal and gastric cancers are the most common cancers found in men, while esophageal cancer ranks third among women.[6]

Endoscopy of gastrointestinal tract is a simple safe and well tolerated procedure. The visualization of proper site of mucosal lesion with biopsy leads to early detection of pathologic process and institution of early treatment.[5]

OBJECTIVES OF THE STUDY

To study the histomorphological patterns and frequencies of lesions in upper GI endoscopic biopsies.

REVIEW OF LITERATURE

Esophagogastroduodenoscopy is a procedure that visualizes the mucosal surfaces of the esophagus, stomach, and proximal duodenum and plays a major role in diagnostic and therapeutic modalities.[7]Endoscopic technology has evolved significantly in past 20 years with widespread availability of videoscopes.[8]

Adolf Kussmaul in 1868 at a meeting of the Freiburg Society of Naturalists with the good sense to use a professional sword-swallower for the demonstration – passed a hollow, rigid metal tube – the first gastroscope through the oesophagus into his subject's stomach. Illumination was provided by a Desormeaux lamp attached proximally, but visibility was poor. Leiter and Nitze developed a successful cystoscope and a crude gastroscope using the same technique. Leiter with von Mikulicz shifted the light to the distal end of the tube but retained the angulation of the shaft. The first semi flexible instrument which could be inserted into the stomach was developed by Schindler & Wolf in Germany in 1932.[9] Hirschowitz , then embarked on the construction of a flexible gastroscope in1957.[10]

Japanese surgeons working in Tokyo were concerned about the high incidence of gastric cancer in their community. Hence they developed a gastrocamera in association with the Olympus Company in early 1950s for the purpose of early diagnosis of carcinoma of stomach. A community of physicians and surgeons, as well as a commercial organization in Japan was highly sympathetic to the use of new techniques in the investigation of gastro-intestinal disease. In 1962, Professor Tadayoshi Takemoto together with the Machida and with Olympus company, developed a new generation of fibre optic instruments for endoscopy that have swept the world.[11] Various methods such as narrow-band imaging, autofluorescence imaging, Raman spectroscopy, confocal endomicroscopy, endoscopic optical

spectroscopy, and magnifying endoscopy have been developed and are under trial. These endoscopic detection methods have enabled endoscopists to collect real-time in vivo histological images or "virtual biopsies" of the GI mucosa during endoscopy.[12]

Indications of UGE biopsies include esophageal, gastric and duodenal ulcers, esophagitis, gastritis, duodenitis, polyps of upper gastrointestinal tract(UGIT), esophageal strictures, precancerous conditions - Barrett's esophagus, tumors of UGIT and in evaluation of malabsorption, iron deficiency anemia, celiac disease, AIDS enteropathy.[1,7,13]

UGE is usually performed on an outpatient basis. The patient is advised not to eat, drink or smoke during the 8 hours before the procedure to ensure that the UGIT is clear. The throat is anesthetized by local anesthetic and I.V. sedation is given to relax the patient. The patient is made to lie on the back or side of the examination table. An endoscope is carefully fed down the esophagus into the stomach and duodenum. Tract is visualized and other instruments are passed through the endoscope to perform additional procedures like biopsy or removal of a polyp or a tumor.[1,14] Tissue sampling has become an integral part of endoscopy procedure and is used to compliment endoscopic imaging. It is generally safe and effective .Various techniques include fine needle aspiration cytology, brush cytology, snare excision, and pinch forceps biopsy.[15]

Various biopsy forceps are available. Single-bite cold-biopsy forceps allow sampling of only a single specimen at a time. Double-bite forceps, are most commonly employed because they enhance directed lesion sampling via impalement of the tissue and stabilization of the forceps cups. Large-capacity or ''jumbo'' biopsy forceps sample a larger volume of tissue encompassing 2 to 3 times the surface area

compared to standard forceps. Multiple bite sampling have been developed that can obtain up to 4 or more specimens on a single pass.[15]

Danesh BJZ *et al* [16] did a comparative study of weight, depth, and diagnostic adequacy of specimens obtained with 16 different biopsy forceps. They concluded that the precise shape, design, and make of the forceps used were not of practical importance. In their study they found that bigger, deeper, and more adequate specimens were obtained by using the standard sized forceps and by applying pressure at the time of biopsy.

A study done by Fantin AC *et al* [17] to assess and compare the diagnostic quality of biopsy specimens obtained with a conventional forceps and a multibite forceps found that the quality of biopsy specimens obtained with the multibite forceps is same as that of specimens taken with a conventional forceps. They concluded that the use of multibite forceps saves time. With multibite forceps 4 specimens can be obtained in 1 pass hence is useful in situations where a large number of specimens are needed or when the potential for transmission of infection is of concern.

A study done by Kim CG [18] on tissue acquisition in gastric epithelial tumor prior to endoscopic resection showed that multiple deep biopsies can induce mucosal ulceration in early gastric cancer and they also found that ulcerative early gastric cancer was associated with piecemeal and incomplete resection. It is also associated with a higher risk of procedure-related complications such as bleeding and perforation.

Malhotra V *et al* [19] studied endoscopic techniques in the diagnosis of upper GI malignancies. They used brush biopsy, forceps biopsy, FNAC and suction cytology and concluded that forceps biopsy is the single most reliable and accepted technique when combined with any cytologic technique, the accuracy reaches 100%.

NORMAL HISTOLOGY OF UPPER GASTROINTESTINAL TRACT

Esophagus is composed of four layers namely mucosa, submucosa, muscularis propria and adventitia. Mucosa has has three components

a) Non-keratinizing stratified squamous epithelium.
b) Underlying lamina propria contains loose areolar connective tissue and scattered inflammatory cells. Finger like extensions of lamina propria, termed papillae extend into the epithelial layer usually up to one-third to one half of the thickness of the epithelial layer .In its distal portion, esophagus contains mucosal glands called as esophageal cardiac glands.
c) Muscularis mucosae is absent in upper part, distinct in the lower part of the esophagus and is thickest near the esophagogastric junction. Submucosa consists of loose connective tissue, occasional lymphoid follicles and submucosal glands.[20] Muscularis propria consists of striated skeletal muscles fibres in upper third, striated and smooth muscle fibres in the middle third and exclusively smooth muscle in the lower third of the organ. It lacks a serosal layer except in the most distal portion.[21]

Esophago-gastric junction is formed where the esophagus joins the stomach. The non-keratinized stratified squamous epithelium of the esophagus abruptly changes to the simple columnar mucin secreting gastric epithelium of the cardiac region of the stomach. At this junction esophageal glands proper may be seen in submucosa. Lamina propria of the esophagus continues into the lamina propria of the stomach where it becomes filled with gastric and cardiac glands and with diffuse lymphatic tissue.[21]

Gastric wall consists of the mucosa, submucosa, muscularis propria and serosa. Mucosa consists of lining epithelium, lamina propria and muscularis mucosae.

Mucosa is lined by simple columnar surface epithelium extending into the gastric pits into which the tubular glands open. Lamina propria is made up of loose connective tissue and fills up the spaces between the gastric glands. Muscularis mucosae is made up of a thin layer of smooth muscle fibres and consists of inner circular and outer longitudinal layer.[22] Submucosa consists of loose connective tissue with numerous elastic fibres. Muscularis externa is composed of three layers: outer longitudinal, inner circular and innermost oblique. Serosa consists of a thin outer layer of connective tissue and is covered by a simple squamous mesothelium of visceral peritoneum.[21, 22]

All the gastric glands have two major components: foveola/crypts/pit and secretory portion known as adenomere. The foveolae represent the most important area for genesis of gastric carcinoma. Gastric glands vary in different anatomic regions of stomach. In Cardia of stomach, foveolae occupy the upper half. In the lower half of cardia, glands contain either pure mucus cells or a mixture of mucus and oxyntic cells. At fundus, the foveolae occupy only 1/4th of the thickness and glands of composite cell distribution which include chief cells, parietal cells (acid secreting), endocrine cells and mucus neck cells. In antral and pyloric glands, foveolae occupy the upper half. Glands contain both mucus secreting and endocrine cells. Cytoplasm of the pyloric cells can be bubbly, vacuolated, granular or glassy.[21, 22]

Duodenum is composed of mucosa, sub mucosa, muscularis externa and serosa. Mucosa is lined by villi which are short and stubby (leaf like) in duodenum. Villous epithelium is composed of tall columnar absorptive cells (enterocytes) lined with microvilli (brush border) admixed with the lighter staining goblet cells. Between bases of villi are pit like crypts of Lieberkuhn, which contain stem cells that replenish and regenerate the epithelium.[4, 22] Normal villous to crypt height ratio varies from 3:1

to 5:1.[13] Lamina propria contains a loose connective tissue matrix containing lymphocytes, plasma cells and occasional eosinophils, macrophages, mast cells and neutrophils. Smooth muscle fibres from muscularis mucosae extend into core of individual villi and are responsible for their movements. Microvilli are cytoplasmic extensions that cover the apices of intestinal absorptive cells. Submucosa contains connective tissue and the submucosal Meissner's plexus along with numerous mucus secreting glands known as Brunner's glands. Muscularis externa is made up of an inner circular and outer longitudinal layer with the myenteric (Auerbach's) plexus, ganglion cells and perineural fibroblasts.[21, 22]

NON-NEOPLASTIC LESIONS OF ESOPHAGUS

Reflux esophagitis/ GERD is esophagitis resulting from reflux of gastroduodenal contents into esophagus. Various conditions that causes mucosal injury are hiatus hernia, defective or weak lower esophageal sphincter (LES), impaired esophageal peristalsis with transient LES relaxation, delayed gastric emptying, decreased salivary gland secretions, increased gastric acid production and bile reflux.[23]

In mild forms little or no abnormality may be seen. Mucosal erosions, ulcerations, intramural thickening, strictures or Barrett's esophagus were noted in more severe disease.[24] Many patients with clinical diagnosis of GERD have no abnormality on endoscopic examination and are labeled as endoscopic negative reflux GERD (ENRD). In such patients esophageal biopsy is useful in diagnosing the reflux disease.[25]Microscopically, intraepithelial edema, necrosis, infiltration by neutrophils and eosinophils are seen in acute cases. Chronic cases have basal cell hyperplasia, elongation of papillae and intraepithelial eosinophils. More severe cases show ulceration, granulation and submucosal fibrosis.[26]

A study done by Fiocca R *et al* [27] revealed that more number of biopsies and distal biopsies are the more informative and has higher diagnostic sensitivity in diagnosing microscopic esophagitis. They also found that the assessment of basal cell hyperplasia and papillae elongation requires well oriented biopsies.

Another study done by Brindley N *et al* [28] concluded that proper orientation of esophageal pinch biopsies improves histologic appraisal and increases the yield of esophagitis in children with GERD.

Kasap E *et al* [29] studied the correlation among standard endoscopy, narrow band imaging and histopathological findings in the diagnosis of non-erosive reflux disease and found that histopathological evaluation is most sensitive. Therefore taking a biopsy will remain useful.

Eosinophilic Esophagitis is a clinico-pathological condition characterized by esophageal and/or upper gastrointestinal symptoms (dysphagia,food impaction, GERD-like symptoms, etc); frequent association with a history of bronchial asthma; normal pH values; absent/poor response to high-dose proton pump inhibitor.[27] It occurs more frequently in young children with atopic symptoms such as eczema, asthama and food allergies.[26]

Endoscopically, mucosal rings, furrows, granularity, exudates, and mucosal fragility is seen. In long standing cases, strictures can be seen.[26] Eosinophilic esophagitis show prominent intraepithelial eosinophilia and its diagnostic criteria is presence of more than or equal to 15 intraepithelial eosinophils/HPF, especially forming microabscesses in the superficial layers of the epithelium. Other disorders which may show similar clinical, histological, or endoscopic features such as GERD should be excluded.[27, 30]

Most common forms of acute infectious esophagitis are viruses and fungi. Herpes esophagitis occurs primarily in immunosupressed patients. Endoscopically, herpetic ulcers are typically shallow, sharply punched out known as “volcano ulcers”. Microscopic diagnostic criteria for herpes esophagitis includes the presence of Cowdry A intranuclear viral inclusion bodies, ground glass nuclei, nuclear moulding , margination of chromatin and multinucleate synctial squamous cells.[22,26] Cytomegalovirus Esophagitis is also on the rise due to predilection for immunocompromised patients. Endoscopic picture shows discrete superficial ulcers in mid or distal esophagus. These coalesce to form giant ulcers. Histologically it shows cellular enlargement, prominent eosinophilic, intranuclear inclusions and occasional granular basophilic cytoplasmic inclusions.[24]

Candida Esophagitis is most commonly caused by Candida Albicans and Candida Tropicalis. It occurs in patients with AIDS, diabetes, on antibiotic and immunosuppressant therapy. Endoscopically ,esophageal candidiasis typically appears as white plaques. Pseudohyphae and budding yeast forms can be demonstrated histologically in a background of active esophagitis.[23,26] Primary bacterial esophagitis is very rare and if occurs, it is caused by the normal flora of mouth and upper respiratory tract i.e. Staphylococcus Aureus, Staphylococcus Epidermidis, Streptococcus pyogenes and Bacillus species. Histologically, bacterial infections produce a diffuse acute necrotizing process characterized by intense neutrophilic exudates, cellular necrosis and degeneration.[24]

Chagas disease is a well known parasitic infection caused by the parasite Trypanosoma Cruzi. It is a chronic infection, subsequently progressing to megaesophagus.[24] Chemical Esophagitis is caused by a variety of irritants such as

alcohol, corrosive acids or alkalis. It may also be caused when medical pills lodge & dissolve in the esophagus i.e. Pill Esophagitis.[26]

PRE NEOPLASTIC LESION

Barrett's esophagus(BE) is recognized endoscopically by columnar metaplasia of any length and histologically it shows specialized columnar epithelium lining a segment of distal esophagus above the level of the lower esophageal sphincter. It is seen most commonly in adults due to ulceration caused by GERD and subsequent re-epithelialisation of esophageal squamous mucosa by columnar cells. These columnar cells differentiate from multipotential stem cells lying in the base of the mucosa. BE can also result from other noxious substances such as reflux of bile salts, lysophospholipids and pancreatic enzymes.[24] Metaplastic change from squamous to columnar epithelium goes through an intermediate stage known as multilayered epithelium which is characterized by basally located squamoid cells and superficial mucinous columnar cells.[31] Endoscopically, patches of red, velvety mucosa extending upward from the GEJ are seen which are described as salmon colored mucosa. The columnar mucosa extends proximally circumferentially in a continuous sheet, in the form of finger like projections or as isolated islands. Depending on the length of the mucosa involved, BE is sub classified as long segment BE (>3 cm) and short segment BE (<3cm, difficult to detect on endoscopy).[23, 24]

Microscopically, Barrett's esophagus is of three major types. a) Fundic type containing parietal cells and chief cells. b) Junctional type or cardiac type containing mucous secreting columnar cells of cardiac type. c) Distinctive type with specialized intestinal epithelium containing goblet cells. It is sometimes characterized by a villiform surface and crypts with a mixed population of columnar and goblet cells.[24] When these goblet cells are admixed with gastric type lining cells and is devoid of

absorptive and Paneth cells it is called as incomplete intestinal metaplasia.[27,31] Distinctive type Barrett's esophagus are most susceptible to development of dysplasia and adenocarcinoma.[31]

In the study done by Khandwalla HE *et al* [32] they found that most (71%) patients were suspected to have columnar lined esophagus(CLE) on endoscopy. But these patients were negative for intestinal metaplasia on biopsy for 2 years following endoscopy. The findings support withholding BE diagnosis for individuals with suspected CLE.

Grading of dysplasia in Barrett's esophagus is classified in the following categories (a)Negative for dysplasia (b) Indefinite for dysplasia (c) Low grade dysplasia (d) High grade dysplasia (e) Intramucosal carcinoma.[31]

A study done by Sandick JWV *et al* [33] revealed that adenocarcinoma in BE develops through stages of increasing severity of dysplasia and that endoscopic biopsy surveillance permits early detection of malignancy thereby reducing mortality from esophageal adenocarcinoma.

TUMORS OF ESOPHAGUS

Squamous Cell Carcinoma (SCC) is the commonest malignant tumour in the esophagus, affecting males more commonly. Its peak incidence is in the 5th to 6th decade. There is a marked geographic variation in incidence, the highest being in China, South Africa, and central Asia and low in Europe and North America.[22, 34]

The risk factors for esophageal SCC include alcohol, tobacco use, poverty, caustics, esophageal injury, achalasia, tylosis, stricture, Plummer- Vinson syndrome, polycyclic hydrocarbons, nitrosamines and other mutagenic compounds and history of previous irradiation. Recent studies have suggested role for human papilloma viruses, especially types 16 and 18, in the pathogenesis of some esophageal cancers. The

molecular pathogenesis of SCC is not well defined but loss of tumor suppressor genes like P53 and P16/ INK4a have been implicated. The onset of esophageal SCC is insidious and ultimately produces dysphagia, odynophagia (pain on swallowing), and obstruction. The most common site is middle one third of esophagus (50%) followed by lower third.[22, 23, 34]

Esophageal SCC appears as circumferential, often ulcerated growth with sharply demarcated margins. Early lesions often appear as small, grey white plaque like thickenings. Later it may be polypoidal or exophytic and protrude into the lumen. Histologically, SCC of the esophagus show a range of differentiation from abundantly keratinized, well-differentiated lesions containing prominent intercellular bridges to poorly differentiated, anaplastic, large or small cell tumors in which morphologic evidence of squamous differentiation can only be identified after prolonged searching. The variants include basaloid SCC, adenosquamous carcinoma (evidence of both SCC and malignant glandular counterpart), small cell carcinoma, sarcomatoid carcinoma, lymphoepithelioma like carcinoma and verrucous carcinoma.[22, 23, 34]

Adenocarcinoma of esophagus typically arises in a background of BE and long standing GERD. The risk is greater in those with documented dysplasia and further increased by tobacco use, obesity and prior radiation therapy. It is more common in men than women and molecular studies suggest its association with mutation of P53 gene, loss of chromosome 17p allele and C-erb-B2 overexpression. Esophageal adenocarcinoma usually occurs in the distal third of the esophagus and may invade the adjacent gastric cardia.[23, 35]

Endoscopically, they appear as flat or raised patches in an otherwise intact mucosa and may progress to large masses of 5 cm diameter. Microscopically, most examples are tubular or papillary adenocarcinomas of intestinal pattern and show

variable differentiation. Adenocarcinoma of esophagus most commonly produce mucin and BE is frequently present adjacent to the tumor. Some tumors have the pattern of mucinous adenocarcinoma, with prominent extracellular mucus production, but the diffuse type of signet-ring carcinoma is very unusual. Non-Barrett's associated esophageal adenocarcinomas are extremely rare and are derived from heterotopic gastric mucosa located in the upper esophagus.[34, 35]

NON-NEOPLASTIC LESIONS OF STOMACH

Acute gastritis is a transient mucosal inflammatory process that may be asymptomatic or cause variable degrees of epigastric pain, nausea and vomiting. It may result from the ingestion of alcohol, NSAIDs and other anti-inflammatory drugs which impair the mucosal protection mechanisms. Microscopically, the surface epithelium is intact, although scattered neutrophils may be present among the epithelial cells or within mucosal glands. An erosion is denoted by loss of superficial epithelium generating a mucosal defect limited to lamina propria, and is often accompanied by a pronounced neutrophilic infiltrate. Concurrent erosion and hemorrhage is termed as acute erosive hemorrhagic gastritis.[23]

Chronic Gastritis has following two main features: i) Infiltration of lamina propria by inflammatory cells (Plasma cells & lymphocytes) ii) Atrophy of glandular epithelium. If the inflammatory infiltrate is limited to the foveolar region and not accompanied by glandular atrophy, it is termed as chronic superficial gastritis. If the inflammation is more extensive and accompanied by glandular atrophy, it is termed as chronic atrophic gastritis. If there is only thinning of mucosa with absence of inflammation, it is termed as gastric atrophy.[22, 23] Two types of metaplastic change can occur in chronic gastritis, often in combination i.e. pyloric metaplasia of fundic mucosa and intestinal metaplasia. Endoscopically, well developed atrophic gastritis

and gastric atrophy produce a thin, smooth mucosa with undue prominence of submucosal vessels.[22]

Chronic gastritis is divided into 2 types: 1) Type A or Autoimmune gastritis 2) Type B or Non-immune gastritis. Autoimmune gastritis is characterized by antibodies to parietal cells and intrinsic factor. There is reduced serum pepsinogen I concentration, antral endocrine cell hyperplasia, vitamin B12 deficiency and defective gastric acid secretion (achlorhydria) which affects fundus in a diffuse manner. Autoimmune gastritis is associated with loss of parietal cells resulting in megaloblastic anemia and hyperplasia of antral gastrin producing 'G' cells.[23] There is diffuse mucosal damage and atrophy of the oxyntic mucosa resulting in thinning and loss of rugal folds .Microscopically there is megaloblastic change in epithelial cells accompanied by a chronic inflammatory infiltrate and severe cases show intestinal metaplasia.[22, 23]

Non-immune gastritis (type B) affects the antrum mainly and progresses proximally. The most common cause is infection with H.pylori before the discovery of which, other factors like psychologic stress, caffeine, alcohol and tobacco use were considered the primary causes.[22]

H. pylori infection is the most common cause of chronic gastritis. The disease most often presents as a predominantly antral gastritis with high acid production which progresses to pan gastritis. The route of transmission of H. pylori is either oral –oral, faeco-oral, or environmental. H. pylori infection results in increased acid production and disruption of normal gastric and duodenal protective mechanisms.[23, 36] H. pylori plays a significant role in the genesis of several gastric diseases, including acute gastritis, chronic gastritis, chronic active gastritis, follicular gastritis, intestinal

metaplasia, hyperplastic polyps, gastric and duodenal ulcers, gastric adenocarcinoma and gastric lymphoma.[24,37]

H. pylori are slender, curved spirals in the superficial mucous layer, where they tend to be attached to the epithelium at the site of intercellular junctions. In extreme cases, the organisms carpet the luminal surfaces of foveolar and mucous neck cells, and can even extend into the gastric pits. Occasionally, they can be present in the stomach as coccoid forms. These are solid, round, basophilic, dot like structures on routine histology.[36] Special stains to detect H. pylori are Giemsa , Warthin Starry, Gimenez, Toulidine Blue, Genta stains or by IHC.[38,39]

Endoscopically, H. pylori–infected antral mucosa is usually erythematous and has a coarse or even nodular appearance.[23] Microscopically, inflammatory infiltrate mainly neutrophils accumulate within the lamina propria and some assume intraepithelial location and accumulate in the lumen of gastric pits to create pit abscesses. The superficial lamina propria includes large numbers of plasma cells, often in clusters or sheets, and increased numbers of lymphocytes and macrophages. Foveolar hyperplasia, features of degeneration, in severe cases - erosion, hemorrhage, and mucosal necrosis can be seen. Lymphoid aggregates, some with germinal centers, are frequently present and represent an induced form of mucosa-associated lymphoid tissue, or MALT, that has the potential to transform into lymphoma.[22, 23, 37]

Cohen H and Laine N, [40] in their study on endoscopic methods for diagnosis of H. pylori states that when diagnosis of H. pylori is desired, two antral biopsies from non adjacent sites should be taken for rapid urease testing. Two or more additional biopsies should be stored for histological evaluation. They stated that although histological assessment is not free of pitfalls, it is the gold standard.

In the study done by Akanda MR and Rahman AN,[41] the sensitivity of endoscopic biopsy smear examination, rapid urease test, Haematoxylin & Eosin stain and modified Giemsa stained sections are 86.2%, 96.6%, 77.6% and 86.4% and the specificity is 100%, 97.7%, 97.7% and 97.6% respectively.

A study done by Ahsan K *et al* [42] concluded that the diagnostic accuracy of crush smear cytology for detection of Helicobacter pylori in gastric biopsy material is comparable to histopathology and the technique is very simple, less expensive and less time consuming.

A study done by Ahluwalia C *et al* [43] revealed that use of brush cytology than conventional biopsy for detection of H. Pylori is recommended as it is rapid, simple and easy to perform with a high degree of sensitivity and specificity.

Another study by Seth AK *et al* [44] reveals that biopsy of gastric antrum should always be combined with biopsy from the gastric corpus especially in endoscopic gastritis of corpus for diagnosing H. Pylori in patients with peptic ulcer on acid suppression therapy.

Other rare types of gastritis include suppurative gastritis, emphysematous gastritis, hemorrhagic gastritis, collagenous gastritis ,lymphocytic gastritis, allergic gastro-enteritis, diffuse eosinophilic gastritis, granulomatous gastritis , syphilis , malakoplakia, cytomegalovirus infection, herpes virus infection, candida infection, cryptococcosis, , and graft versus host diseases.[24,37] Metaplasias in gastritis are of four major types - pyloric metaplasia, intestinal metaplasia, ciliated cell metaplasia, pancreatic(acinar) metaplasia.[24,45]

Peptic Ulcer Disease (PUD) is most often associated with H. Pylori induced hyperchlorhydria and chronic gastritis. It’s most common site is gastric antrum and 1st portion of duodenum. Imbalance of mucosal defenses and damaging forces are

responsible for peptic ulcer disease. The primary underlying causes are H. pylori and NSAIDS which cause hypergastrinemia resulting in PUD. Duodenal ulcers are common with alcoholic cirrhosis, COPD, CRF, hyperparathyroidism.[22, 23]

Endoscopically, lesions less than 0.3cm in diameter are shallow while those over 0.6 cm are likely to be deeper ulcers. The ulcer is usually round to oval with sharply punched out defect with overlying of mucosal margins. Hemorrhage and fibrin deposits are often seen in the gastric serosa. The base is smooth and clean and in active ulcers, neutrophilic infiltration along with granulation tissue is seen.[22, 23]

Gastric Polyps

Gastric polyps may develop as a result of epithelial or stromal cell hyperplasia, inflammation, ectopia or neoplasia.[23]

Peutz – Jeghers polyps most commonly present in childhood, are 1-3cm in size with a coarsely lobulated surface and a short, broad stalk. The most useful diagnostic feature is the presence of a core of family arborizing branches of smooth muscle from muscularis mucosa which is covered by abundant but disorganised gastric mucosa. Juvenile polyps, sometimes called retention polyps are round, smooth surfaced lesions of 1-2 cm diameter consisting principally of lamina propria rarely confined to the stomach.[36]

Hyperplastic Polyps are common in both children & adults typically occurring at the junction of pyloric and corpus mucosa, generally varying from 0.5-2.5 cm in diameter having a coarsely lobulated surface. Smaller polyps are sessile while the larger ones have a stalk. The presumed histogenesis is an exaggerated regenerative response to mucosal damage. The histology is variable but basically they consist of elongated, distorted and branched gastric pits with inflamed & edematous lamina

propria. The pit lining cells are frequently hypertrophic and nuclei are typically bland.[36]

Fundic gland polyps has been described in two clinical situations –sporadic following widespread use of PPIs and syndromic in familial adenomatous polyposis(FAP) where hundreds of gastric polyps are present. They are present exclusively in the body or fundus and are multiple in clusters. Endoscopically they are soft, sessile, smooth, translucent and appear as minute mucosal lumps 1-7 mm in diameter. Microscopically, fundic gland polyps consist of proliferation of oxyntic mucosa with cystically dilated fundic glands, lined by an attenuated layer of chief cells, parietal cells and mucus neck cells.[37]

Inflammatory fibroid polyp are polyps whose pathogenesis is unknown but is widely assumed to be related to minor trauma and a myofibroblastic origin has been proposed. It involves the antrum. Grossly, it is sessile. Microscopically, it is centered in the submucosa characterized by vascular & fibroblastic proliferation & inflammatory cells especially eosinophilic infiltration.[36,37] Cronkhite-Canada syndrome is extremely rare condition and is characterized by diffuse GI polyposis, alopecia, hyperpigmentation, and dystrophic changes in fingernails and toe nails. Endoscopically, the polyps are sessile. Histologically, they consisting of hyperplastic, edematous mucosa with epithelial cysts. They resemble hyperplastic polyps and juvenile polyps.[36]

Adenomas comprise 7-10% of all gastric polyps and are sessile or pedunculated and grow in a tubulovillous or a pure villous pattern.[36] They occur throughout stomach with antrum being the most common site. They range from few milimeters to several centimetres. Histologically, they are of two types: showing intestinal differentiation and gastric differentiation. The gastric adenomatous polyps

are composed of gastric foveolar cells. Intestinal type defined by presence of goblet cells or paneth cells.[37] GI adenomas have epithelial dysplasia that can be classified as low or high grade. Both grades may include enlargement, elongation, and hyperchromasia of epithelial cell nuclei, epithelial crowding, and pseudostratification. High-grade dysplasia is characterized by more severe cytologic atypia and irregular architecture, including glandular budding and gland-within-gland, or cribriform, structures.[23]

A study done by Carmack *et al* [46] on gastric polyps revealed that a variety of gastric lesions might present as a polyp, and the need to obtain a biopsy specimen from the gastric mucosa adjacent to a lesion is critical.

TUMORS OF STOMACH

Gastric adenocarcinoma is the most common malignancy of the stomach seen in low socio-economic groups and in individuals with multifocal mucosal atrophy and intestinal metaplasia. Few studies also show an association with H. pylori infection. Gastric cancer incidence varies markedly with geography, being highest in Japan, Chile, Costa Rica, and Eastern Europe. All gastric carcinomas arise from the generative or basal cells of the foveolae. Loss of ECadherin function seems to be the key step in development of diffuse cancer whereas mutations in Catenin, microsatellite instability and accumulation of p53 are associated with intestinal type gastric carcinoma. Any condition causing hypochlorhydria decreases the gastric pH favouring bacterial growth which reduces nitrates to N-Nitroso compounds which are carcinogenic. Patients of Menetrier's disease, gastric polyp, gastric peptic ulcer, gastric stump, irradiation and chemotherapy are at increased risk for developing carcinoma.[22, 23, 34]

The most common site is anterior wall then the posterior wall and lesser curvature more than greater curvature. Endoscopically, it may appear polypoid, fungating, ulcerated or diffusely infiltrating so-called linitis plastica types, or may show a combination of these.[34] With adequate biopsy material, the diagnostic accuracy of gastric biopsies for cancer is 83% .[36]

Microscopically, the World Health Organization classification subdivides gastric carcinoma into five subtypes: papillary, tubular, mucinous, signet-ring cell adenocarcinomas and undifferentiated carcinoma, in which no definite glandular structures or any other specific differentiation is present.[34] Adenocarcinomas may also be graded as well, moderately or poorly differentiated. Papillary adenocarcinoma is characterized by numerous papillary processes with fibrovascular cores. Tubular adenocarcinoma is composed predominantly of neoplastic tubules often showing irregular branching and anastomosis. Mucinous adenocarcinoma (colloid or mucoid carcinoma) is characterized by conspicuous amounts of extracellular mucin (more than 50% of the tumor). Signet-ring cell carcinoma consists predominantly of single cells or small clusters of cell containing intracytoplasmic mucous vacuoles and accounting for more than 50% of the tumor.[34]

The histologic classification of Lauren divides gastric adenocarcinoma into two main types – intestinal and diffuse.[34]

a) Intestinal type adenocarcinoma arises from metaplastic epithelium with glandular formations. The cells are columnar & mucin secreting, the increased secretion of which may cause formation of mucin- lakes which, when long standing may lead to metastatic calcification & ossification.
b) Diffuse type adenocarcinoma is classically known as Linitis Plastica or signet ring carcinoma. It commonly involves the pre-pyloric area and associated with

submucosal fibrosis with or without ulceration. The tumor is composed of dyscohesive cells, having large mucin vacuoles that expand the cytoplasm and push the nucleus to the periphery forming the signet ring cell. The secretory product of most adenocarcinomas is positive with Meyer's Mucicarmine, Alcian blue, Colloidal iron and shows IHC positivity for MUC1, MUC5AC, and MUC2.[22]

The other microscopic variables include adenocarcinoma with neuroendocrine differentiation, adenosquamous carcinoma, mucinous carcinoma, hepatoid adenocarcinoma, oncocytic (parietal gland) carcinoma, lymphoepithelioma like carcinoma, sarcomatoid carcinoma, adenoma with rhabdoid features and gastric carcinoma with osteoclast – like giant cells.[22]

Early Gastric Cancer is defined as carcinoma confined to the mucosa or to the mucosa & submucosa, most commonly seen in the distal third of the stomach. Most cases are of the intestinal type.[22] Endoscopically, they are classified as superficial protruding or non-protruding lesions, protruding pedunculated or protruding sessile. Non-protruding and non-excavated lesions include slightly elevated, completely flat, slightly depressed, elevated & depressed types. Excavated lesions may be further divided into ulcer and excavated & depressed type.[47]

Well-differentiated neuro-endocrine tumors (WDNETS) are mainly composed of serotonin containing argentaffin cells. There are 2 types of gastric WDNETs-composed of G cells (Gastrinoma) and Enterochomaffin like (ECL) cells. Endoscopically , they tend to be small, sharply outlined and covered by flattened mucosa, usually intramural/submucosal polypoidal lesions. Microscopically, the predominant pattern of arrangement is microglandular, with regular nuclei and normochromatic, scanty mitosis with absent necrosis and florid vascularisation and

exceptionally clear cytoplasm. Immunohistochemically, they are positive for chromogrannin, synptophysin and keratin.[22, 34]

Gastrointestinal Stromal Tumor (GIST) is the most common mesenchymal tumor of the abdomen with more than 50% cases occurring in stomach. It is seen in older age group (60yrs and above). They may occur as part of Carnoy's triad: GIST, Pulmonary chondromas, extra- adrenal parganglioma s. All GISTS have gain – of – function mutations of the gene encoding tyrosine kinase CKit (receptor for stem cell factor). The cell of origin is a common stem cell i.e. interstitial cell of Cajal, located in muscularis propria which serves as pacemaker cells for gut peristalsis.[23] GIST is divided into 4 major categories:

A) Tumors showing differentiation towards smooth muscle cells (Actin +, Desmin+,Calponin+).

B) Tumors showing apparent differentiation toward neural elements (NSE+, Leu -7+, S-100+).

C) Tumors showing differentiation towards smooth muscle and neural elements.

D) Tumors lacking differentiation to either cell type (CD34+) .[22]

The smooth muscle differentiation is identified by spindle tumor cells with acidophilic fibrillary cytoplasm and cytoplasmic vacuoles at both ends of nucleus. An epithelioid appearance may be present. The neural differentiation is identified by spindle cells growing in fascicles, palisades & whorls and presence of skenoid fibres i.e, deposition of extracellular, amorphous collagen. The defining criteria for diagnosis is CD117 positivity (membrane component) .[22, 34]

Malignant lymphoma of the stomach accounts for 10% of gastric malignancies. It may be primary, or secondary to systemic lymphoma. Features favouring a primary tumor are concentration of the major tumor bulk within the stomach and /or the regional lymph nodes without involvement of superficial or mediastinal lymph nodes, liver, spleen, bone marrow or peripheral blood. It is of the MALT type (mucosa associated lymphoid tissue present in lamina propria) and virtually all tumors arise in the background of chronic Helicobacter associated gastritis.[34, 36]

It is divided into 2 large categories:

a) Low grade lymphomas (MALT type) are seen in patients over 50yrs in distal half of stomach. Endoscopically, giant convolutions mimicking hypertrophic gastritis or gastric polyps are present. Microscopically, transmural involvement is seen with focal or extensive plasmacytoid differentiation and dutcher bodies may be present. An important diagnostic sign is the infiltration of the glandular epithelium by the lymphocytes called as lymphoepithelial lesions.

b) Intermediate / high grade lymphomas endoscopically appears as a large lobulated (polypoid) mass with superficial /deep ulceration. Histologically, it is composed of cells resembling large non-cleaved cells (centroblasts) but with a slightly more abundant cytoplasm, plasmablastic or immunoblastic appearance. MALT lymphoma cells express B-cell antigens CD20, and CD79a, but not CD5, CD10 or CD23. Endoscopic and histological examination combined with flow cytometry has significance for the diagnosis of GI B- cell lymphoma as a screening tool.[22]

NON-NEOPLASTIC LESIONS OF THE DUODENUM

Chronic duodenitis induced by H. pylori endoscopically shows duodenal gastric metaplasia (DGM) . H. pylori in duodenum may produce chronic gastritis, duodenal ulcer, duodenal bulb deformity and scarring. DGM and H. pylori are usually found in proximal duodenum and H. pylori colonizes the duodenal mucosa only in areas of gastric metaplasia.[13, 22]

A study done by Chu KM *et al* [48] revealed that 90% of the patients with duodenal ulcer are infected by H pylori.

Eosinophilic Duodenitis is diagnosed by presence of gastrointestinal symptoms, biopsy specimens showing eosinophilic infiltration of one or more areas of duodenum and no evidence of parasitic, intestinal or extraintestinal disease. Duodenum may be affected along with other segments of the intestine.[13]

Gonul CD *et al* [49] studied the clinical significance and histopathologic features of duodenal nodularity in children. Their study revealed that the most demonstrative histomorphology in duodenal mucosa is increased lymphocyte and eosinophil infiltration in children with duodenal nodularity.

Gluten-sensitive enteropathy (GSE) also known as celiac disease or celiac sprue, is seen due to ingestion of gluten containing cereals, such as wheat, rye or barley in genetically predisposed individuals. Endoscopically , the duodenal folds appear to be reduced or absent. Microscopically, the villi are atrophic/ absent; there is crypt hyperplasia and intraepithelial lymphocytosis. There is increase in the number of lymphocytes, plasma cells in lamina propria & accumulation of large fat globules in the surface epithelium.[22, 23]The combination of histology and serology is most specific for diagnosis of celiac disease. However a single duodenal biopsy, followed by a favourable response to the gluten free diet, is sufficient to confirm the

diagnosis.[50]The most sensitive tests are the presence of IgA antibodies to tissue transglutaminase or IgA or IgG antibodies to deamidated gliadin. Anti-endomysial antibodies are highly specific but less sensitive.[23]

Marsh Classification for histological grading of celiac disease consists of a four-stage grading system with 40 intraepithelial lymphocytes (IEL) per 100 epithelial cells as the normal upper limit: Type I: infiltrative lesion, characterized by intraepithelial lymphocytosis and a normal villous architecture of the duodenal mucosa. Type II: hyperplastic lesion, characterized by intraepithelial lymphocytosis and crypt hyperplasia, with a normal villous architecture. Type III: destructive lesion, characterised by intraepithelial lymphocytosis, crypt hyperplasia and villous atrophy. Type IV: hypoplastic lesion, characterised by a normal IEL count, normal crypt length and villous atrophy.[51]

Oberhuber *et al* [52] modified this classification by splitting the type III lesions in three substages: mild villous atrophy, marked villous atrophy and completely flat mucosa.

A study done by Prasad KK *et al* [53] revealed that due to different concentrations of toxic gliadin fragment during its passage to different part of duodenal mucosa, a high frequency of histological lesion variability of the duodenal mucosa is seen in Indian children with celiac disease. Therefore, during upper gastrointestinal endoscopy at least 4 duodenal biopsies (2 in the distal duodenum and 2 in the duodenal bulb) should be obtained to avoid the risk of underdiagnosis or misdiagnosis.

A study done by Mee AS *et al* [54] reveals that the most reliable method for diagnosing or excluding villous atrophy is endoscopic forceps biopsy of the

descending duodenum, provided that at least four specimens are obtained with standard size forceps.

Mouzan MIE *et al* [55] stated that endoscopic duodenal biopsy in children is adequate not only for the diagnosis of villous atrophy but also for detection of other gastroenteropathies.

Refractory sprue is defined as symptomatic, severe small intestinal villous atrophy mimicking GSE, not responding to at least 6 months of gluten free diet. Architectural changes in duodenal biopsy specimen range from partial to total (grade IV) villous atrophy with a diffuse and dense mononuclear infiltrate mainly plasma cells in the lamina propria and a massive increase in IEL count. [13]

Cow's milk protein-sensitive Enteropathy (CMSE) may affect school-aged children and in young adults with unexplained GI symptoms. The duodenal villous changes are not seen, however, duodenal IEL count expressing T-cell intracellular antigen I, perforin and granzyme A is increased. Lymphonodular hyperplasia, without villous atrophy found in bulb of duodenum, characterizes CMSE endoscopically.[13]

Whipple's disease is a multisystemic disorder caused by bacterium Tropheryma whipplei. It is an intracellular bacterium. Thickening of duodenal villi is seen with the lamina propria packed with foamy macrophages with numerous PAS positive, diastate resistant intra-cytoplasmic granules. HIV Enteropathy may be defined as atrophy with epithelial hypoproliferation and dysmaturation of enterocytes, which in turn impairs small bowel absorption. The histological features include normal villous architecture to partial villous atrophy, marked depletion in mucosal CD4 T lymphocytes, an increase in CD8 lymphocyte count and increased crypt depth with normal mitoses per crypt. Also, GI opportunistic infections like microsporidiosis,

cyclosporidiosis, isosporidiosis, cryptosporidiosis, mycobacteriosis, cryptococcosis, visceral leishmaniasis, etc. may be detected on endoscopic biopsy.[13]

TUMORS OF DUODENUM

Brunner's gland adenoma (Polypoid Hamartoma / Brunneroma) is most commonly located in the posterior wall of the duodenum at the junction between the first and second portions. It can be associated with duodenitis and erosions. It is characterized by a nodular proliferation of histologically normal Brunner's glands accompanied by ducts and scattered stromal elements. It may be accompanied by ciliated cysts and adipose tissue and can be focal, multifocal or diffuse.[22]

Adenomas of the small intestine are uncommon. The periampullary region is a site of predilection. Multiple duodenal adenomas are a frequent complication of FAP but usually remain small. Morphologically they are sessile or pedunculated. Adenomas are composed of tubular and/or villous structures lined by dysplastic epithelium. Mitotic activity is not limited to the basal zone and is often accentuated within the upper crypt and surface epithelium. The crypts show architectural irregularities. Based on their architecture, they are classified as tubular, tubulo-villous and villous.[22, 56]

Primary duodenal adenocarcinoma is a rare tumor with a poorly defined natural history and prognostic factors. It represents 0.3-1% of all GI tumors and 25-35% of malignant tumors of the GI.[57] Although most cases are sporadic, associations with FAP, crohn's disease, peutz-jegher's syndrome and neurofibromatosis I have been reported.[58]

Duodenal carcinoma tends to have a papillary configuration and is hence amenable to brush cytologic diagnosis.[22] The periampullary region of the duodenum is the most common site and patients present with painless jaundice and bleeding.

They present as polypoid or ulcerated tumors often with a co-existing adenoma. Microscopically, most adenocarcinomas are well or moderately differentiated. Mucinous adenocarcinomas occur, but signet-ring cell carcinoma is rare and should be distinguished carefully from secondary spread from other sites, notably the stomach. Ampullary adenocarcinomas are mainly intestinal in type.[56]

Duodenal endocrine tumors or primary duodenal carcinoids account for only 2.6% of carcinoid tumors in the US.[59] Endoscopically, duodenal endocrine tumors appear as smooth, round elevations, usually measuring 5-20 mm in diameter. Microscopically, they rarely have the features of a classic carcinoid tumor, many cases containing either G or D cells, but both have a well developed glandular component and in addition, the D cell tumors have numerous psammoma bodies usually within the glandular lumina.[22]

Stromal tumors have been thought to arise from an uncommitted mesenchymal cell. New category i.e gastrointestinal autonomic nerve tumors (GANTs) appear to occur in the small intestine. They can be classified as either non-myogenic stromal tumors(activating c-kit mutations positive) or true smooth muscle tumors{ c-kit (CDll7) immuno-negativity and positivity for smooth muscle actin and desmin}. Macroscopically, tumors may grow into the lumen, outwards through the serosa, or in both directions producing a dumbbell growth. GANTs typically extend into the mesentery or retroperitoneum. Microscopically, cells may be spindled or epithelioid. GANTs are described as having a well-developed microvasculature that is prone to focal hemorrhage. Nuclear palisading giving a plexiform appearance with a marked inflammatory component may favor a diagnosis of GANT.[56]

Primary lymphomas are uncommon but account for about 30% of small bowel malignancies. They are divided into B-cell and T-cell malignancies. B-cell

malignancies include MALToma, immunoproliferative small intestinal disease, Mediterranean lymphoma, Burkitt's lymphoma, mantle cell lymphoma, follicular lymphoma, plasmacytoma. T- cell lymphomas include lymphoma arising in celiac disease and T-cell lymphoma with eosinophilia.[56]

MATERIALS AND METHODS

Source of data:

The present study included endoscopic biopsies of upper gastrointestinal tract received in the Department of Pathology, B.L.D.E. University's Shri B.M.Patil Medical College, Hospital & Research Centre, Bijapur.

Study period - Two years retrospective and two years prospective endoscopic biopsies received from July 2010 to July 2014 were included in the study.

Inclusion criteria:

Endoscopic biopsies done for lesions in esophagus, stomach, first part and second part up to opening of common bile duct in the duodenum were included.

Exclusion criteria:

Biopsies done for lesions of the oropharynx were excluded.

Method of Collection of data:

The biopsy specimens received were fixed in 10% buffered formalin followed by tissue processing and embedding in paraffin. Then sections of 3-5 micron thickness were prepared and stained with routine Haematoxylin and Eosin. Other special stains like Periodic Acid Schiff (PAS), Giemsa stain and IHC were performed wherever necessary.

Sample size:

In any statistical analysis, in case of non- availability of prevalence or incidence rate, a sample size of 30 or more are generally considered adequate with the assumption that the sampling distribution of mean is approximately normal.

Hence a total of 196 UGE biopsies were studied.

Statistical methods:

- Diagrammatic presentation
- Percentage of various histomorphological patterns.

RESULTS

A total of 196 UGIT biopsies were obtained and studied over a period of four years from June 2010 to July 2014. Out of 196 cases, 6 cases were inadequate for opinion, 4 were esophageal biopsies and 2 were duodenal biopsies.

TABLE NO.1: DISTRIBUTION OF CASES ACCORDING TO AGE AND SEX

AGE IN YEARS	MALE	FEMALE	TOTAL
1-10	02	00	02
11-20	05	01	06
21-30	16	06	22
31-40	14	03	17
41-50	26	15	41
51-60	25	12	37
61-70	30	14	44
71-80	16	08	24
81-90	02	01	03
TOTAL	**136(69.38%)**	**60(30.62%)**	**196**

UGE biopsies were obtained from patients of 4 years to 87 years of age. Majority of cases were between 40 to 70 years of age. Out of 196 cases 136 (69.38%) were from males and 60(30.61%) were from females with a male to female ratio of 2.26:1.

DISTRIBUTION OF CASES ACCORDING TO AGE AND SEX

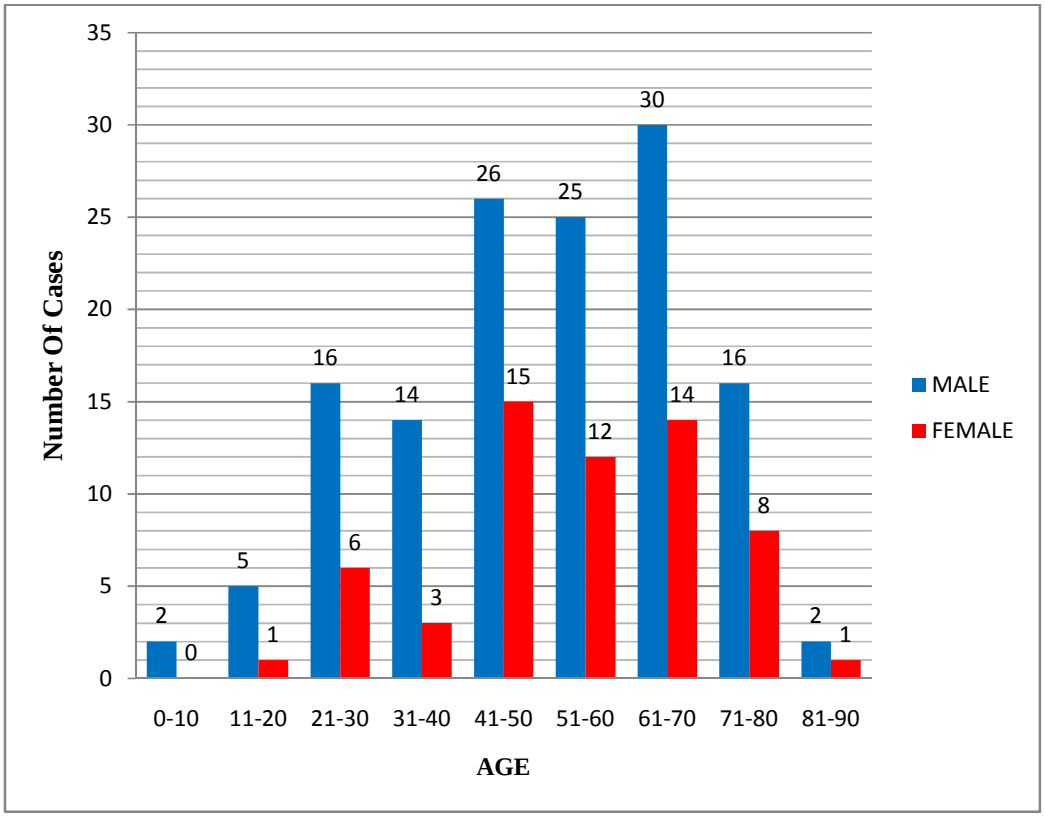
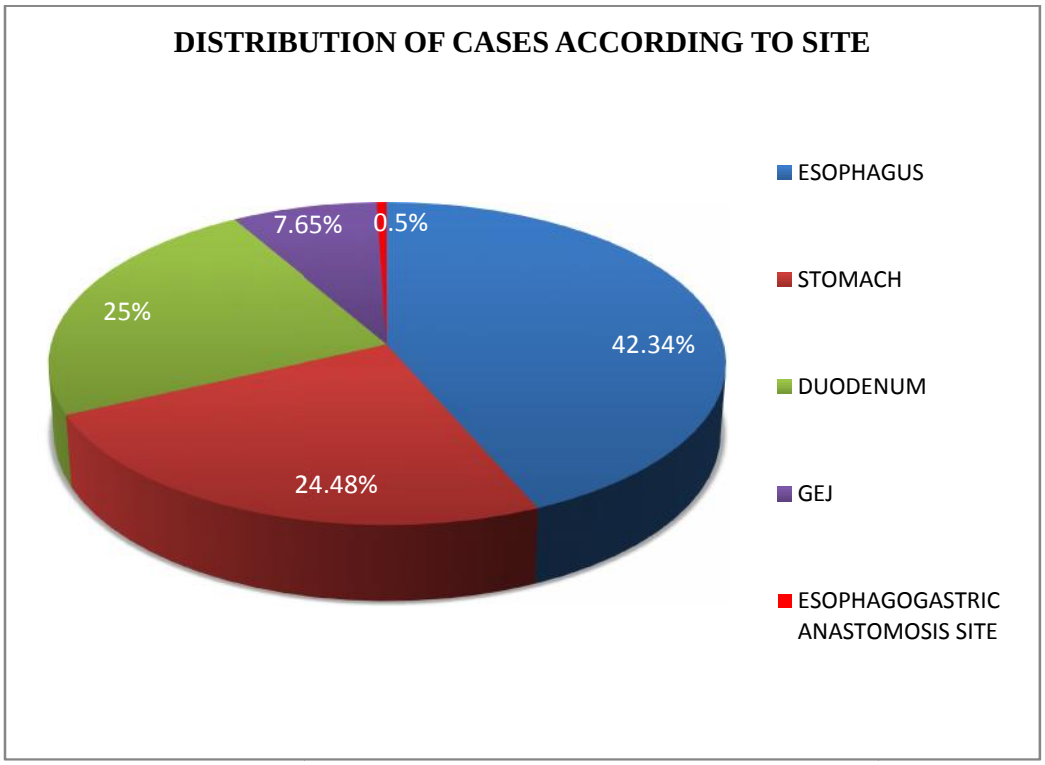


TABLE NO.2: DISTRIBUTION OF CASES ACCORDING TO SITE

Sl No.	SITE	NO. OF CASES	PERCENTAGE%
1.	Esophagus	83	42.34%
	Upper	05	6.02%
	Middle	47	56.62%
	Lower	31	37.34%
2.	Stomach	48	24.48%
	Fundus	07	14.58%
	Corpus	03	6.25%
	Antrum	02	4.16%
	Pre pyloric	18	37.5%
	Pyloric	18	37.5%
3.	Duodenum	49	25%
4.	Gastroesophageal junction	15	7.65%
5.	Esophagogastric anastomosis site	01	0.5%
	TOTAL	**196**	**100%**



Out of 196 cases, 83(42.34%) were from esophagus, 47(23.97%) cases were from stomach, 49(25%) were from duodenum and 15(7.65%) were from gastroesophageal junction. One case was from esophagogastric anastomosis site in a case of post trans-hiatal esophagectomy accounting for 0.5% .In esophagus, mid esophagus was the most common site of biopsy. In stomach, pylorus and pre-pyloric region were the most common sites.

TABLE NO. 3: DISTRIBUTION OF NEOPLASTIC & NON-NEOPLASTIC UGIT LESIONS ACCORDING TO SITE

SITE	NON-NEOPLASTIC	NEOPLASTIC	INADEQUATE	TOTAL
Esophagus	22(26.50%)	57(68.67%)	04(4.81%)	**83**
Stomach	23(48.93%)	25(52.08%)	00	**48**
Duodenum	46(93.87%)	01(2.04%)	02(4.08%)	**49**
Gastroesophageal junction	04(26.66%)	11(73.33%)	00	**15**
Esophagogastric anastomosis site	00	01(1.06%)	00	**01**
Total	**95(48.46%)**	**95(48.46%)**	**6(3.09%)**	**196**

In esophagus, neoplastic lesions (68.67%) were more common than non neoplastic lesions(26.5%). In stomach, neoplastic lesions were 52.08% and non-neoplastic lesions were 48.93%. In duodenum, non-neoplastic lesions (93.87%) were far more common than neoplastic lesion (2.04%). In GEJ, 26.66% cases were non-neoplastic and 73.33% cases were neoplastic. The biopsy from esophagogastric anastomosis site showed moderately differentiated SCC which recurred following transhiatal esophagectomy. 6 cases (4 cases from esophagus and 2cases from duodenum) were inadequate to opine as there was only scant tissue or only epithelium without subepithelial tissue or only fibroconnective tissue.

TABLE NO. 4: DISTRIBUTION OF NON-NEOPLASTIC LESIONS OF ESOPHAGUS

Sl. No	HISTOMORPHOLOGICAL PATTERN	NO. OF CASES
1.	Chronic non-specific esophagitis	11(50%)
2.	Hyperplastic polyp	01(4.54%)
3.	Ciliated metaplasia	01(4.54%)
4.	Ulcer with granulation tissue	01(4.54%)
5.	Barrett's esophagus	04(18.18%)
6.	Chronic esophagitis with mild dysplasia	01(4.54%)
7.	Chronic non specific esophagitis with moderate dysplasia	01(4.54%)
8.	Mild dysplasia	02(9.09%)
	Total	**22**

Amongst the non neoplastic lesions of esophagus, chronic esophagitis was most common lesion accounting to 13/22 cases (59.09%). Out of 4 cases of dysplasia, 2 cases showed features of esophagitis. 4 cases were diagnosed as Barrett's esophagus. There was one case each of hyperplastic polyp, ciliated metaplasia and ulcer with granulation tissue.

TABLE No. 5: DISTRIBUTION OF NEOPLASTIC LESIONS OF ESOPHAGUS

Sl. No	HISTOMORPHOLOGICAL PATTERN	NUMBER OF CASES
1.	Squamous cell carcinoma(SCC)	51(89.47%)
	• Well differentiated	15(29.41%)
	• Moderately differentiated	29(56.86%)
	• Poorly differentiated	07(13.72%)
2.	Adenocarcinoma	02(3.5%)
	• Well differentiated	01(50%)
	• Moderately differentiated	01(50%)
3.	Poorly differentiated carcinoma	03(5.26%)
4.	Highly suspicious for malignancy	01(1.75%)
	TOTAL	**57**

Amongst the neoplastic lesions of esophagus, moderately differentiated squamous cell carcinoma was most commonly diagnosed lesion. One case of well differentiated SCC was associated with candidiasis. There were 2 cases of adenocarcinoma, both were from lower segment of esophagus. Out of 3 cases of poorly differentiated carcinoma, for 2 cases differential diagnosis of small cell carcinoma was suggested based on histomorphology. In one case of esophageal biopsy, diagnosis of suspicious for malignancy was suggested for which follow up was not available.

TABLE No. 6: DISTRIBUTION OF NON-NEOPLASTIC LESIONS OF STOMACH

Sl. No	HISTOMORPHOLOGICAL PATTERN	NUMBER OF CASES
1.	Chronic non-specific gastritis	15(65.21%)
2.	H.pylori gastritis	02(8.69%)
3.	Chronic gastritis with intestinal metaplasia	02(8.69%)
4.	Ulcer with suppurative necrosis	01(4.34%)
5.	Candidal infection	01(4.34%)
6.	Inflammatory polyp	01(4.34%)
7.	Hyperplastic polyp	01(4.34%)
	Total	**23**

The most commonly diagnosed lesion amongst non-neoplastic lesions of stomach was gastritis. 2 cases were of H. pylori gastritis and in 2 cases of chronic non-specific gastritis, intestinal metaplasia was noted.

TABLE No. 7: DISTRIBUTION OF NEOPLASTIC LESIONS OF STOMACH

Sl.No.	HISTOMORPHOLOGICAL PATTERN	NO. OF CASES
1.	Adenomatous polyp	01(4%)
2.	Adenocarcinoma	17(68%)
	• Well differentiated	04(23.52%)
	• Moderately differentiated	09(52.94%)
	• Poorly differentiated	04(23.52%)
3.	Signet ring adenocarcinoma	03(12%)
4.	Poorly differentiated SCC	01(4%)
5.	Poorly differentiated carcinoma	02(8%)
6.	Highly suspicious for malignancy	01(4%)
	Total	**25**

Among the neoplastic lesions in stomach, majority of the cases were of adenocarcinoma accounting to 68% of neoplastic lesions of stomach followed by signet ring adenocarcinoma accounting to 12%. There was only 1 case of benign neoplasm i.e. adenomatous polyp. One case of poorly differentiated adenocarcinoma was associated with candidiasis. Out of 2 cases of poorly differentiated carcinoma, in one case differential diagnosis of NHL was given. IHC (CK7 and CK20) staining was done. CK7 showed focal positivity and CK20 showed diffuse positivity indicating diagnosis of poorly differentiated adenocarcinoma of stomach.

TABLE No. 8: DISTRIBUTION OF LESIONS OF GASTROESOPHAGEAL JUNCTION

Sl. No	HISTOMORPHOLOGICAL PATTERN	NUMBER OF CASES
1.	Chronic non-specific inflammation	02(13.33%)
2.	Barrett's esophagus	01(6.66%)
3.	Hyperplastic polyp	01(6.66%)
4.	Moderately differentiated SCC	06(40%)
5.	Well differentiated adenocarcinoma	03(20%)
6.	Moderately differentiated adenocarcinoma	02(13.33%)
	Total	**15**

Most common lesion at GEJ was moderately differentiated squamous cell carcinoma followed by adenocarcinoma.

TABLE No. 9: DISTRIBUTION OF LESIONS OF DUODENUM

Sl. No	HISTOMORPHOLOGICAL PATTERN	NUMBER OF CASES
1.	Chronic non-specific duodenitis	41(87.23%)
2.	H. Pylori duodenitis	01(2.12%)
3.	Celiac disease	01(2.12%)
4.	Villous atrophy with crypt hyperplasia	02(4.25%)
5.	Well differentiated adenocarcinoma	01(2.12%)
6.	Normal histology	01(2.12%)
	Total	**47**

Majority of the cases in duodenum were of chronic non-specific duodenitis amounting to 87.23%.

PHOTOMICROGRAPHS

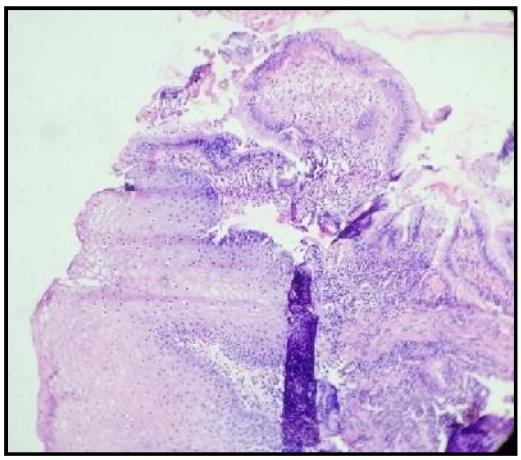


Fig 1: Photomicrograph of Barrett's esophagus (H&E stain 40x)

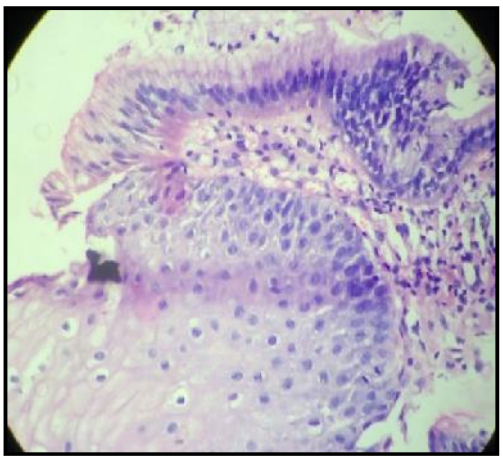


Fig 2: Photomicrograph of Barrett's esophagus (H&E stain 400x)

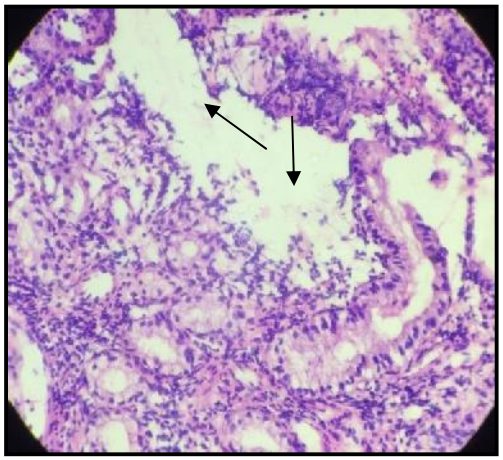


Fig 3: Photomicrograph of chronic gastritis with H. pylori infection (H&E stain 400x)

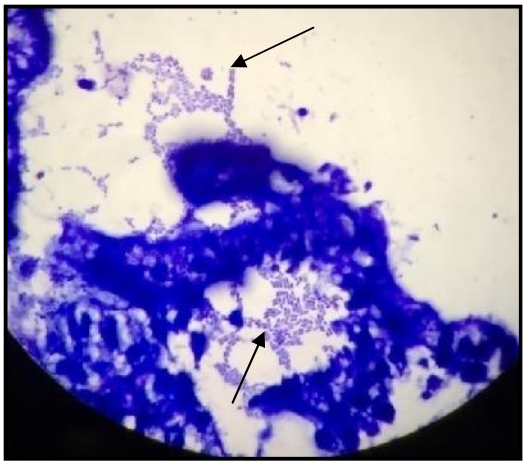


Fig 4: Photomicrograph of H. pylori colonies in stomach (Giemsa stain 1000x)

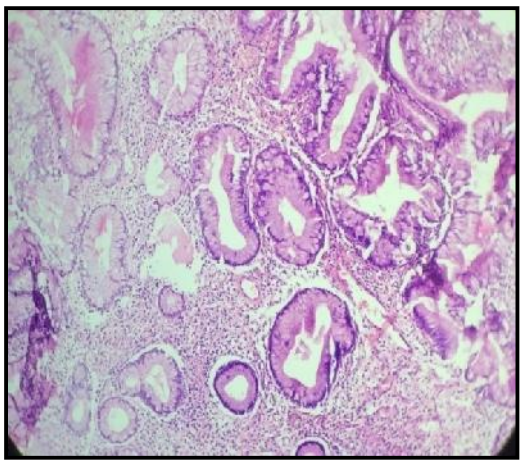


Fig5: Photomicrograph of Inflammatory gastric polyp (H&E stain 100x)

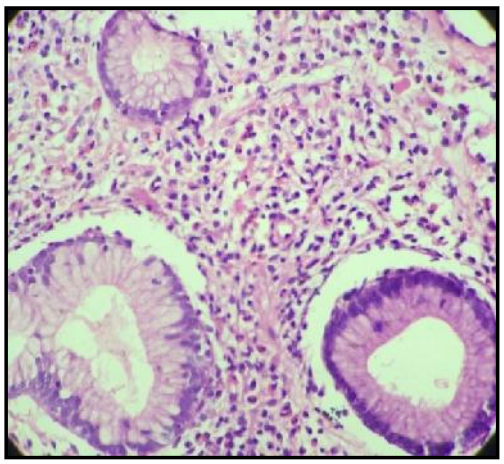


Fig6: Photomicrograph of Inflammatory gastric polyp (H&E stain 400x)

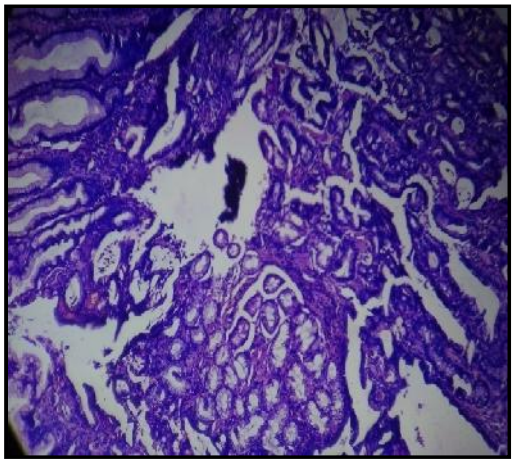


Fig 7: Photomicrograph of Hyperplastic gastric polyp (H&E stain 40x)

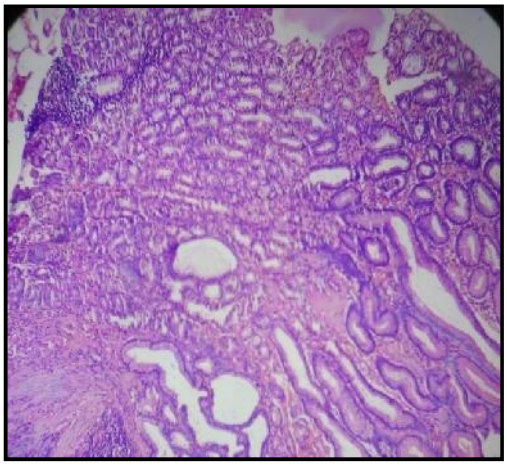


Fig 8: Photomicrograph of Moderately differentiated adenocarcinoma of stomach (H&E stain 40x)

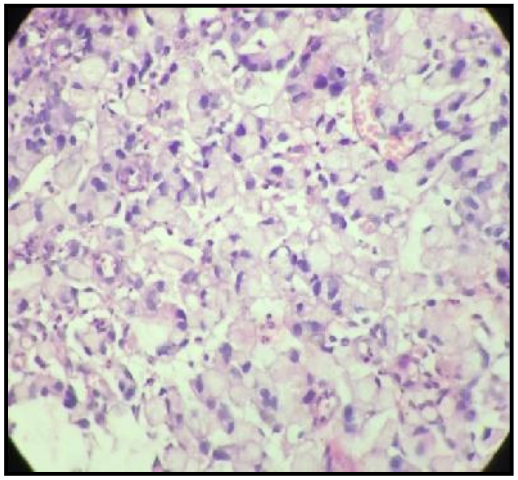


Fig:9 Photomicrograph of Signet ring adenocarcinoma of stomach (H&E stain 400x)

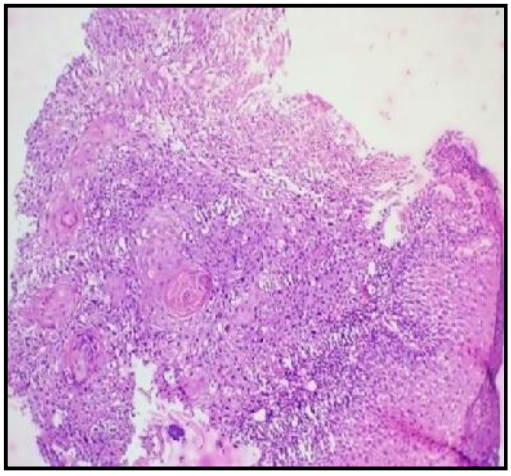


Fig 10: Photomicrograph of Well differentiated SCC of esophagus with Candidiasis(H&E stain 100x)

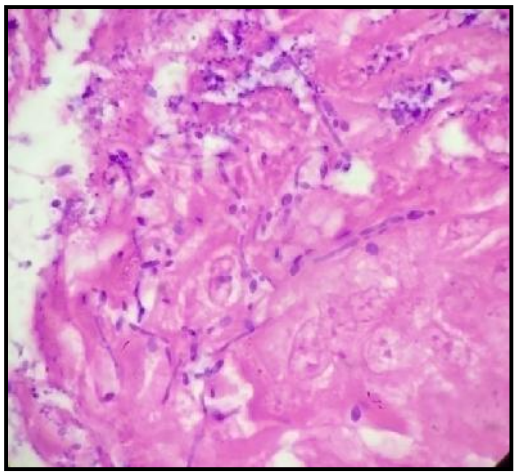


Fig 11: Photomicrograph of Well differentiated SCC – esophagus with Candidal yeast forms and pseudohyphae (H&E stain 400x)

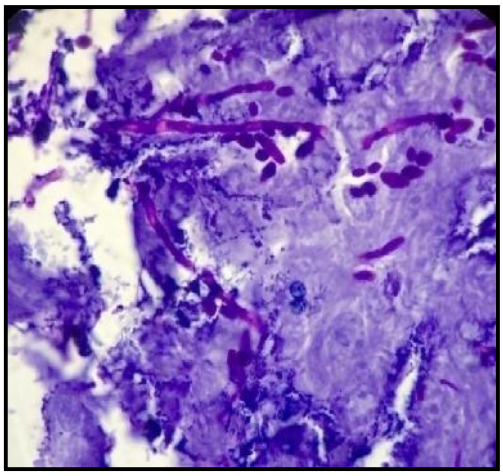


Fig 12: Photomicrograph of Candidal yeast forms and pseudohyphae (PAS stain 1000x)

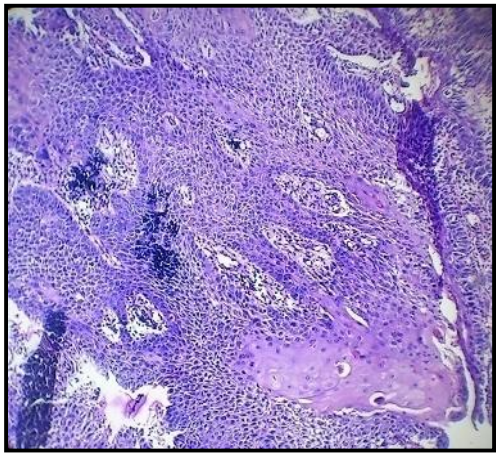


Fig 13: Photomicrograph of Moderately differentiated SCC of esophagus (H&E stain 100x)

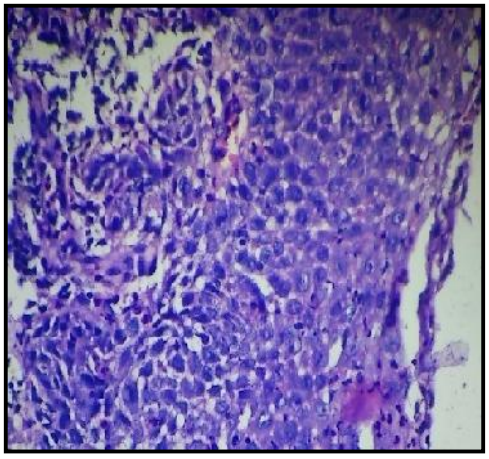


Fig 14: Photomicrograph of Poorly differentiated SCC of esophagus (H&E stain 400x)

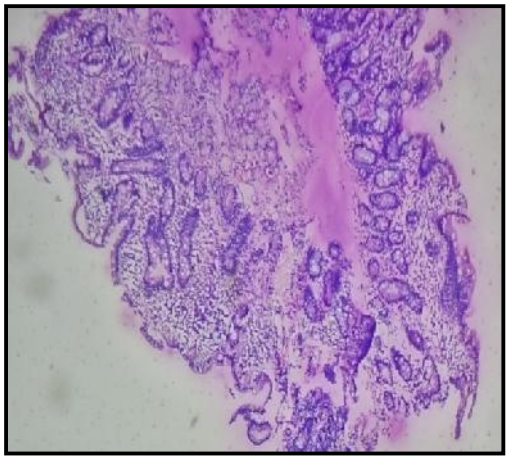


Fig 15: Photomicrograph of Celiac disease - duodenum (H&E stain 40x)

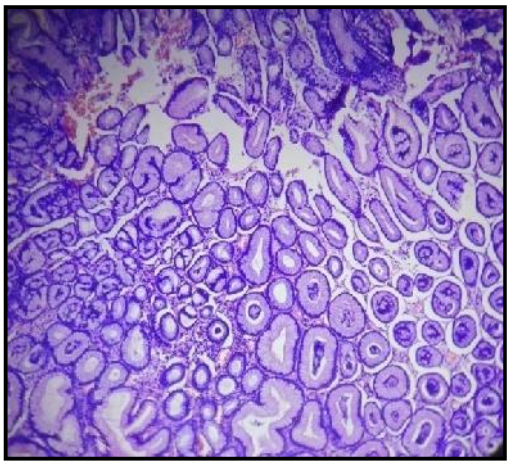


Fig 16: Photomicrograph of Well differentiated adenocarcinoma of duodenum - periampullary region (H&E stain 100x)

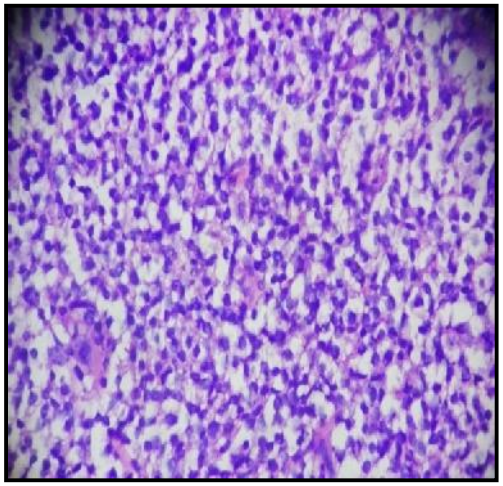


Fig 17: Photomicrograph of Poorly differentiated carcinoma (H&E stain 400x)

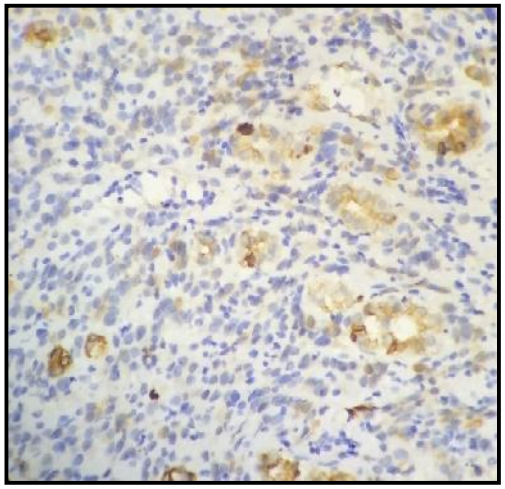


Fig 18: Photomicrograph of Poorly differentiated carcinoma showing focal positivity for CK7 (400x)

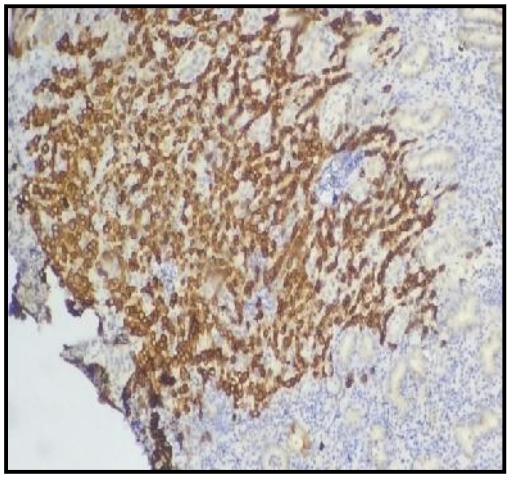


Fig:19 Photomicrograph of poorly differentiated carcinoma showing diffuse positivity for CK20 (40x)

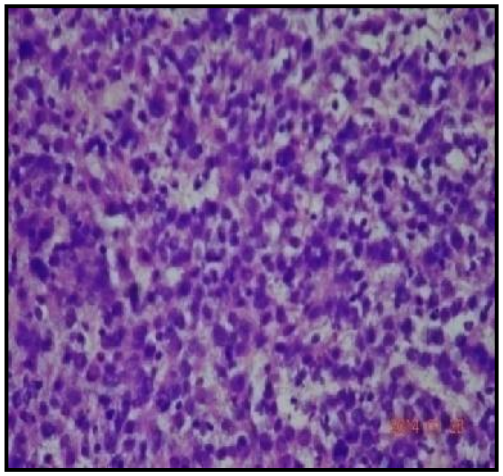


Fig:20 Photomicrograph of MALToma of stomach (H&E stain 400x)

DISCUSSION

Adequate clinical and endoscopic information is a fundamental part of adequacy and it strongly affects in the interpretation of biopsy. In the present study dysphagia, anorexia, vomiting, pain in abdomen, chronic diarrhoea, distension of abdomen and weight loss were the major presenting symptoms. In few cases history of hematemesis was noted. Similar presenting symptoms were reported in a study done Gulia SP *et al.*[5] In their study they also found, presenting symptoms such as melena, constipation and bleeding per rectum, however these complaints were not noted in the present study.

In our study, total of 196 UGE biopsies were studied. Patients with upper GI lesions presented in the age group of 1st to 8th decade, the youngest patient was 04 years old and oldest was 87 years old. The mean age group was 44 years. Most common age group presenting with upper GI lesions was between 40 to 70years accounting to 62%. Male to female ratio obtained in our study was 2.26:1

Gulia SP *et al* [5] studied 192 UGE biopsies. In their study, the youngest patient was of age 19 years and oldest was 75years. Male to female ratio in their study was 1.74:1.

Rashmi K *et al* [60] studied 100 cases of UGE biopsies. In their study, highest incidence of endoscopic biopsies was in 4^{th} and 5^{th} decades and male to female ratio was 2.03:1. Findings of our study are correlating with the results of study done by Rashmi K *et al* .[60]

In our study cases of endoscopic biopsies in male patients were more than in female patients. This could probably be due to the large number of male patients attending the outpatient department compared to the female patients, and increase in

the number of gastrointestinal tract malignancies in males than females as stated by Rashmi k *et al* .[60]

Gulia SP *et al* [5] studied a total of 192 UGE biopsies in which 12cases (6.25%) were from esophagus, 163 cases (84.05%) from stomach and 6 cases (3.64%) were from duodenum.

In a study done by Rashmi K *et al* [60] 25 cases (25%) of esophageal biopsies, 68 cases (68%) of gastric biopsies and seven cases (7%) of duodenal biopsies were reported. In stomach, pylorus was the common site of biopsy.

In our study, out of 196 cases, 83(42.34%) were from esophagus, 47(23.97%) cases were from stomach, 49(25%) were from duodenum and 15(7.65%) were from gastroesophageal junction. One case was from esophagogastric anastomosis site in a case of post trans-hiatal esophagectomy accounting for 0.5%. In esophagus the most common site of biopsy was mid esophagus that comprised of 56.62% of esophageal biopsies. Next common site was lower esophagus. In stomach pyloric and pre-pyloric regions were the common sites of biopsy.

In studies done by Rashmi K *et al,*[60] and Gulia SP *et al,*[5] number of gastric biopsies were more. However in our study, majority of cases were esophageal biopsies.

In our study cases of both non-neoplastic lesions and neoplastic lesions were 48.46% each. Our study showed 68.67% of neoplastic lesions and 26.5% of non-neoplastic lesions in esophagus. In stomach, neoplastic lesions were 52.08% and non-neoplastic lesions were 48.93%. In duodenum, non-neoplastic lesions (93.87%) were far more common than neoplastic lesions (2.04%). In GEJ, 73.33% cases were neoplastic and 26.66% cases were non-neoplastic..

In study done by Gulia SP *et al,* [5] 87.5% cases were inflammatory lesions and 6.25% cases were malignant lesions of esophagus and stomach and 5.62% cases had normal histology. In study done by Rashmi K *et al,* [60] 56% cases were non-neoplastic and 44% cases were neoplastic. In study done by above authors, non-neoplastic lesions were higher than neoplastic lesions. However in our study overall percentage of neoplastic and non-neoplastic lesions was equal.

In our study, in esophagus, neoplastic lesions(68.67%) were more common than non-neoplastic lesions. Amongst the non-neoplastic lesions of esophagus, chronic non-specific esophagitis was the most common lesion accounting to 59.09% of non-neoplastic lesions. Our study findings are similar to the findings of other author's study of histomorphological spectrum of endoscopic biopsies.[60, 61] Two cases of esophagitis in our study were associated with dysplasia, one was mild dysplasia and the other was moderate dysplasia. There were 2 cases (9.09%) of mild dysplasia, 4 cases (18.18%) of Barrett's esophagus and one case (4.54%) each of hyperplastic polyp, ciliated metaplasia and ulcer with granulation tissue.

Amongst the neoplastic lesions of esophagus, most common lesion in our study was SCC- moderately differentiated. The next common malignancy was adenocarcinoma. There were 5.26% cases of poorly differentiated carcinoma.

These findings are similar to study done by Pun CB *et al* [62]. They studied a total of 106 esophageal carcinoma cases (57 endoscopic biopsies and 49 radical esophagectomy specimens). Their study revealed SCC as the most common malignancy occurring in esophagus followed by adenocarcinoma. Among SCC, moderately differentiated SCC was most common. There were 3.78% cases of undifferentiated carcinoma.

In study done by Rashmi K *et al,* [60] all neoplastic esophageal lesions were malignant and all were SCC.

In our study, SCC was most common in the age group of 50 to 75 years and the most common site of occurrence was mid esophagus followed by lower esophagus which is similar to the finding of study done by Pun CB *et al* [62] where esophageal cancer was most common in the age range of 61 to 70 years. But the most common site of occurrence of SCC in their study was distal third of esophagus followed by mid esophagus.

In our study, in stomach neoplastic lesions (52.08%) were more than non-neoplastic lesions (48.93%). However in a study done by Rashmi K *et al,* [60] non-neoplastic lesions (68.33%) in stomach were more than neoplastic lesions (39.7%).

In our study, there were 41.66% cases of adenocarcinoma (including signet ring adenocarcinoma) of stomach, 39.58% cases of chronic gastritis. Two cases of chronic gastritis were associated with intestinal metaplasia.

Pailoor K *et al* [63] conducted a study to correlate histopathological diagnosis with endoscopy of 52 gastric biopsies. In their study, 55.76% cases were of adenocarcinoma of stomach, 34.61% cases were of gastritis. 3 cases of gastritis were associated with metaplasia.

Sultana A *et al* [64] studied 105 cases of gastric biopsies and correlated the endoscopic and histolologic findings. In their study there were 56.19% cases of adenocarcinoma, 36.19% cases of gastritis. Metaplasia was noted in 2 cases.

Our study findings of lesions of stomach were similar to the study done by Pailoor K *et al* [63] and Sultana A *et al* .[64]

In our study there was one case adenomatous polyp and one case of hyperplastic polyp. In study done by Rashmi K *et al* [60] there were 5 cases of adenomatous polyp and 3 cases of hyperplastic polyp.

In our study, Giemsa stain for H. pylori was done in all cases of chronic gastritis and chronic duodenitis. H. pylori was found in only 2 cases (10.5%) of chronic gastritis and in one case (2.38%) of chronic duodenitis. In study done by Rashmi K *et al,*[60] H. pylori positivity was seen in 7% cases of non-neoplastic lesions of stomach. Gulia SP *et al* [5] found H. pylori in 4.57% cases of non neoplastic lesions of stomach. Observations of our study were correlating with study done by Rashmi K *et al* [60].

According to a study done by Loffeld RJLF *et al* ,[65] the presence of H. pylori is decreasing due to a lower acquisition of the micro organism. According to study done Gulia SP *et al,* [5] H. pylori negative gastritis could be due to therapy for H. pylori eradication or failure to see organism in the tissue specimens.

H. pylori causes predominantly antral gastritis.[36] In many studies the prepyloric antrum was the preferred site of biopsy .[40] Genta & Graham performed a detailed topographic study of H. pylori in the stomach of untreated patients, and reported a sensitivity of 100% for a single biopsy taken from the angle of the stomach. The sensitivity of one distal antral biopsy was 96–97%. The sensitivity of two biopsies from virtually anywhere in the stomach was 100%. [66]

In our study, we received only 2 biopsies from antrum and 3 from corpus of stomach. This might explain for the low rates of H. pylori positivity in our study.

In our study, 2 cases of gastric biopsy were poorly differentiated carcinoma. In one case differential diagnosis of NHL was given. Immunohistochemistry (CK7 and CK20) staining was done. CK7 showed focal positivity and CK20 showed diffuse

positivity indicating diagnosis of poorly differentiated adenocarcinoma of stomach. In one case which was reported as chronic non-specific gastritis on biopsy, gastrectomy was done as clinical features and endoscopic findings were highly suggestive of malignancy. The resected specimen on histopathology showed features of MALToma.

In a study done by Scott BB and Jenkins D,[6] search for gastro-esophageal candidiasis was made by histological examination of all the endoscopic biopsies taken from 465 patients. Nineteen cases of candidiasis were found giving an overall incidence of 4%. There were 12 cases with esophageal candidiasis, two with both esophageal and gastric candidiasis, and five with gastric candidiasis.

In our study, candidiasis was associated with one case of well differentiated SCC of esophagus, one case of poorly differentiated adenocarcinoma of stomach and there was one case of pre-pyloric perforation associated with candidiasis.

The gastroesophageal junction (GEJ) is an anatomic area that represents the junction between the distal esophagus and the proximal stomach (cardia) .[21] In our study there were 15 biopsies from GEJ in which SCC (40%) was the most common lesion followed by adenocarcinoma (33.33%) and chronic non-specific inflammation(13.33%). There was one case of Barrett's esophagus.

In a study done by Rashmi K *et al,*[60] there were seven cases of duodenal biopsies. Four patients had chronic non-specific duodenitis followed by one patient each with duodenal ulcer, well differentiated adenocarcinoma of ampulla of vater and tubular adenoma.

In a study done by Gulia SP *et al,* [5] there were 6 cases of duodenal biopsies and all 6 cases showed features of duodenitis.

In our study, we received 49 cases of duodenal biopsies in which there were 85.7% cases of chronic duodenitis. Giemsa stain was done in all cases of duodenitis

but only one case (2.38%) of duodenitis showed positivity for H. pylori. There were 2 cases of villous atrophy with crypt hyperplasia. In one case of duodenal biopsy, diagnosis of celiac disease was suggested based on histological features of complete villous atrophy, crypt hyperplasia and intraepithelial lymphocytes. In one case endoscopic biopsy was taken from peri-ampullary region which was reported as well differentiated adenocarcinoma.

In our study majority of cases were of duodenitis which was similar to study done by other authors.[5, 60]

CONCLUSION

Upper gastrointestinal tract disorders are one of the most commonly encountered problems in clinical practice. Upper GI endoscopy is a safe and well tolerated procedure. It helps in visualization of specific site of mucosal lesions. Endoscopy is incomplete without biopsy and histopathology is the gold standard for the diagnosis of endoscopically detected lesions. Endoscopic biopsy leads to an early diagnosis of various upper GI lesions and acts as a powerful diagnostic tool for early therapeutic decisions and management of the patients.

SUMMARY

A total of 196 UGIT endoscopic biopsies were studied received in the Department of Pathology, B.L.D.E. University's Shri B.M.Patil Medical College, Hospital & Research Centre, Bijapur from July 2010 to July 2014.

The histomorphological patterns of all the biopsies were studied and divided according to the site and nature of lesions.

Most of the patients with esophageal carcinoma presented with dysphagia and most of the duodenal biopsies were done to rule out malabsorption. Most of the carcinomas of UGIT endoscopically showed ulceroproliferative, nodular growth. Few cases showed stricture. Cases of esophagitis, gastritis and duodenitis did not show any significant finding on endoscopy in majority of the cases.

SCC of esophagus occurred more commonly in elderly age group i.e. between 50 to 75years. Adenocarcinoma of stomach occurred more commonly in middle aged and elderly age i.e. between 30 to 70years. However esophagitis, gastritis and duodenitis occurred in all age groups from 2^{nd} to 8^{th} decade.

Overall the numbers of neoplastic lesions were equal to non neoplastic lesions. Most commonly diagnosed non-neoplastic lesion in esophagus was chronic esophagitis and neoplastic lesion was SCC. Commonly diagnosed non-neoplastic lesion in stomach was chronic gastritis & and neoplastic lesion was adenocarcinoma. In duodenum, majority of cases were of chronic duodenitis.

Diagnostic interpretation limitations on endoscopic biopsies were encountered at times due to tiny biopsy material, handling and processing artifacts. These limitations can be overcome by taking multiple endoscopic biopsies.

Endoscopic biopsy leads to early diagnosis of various UGIT lesions. And thus helps in early therapeutic decisions & management of patients.

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ANNEXURE-I

ETHICAL CLEARANCE

B.L.D.E. UNIVERSITY'S
SHRI.B.M.PATIL MEDICAL COLLEGE, BIJAPUR-586 103
INSTITUTIONAL ETHICAL COMMITTEE

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Ethical Committee of this college met on 18-10-2012 at 3-30pm to scrutinize the Synopsis of Postgraduate Students of this college from Ethical Clearance point of view. After scrutiny the following original/corrected & revised version synopsis of the Thesis has been accorded Ethical Clearance.

Title "Spectrum of histomorphological patterns of upper gastrointestinal tract endoscopic biopsies"

Name of P.G. student Dr. Sneha Tawalkar
Pathology

Name of Guide/Co-investigator Dr Surekha Arakeri
Prof. Pathology

DR.TEJASWINI. VALLABHA
CHAIRMAN
INSTITUTIONAL ETHICAL COMMITTEE
BLDEU'S, SHRI.B.M.PATIL
MEDICAL COLLEGE, BIJAPUR.

Following documents were placed before E.C. for Scrutinization
1) Copy of Synopsis/Research project.
2) Copy of informed consent form
3) Any other relevant documents.

ANNEXURE-II

SAMPLE INFORMED CONSENT FORM

TITLE OF THE PROJECT : SPECTRUM OF HISTOMORPHOLOGICAL PATTERNS OF UPPER GASTROINTESTINAL TRACT ENDOSCOPIC BIOPSIES

GUIDE : Dr. SUREKHA U. ARAKERI
M.D. Pathology

POSTGRADUATE STUDENT: Dr. SNEHA JAWALKAR

PURPOSE OF RESEARCH:

I have been informed that the present study will be done to know the morphological patterns and frequencies of lesions in upper GI endoscopic biopsies.

PROCEDURE:

I understand that after having obtained a detailed clinical history thorough clinical examination will be done and after that endoscopic biopsy will be done & will be sent for histopathological examination.

RISK AND DISCOMFORTS:

I understand that I may experience some pain and discomforts during the endoscopy procedure or during taking the biopsy. This is mainly the result of my condition and the procedures of this study are not expected to exaggerate these feelings which are associated with the usual course of treatment.

BENEFITS:

I understand that my participation in the study will have no direct benefit to me other than the potential benefit of the treatment.

CONFIDENTIALITY:

I understand that the medical information produced by this study will become a part of hospital records and will be subject to the confidentiality. Information of sensitive personal nature will not be part of the medical record, but will be stored in the investigations research file.

If the data are used for publication in the medical literature or for teaching purpose, no name will be used and other identifiers such as photographs will be used only with special written permission. I understand that I may see the photograph before giving the permission.

REQUEST FOR MORE INFORMATION:

I understand that I may ask more questions about the study at anytime; **Dr. SNEHA JAWALKAR** at the Department of Pathology is available to answer my questions or concerns. I understand that I will be informed of any significant new findings discovered during the course of the study, which might influence my continued participation. A copy of this consent form will be given to me to keep for careful reading.

REFUSAL OR WITHDRAWAL OF PARTICIPATION:

I understand that my participation is voluntary and that I may refuse to participate or may withdraw consent and discontinue participation in the study at any time without prejudice. I also understand that **Dr**. **SNEHA JAWALKAR** may terminate my participation in the study after she has explained the reasons for doing so.

INJURY STATEMENT:

I understand that in the unlikely event of injury to me resulting directly from my participation in this study, if such injury were reported promptly, the appropriate treatment would be available to me. But, no further compensation would be provided by the hospital. I understand that by my agreements to participate in this study and not waiving any of my legal rights.

I have explained to _____the Purpose of the research, the procedures required and the possible risks to the best of my ability.

_____ _____

Dr. SNEHA JAWALKAR Date

(Investigator)

STUDY SUBJECT CONSENT STATEMENT:

I confirm that **Dr. SNEHA JAWALKAR** has explained to me the purpose of research, the study procedure, that I will undergo and the possible discomforts as well as benefits that I may experience in my own language. I have been explained all the above in detail in my own language and I understand the same. Therefore I agree to give consent to participate as a subject in this research project.

_____ _____

(Participant) Date

_____ _____

(Witness to signature) Date

ANNEXURE-III

SCHEME OF CASE TAKING

1) Name	**:**	**CASE NO**	**:**
2) Age	**:**	**IP NO**	**:**
3) Sex	**:**	**DOA**	**:**
4) Religion	**:**	**DOD**	**:**
5) Occupation	**:**		
6) Residence	**:**		
7) Presenting Complaints	**:**		

9) Past History :

10) Personal History :

11) Family History :

12) Treatment History :

13) General Physical Examination

Pallor	**present/absent**
Icterus	**present/absent**
Clubbing	**present/absent**
Generalized Lymphadenopathy	**present/absent**
Anasarca	**present/absent**
Built	**Poor/Average /Well**
Nourishment	**Poor / Average /Well**

Ophthalmic examination:

Vitals:-

PR	**:**	**BP**	**:**
RR	**:**	**Temp**	**:**
Weight	**:**		

Systemic Examination:

i. **Respiratory System**

ii. **Cardiovascular System**

iii. **Central Nervous System**

iv. **Per abdomen examination**

Provisional Diagnosis:

Investigations:

Hematological examination

Ultrasonography:

Endoscopy findings:

Histopathological examination:

Macroscopy:

Microscopy:

Special stains:

Final Diagnosis:

KEY TO MASTER CHART

EGAS	-	Esophagogastric anastomosis site
Dys	-	Dysphagia
Dyp	-	Dyspepsia
Vom	-	Vomiting
Drh	-	Diarrhea
Epi Pain	-	Epigastric pain
Epi Full	-	Epigastric fullness
Pain abd	-	Pain abdomen
Hem	-	Hematemesis
Ls st	-	Loose stools
Anx	-	Anorexia
LOA	-	Loss of appetite
Cp	-	Chest Pain
Chr	-	Chronic
Eso	-	Esophagus
Sto	-	Stomach
Mal	-	Malabsorbtion
Diff	-	Differentiated
Mod	-	Moderately
Ca	-	Carcinoma

MASTER CHART

Sl.No.	NAME	OPD/IPD No.	HPR No.	AGE	SEX	SITE OF BIOPSY	Clinical history	Clinical Diagnosis	Endoscopic Findings	HPR Diagnosis
1	Radhabai	12087	1911/10	52	F	E(m)	Dys	?Ca esophagus	Friable growth	Mod diff SCC
2	Nagamma	144443	2095/10	70	F	E(l)	Dys	?Ca esophagus	Ulcerative,nodular,friable growth	Well diff SCC
3	Saliya Maniyar	12700	2106/10	30	F	G(p)	Vom	?Chr gastritis	Erosions	Chr non-specific gastritis
4	Mallamma	153847	2274/10	60	F	E(m)	Dys	?Ca esophagus	Strictures with growth	Well diff SCC
5	Kasturibai	155342	2322/10	26	F	GEJ	Pain abd	?Barrett's esophagus	Grade 2 esophagitis	Chr non-specific esophagitis
6	Madevi	15621	2598/10	55	F	E(m)	Dys	?Ca esophagus	Growth in esophagus	Well diff SCC
7	Basappa	197466	3010/10	70	M	E(m)	Dys	?Ca esophagus	Growth in esophagus	Mod diff SCC
8	Draxayani	20126	3282/10	46	F	D	Drh	Malabsorption	Loss of mucosal folds	Chr non-specific duodenitis
9	Krishnabai	20309	3314/10	60	F	G(p)	Vom	?Ca stomach/Peptic ulcer	Ulcers & nodularity at pylorus	H. pylori gastritis
10	Gurupadappa	223904	3358/10	50	M	GEJ	Dys	?Ca esophagus	Growth at GEJ	Mod diff SCC
11	Sunandabai	256369	3854/10	41	F	G(a)	Dyp	?gastritis	No significant findings	Chr non-specific gastritis
12	L S Choragi	261814	3943/10	58	M	G(p)	Pain abd,Dst,Vom	?Ca stomach	Growth with ulcers	Mod diff adenocarcinoma
13	Gourabai	24254	3953/10	74	F	E(m)	Dys	?Ca esophagus	Ulceroproliferative growth	Poorly diff SCC
14	Neela Pattanshetty	284894	4261/10	65	F	E(l)	Dyp	GERD	Esophageal stricture	Barrett's esophagus
15	Siddawwa	26682	4260/10	60	F	G(f)	Dys	?Ca stomach	Ulcerated nodular friable growth	Mod diff adenocarcinoma
16	UdayKumar	26775	4273/10	4	M	D	Chr drh	CS/CF/IBD	Loss of mucosal folds	Celiac disease
17	Shivappa Hudedar	492	116/11	55	M	G(f)	Epi ful	?Ca Stomach, ?Ca pancreas with mets	Ulcerated nodular friable growth	Poorly diff adenocarcinoma
18	Gurunath	13770	199/11	70	M	E(m)	Dys	?Ca esophagus	Esophageal growth	Poorly diff SCC
19	Mallanagouda Biradar	4737	874/11	50	M	E(m)	Dys	?Ca esophagus	Growth with strictures	Poorly diff SCC
20	Peerappa	4876	890/11	35	M	G(pp)	Pain abd	?Ca stomach	Ulceroproliferative growth	Mod diff adenocarcinoma
21	Dundawwa	5549	1000/11	65	F	E(m)	Dys	?Ca esophagus	Esophageal growth	Mod diff SCC
22	Shivaji M	78423	1192/11	70	M	E(m)	Dys	?Ca esophagus	Esophageal growth	Well diff SCC
23	Moulasaab Walikar	79252	1215/11	65	M	D	Chr drh	?Sprue	No significant finding	Chr non-specific duodenitis
24	Gangawwa	6845	1229/11	48	F	G(c)	Pain abd, vom	?Ca stomach	Ulceroproliferative growth	Signet ring adenocarcinoma
25	Ramu	6904	1274/11	9	M	D	Drh,vom	?Chr malabsorption	Scant duodenal folds in 2nd Part	H. pylori duodenitis
26	Sarubai	84102	1292/11	55	F	E(m)	Dys	?Ca esophagus	Mid esophageal growth	Well diff SCC
27	Jyoti Byokod		1853/11	22	F	E(l)	Vom	Esophagitis?Candidiasis	Tiny white patches over lower esophagus	Chr non-specific esophagitis
28	Umawwa	11933	2127/11	74	F	E(m)	Dys	?Ca esophagus	Esophageal growth	Mod diff SCC
29	Basamma	14294	2557/11	52	F	E(m)	Dys	?Ca Esophagus	Strictures with growth	Mod diff SCC
30	Ambadas Salonke	15246	2736/11	72	M	GEJ	Dys,vom	?Ca Esophagus	Ulcerarated friable growth at GEJ	Mod diff adenocarcinoma
31	Iranna Paddar	135	2939/11	59	M	GEJ	Vom	? Ca esophagus	Growth at GEJ	Mod diff SCC
32	Devi Dayal	208352	3135/11	62	M	E(m)	Dyp	Gastritis	Red patch above squamocolumnar junction	Barrett's esophagus
33	Basavaraj Hugar	19785	3416/11	30	M	D	Chr drh	Pan gastritis	No significant findings	Chr non-specific duodenitis
34	Mallawwa	230498	3417/11	75	F	E(m)	Dys	?Ca esophagus	Esophageal growth	Mod diff SCC
35	Somalingappa	21416	3666/11	64	M	E(m)	Dyp	?Ca esophagus	Ulcerated nodular friable growth	Mod diff SCC
36	Siddaramappa	255541	3819/11	75	M	E(l)	Dys	?Ca esophagus	Growth in esophagus	Mod diff SCC
37	Shankrappa Bandi		3881/11	45	M	E(m)	Dys	?Ca esophagus	Strictures with growth	Well diff SCC
38	Bharat	265534	3995/11	22	M	D	Chr drh	?Sprue	No significant findings	Chr non-specific duodenitis
39	Kalabai	23205	4019/11	48	F	E(m)	Dys	?Ca esophagus	Ulcerated growth	Mod diff SCC
40	Pandurang	24073	4216/11	46	M	G(f&c)	Hem	Leiomyoma	Multiple erosions in body and fundus	Chr non-specific gastritis
41	Shantabai	V/34/11	4285/11	62	F	G(F)	Pain Abd, vom	?Ca stomach	Ulcerative nodular friable growth	Mod diff adenocarcinoma
42	Mallamma Hiremat	25484	4550/11	50	F	E(m & l)	Dys	?Ca esophagus	Ulcerated nodular friable growth	Poorly diff SCC
43	Chaganabai Sadiwala	301591	4709/11	60	F	GEJ	Dys	?Ca esophagus	Growth with strictures	Mod diff SCC
44	Sharanamma	27113	4944/11	75	F	E(l)	Dys	?Ca esophagus	Esophageal strictures with growth	Mod diff SCC
45	Chandrappa Hikkongulli	01-Jan	25/12	70	M	E(m)	Dys	?Ca esophagus	Nodular growth	Mod diff SCC
46	Ranolappa	1611	280/12	63	M	E(m)	Dys	?Ca esophagus	Ulcer	Highly suspicious for malignancy
47	Kashinath	1474	293/12	20	M	D	Vom	Aganglionosis of duodenum	Duodenum dilated upto 3rd part	Inadequate for opinion
48	Pareppa Balabatti	19017	337/12	24	M	D	Chr drh	Tropical sprue	Loss of mucosal folds	Villous atrophy with crypt hyperplasia
49	Asif Nadaf	19108	468/12	18	M	D	Chr drh	?Brunner gland hypertrophy	No significant finding	Chr non-specific duodenitis
50	Maleppa Biradar	19082	427/12	44	M	G(a)	Dyp	Gastritis	No significant finding	Chr non-specific gastritis
51	Sidappa Mandeep	1726	370/12	48	M	G(p)	Paiu abd	Gastritis & duodenal polyposis	Polyp	Inflamatory gastric polyp
52	Kumar Dudgi	2069	376/12	13	M	D	Ls st	?Kochs ?Malabsorption	No significant findings	Chr non-specific duodenitis
53	Vittal Logavi	2099	408/12	62	M	G(p)	Pain abd,vom	?Ca stomach	Ulceroproliferative growth	Poorly diff SCC
54	Sangeetha	11L133	607/12	28	F	E(m)	Dyp	Esophagitis	Grade 2 esophagitis	Chr non-specific esophagitis
55	Jaibunissa	11L152	660/12	65	F	D	Chr drh	?Malabsorption	Loss of mucosal folds	Chr non-specific duodenitis
56	A R Sonagi	11L26	754/12	55	M	G(pp)	Pain abd,vom	?Ca stomach	No significant finding	Chr non-specific gastritis
57	Gangayya	11L93	920/12	62	M	E(m)	Dyp	? Ca esophagus	Nodular growth	Mod diff SCC
58	Bhuvaneshwari	19193	936/12	74	F	D	Chr drh	Malabsorption	No significant findings	Inadequate for opinion
59	Sidamma	11L92	917/12	55	F	E(l)	Dyp	?Ca esophagus	Esophageal stricture	Well diff SCC
60	Hazifa	19196	944/12	65	M	E(m)	Vom	?Ca esophagus	No significant finding	Chr esophagitis with moderate dysplasia
61	Siddappa	19210	986/12	54	M	G(pp)	Pain abd	Prepyloric ulcer /?Ca stomach	No significant finding	Ulcer with suppurative necrosis
62	Gangamma	19328	1290/12	66	F	E(m)	Dys	Ca esophagus	Ulceroproliferative growth	Mod diff SCC
63	Jayashree	19343	1334/12	44	F	D	Chr drh	Malabsorption	No significant findings	Chr non-specific duodenitis
64	Chandrakant	19344	1337/12	55	M	G(c)	Vom,drh	Gastritis	Gastritis	Chr non-specific gastritis
65	Bhagawwa	6746	1431/12	65	F	E(m)	Dys	?Ca esophagus	Growth below upper esophageal sphincter	Poorly diff Ca. DD:Small cell ca

66	Kamanna	19164	1645/12	65	M	E(l)	Dys	Ca esopahgus	Nodular growth	Mod diff SCC
67	RajMohammad	19165	1651/12	71	M	G(f)	Vom	Gastritis	No significant findings	Chr non-specific gastritis
68	Iramma	7856	1686/12	45	F	G(pp)	Pain abd, LOA	Acid peptic disease	Growth in the prepyloric region	Mod diff adenocarcinoma
69	Appasab S	7989	1712/12	56	M	G(p)	Vom,drh	GOO?malignancy	Pyloric stenosis with ulcer and nodularity	Signet ring adenocarcinoma
70	Iranna	19662	1963/12	80	M	G(p)	Pain abd,vom	Gastritis	No significant findings	Chr gastritis with intestinal metaplasia
71	Raneeyraja	19456	2283/12	23	M	D	Chr drh	Malabsortion	No significant findings	Chr non-specific duodenitis
72	Rita Jain	19457	2284/12	35	F	D	Chr drh	IBD	No significant findings	Chr non-specific duodenitis
73	Bhajantri V	119814	2290/12	70	M	GEJ	Anx	Barrett's /Ca Esophagus	Nodule in esophagus	Barrett's esophagus
74	Mallanna	19460	2292/12	49	M	G(p)	Vom	Acid peptic disease	No significant findings	Chr gastritis with intestinal metaplasia
75	Vithal K	19519	2397/12	21	M	D	Chr drh	Malabsorption	No significant findings	Chr non-specific duodenitis
76	Shranappa	19589	2497/12	56	M	G(p)	Vom,drh	Ca stomach	Ulceroproliferative growth	Well diff adenocarcinoma
77	Mallikarjun	19584	2502/12	23	M	D	Ls st	Gastroenteritis	No significant findings	Chr non-specific duodenitis
78	Chandan	19581	2506/12	60	M	D	Chr drh	Duodenitis	No significant findings	Chr non-specific duodenitis
79	Devappa	147545	2564/12	38	M	E(u)	Dys	Esophageal carcinoma	Mucosal thickening	Mild dysplasia
80	Vithal M	19743	2681/12	48	M	D	Ls st	Gastroenteritis	No significant findings	Chr non-specific duodenitis
81	Amruta	19839	2933/12	33	F	D	Chr drh	Malabsorption	No significant findings	Chr non-specific duodenitis
82	Ramesh Pyuti	168437	2937/12	48	M	GEJ	Dys	Ca esophagus	Stricture with growth	Mod diff SCC
83	Gangadhar Badiger	13375	2963/12	65	M	E(m)	Dys COPD	?Ca esophagus	Mid esophageal stricture	Well diff SCC
84	Malliakarjun	19862	3012/12	36	M	E(m)	Dys	Esophageal Ca	Nodular growth	Chr non-specific esophagitis
85	Shantabai	19873	3045/12	45	F	E(l)	Vom	Peptic ulcer disease	Ulcer at squamocolumnar junction	Ulcer with foci of granulation
86	Gangadhar	14221	3085/12	30	M	D	Pain abd	Pain Abd under evaluation	No significant findings	Normal histology
87	Shantabai	178724	3126/12	45	F	EGAS	Vom,Pain abd	?Recurrent Ca esophageal,? Benign Ulceration	Stricture with growth	Mod diff SCC
88	Shivaraj	19922	3177/12	15	M	D	Chr drh	Malabsorption	No significant findings	Chr non-specific duodenitis
89	Bhimashri	180992	3182/12	70	F	E(m)	Dys	Ca esophagus	Growth in esophagus	Well diff SCC
90	Davalat Pujari	14851	3258/12	70	M	E(m)	Dys	Ca esophagus	Fungating mass	Poor diff SCC
91	Manoj K	19966	3325/12	48	M	G(pp)	Pain abd	Gastritis	No significant findings	Chr non-specific gastritis
92	Lalsaab	20006	3412/12	55	M	G(p)	Pain abd, vom	Peptic ulcer disease	Healing ulcer at pylorus	Chr non-specific gastritis
93	Iranna Gouda Dinni	15727	3495/12	45	M	E(l)	Dys	Severe anemia	Erosions in lower eophagus	Barrett's esophagus
94	Hanmanthray Jumaner	199936	3500/12	77	M	G(pp)	Pain abd	Gastritis	No significant findings	Chr non-specific gastritis
95	Chohku	20077	3560/12	60	M	E(m)	Dyp	GERD	Polyp	Hyperplastic polyp
96	Shanawaz	20086	3579/12	25	M	D	Ls st	Gastroenteritis	No significant findings	Chr non-specific duodenitis
97	Sangamesh	20257	3612/12	32	M	D	Chr drh	Malabsorption	Loss of mucosal folds	Chr non-specific duodenitis
98	Laxman	16949	3799/12	65	M	D	Pain abd	Duodenitis	Duodenal Ulcer with Grade A Esophagitis	Chr non-specific duodenitis
99	Kamalabai	17752	3884/12	84	F	GEJ	Dys	Severe esophagitis/ Barett's esophagus	A small patch of nodularity seen at GEJ	Chr non-specific inflammation
100	Humanappa	19314	4154/12	80	M	GEJ	Dys	?Ca esophagus	Ulceroproliferative Mass	Well diff adenocarcinoma
101	M S Kumar	243003	4214/12	39	M	D	Chr drh	Duodenitis	No significant findings	Chr non-specific duodenitis
102	Dhareppa	198441	4237/12	72	M	E(u)	Dys,Dyp	?Ca esophagus	Smooth friable mass	Chr non-specifc esophagitis with mild dysplasia
103	Tukkubai	21464	4528/12	58	F	E(l)	Dys Dyp	Ca esophagus	Ulceroproliferative growth	Well diff SCC
104	Rachappa	278399	4809/12	55	M	E(m)	Dys	Ca esophagus	Ulcerated Nodular Friable Growth	Mod diff SCC
105	Parvatibai	20607	4862/12	78	F	G(pp)	Pain abd	Gastritis	Polyp	Hyperplastic polyp
106	Pandit	23007	4866/12	60	M	G(f)	Pain abd	Ca stomach	Ulcerated Nodular friable growth	Mod diff adenocarcinoma
107	M A Maniyar	20618	4948/12	85	M	E(l)	Dys	Ca esophagus	Ulceroproliferative growth	Well diff adenocarcinoma
108	Shivanna Talikoti	25226	5202/12	55	M	E(m)	Dys	Fungal esophagitis with pulmonary Kochs	Fungating mass	Mod diff SCC
109	Savita	V/95/12	5230/12	20	F	G(p)	Epi ful	Ca stomach	Ulceroproliferative growth	Poorly diff adenocarcinoma
110	Girish Mirajkar	335915	5732/12	38	M	D	Chr drh	?Sprue	No significant findings	Chr non-specific duodenitis
111	Chandrashekar	21036	5848/12	44	F	D	Ls st	Malabsorption	No significant findings	Chr non-specific duodenitis
112	Ballavantray H	21046	5935/12	45	M	D	Ch drh	IBD	No significant findings	Chr non-specific duodenitis
113	Shrishail	21051	6001/12	40	M	D	Ls st	?Sprue	No significant findings	Chr non-specific duodenitis
114	Parasuram	21083	20/13	42	M	E(l)	Dys	Ca esophagus	Growth	Mod diff SCC
115	Malasidappa	21235	160/13	68	M	E(m)	Dys	Ca esophagus	Ulceroproliferative growth	Well diff SCC
116	Iranna	21658	590/13	32	M	E(m)	Dys	GERD	No significant findings	Chr non-specific esophagitis
117	S B Patil	31266	602/13	49	M	E(m)	dys dyp	?Embedded foreign body?Ca esophagus	Nodular swelling	Indequate for opinion
118	GuruNingappa	3900	835/13	46	M	G(pp)	Dys	Gastritis	Antral gastritis	H. pylori gastritis
119	Gerngi	21662	922/13	76	M	E(l)	Dyp	GERD	Red patch above squamocolumnar junction	Barrett's esophagus
120	Lalu	4902	1062/13	70	M	E(u)	Dys	?Ca esophagus	Ulceroproliferative growth	Mod diff SCC
121	Sangappa	21531	1149/13	50	M	G(pp)	Dys	Ca stomach	Ulceroproliferative growth	Poorly diff adenocarcinoma with candidiasis
122	Prakash N	21907	1388/13	41	M	D	Chr drh	Malabsorption	No significant findings	Chr non-specific duodenitis
123	Siddanagouda Patil	31266	1471/13	49	M	E(m)	Dys	?Ca esophagus/Benign lesion	Mucosal thickening	Mild dysplasia
124	Girimallu Julapi	6927	1474/13	65	M	G(f)	Vom,Pain abd	?Ca stomach	Fungating Mass	Mod diff adenocarcinoma
125	Siddaram	8029	1627/13	60	M	GEJ	Vom,Pain abd	?Ca esophagus	Ulceroproliferative friable growth	Well diff adenocarcinoma
126	Ashok Baraknalli	8261	1670/13	54	M	E(l)	Dys	?Ca esophagus	Ulcerated nodular growth	Mod diff SCC
127	Parasuram	21083	1814/13	42	M	E(m)	Dys	Ca esophagus	Ulceroproliferative growth	Mod diff SCC
128	Husanappa	21094	1817/13	60	M	E(m)	Dys	Ca esophagus	Stricture with growth	Poorly diff Ca
129	Revappa	21125	1837/13	48	M	E(l)	Dys Dyp	Ca esophagus	Ulceroproliferative growth	Poorly diff Ca. DD: Small cell ca
130	Jairam	5547	1197/13	80	M	G(pp)	Vom,Pain abd	Acute abdomen	Pre Pyloric Perforation	Candidal infection
131	Annappa	Y/3/13	2046/13	40	M	D	Chr drh	Malabsorption	Loss of mucosal folds	Chr non-specific duodenitis
132	HussainPasha	22070	2066/13	87	M	E(m)	Dys	Ca esophagus	Mucosal thickening	Chr non-specific esophagitis
133	Sangappa	10771	2101/13	60	M	G(pp)	Dys,Dyp	Gastritis	Erosions at prepyloric region	Highly suspicious of malignancy

134	Ambrappa	22084	2168/13	60	M	E(l)	Pain abd	GERD	No significant findings	Chr non-specific esophagitis
135	Sangappa	22219	2288/13	65	M	G(p)	Vom,Pain abd	Gastritis	Polyp at pylorus	Adenomatous polyp
136	Shivappa	12654	2474/13	64	M	D	Chr drh	Malabsorption	No significant findings	Chr non-specific duodenitis
137	Basamma	14090	2711/13	30	F	D	Chr drh	Malabsorption	Loss of mucosal folds	Chr non-specific duodenitis
138	S G Parappannavar	41051	2756/13	75	M	G(pp)	Vom,Drh	Gastritis	Ulceroproliferative growth	Poorly diff Ca
139	Sharu	13686	2808/13	52	M	E(m)	Cough	Tracheoesophageal fistula	Ulceroproliferative growth	Mod diff SCC
140	Gangubai Balikai	166703	2861/13	68	F	GEJ	Pain abd,Regur,nau,	Polyp At GEJ	Polyp at GEJ	Hyperplastic polyp
141	Iramma	22627	2913/13	75	F	E(l)	Dys	Ca esophagus	No significant findings	Chr non-specific esophagitis
142	Amrappa	22152	2934/13	60	M	E(l)	Dys,Dyp	GERD	No significant findings	Chr non-specific esophagitis
143	Barawwa Godekar	15604	2948/13	75	M	E(m)	Dys	Ca esophagus	Ulceroproliferative growth	Mod diff SCC
144	Rajshekar	16433	3094/13	68	M	E(l)	Dyp	Ca esophagus	Ulceroproliferative growth	Mod diff SCC
145	Mayawwa Pujari	16462	3153/13	50	F	D	Chr drh	?Periampullary Ca	Ulceroproliferative growth	Well diff adenocrcinoma
146	Nilamma Hiremath	184226	3159/13	55	F	G(pp)	Vom,Drh	Pan gastritis	Pangastritis	Chr non-specific gastritis
147	Shivanna	17173	3238/13	61	M	D	Cp,Brt	Severe anemia	No significant findings	Chr non-specific duodenitis
148	Shakrappa Nidoni	194833	3302/13	75	M	E(l)	Dys	?Ca esophagus	Ulceroproliferative growth	Mod diff adenocarcinoma
149	Neelamma	192391	3326/13	50	F	E(m)	Dyp	GERD	No significant findings	Chr non-specific esophagitis
150	Chandappa	209565	3561/13	50	M	E(m)	Dys	Ca esophagus	Ulcerated nodular friable growth	Mod diff SCC
151	Rajshekar Mannur	21016	3877/13	70	M	GEJ	Dys,vom	?Benign stricture?Ca esophagus	Strictures with growth	Well diff adenocarcinoma
152	Vishnu Madar	247200	4128/13	30	M	G(pp)	Dys	Healing ulcers in prepyloric region	Ulcers at prepyloric region	Chr non-specific gastritis
153	Kashinath	23726	4245/13	30	M	E(l)	Dyp	Esopahgitis	No significant findings	Chr non-specific esophagitis
154	Loku	20733	4475/13	40	M	G(p)	Dys	Ca stomach	Ulcerated nodular friable growth	Mod diff adenocarcinoma
155	Ramu Rathod	24932	4496/13	40	M	GEJ	Vom,Pain abd	?Ca esophagus	Ulceroproliferative growth	Mod diff adenocarcinoma
156	Shankarppa	26396	4728/13	66	M	E(l)	Dys	Ca esophagus	Stricture	Mod diff SCC
157	Kallawwa M	26613	4758/13	80	F	G(c)	Pain abd, vom	Upper GI obstruction	Growth in Lesser Curvature	Signet ring adenocarcinoma
158	H B Desai	292123	4798/13	72	M	G(pp)	Pain abd	Pangastritis	Pangastritis	Chr non-specific gastritis
159	Hemareddy Biradar	28154	5014/13	55	M	G(p)	Pain abd, vom	GOO?malignancy	Ulceroproliferative growth	Well diff adenocarcinoma
160	Manohar	23415	5237/13	32	M	D	Chr drh	R/o Sprue	No significant findings	Chr non-specific duodenitis
161	Rachappa	23896	5403/13	30	M	D	Chr drh	Malabsorption	No significant findings	Chr non-specific duodenitis
162	Iranna	23912	5424/13	24	M	D	Ls st	R/o Sprue	Loss of mucosal folds	Chr non-specific duodenitis
163	Isarail	20678	5506/13	24	M	G(p)	Vom,drh	Gastritis	Erosions at pyloric region	Well diff adenocarcinoma
164	Siddaramappa	20667	5516/13	45	M	D	Chr drh	Sprue	Loss of mucosal folds	Chr non-specific duodenitis
165	Motiram Rathod	20737	5623/13	55	M	E(m)	Dys	ca esophagus	No significant findings	Chr non-specific esophagitis
166	Sharanappa	519	5745/13	51	M	GEJ	Dys	?Malignancy	Growth at GEJ	Mod diff adenocarcinoma
167	Ashok	20815	5777/13	53	M	E(l)	Dys	Ca esophagus	Ulceroproliferative growth	Well diff SCC
168	B H Pujari	20952	5848/13	63	M	E(l)	Dys	Ca esophagus	Ulcerated growth	Mod diff SCC
169	Kaldappa	20842	5852/13	65	M	E(m)	Dyp	Ca esophagus	Stricture	Mod diff SCC
170	Gurubasappa	20874	5859/13	74	M	E(l)	Dys,Dyp	Ca esophagus	Ulceroproliferative growth	Mod diff SCC
171	Baburao	20872	5860/13	80	M	G(pp)	Dys,vom	Ca stomach	Ulceroproliferative growth	Well diff adenocarcinoma
172	Hanamawwa	20857	5865/13	50	F	G(p)	Dys,vom	Ca stomach	Large ulcer at pylorus	Poor diff adenocarcinoma
173	Sevalal	20940	5866/13	50	M	E(l)	Dyp	Ca esophagus	Stricture with growth	Well diff SCC
174	Shobha	2398	5942/13	40	F	G(p)	Vom, Pain abd	Ca Stomach	Nodules in pylorus	Chr non-specific gastritis
175	Mallapaa	22533	6014/13	62	M	G(pp)	Vom,dys	Ca stomach	Ulceroproliferative growth	Poorly diff ca
176	Chanappa	22421	6081/13	73	M	E(l)	Dys	?Ca esophagus	Ulceroproliferative growth	Inadequate for opinion
177	Kamala	24137	6318/13	65	F	E(m)	Dys, Dyp	Ca esophagus	Ulcerated nodular growth	Poorly diff SCC
178	Ramesh	24135	6319/13	30	M	D	Ls st	Malabsortion	No significant findings	Chr non-specific duodenitis
179	Suresh	24042	6510/13	42	M	D	Chr drh	?Sprue	Loss of mucosal folds	Chr non-specific duodenitis
180	Parvati	23981	6728/13	30	F	D	Chr drh	Malabsorption	loss of mucosal folds	Chr non-specific duodenitis
181	Nirmala	23969	6738/13	42	F	D	Ls st	R/o Sprue	No significant findings	Chr non-specific duodenitis
182	Siddu	23963	42/14	30	M	D	Ls st	R/o Sprue	No significant findings	Villous atrophy with crypt hyperplasia
183	Shivappa	22712	129/14	63	M	D	Pain abd	Gastritis	No significant findings	Chr non-specific duodenitis
184	Hanumant	22757	246/14	30	M	D	Chr drh	R/o Sprue	No significant findings	Chr non-specific duodenitis
185	Mallappa	24244	356/14	32	M	D	Ls st	R/o Sprue	No significant findings	Chr non-specific duodenitis
186	Lingayat	24371	513/14	19	M	D	Ls st	R/o Sprue	Loss of mucosal folds	Chr non-specific duodenitis
187	Siddalingamma	59255	1396/14	68	F	E(l)	Dys	Ca esophagus	Nodular swelling	Ciliated metaplasia
188	Shanta Babanagar	65090	1470/14	38	M	E(l)	Dys,Dyp	Ca esophagus	Stricture with growth	Well diff SCC with candidiasis
189	Mahadev	76419	1684/14	50	M	G(p)	Dys	Ca stomach	Ulceroproliferative growth	Mod diff adenocarcinoma
190	Shikarappa	7841	1900/14	47	M	E(u)	Dyp	Ca esophagus	Strictures with growth	Indequate for opinion
191	Shakuntala	108065	2196/14	50	F	E(m)	Dys	Ca esophagus	Stricture with growth	Poorly diff SCC
192	Siddawwa	126341	2471/14	65	F	E(l)	Dys	Ca esophagus	Ulceroproliferative growth	Mod diff SCC
193	Neelakantaiyya	127039	2494/14	70	M	E(u)	Dys	Ca esophagus	Circumferential ulceroproliferative growth	Indequate for opinion
194	Mrs Saleem D	24552	2507/14	52	F	G(pp)	Vom,Dyp	Gastritis	Erosions at prepyloric region	Chr non-specific gastritis
195	Gouramma	210041	3747/14	65	F	GEJ	Dys	Ca esophagus	Strictures with growth	Mod diff SCC
196	Kamala	213824	3811/14	65	F	E(m)	Dys	Ca esophagus	Growth	Well diff SCC