IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)
e-ISSN: 2279-0853, p-ISSN: 2279-0861.Volume 17, Issue 5 Ver. 7 (May. 2018), PP 77-88
www.iosrjournals.org

Histopathological And Endoscopic Analysis Of Biopsies From Various Lesions Of Upper Gastrointestinal Tract In A Tertiary Health Care Centre.

A.R.Piyush[1],Roobina Khan[1],Hasan Harris[2],Veena Maheshwari[1]
[1](Department of Pathology, Jawaharlal Nehru Medical College, Aligarh Muslim University, India)
[2](Department of Surgery, Jawaharlal Nehru Medical College, Aligarh Muslim University, India)
**Corresponding Author: A.R.Piyush*

Abstract: *A wide spectrum of disorders affect the upper gastrointestinal tract and are responsible for a huge number of morbidity and mortality. Upper GI endoscopy together with histopathology plays an important role in the accurate diagnosis and management of these disorders. Hence we conducted a 2 year prospective study in a tertiary health care centre with the aim of identifying different histopathological lesions occurring at this site.We analysed biopsies of 176 patients with respect to age, gender, site of lesion, possible underlying etiology along with findings of endoscopy and histopathology.*

Date of Submission: 01-05-2018 Date of acceptance: 17-05-2018

I Introduction

Upper Gastrointestinal tract harbours a wide range of neoplastic and nonneoplastic lesions affecting various age group.These disorders lead to broad range of chronic sign and symptoms presentation at indoor and outdoor facilities. Nonneoplastic lesion includes infective,inflammatory, autoimmune, mechanical and other disorders with numerous underlying etiology[1]. An important being, Helicobacter pylori gram-negative, microaerophilic bacteria, associated with gastritis, duodenal ulcer, gastric ulcer, atrophic gastritis, intestinal metaplasia, gastric carcinoma and MALT lymphoma[2,3,4,5,6].Endoscopy started in 1960s not only aids as a diagnostic tool but also helps in monitoring the course, assessing extent of the disease, evaluating response to therapy and helps in early intervention of complications.

II Material And Methods

A prospective study was conducted over a period of 2 years from November 2012 to November 2014 on 176 patients attending the Outdoor & indoor of Department of Surgery, multiple biopsies were taken and processed in Department of Pathology ,Jawaharlal Nehru medical College, AMU, Aligarh.

Study Design: Prospective observational study

Study Location: This was a tertiary care teaching hospital based study done in Department of Pathology ,Jawaharlal Nehru medical College, AMU, Aligarh.

Study Duration: 2 years from November 2012 to November 2014.

Sample Size: 176 patients.

Subjects & selection method: All the adult patients, including both male and female presenting with long standing complaints of upper gastrointestinal symptoms like dysphagia, dyspepsia, nausea and vomiting, bloating, loss of weight, haematemesis were selected. The duration, family history, past history, treatment history ,environmental and hereditary factors, along with preliminary investigations were taken into account. The patients were subjected to endoscopy and multiple biopsies of the suspected lesions were taken . An informed consent was taken before performing endoscopic biopsies.

Inclusion criteria: All patients including both male and female presenting with chronic gastrointestinal complaints.

Exclusion criteria: The patients receiving proton pump inhibitors, antibiotics, or bismuth subsalicylate in the previous 6 wk or those with a history of using nonsteroidal anti-inflammatory drugs and medication for H. pylori infection were excluded from the study.

Procedure methodology: After taking an informed consent ,multiple endoscopic biopsies were taken from the desired lesion along with the documentation of endoscopic appearance of the lesion.The biopsy pieces were fixed in 10% formaldehyde and routinely processed. Approximately 5 micrometer thick sections were cut and histopathological examination was done using Haematoxylin and eosin stain. Special stains like Giemsa, Periodic Acid Schiff stains and Immunohistochemistry was performed wherever necessary. In addition to

classification of lesion as benign or malignant, Haematoxylin and Eosin helped in evaluation of various benign lesions along with severity of inflammation and other significant changes like intestinal metaplasia, atrophy and presence of helicobacter pylori.

III Result

After evaluation of biopsies of 176 patients,following observation was made.

Table: 1 - Distribution of biopsies according to the site.

Site	Number of biopsies	Percentage
Oesophagus	56	32%
Gastric	112	64%
Gastroesophageal junction	08	04%
Total	176	100%

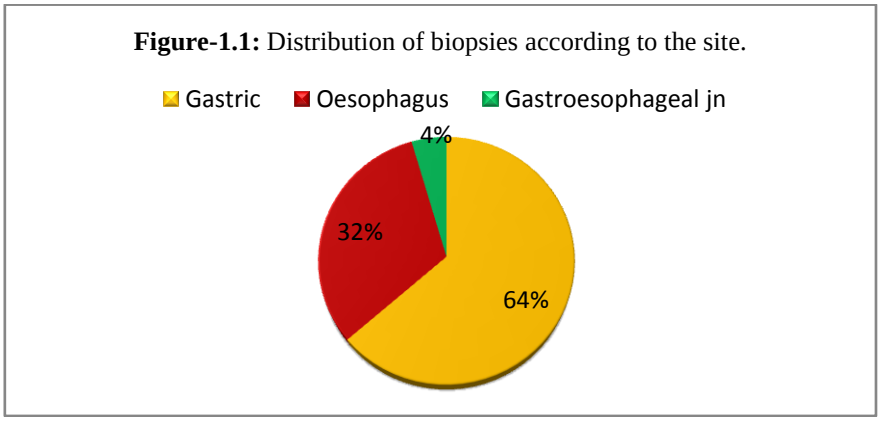


Figure-1.1: Distribution of biopsies according to the site.

Table 1 and Figure 1.1 depicts that out of 176 endoscopic biopsies, most of the biopsies 112(64%) were from gastric region, 56(32%) were from oesophagus and 8 (4%)were from gastroesophageal junction.

Table: 2 - Distribution of patients according to the gender.

Site	Male	Female	Total	percentage
Oesophagus	30(17%)	26(15%)	56	32%
Gastric	75(43%)	37(21%)	112	64%
Gastroesophageal junction	04(2%)	04(2%)	08	04%
Total	109(62%)	67(38%)	176	100%

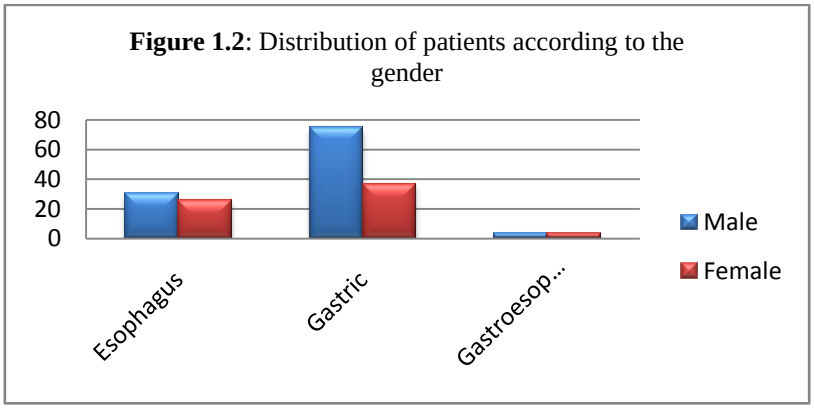


Figure 1.2: Distribution of patients according to the gender

Table 2 and Figure 1.2 shows male preponderance with male patients being 109(62%) and 67(38%) female patients. Gastric lesions had higher male to female ratio of 2:1 while slight male preponderance (M:F :: 1.15 : 1) was noted in oesophageal lesions. Lesions in gastroesophageal junction showed equal predisposition with a ratio of 1:1. Over all male to female ratio was 1.63:1.The mean age calculated in case of both male and female was 47 yrs.

Table 3: Distribution of patients according to the age

Age	Oesophagus	Gastric	Gastroesophageal junction	Total
<20	02(04%)	06(05%)	00	08 (4.5%)
21-30	03(05%)	18(16%)	00	21(12%)
31-40	05(09%)	22(20%)	02(25%)	29(16.5%)
41-50	13(23%)	25(22%)	02(25%)	40(23%)
51-60	16(29%)	22(20%)	02(25%)	40(23%)
61-70	11(20%)	15(13%)	02(25%)	28(15%)
71-80	03(05%)	04(04%)	00	07(04%)
81-90	03(05%)	00	00	03(02%)
Total	56(100%)	112(100%)	08(100%)	176(100%)

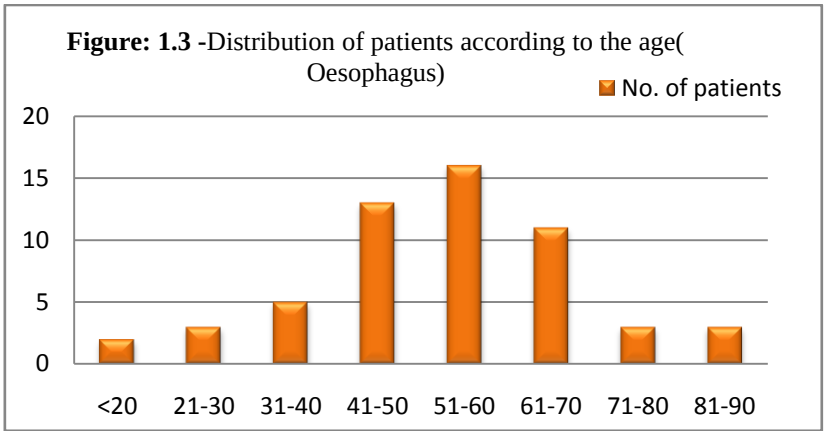


Figure: 1.3 -Distribution of patients according to the age(Oesophagus)

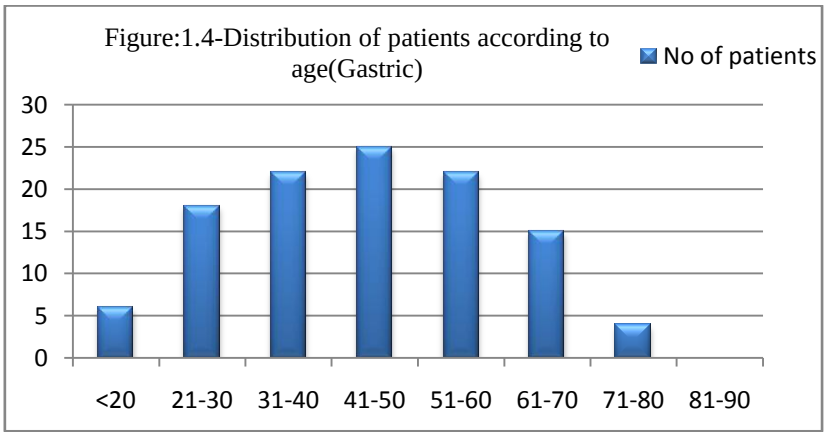


Figure:1.4-Distribution of patients according to age(Gastric)

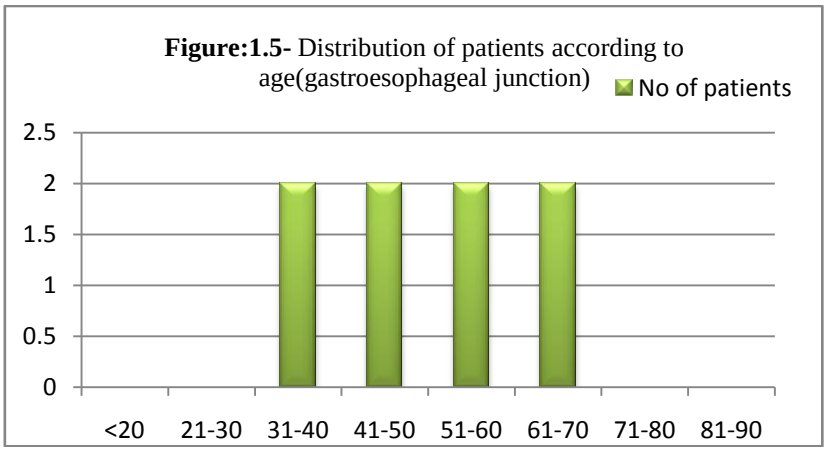


Figure:1.5- Distribution of patients according to age(gastroesophageal junction)

Based on Table 3 and Figure 1.3,1.4 and 1.5, maximum number of patients were in 4th and 5th decade with 23% each. Gastric lesions were maximum in 4th decade(22%) .Second most common age group affected with gastric lesions were between 3rd and 5th decade with 20% cases each. Lesions of oesophagus peaked in 5th

(29%) decade followed by 4th (23%) decade. Gastroesophageal lesions were equally affected between 30-70 yrs.Over all mean age in the lesions of all the sites was 47 yrs.

Table:4- Distribution of presenting complaints in patients according to the site.

Presenting complaints	Oesophagus	Gastric	Gastroesophageal junction	Total
Dysphagia	22(40%)	09(08%)	1(12.5%	32(18%)
Dysphagia with weight loss	15(27%)	04(03%)	1(12.5%)	20(11%)
Dyspepsia	11(20%)	89(79%)	6(75%)	106(60%)
Epigastric pain	07(12%)	78(70%)	5(62.5)	90(51%)
Haematemesis	02(03%)	03(03%)	1(12.5%)	06(03%)
Nausea,vomiting	15(27%)	87(78%)	6(75%)	108(61%)
Total no cases	56(100%)	112(100%)	8(100%)	176(100%)

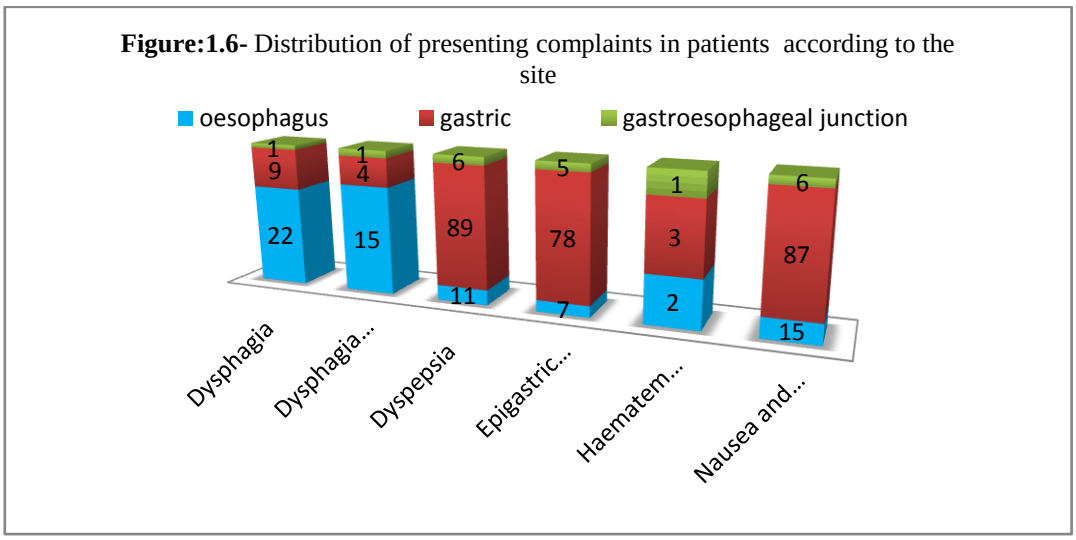


Figure:1.6- Distribution of presenting complaints in patients according to the site

The presenting complaints of the patients is depicted in Table 4 and Figure 1.6, showing that dyspepsia(79%)and nausea,vomiting(78%) were the primary complaints of the patients with gastric lesions followed by epigastric pain(70%). Similar spectrum of complaints were observed in patients of gastroesophageal junction.Dysphagia was seen in 40% of the patients with oesophageal lesions while its association with weight loss was seen in 15 (27%) cases.Haemetemesis was infrequent,accounting for 3% of total number of cases.

Table:5-Histopathological diagnosis of Oesophageal biopsies.

Diagnosis	**Total no of cases**
Oesophagitis	12 (21%)
Squamous papilloma	01 (02%)
Barretts oesophagus	03 (05%)
Dysplasia	16 (29%)
Carcinoma In situ	02 (04%)
Malignancy	22 (39%)
Total	56 (100%)

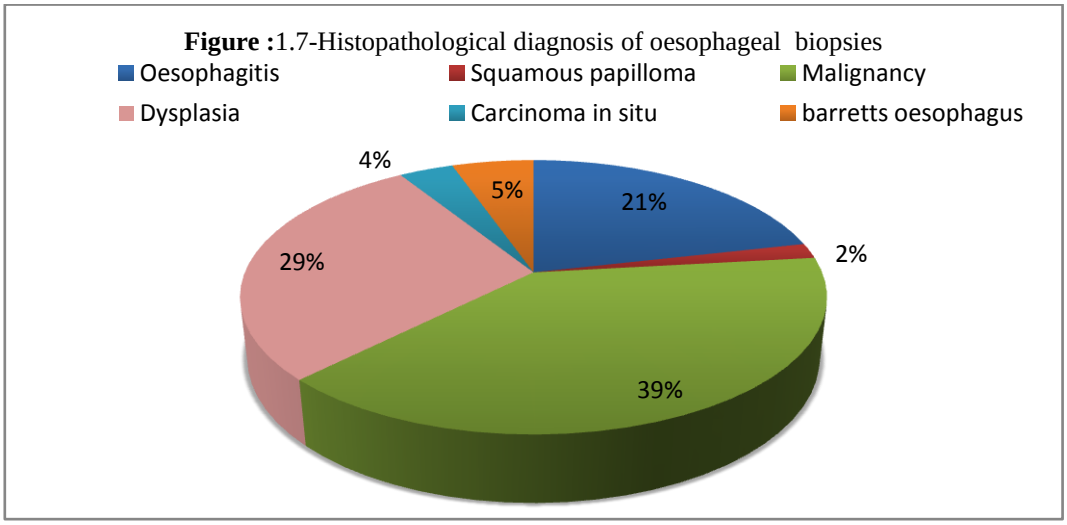


Figure :1.7-Histopathological diagnosis of oesophageal biopsies

The distribution of various oesophageal lesions in the present study is shown in Table 5 and Figure 1.7. A single case of squamous papillom, having stratified squamous lining with underling fibrovascular core was observed.(Fig-2). Barretts oesophagus was seen in 3(5%) of cases one, being associated with mucinous adenocarcinoma (Fig-3).Oesophagitis, was seen in 12(21%) of cases,while 16(29%) cases of dysplasia was observed making it the second most common lesion. Malignancy was the most common lesion consisting of 22(39%) cases. It was composed of 19(34%)cases of Squamous cell carcinoma with well, moderate(Fig-4) , poor(Fig-5) differentiation. It also included a case of Basaloid squamous cell carcinoma. Single case of Mucoepidermoid carcinoma and 2(3%) cases of Adenocarcinoma were also reported.

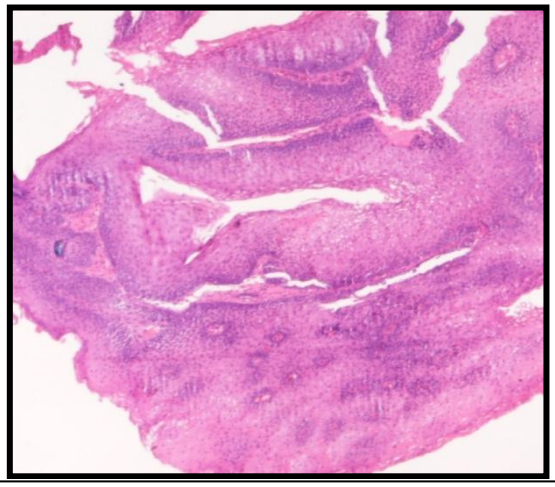


Figure-2: Squamous papilloma of oesophagus showing stratified squamous epithelium with a fibrovascular core (H&E:40X)

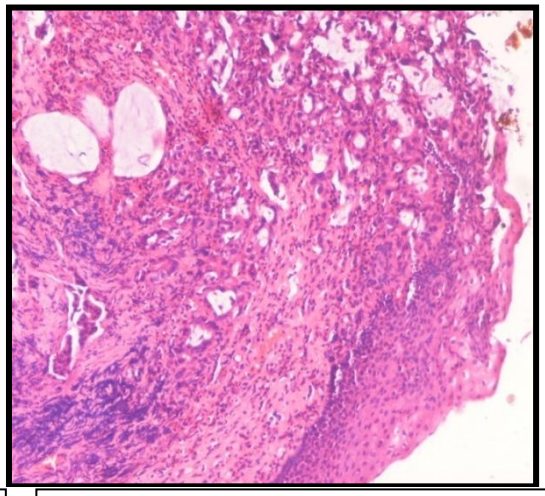


Figure -3: Barrett's oesophagus with associated mucinous adenocarcinoma.(H&E:100X)

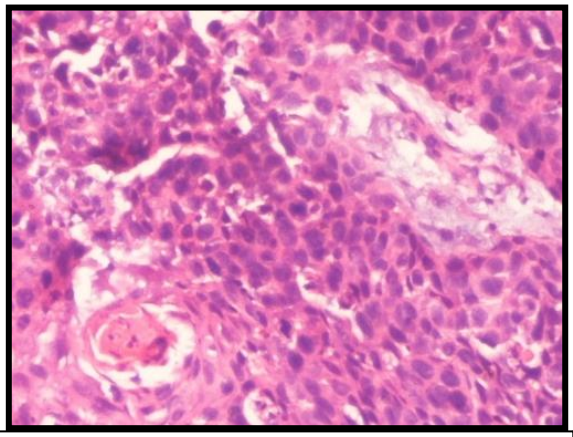


Figure -4: Moderately differentiated Squamous cell carcinoma of oesophagus showing keratinization and malignant squamous cells.H&E:400X)

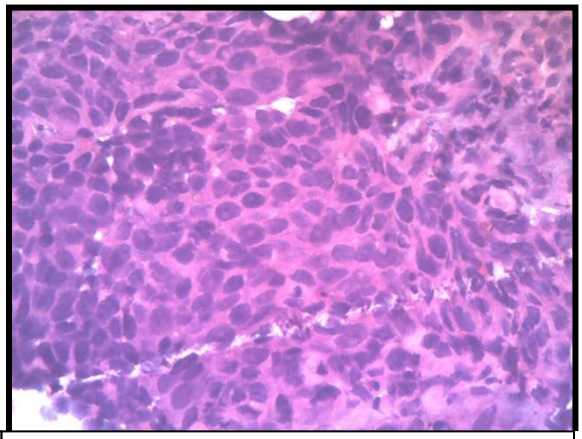


Figure-5: Poorly differentiated Squamous cell carcinoma of oesophagus with minimal evidence of keratinization . (H&E:400X)

Table:6-Histopathological diagnosis of gastric biopsies.

Diagnosis	**Total no of cases**
Acute gastritis	03 (03%)
Chronic non atrophic gastritis	73 (65%)
Chronic atrophic gastritis	23 (21%)
Dysplasia	06 (05%)
Malignancy	07 (06%)
Total	112(100%)

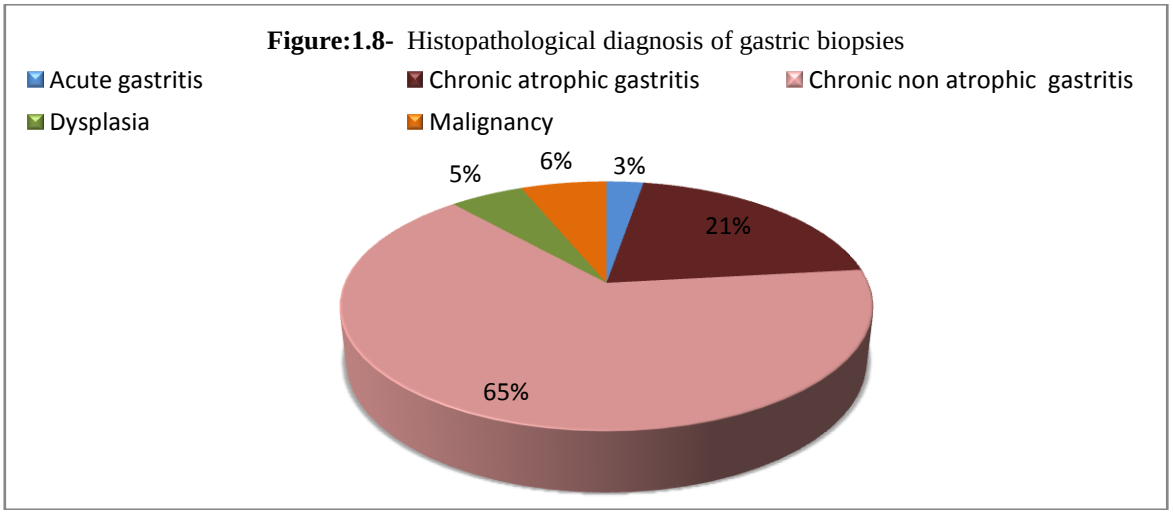


Figure:1.8- Histopathological diagnosis of gastric biopsies

The distribution of various gastric lesion is shown in Table 6 and Figure 1.8 Malignancy was seen in 7(6%) cases which included 5(4%) cases of Adenocarcinoma(Fig-6)out of which 1case was of Signet ring adenocarcinoma.(Fig- 7) and 2 cases of MALT lymphoma (H&E showing monomorphous population of singly scattered atypical cells with features of hyperchromasia, confirmed by application of leucocyte common antigen showing membranous positivity in all the cells-Fig-8).Gastritis was the commonest lesion which included 23(21%) cases of Chronic atrophic gastritis, some of which were associated with Helicobacter pylori infection having lymphoid follicle formation, glandular atrophy and presence of intestinal metaplasia(Fig-9).Rest atrophic gastritis could have autoimmune disorder as underlying pathology(Fig-10).Predominantly Chronic non atrophic gastritis was seen in 73(65%) cases .Chronic non atrophic or chronic active gastritis(Fig-11,12) has features of activity(presence of neutrophilic infiltration), in later stages can even have cryptitis(neutrophilic infiltration within lining), crypt abscess(neutrophilic collection and debris within lumen of gland).Dysplasia and acute gastritis was seen in 6(5%) and 3(3%) cases respectively.

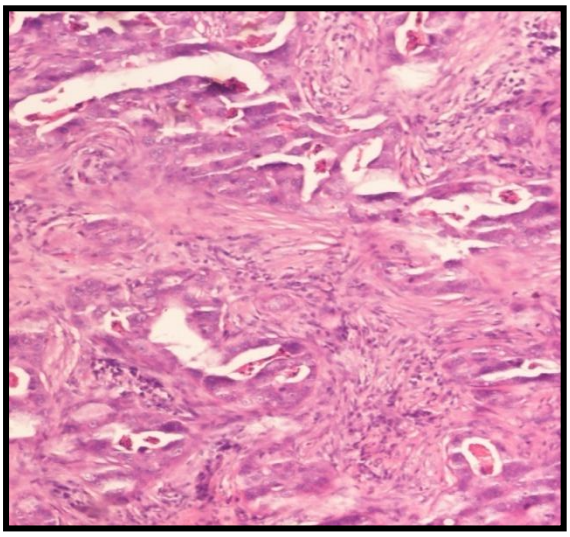


Figure -6: Well differentiated adenocarcinoma of stomach. Showing variable sized atypical glands (H&E:40X ;100X)

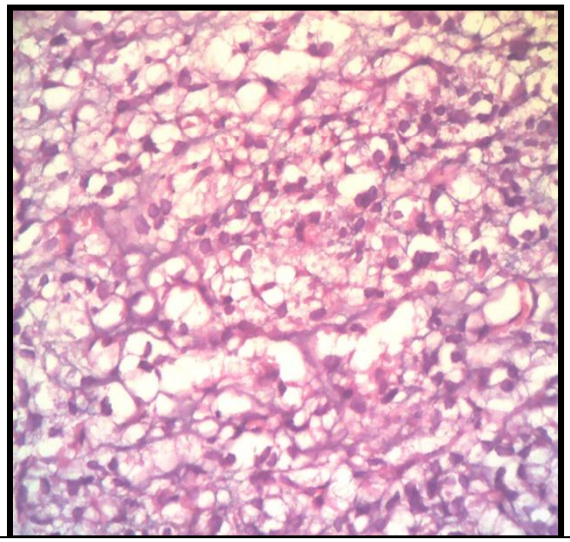


Figure -7: Signet ring adenocarcinoma of stomach showing solid infiltration of atypical vaculated cells with eccentric nuclei (H&E:100X)

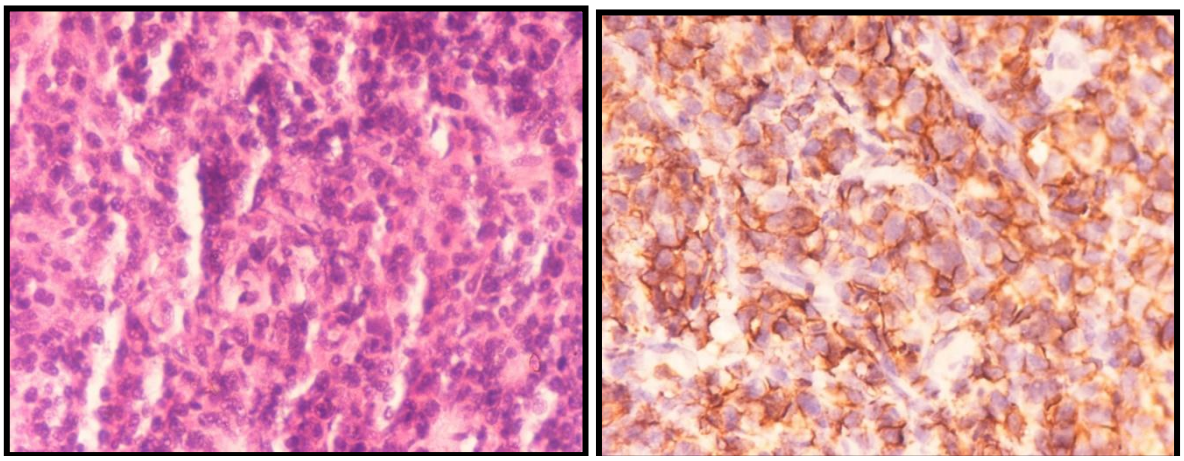


Figure-8: MALT lymphoma of gastrointestinal tract .(H&E:400X; immunohistochemistry of Leucocyte Common Antigen showing membranous positivity in individual cell:400X)

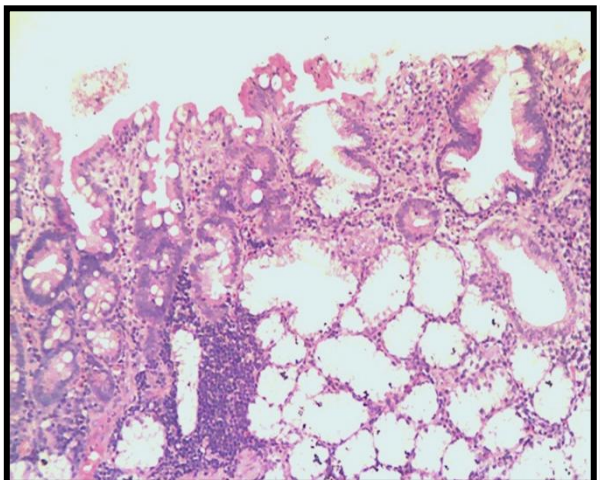


Figure -9: Chronic atrophic gastritis with follicle formation moderate chronic inflammation , atrophy and intestinal metaplasia. (H&E:100X)

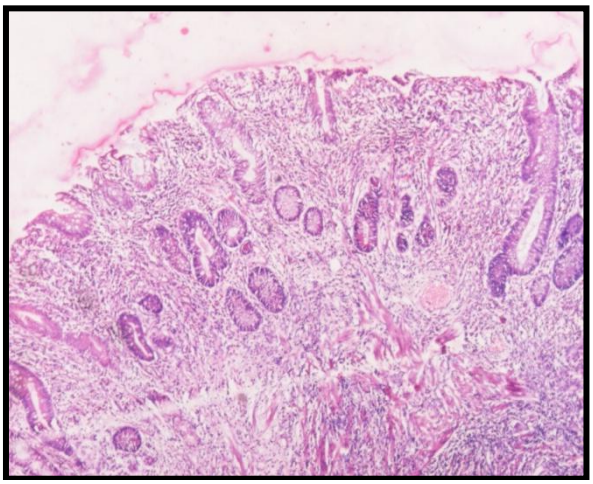


Figure -10: Chronic atrophic gastritis showing marked atrophy of glands. (H&E:100X)

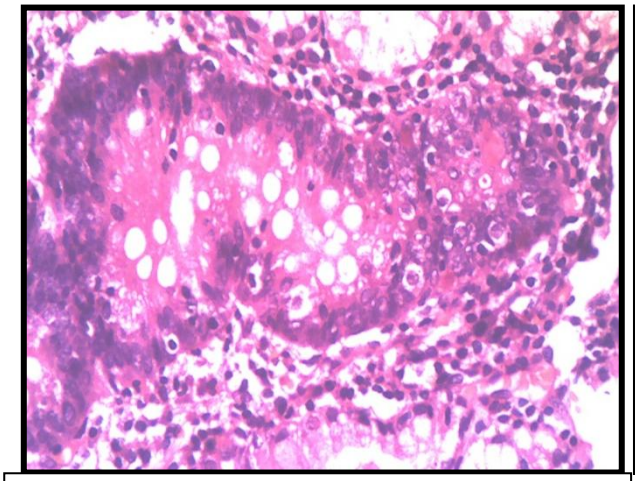


Figure -11: Chronic active gastritis showing cryptitis of the gland(H&E:400X)

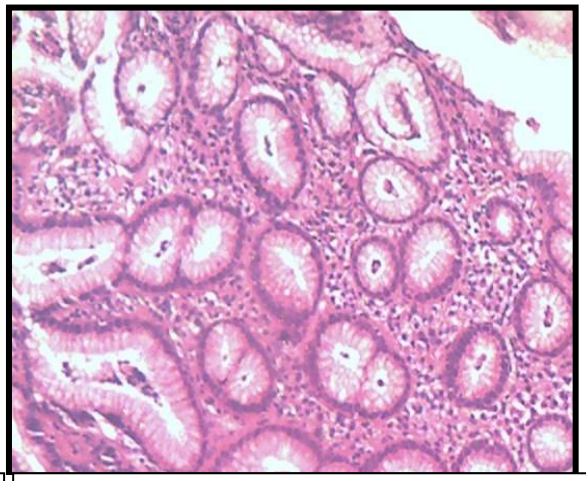


Figure -12: Chronic active gastritis with mild chronic inflammation, moderate polymorphonuclear infiltration and crypt abscess(H&E:100X)

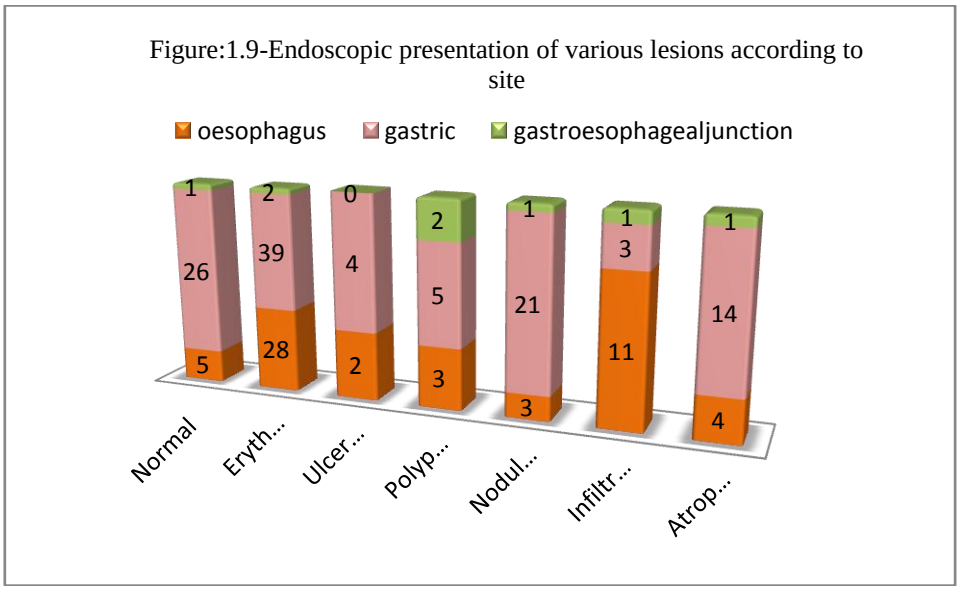
Table:7-Histopathological diagnosis of gastroesophageal junction biopsies

Dysplasia	Malignancy	Polyp	Total
4(50%)	2(25%)	2(25%)	8

Our study included relatively less number of lesions occurring at gastroesophageal junction having dysplasia in 4(50%) of cases. Malignancy consisting of adenocarcinoma and polyp occurred in almost half frequency as that of dysplasia with only 2(25%) cases reported.

Table:8-Endoscopic presentation of various lesions according to the site.

Endoscopic presentation	Oesophagus	Gastric	Gastroesophageal junction	Total
Normal	05(09%)	26(23%)	1(12.5%)	32(18%)
Erythematous mucosa	28(50%)	39(35%)	2(25%)	69(39%)
Ulcerative	02(04%)	04(04%)	0	06(03%)
Polypoidal	03(05%)	05(04%)	2(25%)	10(06%)
Nodular	03(05%)	21(19%)	1(12.5%)	25(14%)
Infiltrative	11(20%)	03(03%)	1(12.5%)	15(09%)
Atrophic	04(07%)	14(12%)	1(12.5%)	19(11%)
Total	56(100%)	112(100%)	8(100%)	176(100%)



Endoscopic presentation of various upper gastrointestinal tract biopsies is shown in Table 8 and Figure 1.9 suggesting that maximum number of lesions 69(39%) presented as an erythematous lesion(Fig-15). Infiltrative and atrophic appearances were seen in 15(9%) and 19(11%) cases respectively .Nodular (Fig-13) growth pattern were seen in 25(14%) cases. Polypoidal growth (Fig-14) and ulcerative pattern was less frequent accounting for 10(6%) and 6(3%) cases respectively. However 32(18%) of total cases had normal endoscopic finding.

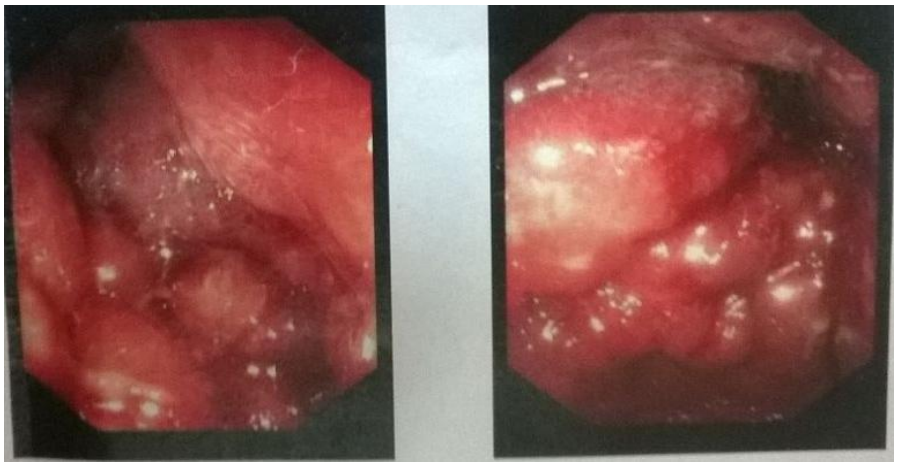


Figure 13: Nodular growth of malignancy in gastric region.

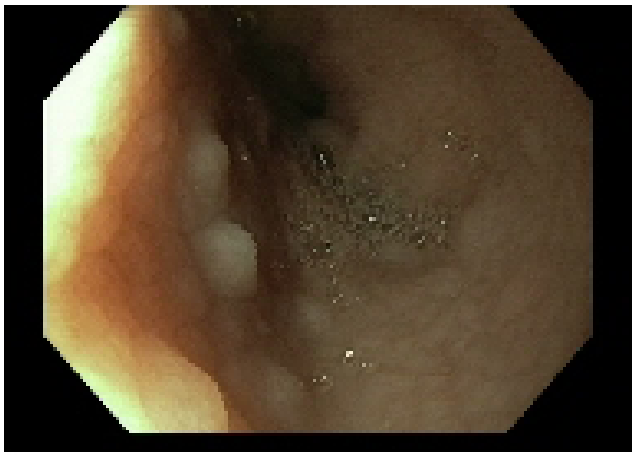


Figure 14: Polypoidal growth of papilloma in oesophagus

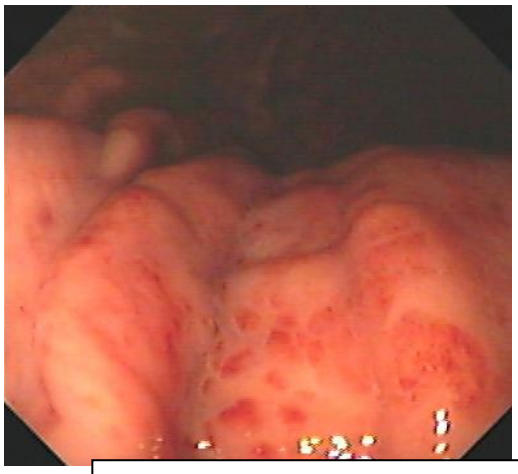


Figure 15:Erythematous mucosa in gastritis

Table14& Figure 1.11: H.pylori positivity in various lesions

Diagnosis	No of cases with H.pylori positivity
Acute gastritis	01 (02%)
Chronic atrophic gastritis	07 (15%)
Chronic non atrophic gastritis	39(83%)
Total no cases with positivity	47(100%)

Above Table-14 shows presence of H.pylori in various upper gastrointestinal tract lesions . They were most commonly seen in chronic non atrophic gastritis in ,39(83%) cases .However H.pylori was seen in chronic atrophic gastritis and acute gastritis in7(15%) and 1(2%) cases respectively. These finding were seen in H& E stain with presence ofH.pylori on surface and within the lumen of the gland(Fig-17). However special stain like Giemsa(Fig-16) and Polyclonal H.pylori antibody, specific for H.pylori(Fig-18) was also applied, having much higher sensitivity and specificity than H&E. H.pyloriwas not observed in oesophageal lesions and in malignancies of gastric and gastroesophageal junction region.

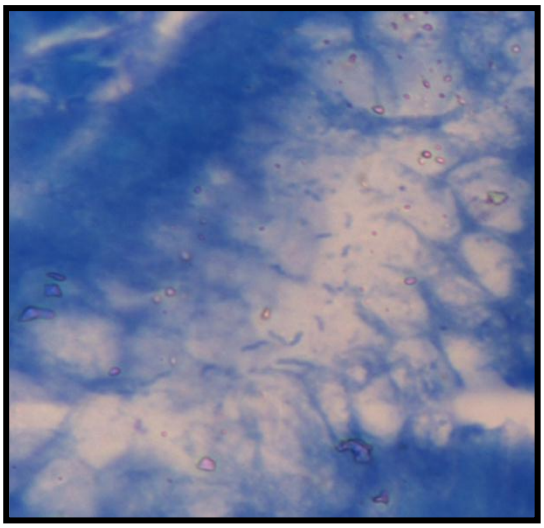


Figure 16 :H.pylori with Moderate positivity.(Giemsa:1000X)

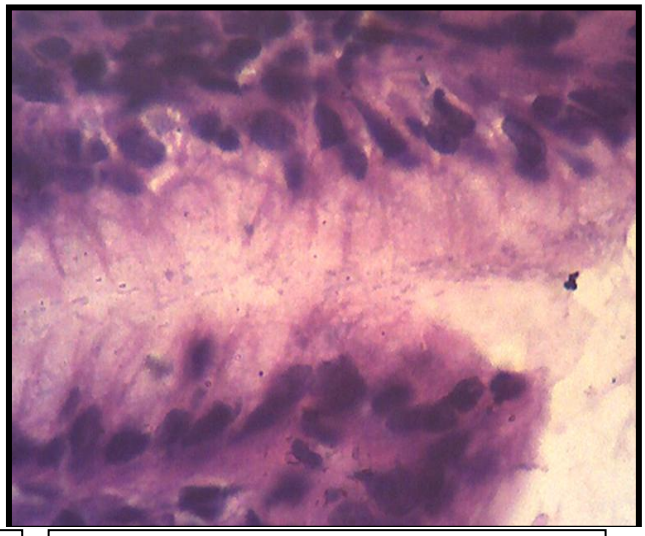


Figure 17 :H.pylori with mild positivity.(H &E:1000X)

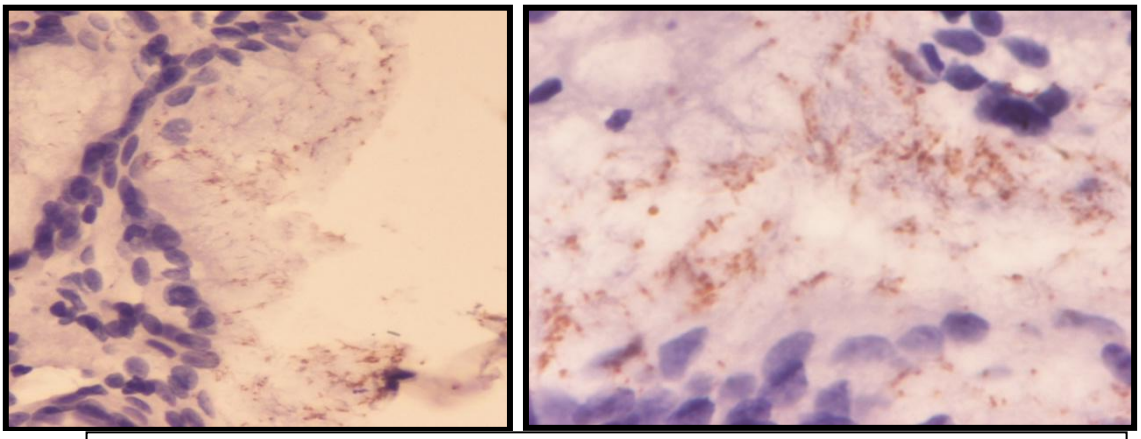


Figure 18:H.pylori positivity by Immunohistochemistry ;Polyclonal H.pylori Antibody:1000X

IV Discussion

Our study included 176 endoscopic biopsies which were studied and evaluated clinically, endoscopically and histopathologically Out of total 176 biopsies,112(64%) biopsies were from gastric region, 56(32%) were from oesophagus and 8 (4%)were from gastroesophageal junction (Table -1)

A comparative study was performed by Suzana et al on 154 gastric biopsies and Smith et al on 168 gastric biopsies respectively[7,8].In contrast few studies included larger number of biopsies like Qureshi et al with 913 endoscopic biopsies, 486(53%) gastric and 428(47%) oesophageal[9]. Similarly Gadour and Ayoola studied 2572 endoscopic biopsies[10].

Our study showed male preponderance with male patients being 109(62%) and 67(38%) female patients. Male to female ratio was 1.63:1. Gastric lesions had higher male to female ratio of 2:1 while slight male preponderance (M:F :: 1.15 : 1) was noted in oesophageal lesions.Comparably Redeen et al conducted similar study on 304 patients, having 161(53%) male and 143(47%) female with a male to female ratio of 1.13:1[11]. Similarly, Islam et al observed a male to female ratio of 1:0.9[12].

Our study had maximum number of patients (46%) in 4^{th} and 5^{th} (23%)decade .Gastric lesions were maximum between 40-50(22%) yrs. Lesions of oesophagus peaked in 5^{th} (29%) decade followed by 4^{th} (23%) decade. Comparably Gadour and Ayoola noted majority of oesophageal lesions in their seventh and eighth decade[10] .

In our study , the mean age affected in lesions of all the site was 47 yrs, which is comparable to the mean age of 49 yrs observed by Russo et al in a study of 110 cases[13]. However Islam et al noted a relatively lower mean age of 38 yrs in his study of 81 patients[12].

In our study,out of total 176 patients maximum number of patients(60%) presented with nausea,vomiting and dyspepsia.Second most common (51%)complain was epigastric pain followed by

Dysphagia(18%). Lesions of gastric region had dyspepsia(79%)and nausea,vomiting(78%) as the primary complaint.While epigastric pain was the third most common complain, prevalent in 70% of the patients with gastric lesion.Above values are explained by the fact that gastritis ,was the most common lesion encountered in the gastric region.Dysphagia presented as the most common symptom in patients with oesophageal lesions(40%) ,while 27% of this patients were associated with weight loss.This may be attributed to thefact that oesophageal malignancies was present in 19(34%) out of 56 cases.Our findings were in concordance with Gadour and Ayoola, who observed dyspepsia and nausea vomiting as the most common symptom in gastric lesions[10].They also documented dysphagia as the most common complaint in 29 (74.1%) out of total 39 cases of oesophageal malignancies , which again simulates our study.However haemetemesis was much more common(20%)than our study (3%) which may be attributed to increased number of cases with bleeding peptic ulcer diseases.Similar results were cited by Qureshi et al showing dysphagia as the primary complaint among 388(42%) patients of oesophageal lesion[9].

Our study had malignancy as the most common oesophageal lesion consisting of 39% of total 56 cases.Out of 22 diagnosed cases of oesophageal carcinomas, squamous cell carcinoma was the found in 19 (86%)cases while adenocarcinoma was seen in 2 (9%) case. Hasan et al showed similar results with squamous cell carcinoma as the predominant (83.5%) oesophageal malignancy[14].Our study was also in concordance with Ali et al documenting 92.5% cases of squamous cell carcinoma and 7.5% cases of adenocarcinoma ,in a study of 69 malignant lesions[15].However Tachibana et al reported 73% cases of adenocarcinoma.This can be attributed to change in epidemiological trend leading to rise of incidence of adenocarcinoma in western world[16].

Second most common oesophageal lesion observed in our study was dysplasia accounting for 16(29%) cases.This was in concordance with Taylor et al who reported 25% prevalence of oesophageal dysplasia in adults in China[17].However Lu et al obtained 19% cases of dysplasia in 2,013 patients[18].

Oesophagitis constituted 12(21%) cases in our study which was quite similar to the study of Qureshi et al who reported it in 20% cases[9] .

We studied 112 gastric biopsies,out of which 96 (86%)were diagnosed as chronic gastritis. Chronic atrophic gastritis was seen in 21% of cases ,which was in concordance with Suzana et al who documented 15% prevalence of chronic atrophic gastritis in their study[7].Our study showed 65% non atrophic gastritis .However Qureshi et al observed 19% cases in their study[9].We observed relatively less number of cases of malignancy, 6% which was similar to that reported by Qureshi et al[9].

Our study had maximum number of cases 69(39%) presenting as an erythematous lesion which lies in the fact that gastritis was the most common lesion(55%) in our study .This was in concordance with Khan et al who obtained maximum number of erythematous lesion with 51 cases[19] .Normal mucosa was second most common(18%) finding in our study.Similar findings were observed by Calabrese et al and Khan et al amounting to 25% and 32% cases respectively[20,19].Atrophic mucosa was very less encountered in our study with 11% of total cases simulating the study of Calabrese et al ,who reported 18% cases[20].

We observed highest percentage of H.pylori in nonatrophicgastritis(83%) which explains their site of origin from antrum, hence associated with increased prevalence of H.pylori**[21]**.This gastritis is synonymus with chronic superficial gastritis or hypersecretary gastritis/chronic active gastritis known to be associated with H.pylori in previous studies**[22].**Following studies obtained almost similar association of h.pylori with chronic active gastritis.

Diagnosis	Hashemi et at[23]	Ahmad et al[24]	Hussein et al[25]
Chronic active gastritis	73%	85%	80%

Out of total H.pylori positive cases ,7(15%) belonged to atrophic gastritis. However almost 30% (7 of 23) cases of atrophic gastritis were associated with H.pylori which suggests their removal from antrum. Atrophic gastritis occurring in corpus is usually autoimmune in nature.Multifocalatrophic gastritis and Pan atrophic gastritis are usually associated with intestinal metaplasia,which do not harbour H.pylori**[21].**

V Conclusion

We studied multiple endoscopic biopsies from 176 patients ,112 being from gastric region encompassing wide variety of non-neoplastic and neoplastic lesions with wide range of age and site distribution. Endoscopic appearance plays an important role in correlation with histopathological diagnosis, however limitations in diagnostic interpretation was encountered due to tiny and superficial biopsy material.Endoscopic intervention at an early stage , like transformation of dysplasia into malignancy can initiate early treatment and results in better prognosis .Similarly an early neutrophilic infiltrate and presence of h.pylori in cases of gastritis induced by the latter, and its progression to atrophy and thereafter to intestinal metaplasia can be halted by mere treatment by antibiotics, if biopsied at an early stage.

References

[1]. Rashmi K, Horakerappa MS, Ali K. article A study on histopathological spectrum of upper gastrointestinal tract endoscopic biopsies.International Journal of Medical Research & Health Sciences.2013;2(3):418-24
[2]. Warren J.R. and MarshallB.J.Unidentified curved bacilli on gastric epithelium in active chronicgastritis.Lancet.1983; 1:1272-73
[3]. Goodwin CS, Armstrong JA, ChilversT.Helicobacter pylori and Helicobactermustelae .International journal of systematic Bacteriology.1989;39:397-405.
[4]. ZantenSJ:Do socio-economic status,marital status and occupation influence the prevalence of H.pylori infection? Aliment.Pharmacol.Ther.1995;9(2):41-4.
[5]. Anand AC, Anand M, Reddy et al. H.pylori cause gastric carcinoma.Indian J Gastroenterol.1997;116:22-35.
[6]. Parsonnet J and Isaacson PG.Bacterial infection and MALT lymphoma. Eng J Med.2004;350:213-15.
[7]. Suzana MK, Skender T, EmineDD.Helicobacter pylori gastritis -updated sydney classification applied in our material. Biol. med. sci.2009;1:45–60
[8]. Smith SI , Fowora MA , OtegbayoJA,etal.Comparison of PCR with other diagnostic techniques for the detection of H. pylori infection in patients presenting with gastroduodenal symptons in Nigeria.Int J MolEpidemiol Genet.2011; 2: 178–184.
[9]. Qureshi NA, HallisseyM T, FieldingJW,etal.Outcome of index upper gastrointestinal endoscopy in patients presenting with dysphagia in a tertiary care hospital-A10 years review. BMC Gastroenterology. 2007; 7:43 .
[10]. Gadour MO and AyoolaEA.The frequency of upper G.I. malignancy in Gizan.Saudi J Gastroenterol.2004;10:16-21.
[11]. Redeen S, Petersson F , TornkrantzE,etal.Reliability of Diagnostic Tests for Helicobacter pylori Infection. Pathology Gastroenterology Research and Practice . 2011;6.
[12]. Islam MDU, Rahman SHZ, Shamsuzzaman SM. A Comparative Study Among Different Invasive Methods For The Diagnosis Of Helicobacter Pylori .Faridpur Med Coll. J. 2010;5:21-24.
[13]. Russo F, Berloco P, Cuomo R,et al. Helicobacter pylori strains and histologically-related lesions affect the outcome of triple eradication therapy. Aliment PharmacolTher. 2003; 17: 421–8.
[14]. Hasan Z, Jayaram M, Devi U,etal.Patterns of oesophageal neoplasms in a referral diagnostic centre of banglore.Journal of Evolution of Medical and Dental Sciences.2013;2:9250-60.
[15]. Ali A, Naseem M, Khan TM et al. Oesophageal cancer in northern areas of Pakistan. J Ayub Med Coll Abbottabad. 2009;21:148-50.
[16]. Tachibana M, Hirahara N, KinugasaS.Clinicopathological features of superficial oesophageal cancer:Results of consecutive 100 patients.AnnSurg Oncol.2007;15:104-6.
[17]. Taylor PR,Abnet CC,DawseySM,et al. Squamous dysplasia--the precursor lesion for esophageal squamous cell carcinoma.CancerEpidemiol Biomarkers Prev. 2013 ;22:540-52.
[18]. Lu XJ, Chen ZF, Guo CL. Endoscopic survey of esophageal cancer in a high-risk area of China.World J Gastroenterol. 2004;10:2931-5.
[19]. Khan MQ, Alhomsi Z, Ahmad M. Endoscopic features of Helicobacter pylori induced gastritis.Saudi J Gastroenterol.1999 ;5:9-14.
[20]. Calabrese C, Febo G, Brandi G,et al. Correlation between endoscopic features of gastric antrum, histology and Helicobacter pylori infection in adults. .Ital J GastroenterolHepatol. 1999;31:359-65.
[21]. Dixon MF, Genta RM, Yardley JH,etal.Classification and grading of gastritis.The American Journal of Surgical Pathology.1996;20:1161-81.
[22]. Correa P. Chronic gastritis: a clinico-pathological classification. Am J Gastroenterol.1988;83:504- 9.
[23]. Hashemi MR, Rahnavardi M, Bikdeli B.H pylori infection among 1000 southern Iranian dyspeptic patients.World J Gastroenterol .2006; 12: 5479-82.
[24]. Ahmad M and AkwaaA.Prevalence of Helicobacter pylori Infection in a Group of Morbidly Obese Saudi Patients undergoing Bariatric Surgery: A Preliminary Report.Saudi journal of gastroenterology. 2010;16:264-67.
[25]. Hussein NR, Napaki SM, Atherton JC,etal.A Study of Helicobacter pylori associated gastritis patterns in Iraq and their association with strain virulence.Saudi J Gastroenterol. 2009;15:125-7.

A.R.Piyush ." Histopathological And Endoscopic Analysis Of Biopsies From Various Lesions Of Upper Gastrointestinal Tract In A Tertiary Health Care Centre."IOSR Journal of Dental and Medical Sciences (IOSR-JDMS), vol. 17, no. 5, 2018, pp 77-88.