HISTOPATHOLOGICAL STUDY OF GASTRIC MUCOSAL BIOPSIES IN CHRONIC GASTRITIS PATIENTS WITH SPECIAL CORRELATION TO HELICOBACTER PYLORI INFECTION AT RIMS HOSPITAL

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ABSTRACT

BACKGROUND
Chronic gastritis is a common condition in general population. Of the many aetiological factors, Helicobacter pylori is one of the primary cause of chronic gastritis.

AIMS
- To study the histopathological features of chronic gastritis associated with H. pylori infection.
- To find out the relationship between severity of inflammatory reaction in gastritis and intensity of H. pylori.

MATERIALS
This cross sectional study was carried out in Regional Institute of Medical Sciences (RIMS) Hospital from October 2013 to September 2015. Patients with symptoms suggestive of chronic gastritis attending RIMS OPD were subjected to upper gastrointestinal endoscopy. One of the biopsy specimens was used for rapid urease test using RUT dry test kit and others were processed for histopathological examination as per standard protocol. All the slides were stained with Haematoxylin and Eosin (H&E) stain and Giemsa stain. Histological variable grading was done using the "Updated Sydney System 1994". All the data thus collected were analysed.

RESULTS
A total of 60 patients (39 males and 21 females) were included in the study with age ranging from 19 to 82 years. Among the 60 patients, maximum endoscopic findings were ulcer (33.3%) followed by erythematous findings (26.7%). The results of Rapid Urease Test (RUT) were positive in 30% (18/60). The histopathology reports for H. pylori detection were positive in 35% (21/60). The majority (81.7%) of the cases were inflammatory on histopathology followed by neoplasia (8.3%), dysplasia (5%) and normal finding (5%). Neutrophilic activity was present in all cases of chronic gastritis, in which 15, 5 and 11 numbers of cases showed mild, moderate and severe grading respectively. Mononuclear cell infiltration also was present in all cases of chronic gastritis and 8, 16, 7 numbers of cases were found to have mild, moderate and severe grading respectively. Only five mild atrophy, four mild Intestinal metaplasia (IM) and one moderate Intestinal metaplasia (IM) were found in gastritis cases. Correlation coefficient of neutrophilic activity with H. pylori density was 0.725 (p=0.000), which was statistically significant.

CONCLUSION
In our study, histopathological features of H. pylori associated chronic gastritis have shown H. pylori density, neutrophilic activity, mononuclear cell infiltration, glandular atrophy and intestinal metaplasia. We also have observed the association between neutrophilic activity and H. pylori density.

KEYWORDS
Chronic Gastritis, H. Pylori, Neutrophilic Activity.

HOW TO CITE THIS ARTICLE: Choudhury S, Laishram RS, Punyabati P, et al. Histopathological study of gastric mucosal biopsies in chronic gastritis patients with special correlation to Helicobacter pylori infection at RIMS Hospital. J. Evid. Based Med. Healthc. 2016; 3(55), 2829-2835. DOI: 10.18410/jebmh/2016/618

INTRODUCTION: Chronic gastritis is the chronic inflammation of the gastric mucosa. It is one of the most common gastrointestinal problems among the general population. Although a variety of aetiological factors are associated with chronic gastritis, Helicobacter pylori are the primary cause. It is a diagnosis based on histology and is either nonatrophic or atrophic. The nonatrophic form is primarily associated with H. pylori; whereas the atrophic form is associated with other factors like environmental factors, autoimmune disease beside H. pylori.[1,2] Chronic gastritis may involve the antral mucosa alone (type B) or the oxyntic mucosa alone (Type A) or both (type AB/pangastritis).

Financial or Other, Competing Interest: None.
Submission 11-06-2016, Peer Review 21-06-2016,
Acceptance 03-07-2016, Published 11-07-2016.
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DOI: 10.18410/jebmh/2016/618

H. pylori is one of the most common infections in the world and it is accepted as a cause of antral gland gastritis, peptic ulcer, and the risk factor for the gastric cancers like gastric MALTomas (mucosa associated lymphoid tissue lymphomas) and adenocarcinoma. It is found in all parts of the world and believed that half of the world's population is infected with H. pylori, with highest prevalence in the developing countries. In India, 80% of the population is infected with this bacterium and most of them have been infected since 10 years of age. Possible modes of transmission generally described are through direct person to person contact between family members and also through the contaminated food and water.[2] Helicobacter pylori were formerly known as Campylobacter pylori and Campylobacter pylorides. Usually, the bacteria are easily seen on well-differentiated H&E (Haematoxylin and Eosin) sections and it is not necessary to use special staining for routine diagnosis. The other special staining for H. pylori are Giemsa, Genta, Warthin-Starry, Steiner silver stain, the Alcian yellow-toluidine blue method or immunohistochemistry. Polymerase chain reaction (PCR) techniques for H. pylori detection are also available. H. pylori is a strict microaerophilic, short curved, spiral or S shaped gram negative bacterium. But it may be present in coccoid form following treatment with proton pump inhibitors, which can be reliably identified by specific immunohistochemical (IHC) staining.[3,4]

As Helicobacter pylori is associated with a variety of gastric pathology i.e. gastritis, peptic ulcer and gastric cancers, this study was carried out to observe the histological features of chronic gastritis, with special emphasis on correlation between H. pylori density and neutrophilic activity of gastric mucosa by applying Updated Sydney classification.[5]

METHODS: This cross sectional study was conducted in the Department of Pathology, Regional Institute of Medical Sciences (RIMS), Imphal, Manipur, in collaboration with Surgical Gastroenterology & Minimal Access Surgery Unit, Department of Surgery (RIMS). The duration of the study was two years (from October 2013 to September 2015). Patients with symptoms suggestive of chronic gastritis irrespective of age, sex, religion and socioeconomic status attending the Surgery OPD, Medicine OPD, in patients in Medicine and Surgery wards were included in the study.

Inclusion Criteria and Exclusion Criteria: Inclusion criteria: All cases both from the outpatient department and in-patient department presenting with the symptoms suggestive of chronic gastritis i.e. pain or discomfort centred in upper abdomen, that might be associated with dyspepsia, bloating, early satiety, postprandial fullness, nausea, anorexia, heartburn, regurgitation, belching, weakness were included in the study.

Exclusion Criteria: Patients who refused to undergo upper gastrointestinal endoscopy, those who had alarming symptoms i.e. gastrointestinal bleeding, patients previously had undergone surgery for gastroenterological or hepatobiliary diseases and patients with history of gastrointestinal neoplasm were excluded from the study.

Age, sex, clinical features, site of lesion, endoscopic findings, rapid urease test positivity, histopathological features of chronic gastritis (H. pylori density, neutrophilic activity, mononuclear cell infiltration, atrophy, intestinal metaplasia, lymphoid aggregates) were taken into account for the study.

Gastrointestinal endoscope (FUJINON EG-265WR), flexible biopsy forceps, RUT dry test kit (Mfd. & Mktd. by Gastro Cure Systems) were used for endoscopy and rapid urease test respectively. A biopsy specimen for RUT Dry has been taken as soon as the endoscopist has examined the stomach. After peeling back the label of the RUT Dry kit, the biopsy specimen was introduced in the exposed yellow media and added one drop of distilled water. Then, the kit was labelled as before. Change of dot colour from yellow to pink or red at 5 to 10 minutes was considered (interval time 24 hours) H. pylori positive result.

In pathology laboratory, other specimens were processed for histopathological studies as per the standard protocol. Paraffin sections of 3-5 µm thickness were made by rotary microtome and stained with Haematoxylin and Eosin (H&E) to study the histology of gastric mucosa. The other specimen was stained with Giemsa staining for identification of H. pylori.

The slides were examined using Olympus CH20i microscope. H. pylori detection by histology was considered gold standard and RUT test results were compared with it. All the cases of chronic gastritis were reported following the guidelines put forward by the Updated Sydney System (1994) of reporting.[5]

Ethical Issues: Informed written consents were obtained from all the participating individuals, and approval of Institutional Ethics Committee, RIMS was taken.

STATISTICAL ANALYSIS: All the collected data were analysed by IBM SPSS statistics version 21. Descriptive statistics and significance testing (Spearman rank correlation test) were carried out.

RESULTS AND OBSERVATION: A total of 60 patients (39 males and 21 females) were included in the study with age ranging from 19 to 82 years with a mean age of 54.17 years (SD 15.76) [Table 1]. There were 39 males and 21 females with a M: F=1.85:1. The commonest clinical presentation was dyspepsia (75%) followed by loss of appetite (33.3%) as shown in table-2. Antrum (90%) was the most common involved site followed by fundus (6.7%) and incisura angularis (3.3%). Among the 60 patients, maximum endoscopic findings were ulcer (33.3%) followed by erythematous findings (26.7%) [Table-3]. The endoscopic findings of the 5 malignant cases were 3 (60%) ulcerative growths [Fig. 1], 1 (20%) fungating growth and 1 (20%) polypoidal growth. The results of Rapid Urease Test (RUT) were positive in 30% (18/60) and negative in 70% (42/60) cases. The histopathology reports for H. pylori detection

were positive in 35% (21/60) and negative in 65% (39/60) cases. Results of Rapid Urease Test and histopathology were compared. Taking histopathological demonstration of H. pylori as Gold standard, the sensitivity, specificity, PPV, NPV of RUT compared to histopathology were 85.7%, 100%, 100% and 92.8% respectively.

The majority (81.7%) of the cases were inflammatory on histopathology followed by neoplasia (8.3%), dysplasia (5%) and normal finding (5%) [Table-4]. Among the inflammatory lesions, chronic gastritis (63.2%) was the most common finding on histopathology. Out of 31 chronic gastritis cases, majority (55.1%) were chronic antral gastritis followed by others. Gastric ulcers comprised 36.8% cases of inflammatory lesions [Fig. 2]. Histological typing of gastric adenocarcinoma was done based on WHO guidelines. Out of 5 gastric adenocarcinomas, 3 (60%) cases were tubular type and 2 (40%) were poorly cohesive (including 1 signet ring cell variant) type.

H. pylori positivity were found in 15 out of 27 cases of chronic antral gastritis (55.6%) and 1 out of 3 cases of chronic fundal gastritis (33.3%). 3 out of 18 cases of gastric ulcer (16.7%) also showed positivity for H. pylori [Fig. 3]. Among the chronic gastritis cases, 51.6% (16/31) were H. pylori positive [Table-5]. Among the gastric dysplasia cases, 100% (1/1) high grade dysplasia and 50% (1/2) low grade dysplasia were H. pylori positive. None of the 5 gastric adenocarcinoma cases showed H. pylori positivity [Table-6].

Chronic gastritis cases were evaluated for histopathological features and the variables were graded according to Updated Sydney System. H. pylori were found in 16 out of 31 chronic gastritis cases; among them 6 and 10 numbers of cases showed mild and moderate density respectively. Neutrophilic activity was present in all cases of chronic gastritis, in which 15, 5 and 11 numbers of cases showed mild, moderate and severe grading respectively. Mononuclear cell infiltration also was present in all cases of chronic gastritis and 8, 16, 7 numbers of cases were found to have mild, moderate and severe grading respectively. Only five mild atrophy, four mild IM and one moderate IM were found in gastritis cases [Table-7].

The percentages of H. pylori positive cases were gradually increasing with higher grade of neutrophilic activity (20% in mild, 60% in moderate and 90.9% in severe neutrophilic activity). Therefore, H. pylori density was correlated with neutrophilic activity (Table-8). Statistically, significance was set at p value <0.05 level. Correlation coefficient of neutrophilic activity with H. pylori density was 0.725 (p=0.000), which was statistically significant. The percentages of H. pylori positive cases were gradually decreasing with higher grade of mononuclear cell infiltration (62.5% in mild, 50% in moderate and 42.8% in severe mononuclear cell infiltration). Therefore, H. pylori density was not correlated with mononuclear cell infiltration [Table-9]. Correlation coefficient of mononuclear cell infiltration with H. pylori density was -0.308 (p=0.092), which was statistically insignificant. The lymphoid aggregates were present in 9.7% of chronic gastritis and 5.6% of gastric ulcers cases. In the histopathological findings, 40% (2/5) of glandular atrophy and 40% (2/5) of intestinal metaplasia were H. pylori positive. The endoscopic findings were compared with histopathological features. 1 out of 4 normal looking mucosa (25%) on endoscopy was histologically gastritis. 1 out of 15 (6.6%) erosions and 2 out of 20 (10%) ulcers on endoscopy were histologically dysplastic. All 16 erythematous findings and 14 erosions on endoscopy were diagnosed gastritis. All endoscopically malignant looking lesions (growth) were diagnosed histologically as gastric adenocarcinoma [Table-10].

Age group (In years)	Number of patients	Percentage
16-25	04	6.7%
26-35	04	6.7%
36-45	08	13.3%
46-55	13	21.6%
56-65	18	30.0%
66-75	10	16.7%
76-85	03	5.0%
Total	**60**	**100%**
Average mean (In years)	54.17+15.76	

Table 1: Distribution of Patients according to Age

Sl. No.	Clinical Features	Number of patients (60)	Percentage
1	Dyspepsia	45	75%
2	Loss of appetite	20	33.3%
3	Weakness	18	30%
4	Pallor	10	16.7%
5	Weight loss	04	6.7%
6	Melaena	02	3.3%
7	Haematemesis	04	6.7%
8	Lymphadenopathy	01	1.7%

Table 2: Presenting Clinical Features

Sl. No.	Endoscopic findings	Number of patients	Percentage
1	Erythematous	16	26.7%
2	Erosion	15	25.0%
3	Ulcer	20	33.3%
4	Growth	05	8.4%
5	Normal	04	6.6%
Total		**60**	**100%**

Table 3: Endoscopic Findings of Gastric Mucosal Lesions

Sl. No.	Nature of lesion	Number of patients	Percentage
1.	Normal (no pathology)	03	5.0%
2.	Inflammatory	49	81.7%
3.	Dysplasia	03	5.0%
4.	Neoplasia (malignant)	05	8.3%
Total		**60**	**100%**

Table 4: Distribution of cases based on Histopathological Diagnosis

Lesions		No. of cases	H. pylori positive	H. pylori negative	Percentage of H. pylori positive
Chronic gastritis	Antral	27	15	12	55.6%
	Fundal	03	01	02	33.3%
	Incisura angularis	01	00	01	0%
Gastric ulcer		18	03	15	16.7%

Table 5: Inflammatory Lesions showing Helicobacter pylori Positivity

Lesions		Total No. of cases	H. pylori positive cases	H. pylori negative cases	Percentage of H. pylori cases
Dysplasia	High grade	1	1	0	100%
	Low grade	2	1	1	50%
Gastric adenocarcinoma	Tubular	3	0	3	0%
	Poorly cohesive	2	0	2	0%

Table 6: Premalignant and Malignant Lesions showing H. pylori Positivity

Variables	Absent	Mild	Moderate	Severe	Total
H. pylori	15	6	10	0	31
Neutrophils	0	15	5	11	31
Mononuclear cells	0	8	16	7	31
Atrophy	26	5	0	0	31
Intestinal metaplasia (IM)	26	4	1	0	31

Table 7: Grading of Chronic Gastritis according to Updated Sydney System

Neutrophilic activity→		Mild (15)	Moderate (5)	Severe (11)
H. pylori ↓	Present	3	3	10
	Absent	12	2	1
	Percentage of H. pylori positive	20%	60%	90.9%

Table 8: Correlation between Neutrophilic activity and H. pylori density

Mononuclear cell infiltration→		Mild (8)	Moderate (16)	Severe (7)
H. pylori ↓	Present	5	8	3
	Absent	3	8	4
	Percentage of H. pylori positive	62.5%	50%	42.8%

Table 9: Correlation between Mononuclear cell Infiltration and H. pylori Density

Histopathological diagnosis → / Endoscopic findings↓	Normal	Chronic gastritis	Gastric ulcer	Dysplasia	Neoplastic (Malignant)
Erythematous (16)		16			
Erosion (15)		14		1	
Ulcer (20)			18	2	
Growth (5)					5
Normal (4)	3	1			

Table 10: Endoscopic and histopathological Diagnosis of all Lesions

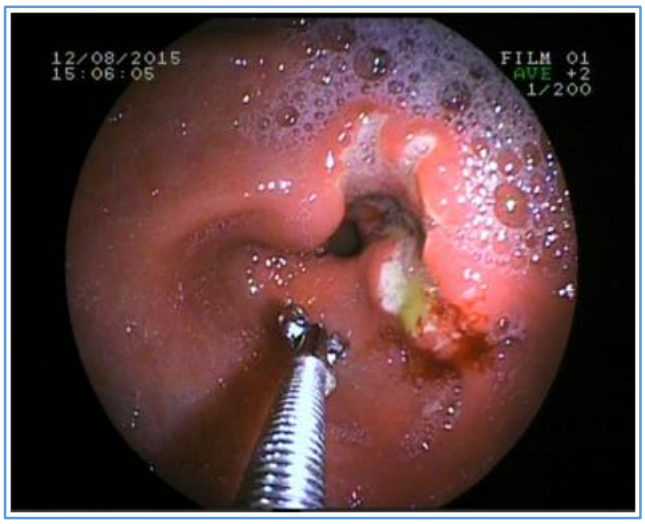

Fig. 1: Picture showing the Endoscopic view of Gastric Ulcer

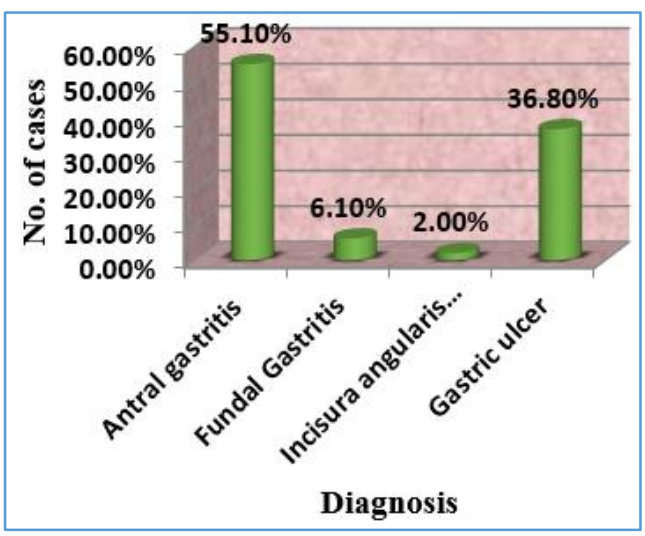

Fig. 2: Bar chart showing the Inflammatory Lesions on Histopathology

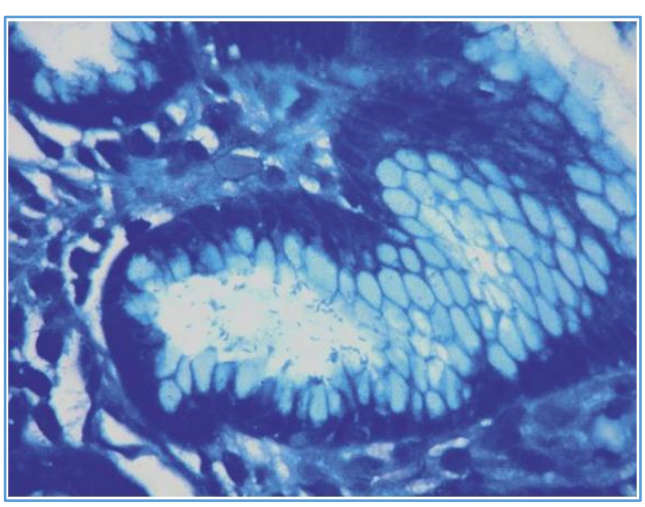

Fig. 3: Microscopic picture of Endoscopic Gastric Biopsy showing H. Pylori. (Giemsa stain, 100X)

DISCUSSION: Chronic gastritis is a common condition in general population. In our present study, the histological features of chronic gastritis and correlation of H. pylori density with inflammatory activity were evaluated and thereafter, results were compared with the findings of various articles by other authors.

The age distribution in the present study showed that maximum numbers of patients were in the age group of 56-65 years and average mean was 54 years, which was comparable to others findings. Muller LB et al,[6] Poudel A et al[7] in their study also showed that the average mean was 52 years. On the contrary, Tanko MN et al,[8] Fareed R et al[9] found the majority of patients in 3rd to 4th decades of life. The maximum number of patients in elderly age group of our study can be explained by reduced mucin synthesis, food habit, life style and geographical variation.

In the studies by Fareed R et al,[9] Mustapha S et al,[10] and Sultana A, et al[11] who obtained male predominance was also seen in our study. The increased incidence of chronic gastritis in males may be due to higher gastric acid production and stress factor. Works carried out by Mustapha S et al,[10] Tanko MN et al[8] and Pruthi S et al[12] showed that the most common presenting symptom of chronic gastritis was dyspepsia which was comparable to the present study.

Pruthi S et al[12] showed that antral ulcer/erosions (46.7%) was the most common endoscopic finding followed by lesions at other sites (Fundus, incisura, lesser and greater curvature). This was consistent with our endoscopic findings. Karki BB et al[13] in their study, found that the sensitivity, specificity, PPV, NPV of RUT for H. pylori detection were 84%, 94.4%, 94.9% and 82.9% respectively. Hence, they concluded that RUT had good sensitivity and specificity for H. pylori detection. Foroutan M et al[14] also obtained the sensitivity and specificity of RUT as 98.57% and 99.29% respectively. This was similar to the present study. Therefore, RUT remains a reliable and quick method for H. pylori detection. On the contrary, Yakoob J et al[15] in their study found that sensitivity, specificity, PPV, NPV of RUT, with or without PPI were 43.3%, 86.4%, 81.3%, 52.8% v/s 71.9%, 80%, 82.1%, 69%. They concluded that exclusive use of RUT for H. pylori detection could not be recommended in patients with prior PPI (Proton pump inhibitors) use. Chen XY et al [16] and Akere A et al [17] in their study observed chronic gastritis as the most common finding in dyspeptic patients similar to our findings.

Yakoob MY and Hussainy AS [2] found 62.5% of H. pylori positive cases among chronic gastritis patients. Park J et al,[18] Dursun M et al[19] also obtained the similar result. Almost similar finding was observed in the present study. On contrary, Kamada T et al[20] found 95.8%, 100% H. pylori positivity in chronic gastritis and gastric ulcers respectively. Although, H. pylori is the most common cause of chronic gastritis and alkaline pH of antrum provides a favourable environment for H. pylori growth; a low H. pylori detection rate in our study may be due to small sample size and drug history (PPI, antibiotics, etc.) which could not be collected properly.

Yakoob MY and Hussainy AS,[2] Garg B et al,[21] Tanko MN et al[8] and Fareed R et al,[9] who also observed statistically significant association between inflammatory activity and H. pylori density (p=0.00) which corroborated with the present

study. Xu XQ et al[22] suggested that neutrophils were the predictive marker of the H. pylori infection, as they showed strong association. This association can be explained by enhanced mucosal level of interleukin-8 mediated by H. pylori, which is a neutrophil chemotactic factor.

Garg B et al[21] and Tanko MN et al[8] in their articles, mentioned about the statistically significant relationship between H. pylori density and chronic inflammation. Pruthi S et al [12] in their study detected H. Pylori in 34.2% cases of mild, 85.7% cases of moderate and 100% of marked neutrophilic activity. They also found H. pylori positivity in 37.5% mild, 33.3% moderate and 80.6% of marked mononuclear cell infiltration. Therefore, they concluded that density of H. pylori increased with the severity of neutrophilic and mononuclear cell activity. In our study, H. pylori positivity found in 20.0% mild, 60.0% moderate and 90.9% severe neutrophilic activity, whereas in mononuclear cell infiltration 62.5% mild, 50% moderate and 42.8% severe cases were H. pylori positive. Hence, H. pylori was correlated well with neutrophilic activity but not with mononuclear cell infiltration. The mononuclear cell infiltration indicates chronicity of infection and this may be the reason behind the absence of any correlation.

Tanko MN et al[8] found glandular atrophy in 38% and intestinal metaplasia in 28% cases of chronic gastritis. In our study, only 16.1% chronic gastritis cases showed both atrophy and intestinal metaplasia. The lesser percentage of atrophy and intestinal metaplasia may be due to sampling error, as we took samples only from the gastric lesions, which was predominantly at antrum. Dinis-Ribeiro M et al[23] mentioned that atrophic gastritis and intestinal metaplasia were often unevenly distributed throughout the stomach.

Correa P and Piazuela MB[24] in their article stated that H. pylori tends to disappear when advanced atrophy and intestinal metaplasia extend, creating an unfavourable environment for H. pylori colonisation. Yoo JY et al[25] also found low detection rate of H. pylori in the presence of mucosal atrophy and intestinal metaplasia. These findings were comparable with our study. Chen XY et al[16] in their study found highest prevalence (89.9%) and density of lymphoid aggregates in gastric ulcers, whereas lowest prevalence (74.6%) was found in chronic gastritis, lowest density was in duodenal ulcers. On contrary, we found lymphoid aggregates in 9.7% cases of chronic gastritis, 5.6% cases of gastric ulcers. Because of our small sample size, we could not show significant difference in the prevalence and density of lymphoid aggregates in various gastroduodenal diseases.

In our study, intestinal metaplasia and atrophy were absent in low grade dysplasia cases, but were present in high grade dysplasia (100%). H. pylori was also present in high grade dysplasia but was absent in low grade dysplasia cases. Martinez-Madrigal F et al[26] in their study also observed that intestinal metaplasia was present in 54% cases of mild dysplasia and in 100% of severe dysplasia. Hence, the study was comparable with our findings.

Correa P and Piazuelo MB[24] stated that H. pylori was associated with gastric adenocarcinoma and was estimated to be responsible for more than 60% gastric cancer cases. Wang C et al [27] in their study found that prevalence of H. pylori was significantly higher with early gastric cancer than in non-neoplasm controls or advanced gastric cancer. They explained that cases of advanced gastric cancer might have lost signs of previous H. pylori infections. In our study, none of the gastric adenocarcinomas showed H. pylori positivity. This may be due to small sample size, sampling error or advanced stage of gastric carcinoma which have lost their previous sings of infections.

In the present study, among the endoscopically normal appearing mucosa, 25% was histologically chronic gastritis. 6.6% of erosion and 10% of ulcer cases were histologically diagnosed as dysplasia. These findings were comparable with others studies. Garg B et al[21] obtained inflammatory activity in 22% of normal looking mucosa on endoscopy. Pruthi S et al[12] found gastritis in all 5 cases of normal appearing mucosa on endoscopy. Sultana A et al[11] also observed that among the endoscopically diagnosed gastric adenocarcinoma, 85.5% were gastric adenocarcinoma and 14.5% were chronic gastritis histologically. 90% of the normal looking mucosa on endoscopy was histologically gastritis and 50% of gastric ulcer was gastric carcinoma. Endoscopy poorly correlated with histological findings and therefore, it should be followed by biopsy for confirmatory result.

CONCLUSION: A study on the relationship of chronic gastritis with H. pylori infection was undertaken in 60 numbers of patients. The degree of severity of inflammatory reaction in gastritis in relation with density and colonisation of H. pylori in the stomach was the second component of the study.

In our study, histopathological features of H. pylori associated chronic gastritis have shown H. pylori density, neutrophilic activity, mononuclear cell infiltration, glandular atrophy and intestinal metaplasia. The findings were graded according to Updated Sydney System. In some cases, features of lymphoid aggregates were detected. In some cases of gastric ulcer and dysplasia, H. pylori bacteria with its inflammatory activity were detected in the background, thereby indicating that the bacteria were responsible for the lesions. We also have observed the association between neutrophilic activity and H. pylori density which was statistically significant in our study.

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