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Original Research Article

Reporting of *Helicobacter pylori* associated gastroduodenal diseases: A clinicopathological approach

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ABSTRACT

Background: *Helicobacter pylori* is one of the most important causes of the varied spectrum of gastroduodenal diseases. It is important to have a collaborative approach between clinicians and pathologists for early initiation of treatment in patients and control the progression of the disease.
Aims: To evaluate the clinico-histopathological spectrum of gastroduodenal diseases with respect to *Helicobacter pylori* infection and provide recommendations for reporting and management of the same.
Materials and Methods: All patients of chronic dyspepsia not responding to conventional treatment were subjected to endoscopy, and mucosal biopsy samples were collected. A rapid urease test (RUT) and histopathology was performed on these samples. A histopathological evaluation was done with special reference to the Updated Sydney System for reporting of chronic gastritis
Results: The specificity, sensitivity, positive predictive value, negative predictive value, and diagnostic accuracy of RUT were 97.22%, 94.04%, 98.75%, 87.5%, and 95%, respectively. The specificity, sensitivity, PPV, NPV and diagnostic accuracy of endoscopy were calculated as 98.68%, 88.5%, 95.83%, 96.15% and 96.07% respectively.
Conclusion: A clinicopathological approach for diagnosis of *Helicobacter pylori* associated gastroduodenal diseases which includes clinical assessment, endoscopic evaluation, RUT and histopathological analysis is of prime importance for effective management of such patients.

1. Introduction

Gastroduodenal diseases are frequent causes of clinical disease. The spectrum of gastroduodenal diseases is wide ranging from inflammatory lesions like gastritis and peptic ulcer disease to frankly malignant ones like gastric carcinoma and lymphoma. With the advent of fibre-optic endoscopy, mucosal biopsy samples can now be obtained from areas which were previously difficult to be sampled. Fibre-optic endoscopy has added to the abundance of tissue available to the pathologists for diagnosis and study of the pathogenesis of gastroduodenal disease.

Of the diverse etiological associations of gastroduodenal disease, the most important is a bacterium named *Helicobacter pylori.*[1] Chronic infection of the gastric mucosa by this bacterium is the most common infection worldwide.[2] Discovered way back in 1983 by Warren and Marshall, this curvilinear bacillus has been seen to play a

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critical role in major gastric and duodenal diseases like:

1. Chronic gastritis
2. Peptic ulcer disease
3. Gastric carcinoma
4. Gastric lymphoma

The study by Wyatt et al shows that *Helicobacter pylori* is present in 90% of the patients with chronic gastritis affecting the antrum, 95% with duodenal ulcers and 70% with gastric ulcers.[3] In 1994, World Health Organisation (WHO) and International Agency for Research on Cancer (IARC) has declared *H.pylori* as a class I carcinogen. (Chronic infection with H. pylori. In: IARC Working Group on the Evaluation of Carcinogenic Risks to Humans. Schistosomes, Liver Flukes and *Helicobacter pylori*. IARC Monographs on the Evaluation of Carcinogenic Risksz n to Humans, 61. Leon, France: International Agency for Research on Cancer, 1994: 177). It has been observed that it is associated with 50% of gastric adenocarcinoma and >80% of gastric lymphomas worldwide.[1]

Chronic gastritis is defined as the presence of chronic mucosal inflammatory changes leading to mucosal atrophy and intestinal metaplasia, usually in absence of erosions. The epithelial changes may become dysplastic and constitute a background for development of carcinoma. This cascade of progression of disease from chronic gastritis to carcinoma is called Correa's cascade of multistep gastric carcinogenesis.[4]

Hence, whenever possible, it is necessary to identify the cause of gastritis in order to check the progression to carcinoma. *H.pylori* being one such cause of chronic gastritis, the diagnosis with the help of chemical tests (rapid urease test) and histopathology, supplemented by special staining procedures (modified Giemsa), is essential to formulate appropriate clinical strategies for better management of patients.

The prevalence of *H.pylori* infection in North East India seems to be high. The aims and objectives of this hospital-based study are:

1. To study the presence of H. pylori infection in patients clinically indicated for upper gastrointestinal endoscopy.
2. To interpret the histopathological changes in endoscopic gastroduodenal mucosal biopsy tissue with special reference to the updated Sydney System.
3. To compare and analyse the histopathological changes occurring in the stomach and duodenum (first part) of *H.pylori* infected and non-infected patients.
4. To ascertain the diagnostic accuracy of endoscopy compared to histopathology in diagnosis of gastroduodenal diseases.

2. Materials and Methods

The study was conducted in a tertiary care teaching hospital over a period of one year from July 2021 till June 2022.

2.1. Inclusion criteria

1. Patients more than 18 years of age, having clinical indications of upper gastrointestinal endoscopy.
2. Patients not under treatment with proton pump inhibitors, bismuth compounds, antibiotics in the last 3 weeks and H_2 blockers in the last 24 hours.

2.2. Exclusion criteria

1. Patients with oesophageal disease.
2. Patients who have been previously treated with anti-*H.pylori* drug regime.
3. Patients who had ultrasonographic evidence of pancreatitis, biliary disease or chronic liver disease.
4. Patients with abnormal coagulation profile.

2.3. Clinical history and routine investigations

1. An informed consent was taken from all the patients who were selected for the study.
2. A thorough clinical history was taken as per the proforma of the present study.
3. Routine blood and stool examinations were done.
4. An ultrasonography (whole abdomen) was done to rule out pancreatic and biliary tract disease.

2.4. Endoscopy and mucosal biopsy sampling

After an overnight fast (6 hours) all the selected subjects underwent upper gastrointestinal endoscopy with flexible fibre-optic endoscope (Fujinon©) and the endoscopic findings were noted. In cases which were endoscopically normal or only with erosions, mucosal biopsies were taken with sterile biopsy forceps (Olympus©) as per the Updated Sydney System (1994). This system recommends that at least 5 mucosal biopsy specimens be taken: 2 each from lesser and greater curvature of cardia of stomach, 2 each from greater and lesser curvature of antrum and 1 from the incisura.

In cases with presence of an ulcer (clinically benign) anywhere in the stomach or first part of duodenum, additional multiple bits of tissue were taken from the different edges of the lesion (over and above the four sites mentioned) for histopathological examination. In cases with presence of a growth (clinically malignant) only multiple bits of tissue from different edges of the growth were taken.

An additional mucosal biopsy was taken from the antrum for the rapid urease test (RUT) for *Helicobacter pylori* in all cases. In cases where the antrum was involved with erosions/ulcer/growth, the biopsy was taken from surrounding normal mucosa for RUT.

2.5. Rapid urease test

The biopsy urease test is a simple and convenient method for diagnosing *H.pylori* infection. It is based on the presence of large amounts of preformed urease enzymes in *H.pylori*. In this study, Pylotest^{TM}kit manufactured and marketed by Halifax Research Laboratories, Kolkata was used. Fresh mucosal biopsy samples from antrum were obtained with biopsy forceps and put in the urea gel media with the help of a needle and crushed. The results are usually obtained by 6 to 9 hours but can be interpreted upto 24 hours.[5] In this study, however, the positive results were obtained within 6 to 9 hours, in majority of the cases. A known urease positive culture colony (Klebsiella) was taken as control for the test.

2.6. Routine processing and staining

All the mucosal biopsy samples obtained for histopathology were put in different bottles containing 10% formalin and properly labelled mentioning the site of biopsy. These tissue samples were routinely processed and stained with Haematoxylin and Eosin for light microscopic examination.[6] In some cases, a modified Giemsa stain was performed to demonstrate *H.pylori* in the tissue sections.[7]

2.7. Histopathological interpretation and reporting

Cases were considered to be *H.pylori* infected when both rapid urease test and histology were positive.

All the cases of chronic gastritis were reported following the guidelines put forward by the Updated Sydney System (1994) of reporting, with an additional note on the pathological involvement of the first part of duodenum.

The malignant cases were diagnosed and classified according to the guidelines put forward by WHO classification.[8]

The updated Sydney System (1994) recommends that at least 5 mucosal biopsy specimens be taken: 2 each from lesser and greater curvature of cardia of stomach, 2 each from greater and lesser curvature of antrum and 1 from the incisura. However, due to constraints of resources and supplied material we followed the recommendations put forward by Genta et al in the present study.[9]

3. Results and Observation

In this study, total 120 cases were evaluated of which majority (31 out of 120 i.e 25.83%) were in the age group of 38-47 years; the Male:Female ratio being 1.22:1.

Majority of the patients presented with upper abdominal pain and discomfort (106 out of 120) followed by acid reflux and other symptoms being heart burn, vomiting, constipation belching etc. Majority of the patients (44/120 i.e 36.67%) had symptoms of 4 to 5 months duration. (Figure 2).

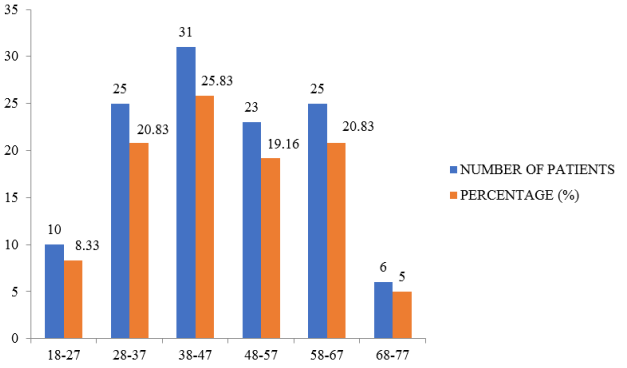

Fig. 1: Bar-diagram showing distribution of patients (clinically indicated for endoscopy) according to age

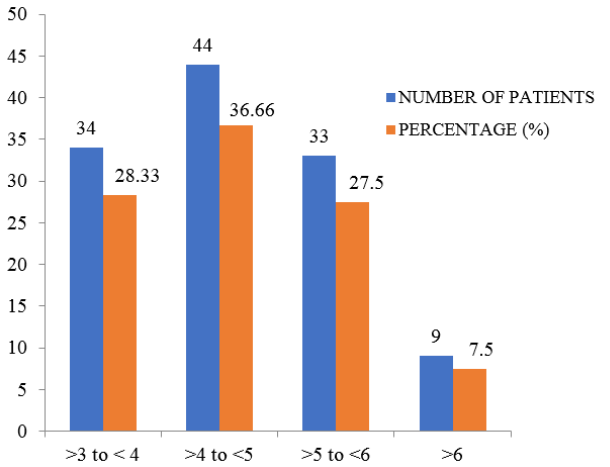

Fig. 2: Bar-diagram showing distribution of patients according to the duration of symptoms.

On endoscopy, the most commonly encountered lesion was an ulcerative growth (21.66%) followed by duodenal ulcers (20.83%) (Figure 3).

The following table shows the results of rapid urease biopsy test compared to histopathology in the patients under study. Five false negative cases (negative on RUT, positive on histopathology) were obtained and only one false positive case (positive on RUT, negative on histopathology) was noted.

Taking histological demonstration of *Helicobacter pylori* as standard the following results are obtained:

1. True positive (TP) = 79
2. True negative (TN) = 35
3. False positive (FP) = 1
4. False negative (FN) = 5

Therefore,

1. Specificity of RUT = TN/TN+FP = 35/35+1 = 35/36 = 97.22%.

Table 1: Results of rapid urease test confirmed by histology

Serial number	Endoscopic findings	Number of cases	Number of cases showing rapid urease test positive	Number of cases showing histological demonstration of *H .pylori*	Remarks
1.	Ulcerative growth	26	13	13	
2.	Duodenal ulcer	25	24	24	
3.	Antral ulcer	21	13	14	1 case was false negative on RUT
4.	Antral erosions	21	17	20	3 cases were false negative on RUT
5.	No abnormality	12	5	6	1 case was false negative on RUT
6.	Duodenal erosions	9	7	7	
7.	Polyp	3	1	0	1 case was false positive on RUT
8.	Fundal erosions	2	0	0	
9.	Fundal nodules	1	0	0	

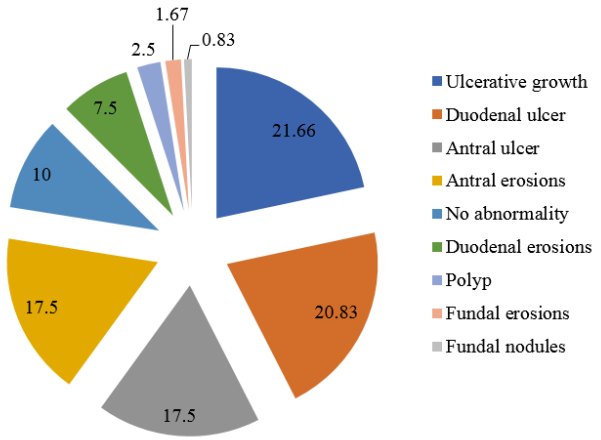

Fig. 3: Pie-diagram showing distribution of cases based on endoscopic findings.

2. Sensitivity of RUT = TP/TP+FN = 79/79+5 = 79/84 = 94.04%.
3. Positive predictive value (PPV) = 79/79+1 = 79/80 = 98.75%.
4. Negative predictive value (NPV) = 35/35+5 = 35/40 = 87.5%.
5. Accuracy of RUT = TP+TN/Total cases = 79+35/120 = 114/120 = 95%.

The above table shows that the maximum number of cases (70.83%) was inflammatory on histopathology. 21.67% cases were malignant, 5% dysplastic and 1.67% cases had no pathology.Table 2

hows that the most common inflammatory lesion obtained was chronic gastritis (29 out of 86 cases i.e. 33.72%) of which chronic antral gastritis was the commonest (24 out of 29 i.e. 82.75%). Only 1 benign neoplastic case was obtained which was a hyperplastic polyp.Table 3

Table 4 shows that there were 6 cases of gastric dysplasia of which 4 (66.67%) were low grade and 2 (33.33%) high grade.

The preceding table shows that of the neoplastic cases on histopathology, 92.6% were gastric adenocarcinoma, 3.70% each of duodenal adenocarcinoma and hyperplastic polyp.Table 5

The above table demonstrates that the most common type of gastric adenocarcinoma was Tubular type (56%), followed by Diffuse type (36%) and 4% each of Papillary and Mucinous type.Table 6

Shows that 100% cases of each of chronic antral gastritis with duodenitis/ duodenal ulcer and chronic atrophic gastritis were positive for *H.pylori*. 18 out of 29 cases (62.06%) of chronic gastritis were positive for *H.pylori* of which chronic pangastritis showed 100% positivity.

The above table shows that among the endoscopically diagnosed cases of ulcerative growth, 23(88.46%) were diagnosed as adenocarcinoma, 2(7.7%) as gastric dysplasia and 1(3.84%) as chronic gastritis, on histopathological examination**.**

The preceding table shows that among the endoscopically normal cases, 7(58.33%) had chronic gastritis, 2(16.67%) had chronic gastritis with duodenitis/duodenal ulcer, 1(8.33%) had chronic atrophic gastritis and 2(16.67%) were normal on histopathological examination.

Statistical considerations of table 21

Taking histopathology as gold standard in diagnosis of benign and malignant diseases, the following results are obtained:

1. True positive (TP) = 23
2. True negative (TN) = 75

Table 2: Distribution of cases based on histopathological diagnosis

Serial number	Nature of lesion		Number of cases (n=120)	Percentage (%)
1.	Normal (no pathology)		2	1.67
1.	Inflammatory		85	70.83
2.	Dysplasia		6	5
2.	Neoplasia	Benign	1	0.83
		Malignant	26	21.67

Table 3: Inflammatory lesions on histopathology (n=85)

Serial number	Diagnosis		Number of cases	Percentage (%)
1.	Chronic gastritis	Antral	24	27.90
		Pangastritis	4	4.65
		Fundal	1	1.16
2.	Chronic gastritis with duodenitis/duodenal ulcer		27	31.39
3.	Chronic atrophic gastritis		24	27.90
4.	Duodenitis/duodenal ulcer		4	4.65
5.	Granulomatous lesion		1	1.16

Table 4: Dysplastic lesions on histopathology (n = 6)

Histopathological diagnosis		Number of cases	Percentage (%)
Dysplasia	Low grade	4	66.67
	High grade	2	33.33

Table 5: Neoplastic lesions on histopathology (n = 27)

Serial number	Nature of lesion	Diagnosis	Number of cases	Percentage (%)
1.	Benign	Hyperplastic polyp	1	3.70
2.	Malignant	Gastric adenocarcinoma	25	92.6
		Duodenal adenocarcinoma	1	3.70

Table 6: Histological typing of gastric adenocarcinoma based on WHO guidelines (2000) (n = 25)

Serial number	Histological type	Number of cases	Percentage (%)
1.	Tubular	14	56
2.	Diffuse/Signet ring	9	36
3.	Papillary	1	4
4.	Mucinous	1	4

Table 7: Inflammatory and benign histopathological lesions showing *Helicobacter pylori* positivity

Lesions		Total number of cases	*H.pylori positive*	*H.pylori* negative	Percentage of *H .pylori* positive cases (%)
Chronic Gastritis	Antral	24	14	10	58.33
	Fundal	1	0	1	0
	Pangastritis	4	4	0	100
Chronic Antral Gastritis with duodenitis/duodenal ulcer		27	27	0	100
Duodenal ulcer/duodenitis		4	1	3	25
Chronic Atrophic Gastritis		24	24	0	100
Granuloma		1	0	1	0
Hyperplastic polyp		1	0	1	0
Total		86	70	16	81.39

Table 8: Grading of variables (according to updated Sydney system) of the *H.pylori* positive inflammatory cases (n=70)

Variables	Absent (score 0)	Mild (score 1)	Moderate (score 2)	Severe (score 3)	Total
H.pylori	0	68	2	0	70
Neutrophils	0	61	8	1	70
Mononuclear cells	0	12	58	0	70
Atrophy	46	21	3	0	70
Intestinal metaplasia	46	21	3	0	70

Table 9: Premalignant and malignant lesions showing *H.pylori* positivity

Lesions	Total number of cases	*H.pylori* positive cases	*H.pylori* negative cases	Percentage of *h.pylori* positive cases
Dysplasia (6)	Low grade (4)	3	1	75
	High grade (2)	1	1	50
Gastric Adenocarcinoma (25)	Tubular (14)	9	5	64.28
	Diffuse (9)	4	5	44.44
	Papillary (1)	0	1	0
	Mucinous (1)	0	1	0

Table 10: Histopathological diagnosis of endoscopicaly suspected malignant cases

Endoscopy (number of cases)	Histopathology (number of cases)		
	Chronic gastritis	Dysplasia	Adenocarcinoma
Ulcerative Growth (26)	1	2	23

Table 11: Histopathological diagnosis of endoscopicaly normal cases

Endoscopic diagnosis (number of cases)	Histopathologic diagnosis (number of cases)			
	Chronic gastritis	Chronic gastritis with duodenitis or duodenal ulcer	Chronic atrophic gastritis	Normal (no pathology)
No Abnormality (12)	7	2	1	2

Table 12: Results of endoscopy and histopathology in diagnosing benign and malignant lesions

Histopathological diagnosis	
Benign	**Malignant**
75	3
1	23

3. False positive (FP) = 1
4. False negative (FN) = 3

Therefore,

1. Specificity of endoscopy = 75/75+1 = 75/76 = 98.68%
2. Sensitivity of endoscopy = 23/23+3 = 23/26 = 88.5%
3. Accuracy of endoscopy = 75+23/108 = 98/102 = 96.07%

(Note: Twelve cases that were endoscopically normal and 6 cases that were histopathologically dysplastic have been excluded from the calculation)

4. Discussion

Of the 85 inflammatory lesions, most common diagnosis was chronic gastritis (in 29 cases i.e. 33.72%) followed by chronic gastritis with duodenitis (in 27 cases i.e. 31.39%), chronic atrophic gastritis (in 24 cases i.e. 27.9%) and chronic duodenitis (4 cases i.e. 4.65%). One case was diagnosed as granulomatous lesion. However, further biopsy sample was not available for staining for acid fast bacilli (AFB) and the patient was advised further investigations to rule out tuberculosis.

The above results were comparable to previous studies by Johnsea R et al and Jonsson et al.[10,11] Johnsea et al found 43.2% cases of chronic gastritis as the most common cause of dyspepsia in adults.[10] A slight higher incidence as compared to our study might be because the study by Johnsea et al was conducted on 309 patients and we had only 120 patients.[10] However, Jonsson et al found chronic gastritis in 33% cases of dyspepsia which was quite similar to our data.[11] But the percentage of chronic gastritis

with duodenitis (65%) and chronic duodenitis (14%) were higher than the present study. These variations might be due to different food habit and food constituents in different regions and countries.

Among the chronic gastritis cases, 14(48.2%) were antral, 9(31.03%) were fundal and 6(20.68%) involved both antrum and fundus. This was in accordance with the study by Karmes et al who obtained antral gastritis in 45%, fundal gastritis in 31% and pangastritis in 24%.[12]

Of the 6 dysplastic lesions, 4 (66.67%) were low grade and 2 (33.33%) were high grade. Of the neoplastic cases, 1 was benign hyperplastic polyp, 25 were gastric adenocarcinoma and 1 duodenal adenocarcinoma.

On classifying all the 25 cases of gastric adenocarcinoma on the basis of WHO guidelines (2000), 14(56%) were tubular adenocarcinoma, 9(36%) were diffuse and 1(4%) each of papillary and mucinous adenocarcinoma. The only case of duodenal adenocarcinoma was moderately differentiated.

Of the 1344 tumours initially described by Lauren, 53% were intestinal and 33% diffuse.[13] The 'tubular' and 'papillary' variety of the WHO classification falls under Lauren's 'intestinal' type.[13] In that case, our incidence of 'intestinal' adenocarcinoma comes out to be (56+4) % i.e. 60%. Lauwyers et al showed the relative frequencies are 50-67% for intestinal and 29-35% for diffuse.[14] Papillary adenocarcinoma accounts for 6-11% and mucinous adenocarcinoma about 10%. It is evident that these data are fairly comparable to the data obtained by the current study.

While evaluating for the presence of *H.pylori* infection on histopathology it was found that 18 out of 29 (62.06% cases) of chronic gastritis were associated with *H.pylori* infection. This is comparable to a thesis study by Atto Ghada et al where he found *H.pylori* infection in 69% patients of chronic gastritis. In chronic gastritis where there was predominant antral involvement (82 cases), *H.pylori* infection was found in 69 i.e. 84.15% cases. Wyatt et al found that 90% case of chronic antral gastritis were associated with *H.pylori* infection.[15] Khan et al (2007) found this association in 84% cases.[16]

A significant observation in the present study has been that 100% cases of chronic antral gastritis which were associated with duodenitis were *H.pylori* infected. Phul et al observed in his study that when associated with duodenitis, 90.5% cases of chronic antral gastritis were positive for *H.pylori*.[17] This was also in accordance with the study by Genta et al.[18] Earlam et al found that chronic antral gastritis in patients with chronic duodenal ulcer is almost invariable and found that this antral gastritis is usually associated with H. pylori infection.[19]

Another important observation in our study was that all cases of chronic atrophic gastritis were positive for *H.pylori* infection. Khan et al also found 100% cases of chronic atrophic gastritis to be positive for *H.pylori*.[16] On the contrary, Lew et al,[20] in an area with low prevalence of *H.pylori*, found that only 6.8% cases of chronic atrophic gastritis were positive for *H.pylori*.[19] But in the present study, the association is high probably because data shows that Indians and Chinese have higher rate of H,pylori infection and also may be due to the fact that other associated causes of atrophic gastritis (e.g. pernicious anaemia) were not taken into account in the current study.[21] This was another drawback of this study.

Seventy inflammatory cases which were positive for *H.pylori* on histology were graded based on five variables (Updated Sydney System, 1994)

1. *H.pylori* colonisation: 97.14% mild and rest moderate.
2. Neutrophils (activity of inflammation): 87.14% mild, 11.4% moderate and 1.4% severe.
3. Atrophy: 87.5% mild and rest moderate.
4. Intestinal metaplasia: 87.5% mild and rest moderate.
5. Chronic inflammation: 17.14% mild, 82.85% moderate.

Suzana et al.,[22] applied updated Sydney system in their material of *H.pylori* gastritis and graded the variables.[23] However, the results vary, probably because environment plays a major role in the topographical distribution and density of *H.pylori* colonisation and hence, inflammatory activity.

Out of the 4 low grade dysplasia cases, 3(75%) were positive for *H.pylori* and out of 2 high grade only 1 (50%) was positive. On evaluation of the cases gastric adenocarcinoma for *H.pylori* infection, 13 out of 25 cases i.e. 52% cases were positive. Among these cases, 9 out of 14(64.28%) cases of tubular adenocarcinoma and 4 out of 9(44.44%) cases of diffuse adenocarcinoma were positive for *H.pylori*. This observation is in accordance with other studies that have shown that *H.pylori*, due to presence of a pro-inflammatory IL-1 genotype, is clearly associated with an increased risk of intestinal (WHO – tubular/papillary) but not with diffuse type.[14]

When the benign endoscopic findings were correlated with their diagnoses, it was seen that 2 cases out of 21 antral ulcers turned out to be dysplastic and 3 cases were finally diagnosed as gastric adenocarcinoma on histopathology. These 3 cases were falsely benign (False Negative) on endoscopy. One case of ulcerative growth on endoscopy turned out to be chronic gastritis on histopathology. This case was falsely malignant (False Positive) on endoscopy. So, in total, there was an incorrect endoscopic interpretation in 6 cases out of 120 i.e. 5%. Dekker et al found incorrect endoscopic interpretation in 7.3% of ulcerous lesions.[23]

Out of the 12 cases which were endoscopically normal, 10 cases (83.33%) had histopathological abnormality. Seven (58.33%) were chronic gastritis, 2(16.67%) were chronic gastritis with duodenitis and 1 (0.83%) was

chronic atrophic gastritis. These results were in accordance with the study by Majeed et al where they found 90% of the endoscopically normal cases turned out to be histopathologically abnormal.[24] However, Khakoo et al, found this observation in 65% cases.[25]

Only 2 cases (16.67%) out of 12 endoscopically normal cases turned out to be histopathologically normal too. Majeed et al found normal looking gastric mucosa in 10.2% of endoscopically normal cases whereas Kassir et al found it in 33% cases.[24,26]

Taking all the benign and malignant cases (excluding the 6 dysplastic and 12 endoscopically normal cases) on endoscopy and histopathology the specificity, sensitivity, PPV, NPV and diagnostic accuracy of endoscopy were calculated as 98.68%, 88.5%, 95.83%, 96.15% and 96.07% respectively. Marco et al found the specificity to be 95% and sensitivity 82%.[27] Todd et al found specificity of endoscopy to be 91.7% and sensitivity to be 81%.[28] This study by Todd et al also mentioned that a repeat endoscopy with biopsy improved the sensitivity up to 100%.[28] Llanos et al found diagnostic accuracy of endoscopy alone to be 86.5%.[29] The accuracy of endoscopic, multiple-directed biopsies was higher than endoscopy alone and reached 94.9%. However Segal et al found the diagnostic accuracy to be 96.5% similar to the present study (96.07%).[30] The difference with Llanos' study can be explained on the basis of the endoscopists' experience and nature of the lesions.

The entire inflammatory cases positive for *H.pylori* infection were treated accordingly to eradicate the organism (Harrison's Principles of Internal Medicine; Current diagnosis and treatment – Gastroenterology, Hepatology and Endoscopy). Cases which were dysplastic on histopathology were advised clinical follow up with a repeat biopsy. These cases were lost to follow up. All the malignant cases were referred for further treatment.

5. Conclusion

Therefore, this extensive study done on *Helicobacter pylori* associated gastroduodenal diseases may be concluded with the following recommendations:

1. Any patient complaining of dyspepsia not responding to initial treatment regimen may be subjected to endoscopy followed by RUT.
2. A histopathological evaluation on the collected punch biopsies may be attempted for confirmation of the *H.pylori* status and also evaluated for any evidence of pre-malignant changes (atrophy or intestinal metaplasia).
3. The Updated Sydney System for evaluation of all cases of chronic gastritis may be useful in planning of treatment regimen for the patients.
4. A collaborative effort of the clinician, endoscopist and the pathologist plays a vital role in management of such patients of gastroduodenal diseases.

6. Source of Funding

None.

7. Conflicts of Interest

There is no conflict of interest.

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