

## Research Paper: Upper Gastrointestinal Endoscopy in Dyspepsia, A Prospective Study



### Medical Science

**KEYWORDS :** Dyspepsia, Upper Gi Endoscopy, Duodenal Ulcer, Helicobacter Pylori

**Dr. Ronak R. Modi**

Senior Resident In General Surgery Department, Amc Met Medical College, Ahmedabad - 380008

**Dr. Krushna R. Modi**

Tutor In P&Sm Department Amc Met Medical College, Ahmedabad. 380008

### ABSTRACT

*Dyspepsia, also known as Upset Stomach or Indigestion, refers to a condition of impaired digestion. Common causes of dyspepsia include various upper gi pathologies. Helicobacter Pylori is now believed to be a major factor in the pathogenesis of duodenal ulcer disease and gastritis. Upper gastrointestinal endoscopy, along with biopsy, is the efficacious way to diagnose underlying organic pathology. This prospective study was done on 240 patients and biopsies were taken as and when required in 108 patients. The main endoscopic diagnoses were duodenal ulcer disease, erosive duodenitis, and erosive antral gastritis. Abnormal endoscopic findings were present in 77.5% of the cases, while 22.5% were endoscopically normal. Identification of H. Pylori were made in gastric biopsies of 26 (76.47%) out of 37 patients with erosive gastritis and in 31 out of 35 patients with duodenal ulcer disease (87.5%). H. Pylori may prove to be an important etiological agent in the pathogenesis of chronic erosive gastritis and non-ulcer.*

### INTRODUCTION

Fiberoptic upper gastrointestinal (UGI) endoscopy is now a well-established procedure for the investigation and management of UGI disorders. It has become the procedure of choice for the majority of patients, perhaps for its diagnostic superiority over barium studies. Dyspepsia seems to be the most commonly presenting symptom of majority of the upper gastrointestinal pathologies, and is frequently associated with, gastro-esophageal reflux disease (GERD) or gastritis.

Dyspepsia, also known as Upset Stomach or Indigestion, refers to a condition of impaired digestion. It is a medical condition characterized by chronic or recurrent pain in the upper abdomen, upper abdominal fullness and feeling full earlier than expected when eating. The characteristic symptoms of dyspepsia are Epigastric Pain, Epigastric Burning, Post-prandial Fullness & Early Satiating.

On investigation, organic disease e.g. duodenal or gastric ulcers are likely to explain the dyspepsia will be found in some patients. In others, no such causal pathology or disease is identified: these patients are said to have functional dyspepsia.

Common causes of dyspepsia include Duodenal ulcer, Gastric ulcer, Oesophageal/Gastric Cancer, Oesophagitis, Gastritis, Duodenitis, Hiatus Hernia, Normal mucosa.

Helicobacter pylori is now believed to be a major factor in the pathogenesis of duodenal ulcer disease and gastritis. Chronic gastritis due to Helicobacter pylori infection, possibly together with exposure to dietary and environmental mutagens, may ultimately lead to gastric cancer. Recent reports have suggested an association between an unusual lymphoma of the stomach, gastric mucosa-associated lymphoid tissue (MALT) lymphoma, and Helicobacter pylori infection. In this study the objective was to delineate the patterns of UGI disease in patients presenting with UGI tract-related symptoms to the GI and Liver units of Smt. S.C.L. Hospital located in Ahmedabad, over a two-year period.

### MATERIAL & METHODS

The study involved 240 patients seen over a 10-year-period from June 2010 to October 2012. Excluded from the study were patients with gallbladder, liver, pancreatic, renal and peritoneal diseases, as well as parasitic infestations.

Presenting symptoms included dysphagia, Epigastric pain and/or upper abdominal discomfort related to meals, heartburn, nausea, vomiting, and any of these symptoms with weight loss. Patients with lactase deficiency symptoms, lower abdominal pain, irritable bowel syndrome and hematemesis or melena were not included in the study. An upper

abdominal ultrasound, complete blood count, erythrocyte sedimentation rate, prothrombin time, partial thromboplastin time, platelets count, random blood sugar, urine and stool analysis were performed on all patients prior to their UGI endoscopic examination.

### PROCEDURE

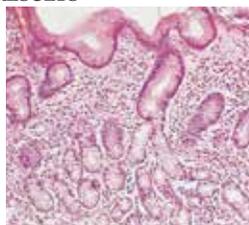
The endoscopy was performed after applying local lidocaine spray to the throat. A total of 36 (15%) patients were given midazolam 1-10 mg intravenously before endoscopy. The remaining 204 (85%) patients were endoscoped without sedation upon their request. The UGI endoscopy included a general examination of the oesophagus, stomach and duodenum. The endoscopic diagnosis of hiatal hernia, esophagitis, gastritis, gastric ulcer, duodenitis and duodenal ulcer were made according to the accepted standard criteria. Erosions with papillomatous or granulated changes of the stomach mucosa were grouped under chronic erosive gastritis as endoscopic diagnosis.

Biopsies for histopathological diagnosis and Helicobacter pylori detection were obtained in all cases with suspected malignant disease, and in patients with erosive antral gastritis, gastric and duodenal ulcer diagnoses.

Additionally, biopsies were obtained from patients with polypoid lesions, post-bulbar duodenal lesions, as well as those with erosive esophagitis, and all patients with esophageal erosions and ulcers of those suspected to have Barrett's esophagitis.

The biopsies taken were fixed in 10% buffered formaldehyde and processed in the usual manner for histological examination. For the histological evaluation, routine hematoxylin and eosin (H&E) stained paraffin embedded sections were used. Where needed, more sections were cut and stained with H&E, D-pas for mucin, Ziehl-Neelsen for acid-fast bacilli (AFB), and Warthin-Starry silver impregnation for identification of Helicobacter pylori organisms. For the diagnosis and classification of gastritis, the system adopted by Whitehead et al. was used. The procedure and its possible complications were explained to each patient and their consent obtained prior to endoscopy.

### RESULTS



**Figure 1: H & E Staining In H. Pylori Positive Biopsy Sample**

The main indications for endoscopy were epigastric pain (88%), heartburn (31%), nausea and vomiting (23%),dysphagia (7.8%), weight loss (6.4%), and distention(4.5%). Out of the 240 patients subjected to UGIendoscopy, 50% were male, and 50% were female. Ageand sex distribution were as shown in Table 1. Theendoscopicfindings are summarized in Table 2.

Of all the esophageal disorders, chronic esophagitis was the most common endoscopic finding (8.75%), followed by hiatal hernia (4.17%) and esophageal ulcers (1.25%). In the stomach, the main finding was chronic gastritis, with erosions amounting to 15.42% of all endoscoped patients, while benign gastric ulcer was found in 5% of patients, and malignant gastric ulcer in only 1.66%. Duodenal ulcers were encountered in 24.17% of all patients, while those with duodenitis, with or without erosions, constituted 16.66%. Gastric/duodenal ulcer ratio was 1:4.83.



Figure 2:Gastric Ulcer in the Antrum

Table 1: General information of patients

Age group	Total	Male	Female
20-30	36	12	24
30-40	64	44	20
40-50	40	12	28
50-60	48	32	16
60-70	44	20	24
70-80	8	0	8
Mean Age	40	20	20
Std. Deviation	16.81	14.42	6.53
Total	240	120	120

Table 2: Upper GI Scopy Findings in Dyspepsia

GI Scopy Findings	Total	(%)
Normal	54	22.5
Erosive esophagitis	21	8.75
Esophageal ulcers	3	1.25
Esophageal neoplasm	1	0.42
Hiatus hernia	10	4.17
Erosive gastritis	37	15.42
Gastric ulcer	12	5
Malignant gastric ulcer	4	1.66
Erosive duodenitis	40	16.66
Duodenal Ulcer	58	24.17
Duodenal malignant lesion	0	0
Total	240	100

The mean age for patients with duodenal ulcer was 38.2 years, and that for those with gastric ulcers was 52.4 years. Out of the 240 endoscoped patients, 108 (45%) patients were biopsied. The biopsies were from the stomach in 75 (69.44%); from the oesophagus in 23 (21.3%); and from the duodenum in 9 patients (8.33%).

The histological diagnoses of the biopsied patients are summarized in Table 3. Of interest was the identification of Helicobacter bacilli in gastric biopsies of 26 (76.47%) out of 37 patients with erosive raised localized antral gastritis. In contrast, bacilli were identified in only 7 of 11 (63.64%) non-ulcer dyspeptic patients with histologically normal mucosa. Helicobacter pylori was seen in 31 patients out of 35 consecutive patients with duodenal ulcer disease (including patients with duodenitis) (87.5%), 9 patients out of 12 patients with gastric ulcer disease (75%).

Malignancy was diagnosed in 5 patients (2.08%). The most common was gastric adenocarcinoma in 4 patients (80%) from which 3 patients (75%) had positive Helicobacter pylori. Esophageal squamous cell carcinoma was seen in 1 patient (20%).

Table 3 : Histological diagnosis in 108 Patients

Diagnosis	Patients	(%)
Chronic esophagitis	9	8.33
Esophageal benign ulcer	1	0.93
Esophageal carcinoma	1	0.93
Chronic superficial gastritis	34	31.48
Chronic atrophic gastritis	1	0.93
Gastric adenocarcinoma	4	3.7
Gastric benign ulcer	12	11.11
Chronic duodenitis	20	18.52
Duodenal ulcer	15	13.88
Normal Histology	11	10.19
Total	108	100

DISCUSSION

UGI endoscopy is an easy, safe and cost-effectivemore specific than bariummeal studies.<sup>2,11,12</sup> In our study,abnormal endoscopic findings were present in 77.5% of the cases, while 22.5% were endoscopically normal. Great proportions of the latter group of patients were female and were probably suffering from functional dyspepsia.<sup>13</sup>

Adenocarcinoma of the stomach in our study showed a higher prevalence than what was reported. Carcinoma of the stomach is less common than in Southeast Asia, Japan and Europe.<sup>14</sup>The results of our limited survey do not support or negate such claims.

Helicobacter pylori in patients with different upper gastrointestinal tract pathologies hasbeen documented very frequently. This study matches the overall incidence rate of H. Pylori all over the world.<sup>15-19</sup>

The absolute prevalence of duodenal ulcer is not known. It is estimated to occur in about 14% to 25% of the population.<sup>16, 20</sup>In this study, upper gastrointestinal tract peptic disorders were documented in 70% of our patients, from which duodenal peptic ulcer disease was encountered in 24.17%. Duodenal ulcer disease was similar, but gastric ulcer disease was less than that observed in Western countries such as the UK. <sup>20</sup>In our study, simple Barrett's esophagitis was nothistopathologically documented in patients.

**CONCLUSION**

In conclusion, we found that highest incidence of dyspepsia occurs in the age group 30-40 years of age. We also found that, fiberoptic endoscopy is a safe, acceptable and mandatory diagnostic procedure. Early endoscopy has a crucial role in making early diagnosis. The main endoscopic diagnoses were duodenal ulcer disease, erosive duodenitis, and erosive antral gastritis. Comparative analysis showed notable variations in the incidence of various endoscopic findings, in particular peptic ulcer disease and malignancies of the stomach and esophagus. *Helicobacter pylori* may prove to be an important etiopathological agent in the pathogenesis of chronic erosive gastritis and non-ulcer.

For the patients having persistent symptoms even after all investigations and all drug trials, they're diagnosed to have 'Functional Dyspepsia'.

In General, the "Prompt Endoscopy and Directed Treatment" should be the approach for choice, although it is a bit costly approach, but straight away gives the accurate diagnosis in most of the cases.

**REFERENCE**

- Bell GD. Monitoring and safety in endoscopy. *Baillieres Clin Gastroenterol* 1990; 5:79-98. | 2. Rogers IM, Solchi GS, Monle B, Joaffa SN. Endoscopy and routine double contrast barium meal in diagnosis of gastric and duodenal disorders. *Lancet* 1976;4:901-3. | 3. Marshal BJ. *Campylobacter pylori*: its link to gastritis and peptic ulcer disease. *Rev Infect Dis* 1990;12:587-93. | 4. Soll H. Pathogenesis of peptic ulcer and implications of therapy. *N Eng J Med* 1990;322:909-16. | 5. Graham DY, Go MF. *Helicobacter pylori*: current status. *Gastroenterology* 1993;105:279-81. | 6. Peura DA. *Helicobacter pylori*: gastritis, ulcers and cancer. *Pract Gastroenterol* (supplement) 1994;18:538-48. | 7. Parsonnet J. *Helicobacter pylori* and gastric cancer. *Gastroenterol Clin N Am* 1993;22:89-104. | 8. Stolte M, Edit S. Healing gastric MALT lymphomas by eradicating *H. pylori* (editorial). *Lancet* 1993;342:568. | 9. Kasugai T. Endoscopic diagnosis in gastroenterology. Tokyo: Igakushoin, 1982. | 10. Whitehead R, Truelove SC, Gear ML. The histological diagnosis of chronic gastritis in fiberoptic gastroscopy biopsy specimens. *J Clin Path* 1972;25:1-11. | 11. Wiliasalo M, Tallroth K, Korhola O, Ihamaki TA. Comparison of double contrast barium meal and endoscopy. *Diagn Mag* 1980;49:1-5. | 12. Fedail SS, Araba BMO, Homeida MM, Ghandour ZM. Upper gastrointestinal endoscopy experience in the Sudan. *Lancet* 1982;2:297-9. | 13. Harvey RF, Salih SY, Read AE. Organic and functional disorders in 2,000 gastroenterology outpatients. *Lancet* 1983;632-4. | 14. Doll R. Cancer in five continents. *Proc R Soc Med* 1972;65:49-55. | 15. Al-Moagel MA, Evans DG, Abdulghani ME, Adam E, Evans DJJR, Malaty HM, Graham DY. Prevalence of *Helicobacter pylori* infection and comparison of those with and without upper gastrointestinal symptoms. *Gastroenterology* 1990;85:944-8. | 16. McGuigan JE. Peptic ulcer and gastritis. In: Harrison's Principles of Internal Medicine. Isselbacher, Braunwald, Nilson, Martin, Fan CI, Kasper, editors. 13<sup>th</sup> ed. New York: McGraw-Hill Inc. 1994:1363-82. | 17. Mourad NA, Ahmed MBK, Al-Wabd A, Foli AK. *Helicobacter pylori*-associated dyspepsia in 208 patients from southern Saudi Arabia. *Ann Saudi Med* 1993;13:340-3 | 18. Graham DY, Shabib SM, Al-Mofleh I. It should be possible to eliminate peptic ulcer disease and gastric carcinoma from Saudi Arabia (editorial). *Ann Saudi Med* 1994;14:179-82. | 19. Ibrahim BH, Anim JT, Sarkar J. *Helicobacter pylori*-associated chronic antral gastritis in Kuwait: a histopathological study. *Ann Saudi Med* 1995;15:570-4. | 20. Langman MJS. Peptic ulcer in the epidemiology of chronic digestive disease. First edition. London: Edward Arnold, 1970:9-39 |