

Original research article**Study of Prevalence of H. Pylori Infection in Patients Undergoing Upper GI Endoscopy****Dr. Neha Singh¹, Dr. Abhay Kumar², Dr. Rajeev Kumar³**¹ Senior Resident, Department of Surgery, AIIMS Patna² Senior Resident, Department of Medicine, AIIMS Patna³ Senior Resident, Department of Medicine, AIIMS Patna**Corresponding Author: Dr. Rajeev Kumar****Abstract**

Background: prototypical developing country as far as H. pylori infection is concerned and more than 20 million Indians are estimated to suffer from peptic ulcer disease. There is very limited data regarding its prevalence in our country. So this study aims to ascertain the prevalence of the H. pylori infection in the patients.

Methodology: The study was conducted with both out patients and in-patients of All India Institute of Medical Sciences, Patna. The study conducted was a cross-sectional prospective study, Study duration of two years. The upper gastro-intestinal endoscopy was done with flexible, fibro-optic endoscope with patients in left lateral positions. Two endoscopic biopsy fragments were obtained from each patient from the antrum. One biopsy fragment was sent to histopathology.

Conclusion: The prevalence was 45% in the present study. It corroborates as there is improvement in the socioeconomic status, living conditions, as India strives on the path of being a developed country, mirroring the prevalence of a western country. There is no statistically significant association between H. pylori infection and gender, age. Among the endoscopic findings, prevalence was highest in gastric polyp, duodenitis, Gastric ulcer, Ca stomach, antral gastritis.

Keywords: Alcoholic Liver Disease, Biliary Gastritis; Percutaneous Endoscopic Gastrotomy; Portal Hypertensive Gastropathy.

Introduction

Helicobacter pylori (H. pylori) infection is associated with a number of common upper GI disorders, but most infected individuals are asymptomatic. About 50% of the world's population is infected with H. pylori, a major cause of chronic gastritis^[1] and the prevalence varies from country to country and region to region. H. pylori infection is a major problem in developing countries, almost always present in the setting of active chronic gastritis and is present in most duodenal (>90%) and gastric (60% to 90%) ulcer patients^[2]. H. pylori has been recognized as a major cause of gastritis and is associated with duodenal ulcer disease, gastric ulcer disease, gastric lymphoma, and gastric cancer in humans^[3]. *H. pylori* is currently the only bacterium to be recognized as a carcinogen^[4]. Helicobacter also clearly

has an etiologic role in the development of gastric lymphoma^[5]. India is a prototypical developing country as far as *H. pylori* infection is concerned and more than 20 million Indians are estimated to suffer from peptic ulcer disease^[6]. There is very limited data regarding its prevalence in our country. So this study aims to ascertain the prevalence of the *H. pylori* infection in the patients undergoing upper GI endoscopy in Aiiims, Patna. Phulwari sharif.

Objectives

To study the association of *H. pylori* with various upper GI disorders, To compare the results of rapid urease test with histopathological examination of antral mucosal biopsy. To study the relationship between Age and Sex with prevalence of *H. pylori* infection.

Review of Literature

The majority of endoscopes in use today are videoscopic, although in many parts of the world, fiberoptic systems are still the standard. In these videoscopic systems, the visualized image is created from reflections onto a charge coupled device (CCD), which is a chip mounted at the end of the endoscope rather than via the fiberoptic bundles. The CCD chip has thousands of pixels (light-sensitive points), which directly increase image resolution^[7] The most common variety of benign gastric pathology is inflammatory disease. Acute inflammation of the stomach, otherwise termed gastritis, can be caused by a variety of inciting factors.^[8] Nonatrophic gastritis is usually caused by *Helicobacter pylori* infection and presents as inflammation that predominantly affects the antrum of the stomach. Chronic *H. pylori* infection can also cause a multifocal atrophic gastritis with patchy atrophy and metaplasia beginning at the incisura and the transitional zone between antrum and body and affecting predominantly the antrum.^[9] Atrophic gastritis due to pernicious anemia is of autoimmune origin and results in the destruction of parietal cell mass. This usually affects the body of the stomach with relative sparing of the antrum. Patients with atrophic gastritis are considered at high risk for gastric adenocarcinoma. A chemical gastritis can also occur where irritation results from non steroidal anti inflammatory drugs (NSAIDs), bile reflux, or alcohol. Peptic ulcer disease may also result from *H. pylori* infection. Endoscopic testing for *H. pylori* involves biopsy of the antrum and body of the stomach for identification of organisms on histology, polymerase chain reaction, or rapid urease test. Culture and sensitivity can also be obtained for resistant strains. Other nonendoscopic options for *H. pylori* testing include the carbon urea breath test, serologic antibody tests, and fecal antigen test. *H. pylori* has been implicated in the pathogenesis of gastritis, gastric and duodenal ulcers, gastric adenocarcinoma, and gastric mucosa-associated lymphoid tissue (MALT) lymphoma. As such, when it is identified, it should be eradicated with pharmacotherapy and its eradication should be documented with repeat testing.^[10] A subset of angiodysplasia, gastric antral vascular ectasia (GAVE) or watermelon stomach, refers to the presence of vascular ectasia in a linear fashion in the antrum of the stomach.^[11] This gives the antrum a striped appearance similar to that of a watermelon. Vascular abnormalities are a significant cause of acute or chronic GI blood loss. Polypoid lesions of the stomach can be classified as benign, potentially malignant, and malignant. By order of prevalence, these include fundic gland polyps, hyperplastic polyps, adenomatous polyps, and malignancies.^[12] The most common type of gastric polyp is the fundic gland polyp, which are usually numerous, sessile polyps found in the fundus of the stomach. Fundic gland polyps can develop in the context of familial adenomatous polyposis, Zollinger-Ellison syndrome, or chronic proton pump inhibitor use, and though they may show signs of metaplasia, they are not considered premalignant.^[13] Hyperplastic polyps are caused by inflammation and can grow extremely large. They typically have a reddish appearance and

are more common in the antrum of the stomach. They are often associated with inflammation of the surrounding mucosa. Hyperplastic polyps are not classically considered malignant, but they often arise in the context of metaplastic chronic gastritis and may contain metaplasia or dysplasia.^[14] Gastric adenocarcinoma is the most common form of gastric malignancy and can take on various endoscopic appearances. It may present as an exophytic mass lesion, a nonhealing gastric ulcer, or an infiltrative process in the submucosa termed linitis plastica. Diagnosis is made with multiple mucosal biopsies. Leiomyomas and gastrointestinal stromal tumors (GISTs) are the most common mesenchymal tumor of the stomach. They are rounded submucosal lesions that can present anywhere in the stomach but most commonly found in the cardia or fundus adjacent to the gastroesophageal junction. Malignant potential is determined by the size, irregular borders, and mitotic index. Due to their submucosal location, tissue may be difficult to obtain without an endoscopic ultrasound assessment and core biopsy.^[15]



Figure 1: Full retroflex view of the GEJ showing a small submucosal tumor at angle of His

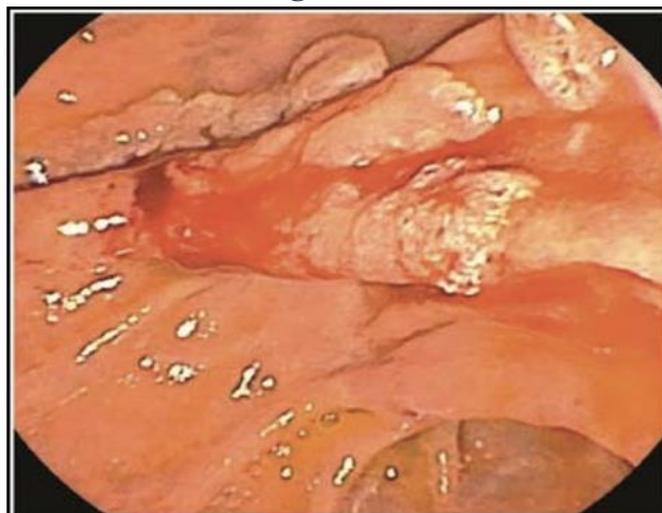


Figure 2: A large sessile duodenal periampullary polyp

H. pylori is an infectious disease that is typically treated with combinations of 2–3 antibiotics along with a PPI, taken concomitantly or sequentially, for periods ranging from 3 to 14 days. In clinical practice, the initial course of eradication therapy, heretofore

referred to as “first-line” therapy, generally offers the greatest likelihood of treatment success. Thus, careful attention to the selection of the most appropriate first-line eradication therapy for an individual patient is essential. There is no treatment regimen which guarantees cure of *H. pylori* infection in 100% of patients. Indeed, there are currently few, if any regimens which consistently achieve eradication rates exceeding 90% [16, 17].

Material and methods

The study was conducted with both out patients and in-patients of All India Institute of Medical Sciences, Patna, Bihar. The study conducted was a cross-sectional prospective study, Study duration of two years. The upper gastro-intestinal endoscopy was done with flexible, fibro-optic endoscope with patients in left lateral positions. Two endoscopic biopsy fragments were obtained from each patient from the antrum. One biopsy fragment was sent to histopathology.

The upper gastro-intestinal endoscopy was done with flexible, fibro-optic endoscope with patients in left lateral positions. The patients were taken for upper gastrointestinal endoscopy after making them fast overnight. The cases admitted with gastric outlet obstruction were given stomach wash the night before and the morning on the day on which the procedure scheduled. Two endoscopic biopsy fragments were obtained from each patient from the antrum. One biopsy fragment was sent to histopathology department in formalin container. Two sections were cut from each block and mounted on two slides, one each on a slide. Slides were stained with normal H and E, stain and Giemsa stain. In positive cases, *H. pylori* appeared as light bluish rods in H and E stained slides with varying sizes on the luminal surface of mucosal cells. In Giemsa stain, *H. pylori* appeared dark blue in a light background. Another biopsy fragment was sent to microbiology department for Rapid urease test. Positive test for *Helicobacter pylori* was indicated by change in colour of the medium from yellow to pink or red. The case was considered as positive when histopathological examination by Giemsa stain was positive.

INCLUSION CRITERIA

Patients undergoing upper GI Endoscopy at AIIMS, Patna.

EXCLUSION CRITERIA

Patients who have already taken anti *H. pylori* medication, Patients who are unwilling for biopsy.

The Descriptives procedure displays univariate summary statistics for several variables in a single table and calculates standardized values (z scores). Variables can be ordered by the size of their means (in ascending or descending order), alphabetically, or by the order in which the researcher specifies.

Results

Prevalence of *H. pylori*

H PYLORI		Frequency	Percent
	Present	54	45.0
	Absent	66	55.0
	Total	120	100.0

In the study, of the total 120 cases, 54 were positive for H.pylori. The prevalence was found to be 45%. , there was one patient below 20 years, 12 patients between 21-30, 29 patients between 31-40, 32 patients between 41-50, 23 patients between 51-60, 14 patients between 61-70, 9 patients above 71 years. Majority of the patients were between the age of 41-50 years. antral gastritis was present in 55 cases, it was normal in 12 cases, diffuse gastritis was present in 10 cases, esophagitis was present in 10 cases, Ca stomach in 10 cases, gastric ulcer in 5 cases, gastric polyp in 4 cases, hiatus hernia in 4 cases, Ca esophagus in 3 cases, PHG in 2 cases, Duodenitis in 1 case, biliary gastritis in 2 cases, duodenal ulcer in 1 case and esophageal varices in 1 case. Antral gastritis was the most common endoscopic finding. It was followed by diffuse gastritis and esophagitis.

In the study, chronic gastritis was present in 86 patients, Adenocarcinoma- Diffuse Type was present in 4 cases, Adenocarcinoma-Intestinal Type was present in 6 cases, Adenocarcinoma-Indeterminate Type was present in 2 cases, Gastric Polyp was present in 4 cases, Gastric Ulcer was present in 2 cases, Intramucosal Carcinoma was present in 2 cases, Normal in 8 cases. Chronic gastritis was the most common histopathological finding.

Prevalence according to Age

Ages	GEIMSA		PREVALENCE	Total
	Present	Absent		
<20y	1	0	100%	1
21-30y	4	8	33.3%	12
31-40y	9	20	31%	29
41-50y	15	17	47%	32
51-60y	11	12	48%	23
61-70y	9	5	64%	14
71 above	5	4	55%	9
Total	54	66	45%	120

		Value	Approx. Sig.
Nominal by Nominal	Phi	.238	.340
	Cramer's V	.238	.340
N of Valid Cases		120	

In the study, excluding the <20year group which had only one sample, the highest prevalence was found in the age group between 61-70 which was 64%, followed by 55% in 71 and above, 48% in 51-60, 47% in 41-50, 33.3% in 21-30 years. As the p value is .304, there is no statistically significant association with age group and H.pylori infection. In the study, the prevalence was highest in gastric polyp and duodenitis, which was 100%, it was followed by 80% in Gastric ulcer, 70% in Ca stomach, 60% in esophagitis, 50% in PHG, 50% in Biliary Gastritis, 47% in antral gastritis, 33% in caesophagus. None of the hiatus hernia, esophageal varices, duodenal ulcer cases were positive for H.pylori. As the p value is .009, there is statistically significant association with endoscopic findings and H.pylori infection. the prevalence was highest in gastric polyp and gastric ulcer, which was 100%, it was followed by 66.7% in adenocarcinoma, 57% in dysplasia, 40% in chronic gastritis, 12.5% in normal cases. As the p value is .014, there is statistically significant association with histopathological findings and H.pylori infection.

Discussion

Prevalence of H.pylori- Comparison with other studies

STUDY	SAMPLE SIZE	TOTAL POSITIVE	PREVALENCE
Adlekha	530	329	62%
Singh	147	87	59%
Bapat	96	50	55%
Rastogi	208	92	44%
Present	120	54	45%

According to a metaanalysis conducted by James K.Y. Hooi, Wan Ying La et al, Regions with the highest reported HP prevalence were Africa (70.1%; 95% CI, 62.6%–77.6%), South America (69.4%; 95% CI, 63.9%–74.9%), and Western Asia (66.6%; 95% CI, 56.1%–77.0%). Regions with the lowest reported HP prevalence were Oceania (24.4%; 95% CI, 18.5%–30.4%), Western Europe (34.3%; 95% CI, 31.3%–37.2%), and Northern America (37.1%; 95% CI, 32.3%–41.9%)^[18]. Several studies have reported different prevalence rate of *H. pylori* infection between countries, which might be due to diverse contributing factors including socioeconomic status, geographical or living conditions, as well as ethnicity or location of each population. The prevalence of *H. pylori* has declined in recent years, which might be related to the human host factors as well as socioeconomic and hygiene factors. It appears that acquisition and transmission of *H. pylori* can be prevented to a large extent by following improved hygienic practices and standard of living^[169]. In a study conducted by Breken.RK in Norway in 2016, *H. pylori* infection was nearly undetectable (0.6%) among the children, whereas the prevalence increased from 20% in adolescents toward a peak of 45% in the highest age group^[20]

In the study conducted by Adlekha et al in Kerala in 2013, no statistically significant difference in age related distribution.^[21] In the study conducted by Ezeigbo. R. Obiagali in Nairobi in 2016, the highest prevalence for *H. pylori* infection and peptic ulcer occurred within the age group 38-47 years with 56.2% and 49.3% respectively, while ages 18-27 years had the least prevalence for both infections.^[22] the prevalence was highest in gastric polyp and duodenitis, which was 100%, it was followed by 80% in Gastric ulcer, 70% in Ca stomach, 60% in esophagitis, 50% in PHG, 50% in Biliary Gastritis, 47% in antral gastritis, 33% in ca esophagus. None of the hiatus hernia, esophageal varices, duodenal ulcer cases were positive for *H.pylori*. As the p value is .009, there is statistically significant association with endoscopic findings and *H.pylori* infection. In the study conducted by Adlekha et al in Kerala in 2013, The most common endoscopic abnormality was gastritis 69%, followed by duodenitis –16.9%, esophagitis –11.9%, duodenogastric reflux –6.9%, hiatal hernia –6%, gastric ulcer (GU) –2%, duodenal ulcer (DU) –2% and Barrett’s esophagus 2%. The commonest identifiable lesion at endoscopy was gastritis (69%). The correlation of endoscopic abnormality with *H. pylori* infection was statistically highly significant with a P < In the study conducted by Adlekha et al in Kerala in 2013, histopathological features when analyzed, 10.5% (56/530) patients were found to have reactive gastritis. *H. pylori* positivity was seen in 65.9% (292/443) patients with diagnosis of gastritis. Normal gastric mucosa was evident in 8.4% (45/530) cases. Histological features such as intestinal metaplasia and glandular atrophy were seen in 8.1% (36/443) and

18.7% (83/443) respectively in patients with gastritis, whereas these entities were seen in 57.1% (12/21) and 71.4% (15/21) of cases respectively in patients with dysplasia/cancer. The correlation of intestinal metaplasia with *H. pylori* infection was statistically non-significant, with P value of 0.58. In the study conducted by P. Sharma in Kashmir in 2015, chronic gastritis was present in 89 patients with 50.5% positive for *H. pylori*, Duodenitis in 16 patients (68.7% positive), Gastric ulcer in 4 patients (50% positive), Duodenal ulcer in 5 (80% positive), Adenocarcinoma in 5 (40% positive), normal in 5 patients (nonpositive).^[23] In the present study, chronic gastritis was present in 87 patients with 40% positive for *H. pylori*, Dysplasia in 7 patients (57% positive), Gastric ulcer in 2 patients (100% positive), Gastric polyp in 4 (100% positive), Adenocarcinoma in 12 (66.7% positive), Normal in 7 cases (12.5% positive). There is statistically significant association of *H. pylori* with gastric ulcer, gastric polyp, adenocarcinoma, dysplasia. It corroborates the findings of other studies as well. Compared to other studies the prevalence of *H. pylori* in Chronic Gastritis is slightly less in the present study. In the study conducted by S. Redén in Sweden in 2011, the sensitivity was 90% and specificity was 98%, Positive Predictive Value (PPV)- 96% , Negative Predictive Value (NPV)- 95%.^[24]

Conclusion

The prevalence of *H. pylori* was found to be 45%. There is recent trend of decrease in the prevalence of *H. pylori*, which might be related to the human host factors as well as socioeconomic and hygiene factors. It appears that acquisition and transmission of *H. pylori* can be prevented to a large extent by following improved hygienic practices and standard of living. Present study corroborates these findings as there is improvement in the socioeconomic status, living conditions, as India strives on the path of being a developed country.

References

1. Brunicaudi FC. Schwartz's Principles of Surgery, McGraw-Hill, 2015: Schwartz's Principles of Surgery. Bukupedia; 2015 Aug 15.
2. Townsend CM, Beauchamp RD, Evers BM, Mattox KL. Sabiston Textbook of Surgery E-Book. Elsevier Health Sciences; 2016 Apr 22.
3. Brown LM. Helicobacter pylori: epidemiology and routes of transmission. Epidemiol Rev 2000; 22: 283–97.
4. Jones KR, Whitmire JM, Merrell DS. A tale of two toxins: Helicobacter pylori CagA and VacA modulate host pathways that impact disease. Front Microbiol. 2010;1:115.
5. Park, JeongBae, and JaSeol Koo. "Helicobacter Pylori Infection in Gastric Mucosa-Associated Lymphoid Tissue Lymphoma." World Journal of Gastroenterology < : WJG 20.11 (2014): 2751–2759.
6. Thirumurthi S, Graham DY.. Helicobacter pylori infection in India from a western perspective. Indian J Med Res (2012) 136(4):549.
7. Ponsky JL. Endoluminal surgery: past, present and future. Surg Endosc. 2006;20(Suppl 2):S500–S502.
8. Dixon MF, Genta RM, Yardley JH, Correa P. Classification and grading of gastritis. The updated Sydney System. International Workshop on the Histopathology of Gastritis, Houston 1994. Am J Surg Pathol. 1996;20(10):1161- 1181
9. Marshall B, Warren JR. Unidentified curved bacilli in the stomach of patients with gastritis and peptic ulceration. Lancet. 1984;323 (8390):1311-1315.
10. Chey WD, Wong BC. American College of Gastroenterology guideline on the management of Helicobacter pylori infection. Am J Gastroenterol. 2007;102(8):1808-1825

11. Jabbari M, Cherry R, Lough JO, Daly DS, Kinnear DG, Goresky CA. Gastric antral vascular ectasia: the watermelon stomach. *Gastroenterology*. 1984;87(5):1165-1170.
12. Carmack SW, Genta RM, Schuler CM, Saboorian MH. The current spectrum of gastric polyps: a 1-year national study of over 120,000. patients. *Am J Gastroenterol*. 2009;104(6):1524-1532.
13. Genta RM, Schuler CM, Robiou CI, Lash RH. No association between gastric fundic gland polyps and gastrointestinal neoplasia in a study of over 100,000 patients. *Clin Gastroenterol Hepatol*. 2009;7(8):849-854.
14. Jain R, Chetty R. Gastric hyperplastic polyps: a review. *Dig Dis Sci*. 2009;54(9):1839-1846
15. Hiki N, Yamamoto Y, Fukunaga T, et al. Laparoscopic and endoscopic cooperative surgery for gastrointestinal stromal tumor dissection. *Surg Endosc*. 2008;22(7):1729-1735.
16. Ferguson DA, Jiang C, Chi DS, Laffan JJ, Li C, Thomas E. Evaluation of two string tests for obtaining gastric juice for culture, nested PCR detection and combined single and double stranded conformational polymorphism discrimination of *Helicobacter pylori*. *Dig Dis Sci* 1999; 44: 2056–62.
17. Li BZ, Threapleton DE, Wang JY et al. Comparative effectiveness and tolerance of treatments for *Helicobacter pylori* : systematic review and network meta-analysis. *BMJ* 2015;351: h452 .
18. Hooi J.K.Y., Lai W.Y., Ng W.K., Suen M.M.Y., Underwood F.E., Tanyingoh D., Malfertheiner P, Global Prevalence of *Helicobacter pylori* Infection: Systematic Review and Meta-Analysis. *Ng S.C. (2017) Gastroenterology*, 153 (2) , pp. 420-429
19. Bastos J, Peleteiro B, Barros R, Alves L, Severo M, de Fatima Pina M, et al.
20. Sociodemographic determinants of prevalence and incidence of *Helicobacter pylori* infection in Portuguese adults. *Helicobacter*. 2013;18:413–22.
21. Breckan RK, Paulssen EJ, Asfeldt AM, Kvamme JM, Straume B, Florholmen J. The All-Age Prevalence of *Helicobacter pylori* Infection and Potential Transmission Routes. A Population-Based Study. *Helicobacter*. 2016;21:586– 595.
22. Adlekha S, Chadha T, Krishnan P, Sumangala B. Prevalence of *Helicobacter pylori* infection among patients undergoing upper gastrointestinal endoscopy in a medical college hospital in Kerala, India. *Ann Med Health Sci Res*. 2013;3(4):559-63.
23. Ezeigbo, R. Obiageli and Ezeigbo C. Ivan. Prevalence of *Helicobacter pylori* and its associated Peptic Ulcer Infection among Adult Residents of Aba, Southeastern, Nigeria. *Int.J.Curr.Microbiol.App.Sci* (2016) 5(6): 16-21
24. Sharma P et al. Histopathological Spectrum of various gastroduodenal lesions in North India and prevalence of *Helicobacter pylori* infection in these lesions: a prospective study. *Int J Res Med Sci*. 2015 May;3(5):1236-1241
25. Redéen S, Petersson F, Törnkrantz E, Levander H, Mårdh E, Borch K. Reliability of Diagnostic Tests for *Helicobacter pylori* Infection. *Gastroenterol Res Pract*. 2011;2011:940650