European Journal of Molecular & Clinical Medicine (EJMCM)
ISSN: 2515-8260 Volume 09, Issue 03, 2022

Original research article

To Study Prevalance of Helicobacter PyloriInfection in Cases of Dyspepsia

Dr. Pankaj Kumar[1], Dr. Sanjeev Bharti[2], Dr. Shivendu[3], Dr. Rajeev Kumar[4]

[1]Senior Resident, Department of Medicine, AIIMS New Delhi

[2]Senior Resident, Department of Medicine, AIIMS New Delhi

[3]Senior Resident, Department of Medicine, AIIMS New Delhi

[4]Senior Resident, Department of Medicine, AIIMS Patna

Corresponding Author: Dr. Pankaj Kumar

Abstract

Background: Dyspepsia is a chronic or recurrent pain or discomfort centered in the upper abdomen; patients with predominant or frequent (more than once a week) heartburn or acid regurgitation, should be considered to have gastro esophageal reflux disease (GERD) until proven otherwise.[(1)] Helicobacter pylori, a curved rod shaped bacterium, has been consistently associated with patients having acid peptic diseases, which plays a major role in its etiopathogenesis.[2]

Objectives: To estimate the prevalence of Helicobacter pylori infection in patients with dyspepsia undergoing upper gastrointestinal endoscopy.

Methods: Hundred cases of dyspepsia, studied clinically as per the proforma from August 2019 to June 2022, were subjected to upper gastro-intestinal endoscopy under topical anaesthesia, during which biopsies, from the pathological areas were taken. Biopsy specimens, were immediately inoculated into freshly prepared urea broth containing phenol red as the indicator. Positive test for Helicobacter pylori was indicated by change in colourof the medium from yellow to pink or red. The other biopsy specimens were sent for routine histopathology and special staining with Giemsa stain.

Conclusion: H.Pylori is consistently associated with patients of acid peptic disease andhas a high prevalence in cases of ulcer dyspepsia than non-ulcer dyspepsia.

Keywords: Dyspepsia; Helicobacter pylori; Urease;Giemsa; peptic ulcer.

Introduction

Dyspepsia is a chronic or recurrent pain or discomfort centered in the upper abdomen;patients with predominant or frequent (more than once a week) heartburn or acid regurgitation, should be considered to have gastro esophageal reflux disease (GERD) until proven otherwise.[1] Helicobacter pylori, a curved rod shaped bacterium, has been consistently associated with patients having acid peptic diseases, which plays a major role in its etiopathogenesis and high incidence of morbidity.[2] The increased risk of H. pylori infection in Chinese and Indians points

to either an inherent genetic predisposition or to socio-cultural practices peculiar to the particular race which may be responsible for transmission of the infection.[3] Several studies have revealed the association of Helicobacter pylori in 70-75 percent of patients with dyspepsia. Endoscopic studies have shown that, Helicobacter pylori isfound in 80-100 percent of patients with duodenal ulcers and 60-75 percent of patientswith gastric ulcers.[2-4] Amidst these profound variations proposed by different studies, and different etiologies for dyspepsia like NSAIDS induced, stress induced, H.pylori infection is also another important risk factor for dyspepsia. Published studies show conflicting results about the prevalence of H.pylori infection among Indian patients and of other countries due to link between genetic predisposition and hygiene and sanity in a developing country like us; therefore we have attempted to study the prevalence of H.pylori infection in dyspepsia.

Objectives

To estimate the prevalence of Helicobacter pylori infection in patients with dyspepsia undergoing upper gastrointestinal endoscopy, To determine the association of Helicobacter pylori infection with acid pepticdiseases.

Review of Literature

H. pylori strains are genetically highly diverse, and certain markers of virulence in H. pylori have been identified. It is likely that most individuals in the community have infection with avirulent strains of H. pylori, which may in fact be beneficial to the host. For example, heightened gastric acidity produced by many H. pylori may act as a barrier to ingested pathogens. They may produce as yet unknown factors that may stimulate innate immune pathways in the host and protect against other pathogens. They may also exert biochemical effects on the host that are yet to be elucidated.[7] Infection with the organism is most common in populations with poor sanitary and hygiene conditions; in developed societies with better sanitation and hygiene, the levels of infection are lower. Associated with this increasing level of hygiene, there is a higher incidence of allergic and autoimmune diseases including asthma and Crohn"sdisease. It is possible therefore that H. pylori infection may protect populations in countries such as India from the allergic and autoimmune diseases that are increasingly prevalent in the developed world.[7] H.pylori infection is associated with dyspeptic symptoms. For nearly 45 years gastric spiral bacteria were repeatedly observed and then forgottenbut in 1983 Barry Marshal and Robin Warren isolated Helicobacter pylori from gastric biopsies in Perth.[8-9] Freedburg and Barron in 1940 stated that "spirochaetes" could be found in upto 37% of gastrectomy specimens but they failed to confirm these findings in gastric suction biopsies.[10] In 1975, Steer and Colin Jones, observed gram-negative bacilli in 80% of patientswith gastric ulcer that suggested, the bacteria might cause a reduction in gastric mucosal resistance which predisposes to ulceration.[11]

But cause and effect relationship has not yet been established beyond doubt. Seroprevalence of H. pylori is high in developing countries such as India. 80% of Indian adults have antibodies against H.pylori in their sera. However, even subjects without dyspeptic symptomsare frequently positive to H. pylori antibody. Many randomized controls trials evaluated effect of H. pylori eradication on symptoms of dyspepsia. Most studies, however, showed conflicting results.[12] Understanding the route of H. pylori transmission is important if public health measures to prevent its spread are to be implemented. Iatrogenic transmission of H. pylori following endoscopy is the only proven mode. For the general population, the most likely mode of transmission is from person to person, by either the oral-oral route (through vomitus or possibly saliva) or perhaps the fecal-oral route. The person-to-person mode of transmission is supported by the higher incidence of infection among institutionalized children and adults and

the clustering of H. pylori infection within families. Also lending support to this concept is the detection of H. pylori DNA in vomitus, saliva, dental plaque, gastric juice, and feces. Waterborne transmission, probably due to fecal contamination, may be an important source of infection, especially in parts of the world in which untreated water is common. Recentstudies in the United States have linked clinical H. pylori infection with consumption of H. pylori-contaminated well water.[13] Overall, inadequate sanitation practices, low social class, and crowded or high-densityliving conditions seem to be related to a higher prevalence of H. pylori infection.

Material and methods

All the patients presenting to Medicine Department and Medical Gastroenterology department at All India institute of medical sciences, New Delhi. Study duration Period, August 2019 T0 June 2022. Hundred Cases of dyspepsia who attend the Medicine OPD were asked for participatingin the study. Informed written consent was taken from all the subjects. A pre structured proforma was used to collect the baseline data.

Inclusion Criteria

Patients between 18 to 60 years of age, Patients who have chronic upper abdomen pain.
Patients having symptoms of dyspepsia such as early satiety, postprandial fullness,burning sensation in chest, Patients diagnosed aschronic gastritis, gastricor duodenal ulcerson Gastroduodenoscopy.

Exclusion Criteria

Pregnant and lactating women, Patients on proton pump inhibitors, Patients who are a known case of chronic pancreatitis, Patients who are on NSAIDS for greater than one month of duration, Patients who have received Anti H. pylori treatment, Patients with esophageal growths on endoscopy.
After applying the inclusion and exclusion criteria, all the patients underwent upper gastro-intestinal endoscopy. According to the endoscopy findings, the patients were divided into following groups.
Non ulcer dyspepsia:
Normal study, Gastritis / Duodenitis
Ulcer dyspepsia:
Duodenal ulcer, Gastric ulcer, Carcinoma stomach

Procedure

All the patients in this study group, both inpatient and outpatient underwent upper gastro-intestinal endoscopy under topical anesthesia. The patients asked to fast for 12 hours prior to the procedure. Only a few patients were given 5 to 10 mg diazepam intravenously forsedation depending on the preference of the consultants. The upper gastro-intestinal endoscopy was conducted with pentax 29P flexible, fibro optic endoscope with patients in left lateral positions. Two biopsy specimens, one of the antral area and the other of the pathological finding were immediately inoculated into freshly prepared urea broth containing phenol red as the indicator. Positive test for helicobacter pylori was indicated by change in colour of the medium from yellow to pink or red. The test was read as strongly positive when the change incolour occurred within 5-15 minutes following inoculation and weakly positive when the colour change occurred in first 6 hours. Any colour change in between was read as intermediate.

Results

Out of 100 patients, there were 75 male patients and 25 female patients, age ranging from 18 to 60 years (mean 39.25). Out of 100 patients, 68 patients were diagnosed to have infected with helicobacter pylori (68%). All these patients presented to our hospital with upper abdominal pain or discomfort. Patients presented with nausea and vomiting were 94, out of which 65 patients were positive for H.Pylori infection, 15 patients had Hematemesis, out of which 12 patients were infected for H.Pylori infection; 8 patients had history of weight loss and 7 patients were positive for H.Pylori. On examination 6 patients were anaemic, out of which 5 were positive for H.Pylori; 42 patients had Epigastric tenderness on palpation and 3 were having mass per abdomen, out of which 35 & 2 patients were positive for H.Pylori respectively.

Table 1: Age distribution of the study participants (N=100)

Age categories	Number of participants	Mean age (in years)	Standard Deviation
18-30 years	27	25.44	3.31
31-40 years	27	35.93	2.48
41-50 years	31	46.45	2.63
51-60 years	16	55.2	3.49

Table 2:Prevalence of H. pylori infection among the study participants across age categories(N=100)

	H. pylori present		H. pylori absent		Total	
Age categories	**N**	**%**	**N**	**%**	**N**	**%**
18-30 years	16	(23.5)	11	(34.4)	27	(27.0)
31-40 years	18	(26.5)	9	(28.1)	27	(27.0)
41-50 years	22	(32.4)	9	(28.1)	31	(31.0)
51-60 years	12	(17.6)	3	(9.4)	15	(15.0)
Total	68	(100.0)	32	(100.0)	100	

Table 3: Distribution of H. pylori infection based on type of endoscopic findings (N=100)

	H. pylori present		H. pylori absent		Total	
Endoscopicfinding	**N**	**%**	**N**	**%**	**N**	**%**
Ulcer dyspepsia	51	(85.0)	9	(15.0)	60	(100.0)
Non ulcerdyspepsia	17	(42.5)	23	(57.5)	40	(100.0)
Total	68	(68.0)	32	(32.0)	100	

Total patients presenting with ulcer dyspepsia were 60 in which 51 patients (85%) were positive for H.pylori and out of 40 patients with non-ulcer dyspepsia 17 patients (42.5%) wre positive for H.pylori.

Table 4: Type of endoscopic findings among patients with dyspepsia based on gender (N=100)

Endoscopic findings	Male		Female		Total	
	N	**%**	**N**	**%**	**N**	**%**
Ulcer dyspepsia	41	(68.3)	19	(31.7)	60	(100.0)
Nonulcer dyspepsia	34	(85.0)	6	(15.0)	40	(100.0)
Total	75	(75.0)	25	(25.0)	100	

Twenty eight patients (90.3%) were positive for H.pylori out of 31 patients having duodenal ulcer. On Chi square test, χ^2=11.87, p<0.01, hence there is a significant association of

Helicobacter pylori with duodenal ulcers, Seventeen patients (77.3%) were positive for H.pylori out of 22 patients, having gastric ulcer. On Chi square test, $\chi^2 = 1.11$, $p>0.01$, hence there is no significance. Six (85.7%) out of 7 patients of carcinoma stomach were found positive for H.pylori, $p>0.01$, hence there is no significant association of H.pylori with carcinoma stomach. Nine (52.9%) out of 17 patients of gastritis were found positive for H.pylori, Chi square test, $\chi^2=2.13$, $p>0.01$, hence there is no significant association of H.pylori with gastritis, Eight (66.7%) out of 12 patients of duodenitis were found positive for H.pylori, Chi square test, $\chi^2=2.13$, $p>0.01$, hence there is no significantassociation of H.pylori with duodenitis

Table 5: Comparison of Rapid Urease Test with Histo-Pathological Examination for the diagnosis of H. pylori infection

	Histo-Pathological Examination				Total	
	H. pylori present		H.pylori absent			
	N	%	N	%	N	%
H. pylori present by RUT	35	(51.5)	0	(0.0)	35	(35.0)
H. pylori absent by RUT	33	(48.5)	32	(100.0)	65	(65.0)
Total	68	(100.0)	32	(100.0)	100	

Sensitivity of RUT: 51. 7%, Specificity of RUT: 100%

Discussion

After the discovery of Helicobacter Pylori by Marshall and Warren in 1983, many studies were conducted to confirm the association of Helicobacter Pylori with various acid-peptic diseases and carcinoma stomach. The following observations were made:

The treatment of H.Pylori led to the reversal of gastritis in patients with chronic nonspecific gastritis. The eradication of H.Pylori decreases the relapse of ulcer dyspepsia to 1-3% when compared to 80% relapses in patients with persistent H.Pylori infections after medical management. In spite of the above findings, the cause and the effect relationship between H.Pylori and peptic ulcer disease is not proved and furthermore many people infected with H.Pylori did not develop peptic ulceration. The association of H.Pylori with non-ulcer dyspepsia is controversial. Therapeutictrails in non-ulcer dyspepsia patients with H.Pylori infections produced conflictingresults. Thus at this stage in the history of acid-peptic disease and its association with H.Pylori, the causation or association between the two is still unclear.Thus we at the „ All india institute of medical sciences New Delhi. ‟ have made a sincere attempt to explore the possibility of association between H.Pylori and ulcer dyspepsia and its contribution to non- ulcer dyspepsia. Marshall and Warren (1984) observed that 18 out of 22 patients with gastric ulcer and all the 13 patients with duodenal ulcers were positive for H.Pylori. In 59 patients with gastro/duodenitis, 32 were positive for H.Pylori. In patients with normal upper G.I endoscopy, 8 out of 16 were positive for H.Pylori.[8] In their study of 180 patients, Von Wulfen et al (1986), found an overall positivity in 98 patients. They observed that in patients with duodenal ulcers,45 out of 54 patients showed H.Pylori, while 13 out of 18 patients with gastric ulcers showed H.Pylori. 79 out of 127 patients with gastritis/duodenitis were positive for H.Pylori.[14] Three studies (Vaira et al, 1994, Sobala et al 1991, Patel et al, 1994) were generally similar in design. Combining the studies provided a much larger sample of 631 patients, in which overall 351 patients were positive for H.Pylori. In these studies, high association of H.Pylori was found out with Duodenal Ulcer.[15] In the present study, the overall positivity of H.Pylori was 68 out of 100 patients. The incidence of H.Pylori is higher in patients

with ulcer dyspepsia when compared to patients with non-ulcer dyspepsia. This result is comparable to those of other studies. Patients of duodenal ulcer were found to be positive for H.pylori were 90.3% and on chi square testing p value was significant which showed that H.pylori infection is highly associated with duodenal ulcers when compared to gastric ulcer. This might be because of the hyperacidic conditions of the duodenum which offers a favorable environment for the organism to thrive. In 68 patients positive for H.pylori; 51 (85%) patients had ulcer dyspepsia and the other patients with non-ulcer dyspepsia were 17 (42.5%). The development of ulcers in these patients may be because of infection with virulent strains of H.pylori.in non- ulcer dyspepsia patients with duodenitis had high prevalenceof dyspepsia comapared to patients with gastritis and normal study.it was also observed that male patients were more affected with H.pylori positive ulcer dyspepsia compared to feamale patients; this might be because of increased smoking habits, decresed hygiene and sanitation habits. In our study rapid urease test had sensitivity of 51.7% and specificity of 100% when compared with histopathology

Conclusion

This was a cross sectional study conducted to determine the role of H.Pylori in acid- peptic diseases. This study design was based on clinical study and endoscopic biopsy of gastric mucosa (and duodenal mucosa whenever necessary) in 100 patients with a history of dyspepsia. Endoscopy confirmed the diagnosis. Rapid urease test and Giemsa staining were conducted on endoscopy biopsy specimens and H.Pylori positivity was based on either Rapid urease test and/or histopathological examination was positive.

References

1. Nicholas J. Talley, Nimish Vakil. Guidelines for the Management of Dyspepsia and the Practice Parameters. American Journal of Gastroenterology. 2005. 100: 2324-2337.
2. Freston J W . “Helicobacter pylori negative peptic ulcers: frequency and implications for management”. Journal of Gastroenterology . 2000. 35(12): 29- 32.
3. Goh K L. Prevalence of and risk factors for Helico bacter pylori infection in a multi-racial dyspeptic Malaysian population undergoing endoscopy. J Gastroenterology Hepatology. 1997 Jun. 12(6): 29-35.
4. Kidd M, Louw J A et al. “Helicobacter pylori in Africa: Observations on anEnigma within an enigma“. Journal of Gastroenterology & Hepatology, 1999 Sept. 14(9): 851-8.
5. Jain A, Buddhiraja S et al. “Risk factors for duodenal ulcers in North India”.Tropical Gastroenterology, 1999 Jan-Mar. 20(1): 36-39.
6. Perri F, Festa V et al. “Dyspepsia and Helicobacter pylori infection: a prospective multicentric observational study.” Digestive and Liver Disease, 2 Mar. 35(3): 157-64.
7. B S Ramakrishna. Helicobacter pylori infection in India: The case against eradication. Department of Gastrointestinal Sciences, Christian Medical College, Vellore. Indian Journal of Gastroenterology. 2006. Volume 25. 25-28.
8. Marshall B J, Warren J R. “Unidentified curved bacilli in the stomach of patients with Gastritis and peptic ulceration.” TheLancet, 1984 Jun16. 1311-15.
9. Fung W P, Papadimitriou J M, Matz L R. “Endoscopic, histological and ultrastructural correlations in chronic gastritis.” Am. J. Gastroenterology, 1979. 71: 269-79.
10. Freedburg A S, Barron L E. “The presence of spirochaetes in human gastric mucosa.”Am. J. Dig Dis, 1940. 7: 443-45.
11. Steer H W, Colin-Jones D G. “Mucosal changes in gastric ulceration and their response to carbenoxolone sodium.” Gut, 1975. 16: 590-97.
12. Ghoshal U C, Tiwari S, Dhingra S, Pandey R, et al. Frequency of Helicobacter pylori and CagA antibody in patients with gastric neoplasms and controls. The Indian Enigma. 2008

May. 53(5): 1215-22.
13. 13.Brown LM "Helicobacter pylori: epidemiology and routes of transmission". Epidemiology Rev. 2000. 22 (2): 283-97.
14. WulfenVon, Heeseman J, Butzow et al. "Detection of C.pyloridis in patients with antral gastritis and peptic ulcers by culture, compliment fixation test and immune blot."Journal of Clin Microbiology, 1986. 24:716-19.
15. Moore R A. "Helicobacter Pylori and Peptic Ulcer: A systematic review of effectiveness and an overview of the economic benefits of implementing what is known to be effective." Pain Research, The Churchill Headington Oxford.1994 Dec.