Original Article

A prospective study of helicobacter pylori in gastroduodenal ulcer and gastric disease

S Abijith Sudhakar Shetty[1], S Poovarasan[2*], M S Moosabba[3]

[1]Assistant Professor, [2]Post Graduate, [3]Professor and HOD, Department of General Surgery, Yenepoya Medical College, Deralakatte, Mangalore-575018, Tamil Nadu, INDIA.
Email: arsan.9595@gmail.com

Abstract

Background and Objective: Dyspepsia is an extremely common symptom with prevalence in the community of approximately 30%. Approximately 50% of world's population is estimated to be infected with H. pylori. The prevalence of H. pylori is higher in developing than in developed countries. H. pylori prevalence differs from one country to another and may differ between different ethnic, social, or age groups within the same country. **Objective:** 1. To study the presence of H.pylori infection in patients with dyspepsia 2. Relationship between ulcer / non ulcer dyspepsia and H.pylori infection. 3. Gastroscopic findings in patients presenting with dyspepsia. **Methods:** A prospective clinical study of 100 patients visiting the Hospital for dyspeptic symptoms meeting the inclusion criteria were included in the study. Detailed history was taken and patients underwent upper GI endoscopy and blood was withdrawn for estimation of haemoglobin, blood group, Rh factor and IgG ELISA for H.pylori. **Results:** 66 males and 34 females participated in the study with mean age of 39.6 years (SD - 12.10). 58% of study population tested positive for H.pylori infection by detection of IgG antibodies against H.pylori in blood by ELISA technique. 37% of the study population had ulcer in stomach or duodenum as detected by upper GI endoscopy. **Interpretation and Conclusion:** Data from this study shows that H.pylori infection is detected in a significant number of patients presenting with dyspeptic symptoms with no other specific risk factors for acid peptic disease (eg: alcohol, smoking, usage of NSAID's).
Key Word: Dyspepsia, H.Pylori, IgG ELISA, Ulcers

[*]**Address for Correspondence:**
Dr. S Poovarasan, 5/318,Ramani,TNHB, Mullai nagar, Mohanur road, Namakkal-637001, Tamil Nadu, INDIA.
Email: arsan.9595@gmail.com
Received Date: 02/10/2015 Revised Date: 29/10/2015 Accepted Date: 22/11/2015

Access this article online

Quick Response Code:

Website: www.statperson.com

DOI: 02 December 2015

INTRODUCTION

Before the discovery of Helicobacter pylori, ulcer disease was considered as the result of a conflict between gastric acid and pepsin, on one side, and protection afforded by gastric mucosal barrier, on the other side. H. pylori is involved in nonulcer dyspepsia, nonsteroidal anti-inflammatory drug ulcers, gastric cancer, MALT lymphoma and, oesophageal adenocarcinoma. The pandemic nature of the H. pylori infection, particularly within developing countries, combined with emerging resistances to antibiotics makes a study like this necessary. The relationships between the human host and the bacterium remains mostly unknown. Dyspepsia is an extremely common symptom with a prevalence in the community of approximately 30%.[1] Dyspepsia is defined as any pain, discomfort, or nausea referable to the upper alimentary tract which may be intermittent or continuous, has been present for one month or more, and is not precipitated by exertion and not relieved within five minutes by rest.[1,2,3.4] H. pylori is a spiral, gram-negative, microaerophilic bacterium. It was established in 1982 ,by Robin Warren and Barry Marshall, as the causative[5,6] agent of gastritis and peptic ulcer a discovery that revolutionized gastroenterology. Before Warren and Marshall, the human stomach was believed to be a sterile area. Helicobacter pylori, formerly known as Campylobacter pyloridis then Campylobacter pylori, is one of the human pathogens with highest prevalence around the world; yet, its exact mode of

How to site this article: S Abijith Sudhakar Shetty, S Poovarasan, M S Moosabba. A prospective study of helicobacter pylori in gastroduodenal ulcer and gastric disease. *International Journal of Recent Trends in Science and Technology*. December 2015; 17(2): 12-16 http://www.statperson.com (accessed 04 December 2015).

transmission is still uncertain. Today, H. pylori is recognized as the most common cause of gastritis, which in turn leads to the development of more gastrointestinal complications such as peptic and duodenal ulcers. Additionally, the organism is classified as a class 1 carcinogen because of its causal relationship to gastric adenocarcinoma, one of the world's deadliest cancers.[7] The growing attention given to *H. pylori* by academicians and clinicians is not surprising since this pathogen colonizes more than half of the world's inhabitants[8], with an evident geographic variation in its epidemiology. Helicobacter pylori is:[9] Gram negative bacilli.Spiral shaped with flagella. Motile. Non-invasive and lives in gastric mucosa. Microaerophilic. Slow growing. In the gastrointestinal tract, the gastric mucosal barrier represents the first line of defense against aggressive factors.[10] Bacteria normally become trapped in the mucus and are excreted in feces. However, urease expression and motility permit H. pylori to survive transiently in an acid environment and to colonize persistently the mucous layer. H. pylori is usually located within the thick mucus layer in close proximity to gastric epithelial cells. A weakening of the mucous barrier by H. pylori, leading in some cases to its collapse, has been proposed as H. pylori possesses a gene that is almost identical to a mucinase gene of Vibrio cholerae. Such mucinase activity may be responsible for the dissolution of the net-like structure of the mucus and the variously sized cave-like clear areas surrounding H. pylori as observed in vivo with electron microscopic technique. However, studies in vitro suggest that the loss of gel structure might also arise from high local pH generated by the urease activity of H. Pylori rather than by mucolytic activity. Furthermore, H. pylori can inhibit the secretory response of mucous cells in vitro, indicating a potential deleterious effect on the quantity of this primary defence mechanism of the gastric mucosa. Several virulence factors for gastric colonization, tissue damage and survival have been identified in H.pylori. Flagella, urease and adhesins are all essential factors for H.pylori to colonize the gastric mucosa. Mutants of H.pylori without flagella or urease are unable to colonize the gastric mucosa in laboratory animals.[11]

MATERIALS AND METHODS

Method f Study: This is a prospective study of patients admitted in Yenepoya Medical College Hospital.

Methodology: Detailed history was taken from the patients. A thorough General Physical Examination and systemic examination was carried out in each patient and entered in a proforma. All the patients underwent upper GI endoscopy and the findings were noted, biopsy was taken from suspicious areas or the ulcer or in normal cases from the antrum. Blood was drawn for estimation of Haemoglobin, Blood grouping, Rh typing and IgG ELISA for H.pylori.

Sample Size: 100 cases

Method of collection of data:

Inclusion Criteria: All patients with upper abdominal pain coming to the department of General Surgery, Yenepoya Medical College Hospital, Derlakatte, Mangalore are included in this study.

Exclusion Criteria: 1. Patients who have been treated previously for H. Pylori. 2. Patients who have undergone gastric or oesophageal surgeries. 3. Patients refusing to partake in this study.

RESULTS

Mean age of the study population is 39.6 years (SD - 12.10). 71% (male - 65.1% and female - 82.4%) of study population were in the age group of 31-60 years. 66% of the study population were males and 34% females. Majority of the patients presented with epigastric pain (77%), followed by abdominal bloating sensation (70%), retro-sternal burning sensation and belching (50% each) and other symptoms present in less than 50% of patients. At an average, each patient presented with 4 dyspeptic symptoms of varying duration of time. 47 % of patients had past history of NSAID use,57% alcohol intake and 45% had history of smoking.Clinically 9 % of the patients had diffuse abdominal tenderness, 74% epigastric / hypochondriac tenderness and 17% diffuse abdominal tenderness. 64.7% of ulcer positive patients (male - 64.3% and female - 66.6%) had gastric pyloric ulcer, followed by gastric body ulcer (27%), duodenal ulcer (21.6%) and gastric fundic ulcer (8.1%). 37 patients (28 males and 9 females) had gastric and duodenal ulcers ($p = 0.12$). 8 patients had ulcers at multiple site (6 males and 2 females). 53.5% patients with H.pylori infection had dyspeptic symptoms for a period of 1-3 years, 31% had symptom duration < 1 year and 15.5% with symptom duration > 3 years. In both males and females with IgG H.pylori positivity, Gastric pyloric ulcer was the commonest site of ulcer (57.1% in males and 66.7% of females). In patient with risk factors of alcoholism, smoking, NSAID's and other drugs. All the three tests were positive in significant number of cases, two test IgG ELISA and urease or histopathology were significant, IgG alone was also significant . The combination of other tests were not significant . IgGELISA, according to our study, is important test for detecting H pylori. the sensitivity does not improve in combination improves in combination with Urease and histopathology or both.

Table 1: Frequency of H pylori positivity

	Number	P Value
IgG ELISA	8	<0.0001
Histopathology	0	1
Urease	0	1
IgG ELISA+Urease	18	<0.0001
IgG ELISA+Histopathology	5	<0.0001
Urease+Histopathology	0	1
IgG ELISA+Urease+Histopathology	27	<0.0001

Table 2: Symptoms and IG elisa+Histopathology+Urease positive patient

Symptoms	No. of patient		Total
	Male	Female	
Epigastric pain	12	8	20
Diffuse abdominal pain	4	1	5
Retrosternal burning sensation	5	9	14
Early satiety	6	5	11
Abdominal bloating	6	2	8
Belching	2	3	5
Anorexia	2	3	5
Nausea	0	2	2
Vomiting	0	2	2
Hematemesis	2	0	2
Malena	2	0	2

Table 3: Symptoms in H.pylori positive patients

Symptom	Male	Female	Total	P value
Epigastric pain	30	16	46	0.228
Diffuse abdominal pain	8	2	10	0.407
Retrosternal burning sensation	12	10	22	0.064
Early satiety	7	6	13	0.182
Abdominal bloating	20	12	32	0.238
Belching	18	9	27	0.724
Anorexia	4	4	8	0.212
Nausea	8	10	18	0.007
Vomiting	1	6	7	<0.001
Hematemesis	1	-	1	0.499
Malena	4	4	8	0.212

DISCUSSION

Dyspepsia (any pain, discomfort, or nausea referable to the upper alimentary tract which may be intermittent or continuous) is common symptom with a prevalence in the community of approximately 30%[1] with a mean work loss of 36 weeks.[12] In 1905 Lord Moynihan declared that most cases of dyspepsia could be diagnosed by the symptoms alone.[13] Recent studies have suggested, however, that patients who present with dyspepsia are often misdiagnosed.[14] In our study, all patients complaining of dyspepsia or other upper abdominal symptoms underwent UGI scopy, and in all cases a search was conducted for H.pylori by biopsy for histopathology and ureas test and blood for IgG ELISA.

This study group consists of 100 patients .The mean age of the study population is 39.6 years with minimum age being 18 years and maximum 60 years. 28 years was the median age of individuals included in a similar study conducted by Katelaris PH *et al.*[15] in India with almost the same exclusion. In our study epigastric pain(77%), abdominal bloating (70%) and belching(50%) were the most common symptoms for hospital visits. Many of them were on NSAID's (47%) or other drugs(22%) or have had other risk factors like alcoholism(57%), smoking(45%) etc. In our study group, the main symptoms noted were epigastric pain 77% followed by abdominal bloating sensation (70%), retrosternal burning sensation and belching (50% each) and other symptoms were present in less than 50% of patients. In comparison with GP Crean *et al.*[12] in his study noted; Epigastric pain was recorded by more than half the patients (755),and reported by 60-70 % of all patients with gastric ulcer disease, duodenal ulcer disease, gastric carcinoma, and gall stone disease, 40-50% of those with functional dyspepsia, alcohol related dyspepsia or oesophageal disease and by about 20% of patients with IBS or organic disease. A similar finding was noted by JC Horrocks *et al.*[16] wherein he noted most patients suffered from epigastric pain (286 / 360 patients - 79.4%).

Comparison with J C Horrocks

Sign JC Horrocks	Our study (%)	JC Horrocks *et al.* (%)[16]
Diffuse Abdominal Tenderness	7	1
Epigastric / Hypochondriac Tenderness	74	78
Periumbilical Tenderness	10	4
No Tenderness	9	15.2

Most patients in our study group had dyspeptic symptoms for a period of 1-3 years (47%), followed in by less than 1 year (30%) of the patients and more than 3 years (23%) of the patients. In comparison a study conducted by GP Crean *et al.*[12] considered duration of dyspeptic symptoms for more than 3 years to be a common finding. 66% of our study group comprised of males and 34% females. In our study H.pylori was directly observed by histopathology and indirectly inferred by urease and IgG ELISA. As evidenced by histopathology H.pylori was seen in 38 cases, by urease test 45 cases and ELISA IgG in 58 cases. In 27 patient all three test were positive. All patient with urease test positive were ELISA IgG positive. Only histopathology positive was not seen. ELISA and histopathology positive and urease negative was noted in 5 cases. The statistically significant tests to detect H pylori positivity are Ig ELISA alone, or in combination with urease or histopathology, Urease and

histopathology together were insensitive ($p < 0.05$). In comparison with Manjunath SM,*et al* conducted a study to estimate the prevalence of H.pylori in various gastroduodenal diseases and compared the sensitivity of IgG anti H.Pylori (ELISA) with rapid urease test at LTM Medical College, Mumbai and found that estimation of IgG anti H.pylori antibody is more sensitive than Rapid Urease Test (RUT) in detecting H.pylori infection and the combination of two tests does not increase the sensitivity significantly.[17] The positivity of different test had no significance to the age and gender of the patient. Highest incidence of H.pylori positivity in this study population was found in the age group of 46-60 years in males (40%) and 31 - 45 years in female (44.4%). The duration of symptoms did not vary with the sensitivity of different test, 53.5% patients with H.pylori infection had dyspeptic symptoms for a period of 1-3 years, 31% had symptom duration < 1 year and 15.5% with symptom duration > 3 years. In patient with risk factors of alcoholic, smoking, NSAID's and other drugs. All the three tests were positive in significant number of cases, two test IgG ELISA and urease or histopathology were significant, IgG alone was also significant. The combination of other tests were not significant . IgGELISA, according to our study, is important test for detecting H pylori. the sensitivity does not improve in combination improves in combination with Urease and histopathology or both. In the group of patients with H.pylori positive by three tests, the common symptoms were epigastric pain (71.42%), retrosternal burning sensation in (50%), early satiety(39.9%) , abdominal bloating (28.9%) and other were not commonly absorbed. In patients wherein two tests were done, ELISA and histopathology showed epigastric pain (80%), retrosternal burning sensation in (40%), early satiety(60%) over the other symptoms. In patient with IgG and urease positive showed epigastric pain (72.2%), early satiety (72.2%), retrosternal burning sensation (55.55%) and anorexia (44.44%). In cases where only IgG ELISA was positive and other tests negative ,epigastric pain (100%), abdominal bloating (50%) and retrostenal burning sensation (40%) were noted in that order. In H pylori positive cases, epigastric pain (75%:89%), abdominal bloating sensation (50%:66.6%), Belching (45%:55.5%) were noted more in females than males, where as retrosternal burning sensation (30%:25%) and other complaints with a prevalence less than 25% were observed more in males than females. In our study, 20 of the H,pylori positive patient (34.4%) had ulcers, of which majority were in the pylorus (60%) and in the rest of the patients it was equally present in the fundus and duodenum, followed by gastric body-27% . It is mentioned in literature[18] that gastric antrum-corpus transition zone is peculiarly susceptible to acid peptic attack of H.pylori and therefore principle site of peptic ulceration.Our study confirms this. Majority of the patients (85%) in the study group were consuming mixed diet. 33.3% vegetarian patients had ulcer disease on gastroscopy ($p = 0.26$; insignificant) and 37.7% patients consuming mixed diet had ulcer on gastroscopy ($p = 0.03$; statistically significant). In this study, 69% of H.pylori infected patients did not have family history ofdyspeptic symptoms. 31% of H.pylori infected patients had family history of dyspeptic symptoms. However the data is statistically insignificant with $p = 0.80$.Majority (37.5% males and 44% females) of H.pylori infected patients had their blood group O Positive ($p=0.026$; significant).

SUMMARY

Dyspepsia is a common complaint amongst people. Epigastric pain, bloating sensation of the abdomen and retrosternal burning were the most common symptoms. This occurence was observed more in the female population. ELISA by itself was very sensitive in recognising H.Pylori. ELISA in combination with urease was also sensitive in detecting H. Pylori.

H. pylori was noted in 58% of the people. 34.4% of these patient had ulcers mainly in the pylorus. Of the 37 patients with ulcers ,28 were infected with H pylori. H. pylori incidence was equal in both the sexes. No significant family history of H.pylori infection was noted in the affected population. H.pylori infection was common amongst people of "O" positive group.

CONCLUSION

A study of hundred cases of dyspepsia was conducted. They were investigated by upper gastroduodenoscopy, histopathology and urease test was conducted of the specimen .Serological IgG ELISA test was conducted in these patients.

Based upon the observation and analysis of our study, we conclude that

- IgG ELISA test alone is sensitive enough to explain the symptoms.
- IgG ELISA in combination with urease and histopathology was also effective, histopathology and urease test alone by themself were insensitive.
- Epigastric pain, belching and bloating sensation were the most common symptoms observed in our study group.
- The incidence of this symptoms was more in female than male.
- In females H.pylori was noted in younger age group than male.

- A significant number of cases with risk factors like NSAIDS, other drugs, alcohol and smoking had H.pylori infection.
- H.pylori infection was noted more commonly among those consuming mixed diet (85%).
- A family history of H pylori infection was not a prerequisite for infection.
- 34% of patients with ulcer were noted to be infected by H. Pylori and most of these ulcers were in the pylorus.
- H.pylori infection was common amongst people of "O" positive group. Most of the patients had the symptoms for < 3 yrs.

REFERRENCES

1. Krag E. Non-ulcer dyspepsia. Introduction: epidemiological data. Scand J Gastroenterol 1982; 17: Suppl 79:6-8.
2. Talley NJ, Piper DW. The association between nonulcer dyspepsia and other gastrointestinal disorders.Scand J Gastroenterol 1985; 20: 896-900.
3. Talley NJ, Fung LH, Gillian IJ, McNeil D, Piper DW.The association of anxiety, neuroticism and depression with dyspepsia of unknown cause: a case control study.Gastroenterology 1986; 90: 886-92.
4. Crean GP, Card WI, Beattie AD, *et al*. 'Ulcer-like dyspepsia'. Scand J Gastroenterol 1982; 17: Suppl 79:9-15.
5. Warren JR, Marshall B: Unidentified curved bacilli on gastric epithelium in active chronic gastritis. Lancet 1983,321:1273-1275.
6. Marshall BJ, Warren JR. Unidentified curved bacilli in the stomach of patients with gastritis and peptic ulceration.Lancet 1984 , 1:1311-1315.
7. Peter S, Beglinger C: Helicobacter pylori and gastric cancer: the causal relationship. Digestion 2007(75):25-35.
8. The EUROGAST Study Group: Epidemiology of, and risk factors for, Helicobacter pylori infection among 3194 asymptomatic subjects in 17 populations. Gut 1993(34):1672-1676.
9. Anthony SF, Dennis LK, Dan LL, Eugene B, Stephen LH, Larry J, *et al*. Harrison's principles of internal medicine. 17th ed. NewYork (USA): The McGraw-Hill companies,Inc.;2008.p.946.
10. Harry LTM, George LM, Stuart LH. Helicobacter pylori-physiology and genetics. 1st ed. Washington DC (USA): ASM Press;2001.p.459-470.
11. Harry LTM, George LM, Stuart LH. Helicobacter pylori-physiology and genetics. 1st ed. Washington DC (USA): ASM Press;2001.p.27-38.
12. Crean GP, Holden RJ, Knill RP, Beattie AD, James WB, Marjoribanks FM, *et al*. A database on dyspepsia. Gut 1994;35:191-202.
13. Moynihan BGA. On duodenal ulcer: with notes of 52 operations. Lancet 1905; 1: 340-6.
14. Dombal FT. Analysis of foregut symptoms. In:Baron JH, Moody FG, eds.Butterworth's International Medical Review. Gastroenterology: I Foregut.London: Butterworths, 1981: 49-66.
15. Katelaris PH, Tippett GH, Norbu P, Lowe DG, Brennan R, Farthing MJ. Dyspepsia, Helicobacter pylori, and peptic ulcer in a randomly selected population in India. Gut 1992;33:1462-1466.
16. JC Horrocks, FTD Dombal. Clinical presentation of patients with "dyspepsia". Detailed symptomatic study of 360 patients. Gut 1978; 19: 19-26.
17. Manjunath SM, Jacob A, Nutan D, Peeyush M, Bhagwan ST, Prabha S. Serological estimation of Cag 'A' and H.pylori IgG antibodies in nonulcer dyspepsia and gastro-duodenal disease. Indian J Gastroenterol. 2003 Nov;22(1):A27.
18. Harry LTM, George LM, Stuart LH. Helicobacter pylori-physiology and genetics. 1st ed. Washington DC (USA): ASM Press;2001.p.459-470.

Source of Support: None Declared
Conflict of Interest: None Declared