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ORIGINAL ARTICLE

PREVALENCE OF HELICOBACTER PYLORI INFECTION IN PATIENTS WITH DYSPEPSIA

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ABSTRACT: Dyspepsia is synonymous with commonly used non-medical term indigestion. It includes symptoms like pain, bloating, nausea & early satiety. It is now recognized that the large majority of duodenal and gastric ulcers are caused by H. pylori infection and/or NSAID use. H. pylori infection is associated with poverty, household crowding & limited education. Colonization rates exceed 70% in some groups and vary from less than 10% to more than 80% worldwide. Several studies have revealed the association of H. pylori in 70–75% of patients with dyspepsia. The aim of this study is to study the prevalence of H. pylori infection in dyspeptic patients. To study the various upper GI endoscopy findings in dyspeptic patients.

KEYWORDS: H. pylori, Dyspepsia.

INTRODUCTION: Dyspepsia is synonymous with commonly used non-medical term indigestion. It includes symptoms like pain, bloating, nausea & early satiety.[1] It is also defined as persistent/recurrent pain or discomfort localized to upper abdomen, may or may not be related to meals. Once the patient has been evaluated for dyspepsia he/she will be categorized as having either organic dyspepsia (e.g., APD, gastric carcinoma, NSAID gastropathy etc.) or functional/non ulcer dyspepsia

Acid peptic diseases comprise a wide spectrum of diseases which cause considerable morbidity. A variety of factors may contribute to the development of APD. Although it is now recognized that the large majority of duodenal and gastric ulcers are caused by H. pylori infection and/or NSAID use.[2,3]

Several studies have revealed the association of H. pylori in 70-75% of patients with dyspepsia. Endoscopic studies have shown that H. pylori is found in 80-100% of patients with duodenal ulcer and 60-75% patients with gastric ulcer.[4]

The study of gastric bacteriology gained significant impetus after the isolation of Helicobacter pylori in 1983 from gastric biopsies, by Barry Marshal and Robin Warren,[5] in Perth. For nearly 45 years prior to that, gastric spiral bacteria were repeatedly observed and then forgotten.[6,7,8]

H. pylori infection is associated with poverty, household crowding & limited education. Risk factors for H. pylori infection are (1) birth or residence in a developing country, (2) domestic crowding, (3) unsanitary living conditions, (4) unclean food or water, and (5) exposure to gastric contents of an infected individual.

The mode of H. pylori transmission is not well defined, but humans are the only known host, making oral-oral, fecal-oral, and environmental spread the most likely routes of infection. H.pylori is a gram negative bacterium, curved 's' or spiral shaped, with blunt rounded ends & 4-6 unipolar sheathed flagella with bulbous tips.

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The organism can also assume coccoidal form after prolonged culture & exposure to oxygen. This form considered to be dormant state may serve to protect the organism in unfavourable environment.[9,10]

Two strains of H.pylori, based on their interaction with PMN.. 1. Virulent strains – induce an oxidative burst in neutrophil.2. Non virulent strains – do not induce this reaction. The virulent strains may be responsible for ulceration as the oxidative burst liberates free radicals which cause cell damage.

Four Features are linked to H. Pylori Virulence:

- Flagella, which allow the bacteria to be motile in viscous mucus.
- Urease, which generates ammonia from endogenous urea and thereby elevates local gastric pH.
- Adhesins that enhance their bacterial adherence to surface foveolar cells.
- Toxins, such as cytotoxin-associated gene A (CagA), that may be involved in ulcer or cancer development by poorly defined mechanisms.

The Mechanisms Responsible for h. Pylori–induced gi Injury Remain to be Fully Elucidated, but Three Potential Mechanisms have been Proposed:

1. Production of toxic products to cause local tissue injury.
2. Induction of a local mucosal immune response.
3. Increased gastrin levels with a resultant increase in acid secretion.

Characteristics of Helicobacter Pylori Associated Gastritis:

	H. pylori–Associated
Location	Antrum
Inflammatory infiltrate	Neutrophils, subepithelial plasma cells
Acid production	Increased to slightly decreased
Gastrin	Normal to decreased
Other lesions	Hyperplastic/inflammatory polyps
Serology	Antibodies to H. pylori
Sequelae	Peptic ulcer, adenocarcinoma
Associations	Low socioeconomic status, poverty, residence in rural areas

Clinical features of H.pylori Infection:

1. Chronic Gastritis: The symptoms associated with chronic gastritis are typically less severe but more persistent. Nausea and upper abdominal discomfort may occur, sometimes with vomiting, but haematemesis is uncommon. Chronic gastritis has been divided into two types having similar histologic features but a presumed different pathogenesis. The first type, which is less common, is designated as type A or immune. It usually affects the fundus in a diffuse manner, spares the antrum. The other type, by far the more frequent, begins in the antrum and progresses proximally so that the fundic–pyloric border rises up gradually.[11] This is referred to as type B or non-immune gastritis.[12] The crucial role played in type B chronic gastritis (and other gastric diseases, such as peptic ulcer,

carcinoma, and lymphoma) by H. pylori.[13,14.] The most common cause of chronic gastritis is infection with the bacillus Helicobacter pylori.

2. Peptic Ulcer: Peptic ulcer disease (PUD) is most often associated with H. pylori–induced hyperchlorhydric chronic gastritis, which is present in 85% to 100% of individuals with duodenal ulcers and in 65% with gastric ulcers. In cases infected with H. pylori, a typical constellation of morphologic changes (Loss of the apical portion and dropout of epithelial cells, epithelial pits, erosions, and cellular tufts) is seen at the ulcer edge.[15]

Considerable evidence has accumulated indicating that H. pylori plays a crucial role in the pathogenesis of this disease.[15,16] The risk for the development of a peptic ulcer is approximately 10-fold higher in patients with non-atrophic H. pylori-positive gastritis than in those with a normal stomach, and the risk is increased further (twofold to threefold) when there is antral atrophy.

3. Gastric Carcinoma: The most important development in the field has been the identification ofH. pylori as an important etiologic factor in gastric carcinoma through its role in the development of chronic gastritis.[17,18,19]H.pylori is associated with intestinal type of gastric carcinoma. It is also associated with gastric MALT lymphoma.

Diagnosis: Diagnostic tests for H. pylori are divided between tests that do or do not require a sample of gastric mucosa.

The Non-invasive Tests Available are:

- Serology
- Carbon -labelled urea breath test.

The invasive tests available are:

- Rapid urease test.
- Histology.
- Culture.
- PCR.

Non-invasive tests do not require endoscopy, whereas invasive tests do.

The main disadvantage of invasive test is potential sampling error because of patchy distribution of the organism in gastric mucosa. This can be minimised by taking multiple samples from different sites and predictive value of diagnostic tests can be improved by combining more than one test.

Indications for Diagnosis and Treatment of Helicobacter pylori; Established Active peptic ulcer disease (Gastric or duodenal ulcer), Confirmed history of peptic ulcer disease, (Not previously treated for H. pylori) Gastric mucosa-associated lymphoid tissue lymphoma (Low grade), After endoscopic resection of early gastric cancer, un investigated dyspepsia (Depending on H. pylori prevalence).

Controversial Indications are:

- Nonulcer dyspepsia.
- Gastroesophageal reflux disease.

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- Persons using NSAIDS.
- Unexplained iron deficiency anaemia.
- Populations at higher risk for gastric cancer.

TREATMENT:

The Clinician has Three Major Goals when Faced with a Patient with Ulcer Disease:

1. Symptoms need to be relieved.
2. The ulcer needs to be healed.
3. Recurrence must be prevented.

Various triple regimens for H. pylori eradication have emerged. Most of these employ a proton pump inhibitor in combination with antibiotics such as metronidazole, clarithromycin, or amoxicillin. These regimens are usually 2 weeks in duration, have the advantage of not containing bismuth, and are only given twice a day. Eradication rates for these new triple regimens are about 90% (Still not 100%). For failures, or in patients with high metronidazole resistance, quadruple therapy with bismuth added to the triple regimen is recommended.

It is also important to remember that the success of therapy for H. pylori depends on the correct use of the regimens. One cannot substitute ampicillin for amoxicillin, and one cannot substitute doxycycline for tetracycline. Appropriate dosages need to be used, the recommended frequency of administration adhered to, and the duration of drug therapy enforced.

Test	Sensitivity	Specificity	Availability	Cost
Invasive				
Histology	88-95%	90-95%	+ + + +	££££
Culture	80-90%	95-100%	+ +	£££
Urease test	90-95%	90-95%	+ + + +	£-££
Non-invasive				
^{13}C-UBT	90-95%	90-95%	+ + + +	£££
^{14}C-UBT	86-95%	86-95%	+ + +	££
Serology:				
ELISA	80-95%	80-95%	+ + +	£
NPT	60-90%	70-85%	+ + + +	££
Stool antigen	90-95%	90-95%	+ +	££

UBT = urea breath test. NPT = near patient test.

METHODOLOGY: 50 cases of dyspepsia were studied clinically as per the proforma over a period of one and half years from July 2013 to October 2014. The inclusion and exclusion criteria were as follows;

Inclusion Criteria:

1. Patients with dyspepsia aged >14 years.
2. Patients having dyspeptic symptoms for more than 6 months.
3. Both males and females are included.

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Exclusion Criteria:

1. Patients with dyspepsia aged <14 years.
2. Patients with gall bladder or pancreatic diseases may present with dyspepsia. They are excluded from the study.
3. Patients who are not willing and unfit for endoscopy.
4. Patients who have already taken anti H.pylori medication.

After applying the inclusion and exclusion criteria, all the patients under went upper gastro-intestinal endoscopy. According to the endoscopy findings the patients were divided into following groups.

Non-ulcer Dyspepsia:

a. Normal study.
b. Gastritis/Duodenitis.

Ulcer Dyspepsia:

a. Duodenal ulcer
b. Gastric ulcer.

PROCEDURE: All the patients in this study group, both in-patient as well as out-patient underwent upper gastro-intestinal endoscopy under topical anaesthesia. The patients were asked to fast for 12 hours prior to the procedure. The cases admitted with gastric outlet obstruction were given stomach wash the night before and the morning of the day on which the procedure was scheduled. Only a few patients were given 5-10mg diazepam intravenously for sedation depending on the preference of the consultants.

Lignocaine viscous or oral lignocaine sprays were given to the patient 5-10minutes before the procedure for the local anaesthetic effect. The upper gastro-intestinal endoscopy was conducted with flexible, fibre-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 other biopsy specimen was sent in formalin solution for Histopathology. Each of the biopsy specimens were fixed in 10% buffered formalin, routinely processed to paraffin and 3µmsections cut. One section of each biopsy specimen was routinely stained with Haematoxylin & eosin stain and examined microscopically for presence of Helicobacter pylori organisms. Histopathology test was given as positive when Helicobacter pylori were detected by routine Haematoxylin & eosin stain. The case was considered as positive when histopathological examination/both histopathological examination and rapid urease test are positive. The case was considered negative when histopathological examination is negative. When histopathological examination is positive and rapid urease test is negative (False negative) then it is considered as positive. When histopathological examination is negative and rapid urease positive (False positive) the case is considered as negative.

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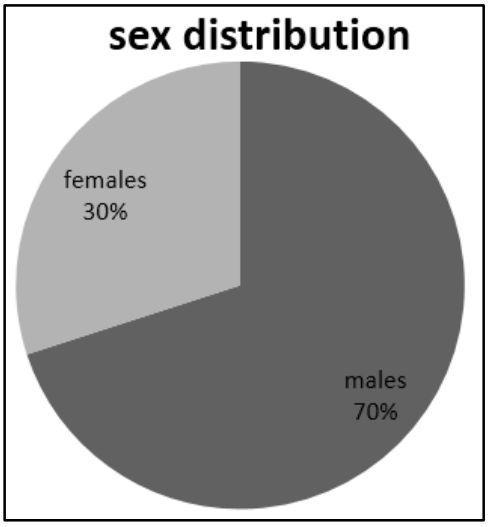
RESULTS: Out of50 patients, there were 35 male patients and 15 female patients, age above 14yrs. Out of 50 patients, 20 patients (14 males and 6 females) were diagnosed to have been infected with Helicobacter pylori. All these patients presented to our hospital with upper abdominal pain or discomfort. 10 patients presented with nausea or vomiting out of which 2 had Helicobacter pylori infection. 15 patients had haematemesis, out of which 6 patients were positive for Helicobacter pylori infection. 1 patients had malena turned out to be Helicobacter pylori negative. On examination of these patients, 12 patients were anaemic out of whom 2 patients were positive for Helicobacter pylori. Of these 50 patients, 30patients had epigastric tenderness on palpation. Of these 30 patients, 12 patients were tested positive for Helicobacter pylori.

Clinical Presentation	Number of Cases	H.pylori Positive	Percentage
Pain abdomen/discomfort	50	20	40%
Nausea/vomiting	10	2	20%
Haematemesis	15	6	40%
Anaemia	12	2	16.6%
Epigastric tenderness	30	12	40%
Malena	1	0	

Table 1



Depending on the Endoscopic Findings, all these Patients were categorized into 2 Groups:

A. Ulcer Dyspepsia.
B. Non-Ulcer Dyspepsia.

	Total Cases	Positive
Ulcer dyspepsia	5	2
Non ulcer dyspepsia	45	18

Table 2

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A]. ULCER DYSPEPSIA: In this group there were 5 patients. This group was further divided into

1. Duodenal Ulcer; 1 patient who was negative for h pylori.
2. Gastric Ulcer; 4 patients. 2 were positive for h pylori.

	Positive	Negative	Total
Duodenal ulcer	0	1	1
Gastric ulcer	2	2	4

Table 3

B].NON-ULCER DYSPEPSIA: In this group, there were 45 patients out of which 18 patients were infected with h pylori.12 males and 6 females.

This Group was Further Divided Into:

1. Normal Study.
2. Gastritis/Duodenitis;

Endoscopy findings	H .pylori positive	H .pylori negative	Total
Normal study	5(all males)	10	15
Gastritis/duodenitis	13(6 females , 7 males)	17	30
	18	27	45

Table 4

CONCLUSION: This was a prospective study conducted to determine the role of Helicobacter pylori in acid-peptic diseases. This study design was based onclinical study and endoscopic biopsy of gastric mucosa (and duodenalmucosa whenever necessary) inpatients with a history of dyspepsia. Endoscopy confirmed the diagnosis. Rapid urease test and histopathological examination were conducted on endoscopy biopsy specimens and Helicobacter pylori positivity was based on either Rapid urease test or histopathological examination or histopathological examination alone was positive.

From the present study it is evident that, there was no specific symptom attributable to H. pylori infection. Helicobacter pylori infection is more common in males than females. Seroprevalence of Helicobacter pylori increases with increasing age. Helicobacter pylori are consistently associated with both peptic ulcer disease & non-ulcer dyspepsia, which is in broad agreement with the studies done earlier. Thus we conclude that, H.pylori infection may have a role in etiopathogenesis of peptic ulcer disease. There appears to be no significant association between Helicobacterpylori infection and unexplained dyspepsia. This finding does not exclude the possibility that a small undefined subset of infected individuals will have symptoms induced by the infection, but only large randomized trials will be able to establish this.

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