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The prevalence of *Helicobacter pylori* infection in Erosive and non-erosive gastro-esophageal reflux disease

Dr. Hasan Salman Al Gharbawi (M.B.Ch.B) (F.I.C.M.S)
Dr. Tarik Mohammed AL-Hadithi (M.B.Ch.B) (C.A.B.M)
Department of Medicine / Al Kadhmiya Teaching Hospital
GIT and endoscopy unit, IRAQ / Baghdad

Corresponding Author: **Dr. Hasan Salman AlGharbawi (M.B.Ch.B) (F.I.C.M.S)**
Department of Medicine / Al Kadhmiya Teaching Hospital
GIT and endoscopy unit, IRAQ / Baghdad
E-mail: *hsalmania@yahoo.com*

Abstract

Objective:
This study was done to determine the prevalence of *H. pylori* infection and symptoms in patients with erosive and non-erosive gastro-esophageal reflux disease.

Patients and Methods:
A total of 54 patients with symptoms of GERD in addition to 10 control subjects (sex and aged – matched), were examined by endoscopy and 2 biopsies were taken, one from the gastric antrum for rapid urease test and the other from esophagus for histopathological determination of GERD.
Severity of GERD was graded by SAVARY –MILLER classification, and patients were grouped into 4 groups:
(Group A = non-erosive GERD) (Group B = erosive GERD) (Group C = symptoms with negative endoscopy) (Group D = control).

Results:
H. pylori infection was positive in 14 patients (73.6%) in Group A , while it was positive in 6 patients (24 %) in group B , 7 patients (70%) in group C , and 5 patients (50%) in group D.
By application of chi square test, P value of this test is significant.
Regarding presenting symptoms, Heartburn is the commonest followed by dyspepsia, regurgitation, water brush, and chest pain.

Conclusion:
Helicobacter pylori prevalence is higher in patients with non-erosive GERD than in normal subjects and in patients with erosive GERD,
There is neither sex predominance nor smoking effect on different grades of esophagitis.
Regarding symptoms, there is no particular predominant symptom in relation to different and severity of esophagitis.

Keywords: *Helicobacter pylori*, GERD, esophagitis, rapid urease test.

Introduction

Gastro-esophageal reflux disease is a common condition affecting 25%-40% of the population. It refers to a spectrum of clinical manifestations resulting from Reflux of stomach and duodenal contents into the esophagus (1).

GERD is applied to patients with symptoms suggestive of reflux or complications thereof, but not necessarily with esophageal inflammation. The distiction between normal and GERD is blurred because some degree of reflux is physiologic (3), which is typically occurs postprandially, short lived and rarely during sleep.

Refulx esophagitis describes a subset of patients with symptoms of GERD who also have endoscopic or histopathologic evidence of esophageal inflammation.

The most common symptoms of GERD are heartburn (pyrosis), regurgitation, and dyspepsia. In addition, a variety of extra-esophageal manifestations have been described including asthma, laryngitis, and chronic coughing.

Although there is a good evidence that infection with *H. pylori* is the principal cause of peptic ulcer disease, there is uncertainty about the organism's role in GERD.

Treating *H. pylori* infection is effective in healing duodenal ulcer (4) . The effect on eradication of the organism in patients with GERD is less clear, with some reports suggesting that this might be counter-productive and that *H. pylori* infection may protect against the disease (5) .

The evidence for the association between *H. pylori* and GERD remains mixed and largely uncertain (6)

There is a clear age related prevalence of *H. pylori* infection in healthy subjects, increasing from 10% in those younger than age 30 to 60% in subjects older than 60.

The majority are infected early in life, although the mode of transmission reains unknown (1)

Helicobacter pylori colonization is more common in blacks, individuals in lower socioeconomic classes, and inhabitants of custodial institutions.(2)

Diagnostic testing for *H. pylori* can be divided into invasive and non-invasive techniques based upon the need for endoscopy.

The technique may be direct (culture , microscopic demonstration of the organism) or indirect (using urease or an antibody response as a marker of the disease).

The diagnosis of the *H. pyolri* can usually be established during endoscopy by one of three methods : biopsy urease test, histology, and less commonly bacterial culure).

General recommendations have been proposed by the American College of Gastro-enterology (8)

-When endoscopy is indicated, the test of first choice is a urease test on an antral biopsy

-If a biopsy urease test is negative, *H. pylori* infection may be diagnosed by histology or serology.

-Biopsy urease test have reduced sensitivity in patient taking PPIs and in patients with recent or active bleeding.

- Histology is generally not necessary and is expensive.

H. pylori is characterized by the production of a strong enzyme called (urease) which has been used to develop rapid urease test (RUT) for rapid laboratory identification, with sensitivity of 99% and specificity of 100% (6).

Regarding Gastro-esophageal reflux disease, esophagoscoy (with biopsy when necessary) should be initial evaluation of suspected GERD because it provides a mechanism for detecting, stratifying, and managing the esophageal manifestations of GERD.

The interoperator variability in assessing the severity of peptic esophaitis spawned many endoscopic grading schemes; of the more than 80 proposed schemes, the most dominant and dependable is SAVARY –MILLER classification for grading of esophagitis.

Grade 1: exhibits one or more supravestibular, non-confluent reddish spots, with or without exudate.

Grade 2: demonstrates erosive and exudative lesions in the distal esophagus which may be confluent, but not circumfrential.

Grade 3: is characterized by circumfrential erosionsin the distal part of esophagus, covered by hemorrhagic and pseudomembranous exudate.

Grade 4: is defined by the presence of chronic complications such as deep ulcers, stenosis, or scarring with Barrett's metaplasia.

Histolgy – even though the esophagus may appear endoscopically normal, it is not necessarily histologically normal.

Mild histologic findings of GERD were described in 1970 and represent the reparative capacity of the esophageal epithelium after cell damage due to acid exposure.

Cellular thickening of the basal cell layer and elongation of the papillae of the epithelium (12)

Other histologic features include the presence of neutrophils and eosinophils, dilated vascular channels in papillae of the lamina propria, and distended, pale squamous (balloon) cells.

Non of these findings are specific for GERD.(13)

Patients and Methods

This study was done on patients attending the endoscopy unit in Al-Kadhmiya Teaching Hospital between April and November 2005.

Fifty four patients with symptoms of Gastro-esophageal reflux disease were included in this study, in addition to 10 individuals (6 males , 4 females) who were taken as control group with an age range 18-50 years .

The total of 54 patients were examined by endoscopy and esophagitis graded by modified Savary – Miller system. then patients grouped into 4 groups:

Group A (N=19) = non-erosive GERD (mild – grade 1)
Group B (N=25) = erosive GERD (grade 2 and 3)
Group C (N=10) = GERD symptoms with negative endoscopy.
Group D (N=10) = control group.

Two biopsies were taken, one from gastric antrum for rapid urease test, and the other from esophagus (5 cm above gastro-esophageal junction) for histopathological study.

All patient were asked about symptoms GERD disease in specific.

All esophageal biopsies were examined by same pathologist who was blinded to the *H. pylori* status of the patients.

The stain used for histopathology was hematoxylin – Eosin stain.

Rate comparisons in this study were made by Chi – Square test.

Results

Sixty four patients were included in this study, 39 (60.9%) of them were male and 25(39.1%) were female as in table (1).

Table (1): shows the sex in relation to four groups of patients.

sex	Group A	Group B		Group C	Group D	Total	
	Grade 1	Grade 2	Grade 3		Control		
Male	12	11	5	5	6	39 (60.9%)	
Female	7	6	3	5	4	25 (39.1%)	P-value
Total	19	17	8	10	10	64	0.9567

Sex difference in different groups was not significant as the P-value of the test is greater than (0.05) which statistically not important

The results of the rapid urease test in comparison to different grades of esophagitis are shown in table (2) and as follows :

Group (A) = 14 (73.6%) positive versus 5 (26.4%) negative

Group (B) = 6 (24%) positive versus 19 (76%) negative

Group (C) = 7 (70%) positive versus 3 (30%) negative

Group (D) = 5 (50%) positive versus 5(50%) negative

Table (2) : shows the result of rapid urease test in relation to four groups of patients.

RUT	Group A Grade 1	Group B Grade 2	Group B Grade 3	Group C	Group D	Total
Positive	14 (73.6%)	4 (23.5%)	2 (25%)	7 (70%)	5 (50%)	32 (50%)
Negative	5 (26.4%)	13 (76.5%)	6 (75%)	3 (30%)	5 (50%)	31 (50%)
Total	19	17	8	10	10	64
p-value	0.0080	0.1900	0.6444	0.0078	0.9670	

P-value of this study is significant in group A and C .
The prevalence of *H. pylori* infection is higher in group A and C , than group B and D.
This means that esophagitis score is higher in the non-infected individuals.

The results of smoking in relation to different groups of patients in this study was shown in table (3).

Table (3) : shows the relation of smoking in different groups of patients in study.

Smoking	Group A Grade 1	Group B Grade 2	Group B Grade 3	Group C	Group D	Total	P-value
Positive	9	12	6	5	5	37 (57.8%)	
Negative	10	5	2	5	5	27 (42.2%)	
Total	19	17	8	10	10	64	0.4845

P-value of the test was not significant as it was greater than (0.05), this means that there is no major difference in smoking and non-smoking groups in relation to different grades of esophagitis.

The results of the presenting symptoms in relation to different grades of esophagitis was shown in table (4)

Table (4) : shows symptoms in relation to different grades of esophagitis.

Symptoms	Group A Grade 1	Group B Grade 2	Group B Grade 3	Group C	P-value
Heartburn	17 (84.4%)	16 (94.1%)	8 (100%)	10 (100%)	0.9866
Dyspepsia	16 (84.2%)	13 (76.4%)	8 (100%)	7 (70%)	0.9876
Regurgitation	10 (52.6%)	11 (64.7%)	6 (75%)	5 (50%)	0.9345
Water brash	10 (52.6%)	6 (35.2%)	7 (87.5%)	5 (50%)	0.0789
Chest pain	4 (21%)	1 (5.8%)	2 (25%)	1 (10%)	0.0876
Total	19	17	8	10	

There is no symptom predominance in relation to different grades of esophagitis, but the commonest presentation is with heartburn and dyspepsia while regurgitation, water brash and chest pain comes later.

Discussion

Gastro-esophageal reflux disease is a clinical condition that result from reflux of caustic fluid from the stomach into the esophagus.

The pathophysiology of GERD is multifactorial with the disease ultimately related to damage (or sensitize) the esophageal mucosa and those tending to preserve it (1).

Suspicion of an interaction between *H. pylori* and GERD stems from epidemiologic data showing that as the prevalence of *H. pyolri* decreased in western societies, the prevalence of GERD and adenocarcinoma of the esophagus increase (11).

For *H. pylori* to exert a direct pathogenic effect on GERD, it must have an impact on one of those primary disease determinants. *H. pylori* has no apparent effect on esophago-gastric junction competence.

There are no data suggesting that *H. pylori* decreases lower esophageal sphincter (LES) pressure or the frequency of transient LES relaxations.

Furthermore, esophageal peristaltic function and acid clearance are unlikely to be affected by *H. pylori*.

In our study, we found that esophagitis score is increased in non-infected group, so H.pylori might give a protection against the disease.

Several reports were done in this issue , suggesting the same result, when the *H. pylori* positive patients are less likely to GERD , and when present, the severity of esophagitis was lower as compared to those who are *H. pylori* negative (12,13)

In the study of Manes-G, Balzano- A et al, which was done in Cardarelli hospital, Napes, Italy, the prevalence of *H. pylori* is higher in non-erosive GERD (62%) than in erosive one (36%) (13).

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Another study which was done by Sung JY, et al. In university of Hongkong, China, showing that esophagitis score was significantly higher in the non-infected group (2.76 +/ - 1.38) than the infected group (1.6 + /- 0.92) and the P-value = 0.0045 (14).

The one primary mechanism by which *H. pylori* might influence the pathogenesis of GERD is by modifying the gastrin refluxate.

H. pylori has a major effect on somatostatin D cells in the gastric antrum such that feedback inhibtion by luminal acid on gastric release is interrupted.

As a result, gastrin levels are higher in *H. pylori* infected individuals and these levels do not exhibit normal feedback inhibition (15).

Lack of feedback inhibition is hypothesized to be ultimately responsible for the increased acid secretion found in patients with duodenal ulcer who are *H. pylori* positive and have antral predominant gastritis.

H. pylori infection associated with corpus-predominant gastritis is also associated with increased gastrin levels, but alters cell function as a result of local inflammation and increased levels of cytokines, such as tumour necrosis alpha.

This can eventually lead to hypochlorhydria and gastric atrophy, making *H. pylori* as protective factor against GERD (16).

Another hypothesized mechanism accounting for worsening reflux disease in patients with *H. pylori* is that urease-mediated ammonia production may exert a buffering effect on gastric juice, however, this effect is likely to be minimal compared to the *H. pylori* effect on parietal cell mass (16).

Several studies had done and suggest the previous mechanism like that which was done by Wu JCY, FKL chan, JY Sung et al , on the effect of *H. pylori* eradication on treatment of GERD, the result of this study reach a conclusion that the eradication of *H. pylori* leads to more difficult control of disease in reflux patients.(17)

The logical predictions from the above discussions would apppear to be that eradication of *H. pylori* in antral-predominant gastritis patients would improve GERD symotoms, on the basis of decreased competence of the esophago-gastric junction (EGJ) , while eradication in patients with corpus-predominant gastritis could worsen GERD in the same set of patients.

Conclusion

Helicobacter pylori prevalence is higher in patients with non-erosive GERD than in normal subjects and in patients with erosive GERD,

There is neither sex predominance nor smoking effect on different grades of esophagitis.

Regarding symptoms, there is no particular predominant symptom in relation to different and severity of esophagitis.

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