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Evaluation of the role of *Helicobacter pylori* in dyspepsia in Al- Ramadi city

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Abstract

Back Ground: *Helicobacter pylori* are the most common chronic bacterial infection, and a significant etiological factor in acid peptic diseases and gastric cancer. Dyspepsia is a common gastrointestinal disorder, and the most common indication for gastroscopy. Detection of *H. pylori* during endoscopy has become standard clinical practice.

Objective: To detect the different underlying OGD causes of dyspeptic symptoms in patients presented to the outpatients clinic of the gastroenterology unit in Al-Ramadi General Hospital, and to evaluate the role of *H. pylori* in relation to the different OGD results.

Methods: One hundred patients with various upper gastrointestinal complaints were investigated for evidence of *H. pylori* infection and its possible relation to the upper gastrointestinal disease. Multiple biopsies from the antrum of the stomach were taken through fiber optic endoscope from these patients. Diagnosis of *H. pylori* infection was done by the urease test.

Results: One hundred patients, (62 males, 38 females), with age range between (9-63) year old and a mean age of 36 year old, were included in this study. Urease test was positive in (48%) of all patients (46% in male, 50% in female). Urease test was positive in 82% of patients with duodenal ulcer, 75% with Gastric carcinoma, 60% with gastric ulcer, 50% with gastritis, 50% with duodenitis, 41% with esophagitis, 37% with normal looking mucosa.

Conclusion: *H. pylori* were more likely associated with DU than GU. There is a high association between *H. pylori* and CA stomach. *H. pylori* was found in about 1/3 of patients with normal OGD and its difficult to tell about its role in this group of people. Most patients with positive OGD and negative urease test were found to have other predisposing factors such as NSAID or cigarette smoking. CA stomach was the least likely cause of upper GI symptoms. About half of the patients with upper GI symptoms have normal OGD which could either indicates functional dyspepsia or other causes of dyspepsia like liver, gall bladder or pancreatic diseases.

Keywords: *H. Pylori*, dyspepsia, urease test

Introduction

Helicobacter pylori (previously named campylobacter pylori) is a spiral organism which lives in human stomach and was first described by krenitz in Germany in 1903^[1]. The first culture of *H. pylori* was performed in Perth, Australia, in April 1982 by Marshal and Warren, and the first report was made in the following year in the Lancet^[2]. Since then evidence has accumulated to link this bacterial organism in the pathogenesis of peptic diseases^[3]. *H. pylori* is a gram-negative, spiral, flagellate bacillus, (0.5×3) µm in size, and is non-invasive, living in gastric mucus^[4], and a small proportion of the bacterial cells are adherent to the mucosa. Initially, *H. pylori* reside in the antrum, but over time, migrate towards the more proximal segments of the stomach. The organism is capable of transforming into a coccoid form, which represents a dormant state that may facilitate survival in adverse conditions^[5]. Its spiral shape and flagella render *H. pylori* motile in the mucous environment^[6], and its efficient urease protects it against acid by catalyzing urea hydrolysis to produce buffering ammonia^[7, 8]. *In vitro*, *H. pylori* are micro-aerophilic and slow-growing and requires complex growth media^[6]. The prevalence of *H. pylori* colonization is about 30% in the united states and other developed countries as opposed to > 80% in most developing countries. In the united states, prevalence varies with age, around 50% of 60-years old persons, as opposed to 25% of 30 years old persons, are colonized^[9, 10].

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The role of *H. pylori* in upper GIT diseases

All *H. pylori* colonized persons have gastric inflammation, but this condition in itself is asymptomatic. Symptoms are due to illnesses such as peptic ulceration or gastric malignancy which develop in fewer than 10% of individuals colonized with *H. pylori*. More than 80% of peptic ulcers are related to *H. pylori*, most of the remainder being due to damage caused by aspirin or NSAID [6]. The main lines of evidence for an ulcer promotion role of *H. pylori* are that eradication of *H. pylori* results in a dramatic drop in the rate of ulcer relapse (from about 80% to 15% in the first year), and that ulcer not induced by NSAID rarely develops in the absence of *H. pylori*. The particular end result of *H. pylori* infection (gastritis, duodenitis, PU, lymphoma, gastric CA) is determined by a complex interplay between bacterial and host factors [5].

1-Bacterial factors: *H. pylori* are able to induce mucosal injury and avoid host defences. Urease which allows the bacteria to reside in the acidic stomach. Generates NH₃, which can damage epithelial cells. The bacteria produce surface factors that are chemotactic for neutrophils and monocytes, which in turn contribute to epithelial cell injury [5, 11]. *H. pylori* make protease and phospholipases that break down the glycoprotein lipid complex of mucus gel, thus reducing the efficacy of this first line of mucosal defence [12]. *H. pylori* expresses adhesions, which facilitate attachment of the bacteria to gastric epithelial cell [5].

2-Host factors: the immune response to *H. pylori* includes both the production of Antibody (local & systemic) and cell mediated response, but are ineffective in clearing the bacteria [13]. The pattern of gastric inflammation is associated with disease risk. Antral gastritis is most closely linked with duodenal ulceration, whereas, pan gastritis is most closely linked with gastric ulcer and adeno-carcinoma. How do gastric *H. pylori* increase risk of duodenal ulceration? One explanation is that antral *H. pylori* colonization diminish the number of somatostatin-producing cells. Since somatostatin mediate inhibition of gastrin release, so there will be hypergastrinemia which leads to increase acid secretion and then increase the risk of duodenal ulceration. Another hypothesis is that gastric metaplasia in the duodenum permit *H. pylori* to bind to it and produce local injury secondary to the host response. After eradication of *H. pylori* from patients with duodenal ulcer disease, the level of acid secretion often falls [6, 14].

Diagnosis of *H. pylori* infection

Several methods may be used to diagnose *H. pylori* infection which can be divided into noninvasive tests and invasive tests which require upper gastrointestinal endoscope [15]. The non-invasive tests include:

1. **Serological tests:** that measure specific *H. pylori* IgG Ab, can determine if a person has been infected.
2. **Urea breath test:** in this test, the patient is given either 13c or 14c labeled urea to drink, *H. pylori* metabolizes the urea rapidly and the labeled carbon is absorbed. This labeled carbon can then be measured as CO₂ in the patient's expired breath.

The invasive tests include

1. **Biopsy urease test:** A colorimetric test based on the ability of *H. pylori* to produce urease.
2. **Histological identification of 6. Organisms:** Considered the gold standard of diagnostic tests.
3. **Culture of biopsy specimens** [16, 17].

Aim of study

To detect the different underlying OGD causes of dyspeptic symptoms in patients presented to the outpatients clinic of the gastroenterology unit in Al-Ramadi General Hospital, and to evaluate the role of *H. pylori* in relation to the different OGD results.

Methods

From January 2004 to May 2004, one hundred patients with various upper gastrointestinal complaints (epigastric pain, heart burn, anorexia, nausea, vomiting...) were evaluated by OGD in the gastro endoscopic unit, Al-Ramadi General Hospital. In all patients after fasting for at least 4 hours an OGD was done using fibroptic (FG29w pentax) endoscope, 2 biopsy specimens were taken from the antrum of stomach about 2cm proximal to the pyloric ring by the endoscope. The specimens were crushed in the agar tube by sterile rod; the tube was incubated at 37 °C, and change of color from yellow to pink within 24h. Is the indicator for the presence of *H. pylori*. The color change occurs when urea in the agar is hydrolyzed to release ammonia. The urea agar base was prepared in the laboratory, which contain: [urea 2%, bacteriological peptone 1.0, dextrose 1.0, di-sodium hydrogen phosphate 1.2, potassium di-hydrogen phosphate 0.8, sodium chloride 5.0, phenol red 0.012, agar (A) 14.0, ph 6.8].

For statistical analysis SPSS _ 11(statistical package for social sciences _version 11) was used to analysis the group percentage in addition to statistical figure.

Results

One hundred patients, (62 males, 38 females), with age range between (9-63) year old and a mean age of 36 year old, were included in this study. All of them were complaining from chronic and/or recurrent abdominal pain and various dyspeptic symptoms. Endoscopically results included peptic ulcer [DU (11%), GU (5%)], CA stomach (4%), gastritis (14%), duodenitis (6%), esophagitis (7%) and normal OGD (53%) as shown in table 1. Urease test was positive in (48%) of all patients (46% in male, 50% in female) as shown in figure 1.

Table 1: Frequency of Endoscopically diagnosis among patients

Endoscopic diagnosis	Total No.	%	Male	Female
DU	11	11%	7	4
CA stomach	4	4%	2	2
GU	5	5%	4	1
Gastritis	14	14%	7	7
Duodenitis	6	6%	5	1
Esophagitis	7	7%	5	2
Normal	53	53%	32	21
Total	100	100%	62	38

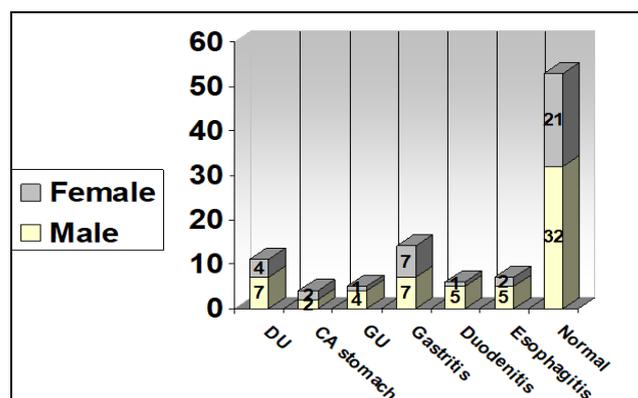


Fig 1: Frequency of endoscopically diagnosis according to sex.

Urease test results among the different endoscopic findings were as follows: out of 11 patients with duodenal ulcer 9 of them were urease positive (82%), out of the 4 patients with CA stomach 3 were urease positive (75%), out of 5 patients with gastric ulcer 3 were urease positive (60%), out of 14 patients with gastritis 7 were urease positive (50%), out of 6 patients with duodenitis 3 were urease positive (50%), out of 7 patients with esophagitis 3 were urease positive (41%), while the 53 patients with normal looking mucosa 20 were urease positive (37%) as shown in table 2 and figure 2.

Table 2: Distribution of patients according to positive urease test

Endoscopical diagnosis	No.	(+ve) urease test	%
DU	11	9	82%
CA stomach	4	3	75%
GU	5	3	60%
Gastritis	14	7	50%
Duodenitis	6	3	50%
Esophagitis	7	3	41%
Normal	53	20	37%
Total	100	48	48%

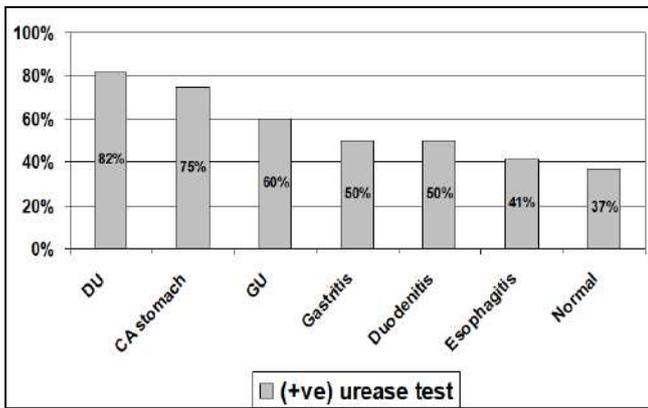


Fig 2: Frequency of positive urease test among patients

Table 3: Comparing the results of positive urease test

Endoscopical diagnosis	positive urease test %		
	In our study	In Ajeal M.D. in Al- Rumady study	In Zuhair, A.K. in Baghdad study
DU	82%	89%	90%
CA stomach	75%	-	-
GU	60%	-	-
Gastritis	50%	87%	68%
Duodenitis	50%	75%	68%
Esophagitis	41%	88%	38%
Normal	37%	63%	55%

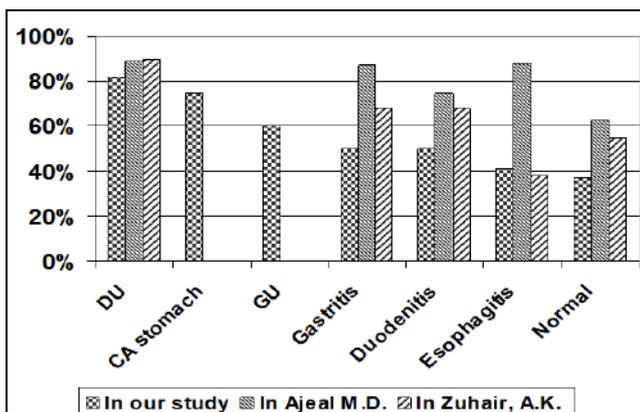


Fig 3: Comparing the results of positive urease test.

Discussion

Our study showed the association of *H. pylori* with the various gastrointestinal disorders, as shown in table (2). We found that about 60% of patients with abnormal OGD have positive urease test, while the other patients who had abnormal OGD and negative urease test could have other predisposing factors other than *H. pylori* infection like NSAID, smoking or else. The highest association was found between *H. pylori* and DU while the least association is between *H. pylori* and esophagitis. Also high association between *H. pylori* and CA stomach was found. These results were compared with that of Zuhair, A.k., in Baghdad city [18], and Ajeal, M.D. Al Ajeal in Al-Ramadi city [19], as shown in table (3). In all these three studies, the highest association was between *H. pylori* and DU. In our, and Zuhair, A.K., study the lowest association was between *H. pylori* and esophagitis, but not in Ajeal. M.D. study in which the association was much higher.

Conclusion

H. pylori were more likely associated with DU than GU. There is a high association between *H. pylori* and CA stomach. *H. pylori* was found in about 1/3 of patients with normal OGD and its difficult to tell about its role in this group of people. Most patients with positive OGD and negative urease test were found to have other predisposing factors such as NSAID or cigarette smoking. CA stomach was the least likely cause of upper GI symptoms. About half of the patients with upper GI symptoms have normal OGD which could either indicates functional dyspepsia or other causes of dyspepsia like liver, gall bladder or pancreatic diseases

Recommendation

All patients with dyspepsia should be screened thoroughly with appropriate tests (invasive or noninvasive) and treated accordingly. Patients with dyspepsia should be advice about dietary habits, drug – administration and smoking cessation.

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