**Helicobacter pylori in Diabetic and Non-Diabetic Patients with Dyspepsia**

Mahshid Talebi-Taher1, Manizheh Mashayekhi2, Mohammad Hossein Hashemi2, and Vanosheh Bahrani2

1 Department of Infectious Disease, Antimicrobial Resistance Research Center, Rasoul-e-Akram Hospital, Tehran University of Medical Sciences, Tehran, Iran
2 Institute of Endocrine and Metabolism, Firoozgar Hospital, Tehran University of Medical Sciences, Tehran, Iran

Received: 19 Feb. 2011; Received in revised form: 3 Jan. 2012; Accepted: 7 Mar. 2012

**Abstract** - Helicobacter pylori (H. pylori) is one of the most common chronic infections in patients with gastrointestinal disorders. Recent reports suggested that H. pylori might have high prevalence among patients with diabetes. The aim of this cross-sectional study was to assess the prevalence of H. pylori infection in diabetes mellitus and to study the relationship between histological findings and H. pylori infection in diabetic patients. Eighty patients with dyspepsia that were referred to our gastrointestinal department between May 2007 and May 2008 were included in our study. We checked fasting blood sugar for all of the study samples. All of patients underwent upper endoscopy and biopsy specimens were obtained from the antrum and the corpus. The specimens for the presence of H. pylori were colored by Giemsa stains. A single pathologist evaluated the histology slides. We found that prevalence of H. pylori infection was significantly higher in diabetics than in non-diabetics ($P=0.001$). Indeed, the prevalence of gastritis did differ significantly between the two groups ($P=0.001$). According to our results diabetes mellitus is one of the risk factor that must be considered in evaluation of H. pylori infection in diabetic patients with dyspepsia.


**Keywords:** Gastrointestinal disorders; Dyspepsia; Helicobacter pylori infection; Diabetes

**Introduction**

*Helicobacter pylori* (H. pylori) is one of the most common chronic infections, etiologically linked to peptic disease (1-3). Recent reports suggested that H. pylori might have a high prevalence among patients with diabetes (4-6). Etiopathogenesis of H. pylori infection in patients with diabetes mellitus have not been defined clearly. Some reports suggested that autonomic neuropathy and poor glycemic control might have a significant role in this field, but other studies had controversial results (7-15). An association between H. pylori infection and changes in gastric motility has been reported in several studies (16). Primary researches performed on patients with diabetes have reported that H. pylori infection is associated with some of the upper gastrointestinal (GI) symptoms improved after eradication of the infection (17,18). The aim of this cross-sectional study was to assess the prevalence of H. pylori infection in patients with diabetes mellitus and to study the relationship between histological findings and H. pylori status in diabetic patients.

**Materials and Methods**

The present study was an analytic observational study, performed through a cross-sectional method. Eighty patients with dyspepsia that were referred to gastrointestinal department of Firoozgar Hospital between May 2007 and May 2008 were included in our study. Our study was confirmed by the Tehran University ethical committee and a signed informed consent was filled by each participant. Dyspepsia as the main inclusion criteria for our participants was confirmed by the Rome criteria as persistent or recurrent pain or discomfort centered in the upper abdomen or epigastrium (19). A structured questionnaire containing demogheraphic data, recent use of antisecretory drugs and past medical history was filled individually.

Patients were excluded in the presence of any of the following histories: 1. Eradication therapy or use of antisecretory drugs or antibiotics in the previous 6 months, 2. Surgery on upper GI tract, 3. Gastric cancer and 4. Using non steroidal anti-inflammatory drugs.
H. pylori in diabetic and non-diabetic patients with dyspepsia

We checked fasting blood sugar (FBS) for all of the study samples with at least 8 hours fasting period. Patients with fasting blood sugar higher than 126 mg/dl in two separated samples were considered diabetic according to the American Diabetes Association criteria (20). All patients underwent an upper endoscopy. Biopsy specimens were obtained from the antrum and the corpus. The specimens were colored by Giemsa stains in order to check the presence of H. pylori. A single pathologist, who was blind to the endoscopic findings of our samples, evaluated the slides histologically. In evaluation of patients with Giemsa coloring system in pathology laboratory, gastritis was classified as mild-chronic gastritis, mild to moderate-chronic inactive gastritis, moderate to severe chronic active gastritis, and normal pathological findings.

Statistical methods
Statistical analysis was done by SPSS for Windows version 14. Chi-square for qualitative and independent sample t-tests for quantitative variables were used in data analysis. Two-tailed significance level at 0.05 was used to detect the difference between variables.

Results
Among the patients in our study, 50 patients (62.5%) were diabetic and 30 patients (37.5%) were non-diabetic. Forty four patients (55%) were female and 36 patients (44%) were male. The overall mean age was 52.10±18.15 years. The mean of age was 53.45±15.74 and 50.44±20.84 years old in female and male patients respectively.

Pathologic findings in diabetic and non-diabetic patients
In the histological evaluation of patients with Giemsa staining system in pathology laboratory, 14(28%) diabetic and 4(13.33%) non-diabetic patients had mild chronic gastritis, 10(20%) diabetic and 12(40%) non-diabetic patients had mild to moderate chronic inactive gastritis, 16(32%) diabetic and 2(6.66%) non-diabetic patients had moderate chronic active gastritis, 2(4%) diabetic patients had moderate to severe chronic active gastritis, and 8(16%) diabetic and 12(40%) non-diabetic patients had normal pathological findings. The prevalence of gastritis did differ significantly between the two groups (P=0.001) (Table 1).

Table 1. Prevalence of H. pylori and gastritis in diabetics (DM) and non-diabetics control group.

<table>
<thead>
<tr>
<th></th>
<th>DM n(%)</th>
<th>Control n(%)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gastritis</td>
<td>42(84%)</td>
<td>18(60%)</td>
<td>0.001</td>
</tr>
<tr>
<td>H. pylori</td>
<td>30(60%)</td>
<td>8(26.66%)</td>
<td>0.001</td>
</tr>
</tbody>
</table>

H. pylori infection in diabetic and non-diabetic patients
The prevalence of H. pylori infection was 60%(30/50) and 26.66%(8/30) respectively, among diabetic patients and non-diabetics and difference between the groups was statistically significant (P<0.001) (Table1).

Discussion
Our data provide direct evidence for a higher prevalence of H. pylori infection in diabetic dyspeptic patients than in non-diabetics. We examined 80 patients with dyspepsia referred to our endoscopic ward. Dyspepsia is a common symptom in individuals with diabetics (21) and this could explain the high proportion of patients with diabetes (50/80, 62.5%) in this study.

The prevalence of H. pylori infection in diabetic patients was different in previous reports. The variability of prevalence rates may be related to the epidemiological distribution of H. pylori or the kind of diagnostic method to detect of infection. A seroprevalence study performed in Netherland reported that the frequency of H. pylori infection was higher in diabetic patients in comparison with the control subjects (4). Another seroprevalence study in United Arab Emirates showed that positive antibody titer for H. pylori infection (IgA>250) in diabetics was 63.3% compared to non-diabetics 48.1% (P<0.001), similarly, according to IgG antibody titer (IgG>300), H. pylori infection was determined in diabetic patients at a rate of 76.7% compared to an infection rate of 64.8% in non-diabetics (P=0.009) (22).

H. pylori infection was documented by histology of gastrointestinal mucosa in 74.4% of the diabetics and in 50% of the controls (P<0.01) (23). Similar data showed by Morrollo et al., they reported that prevalence of H. pylori infection was significantly higher in diabetics than in controls (24). Two other studies showed that the prevalence of H. pylori infection in diabetics by rapid urease test and detection of HpSA (stool antigen positive
in 73% and 51.4% of diabetics and non-diabetics, respectively) was statistically significant (25,26).

There are some studies that showed no association between diabetes mellitus and H. pylori infection. Gasbarrini et al. reported the same prevalence of H. Pylori infection in patients with diabetes type I and the control group (37 vs 34%, respectively) (27). Mallecki et al., in their study found that prevalence of H. pylori infection in diabetic patients was 30% and significantly lower than controls (68%) (28). In a seroprevalence study frequency of H. pylori infection was 33% and 32%, respectively, in patients with diabetes and controls and authors concluded that H. pylori infection appears not to be associated with diabetes (29).

Anastasios et al., study didn’t support an association between H. pylori infection and diabetes (30). The prevalence of H. pylori infection between diabetics was 37.3% and 35.2% in non-diabetics (P=0.78) (30). Demir et al. showed that the prevalence of H. pylori infection was 61.7% and 58.5%, respectively, among type 2 diabetics and non-diabetics and was not statistically significant (P=0.577) (31).

Several hypotheses were presented for confirmation of higher prevalence of H. pylori infection in diabetic patients such as immune system impairment in patients with diabetes mellitus, the reduction of both gastrointestinal motility and acid secretion and higher secretion of pro-inflammatory cytokines related to the H. pylori gastric infection itself (22).

H. pylori infection always exists with inflammation in the stomach and leads to chronic gastritis (32), but more severe diseases such as peptic ulcer and gastric cancer develop in a small proportion of infected individuals (33). Our result was comparable with Morrollo et al. study that found chronic gastritis and H. pylori infection were significantly higher in diabetics (24), and this finding can be attributed to the diabetes-induced achlorhydria. Diabetics might be assumed to be more vulnerable to the harmful effects of H. pylori due to their impaired immune status. In contrast, Anastasios et al. showed that the prevalence gastritis did not differ significantly between diabetics and non-diabetics (30).

In conclusion, results of the present study showed that the prevalence of H. pylori infection in diabetes mellitus patients with dyspepsia is higher than non-diabetics. H. pylori infection was significantly associated both with the presence of chronic gastritis in diabetic patients. According to our results, diabetes mellitus is one of the risk factors that must be considered in the evaluation of H. pylori infection with dyspepsia.

Our study had several limitations such as small number of patients and using one method to detect H. pylori.

Acknowledgment

The authors would like to thank Dr. Leila Zahedi Shoolami for her excellent assistance.

References

H. pylori in diabetic and non-diabetic patients with dyspepsia


