Helicobacter pylori in Diabetic and Non-Diabetic Patients with Dyspepsia

Mahshid Talebi-Taher[1], Manizheh Mashayekhi[2], Mohammad Hossein Hashemi[2], and Vanosheh Bahrani[2]

[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.

Acta Medica Iranica, 2012; 50(5): 315-318.

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.

Corresponding Author: Mahshid Talebi-Taher
Antimicrobial Resistance Research Center, Rasoul-e- Akram Hospital, Tehran University of Medical Sciences, Tehran, Iran
Tel: +98 21 66507056, E-mail:mtalebitaher2000@yahoo.com, m-talebitaher@tums.ac.ir

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 quantitive 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.

	DM n(%)	Control n(%)	*P*-value
Gastritis	42(84%)	18(60%)	0.001
H. pylori	30(60%)	8(26.66%)	0.001

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 be 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 diabetics 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 achlorydria. 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

1. Graham DY. Campylobacter pylori and peptic ulcer disease. Gastroenterology 1989;96(2 Pt 2 Suppl):615-25.
2. Forman D, Newell DG, Fullerton F, Yarnell JW, Stacey AR, Wald N, Sitas F. Association between infection with Helicobacter pylori and risk of gastric cancer: evidence from a prospective investigation. BMJ 1991;302(6788):1302-5.
3. Parsonnet J, Hansen S, Rodriguez L, Gelb AB, Warnke RA, Jellum E, Orentreich N, Vogelman JH, Friedman GD. Helicobacter pylori infection and gastric lymphoma. N Engl J Med 1994;330(18):1267-71.
4. Oldenburg B, Diepersloot RJ, Hoekstra JB. High seroprevalence of Helicobacter pylori in diabetes mellitus patients. Dig Dis Sci 1996;41(3):458-61.
5. Perdichizzi G, Bottari M, Pallio S, Fera MT, Carbone M, Barresi G. Gastric infection by Helicobacter pylori and antral gastritis in hyperglycemic obese and in diabetic subjects. New Microbiol 1996;19(2):149-54.
6. Kojecký V, Roubalík J, Bartoníková N. Helicobacter pylori in patients with diabetes mellitus. Vnitr Lek 1993;39(6):581-4.
7. Schvarcz E, Palmér M, Ingberg CM, Aman J, Berne C. Increased prevalence of upper gastrointestinal symptoms in long-term type 1 diabetes mellitus. Diabet Med 1996;13(5):478-81.
8. Spångéus A, El-Salhy M, Suhr O, Eriksson J, Lithner F. Prevalence of gastrointestinal symptoms in young and middle-aged diabetic patients. Scand J Gastroenterol 1999;34(12):1196-202.
9. Ko GT, Chan WB, Chan JC, Tsang LW, Cockram CS. Gastrointestinal symptoms in Chinese patients with Type 2 diabetes mellitus. Diabet Med 1999;16(8):670-4.
10. Clouse RE, Lustman PJ. Gastrointestinal symptoms in diabetic patients: lack of association with neuropathy. Am J Gastroenterol 1989;84(8):868-72.
11. Lluch I, Ascaso JF, Mora F, Minguez M, Peña A, Hernandez A, Benages A. Gastroesophageal reflux in diabetes mellitus. Am J Gastroenterol 1999;94(4):919-24.

12. Rathmann W, Enck P, Frieling T, Gries FA. Visceral afferent neuropathy in diabetic gastroparesis. Diabetes Care 1991;14(11):1086-9.
13. Annese V, Bassotti G, Caruso N, De Cosmo S, Gabbrielli A, Modoni S, Frusciante V, Andriulli A. Gastrointestinal motor dysfunction, symptoms, and neuropathy in noninsulin-dependent (type 2) diabetes mellitus. J Clin Gastroenterol 1999;29(2):171-7.
14. Jones KL, Horowitz M, Berry M, Wishart JM, Guha S. Blood glucose concentration influences postprandial fullness in IDDM. Diabetes Care 1997;20(7):1141-6.
15. Talley NJ, Young L, Bytzer P, Hammer J, Leemon M, Jones M, Horowitz M. Impact of chronic gastrointestinal symptoms in diabetes mellitus on health-related quality of life. Am J Gastroenterol 2001;96(1):71-6.
16. Simon L, Tornóczky J, Tóth M, Jámbor M, Sudár Z. The significance of Campylobacter pylori infection in gastroenterologic and diabetic practice. Orv Hetil 1989;130(25):1325-9.
17. Gasbarrini A, Ojetti V, Pitocco D, Franceschi F, Candelli M, Torre ES, Gabrielli M, Cammarota G, Armuzzi A, Pola R, Pola P, Ghirlanda G, Gasbarrini G. Insulin-dependent diabetes mellitus affects eradication rate of Helicobacter pylori infection. Eur J Gastroenterol Hepatol 1999;11(7):713-6.
18. Gasbarrini A, Ojetti V, Pitocco D, Armuzzi A, Silveri NG, Pola P, Ghirlanda G, Gasbarrini G. Efficacy of different Helicobacter pylori eradication regimens in patients affected by insulin-dependent diabetes mellitus. Scand J Gastroenterol 2000;35(3):260-3.
19. Talley NJ, Stanghellini V, Heading RC, Koch KL, Malagelada JR, Tytgat GN. Functional gastroduodenal disorders. Gut 1999;45 Suppl 2:II37-42.
20. Report of the Expert Committee on the Diagnosis and Treatment of Diabetes Mellitus. Diabetes Care 1997;20(7):1183-97.
21. Ricci JA, Siddique R, Stewart WF, Sandler RS, Sloan S, Farup CE. Upper gastrointestinal symptoms in a U.S. national sample of adults with diabetes. Scand J Gastroenterol 2000;35(2):152-9.
22. Bener A, Micallef R, Afifi M, Derbala M, Al-Mulla HM, Usmani MA. Association between type 2 diabetes mellitus and Helicobacter pylori infection. Turk J Gastroenterol 2007;18(4):225-9.
23. Gentile S, Turco S, Oliviero B, Torella R. The role of autonomic neuropathy as a risk factor of Helicobacter pylori infection in dyspeptic patients with type 2 diabetes mellitus. Diabetes Res Clin Pract 1998;42(1):41-8.
24. Marrollo M, Latella G, Melideo D, Storelli E, Iannarelli R, Stornelli P, Valenti M, Caprilli R. Increased prevalence of Helicobacter pylori in patients with diabetes mellitus. Dig Liver Dis 2001;33(1):21-9.
25. Agrawal RP, Sharma R, Garg D, Pokharna R, Kochar DK, Kothari RP. Role of Helicobacter pylori in causation of diabetic gastropathies and non-gastrointestinal complications in type 2 diabetes. J Indian Med Assoc 2010;108(3):140-3.
26. Devrajani BR, Shah SZ, Soomro AA, Devrajani T. Type 2 diabetes mellitus: A risk factor for Helicobacter pylori infection: A hospital based case-control study. Int J Diabetes Dev Ctries 2010;30(1):22-6.
27. Gasbarrini A, Ojetti V, Pitocco D, De Luca A, Franceschi F, Candelli M, Sanz Torre E, Pola P, Ghirlanda G, Gasbarrini G. Helicobacter pylori infection in patients affected by insulin-dependent diabetes mellitus. Eur J Gastroenterol Hepatol 1998;10(6):469-72.
28. Małlecki M, Bień AI, Galicka-Latałła D, Stachura J, Sieradzki J. The prevalence of Helicobacter pylori infection and types of gastritis in diabetic patients. The Kraków Study. Exp Clin Endocrinol Diabetes 1996;104(5):365-9.
29. Xia HH, Talley NJ, Kam EP, Young LJ, Hammer J, Horowitz M. Helicobacter pylori infection is not associated with diabetes mellitus, nor with upper gastrointestinal symptoms in diabetes mellitus. Am J Gastroenterol 2001;96(4):1039-46.
30. Anastasios R, Goritsas C, Papamihail C, Trigidou R, Garzonis P, Ferti A. Helicobacter pylori infection in diabetic patients: prevalence and endoscopic findings. Eur J Intern Med 2002;13(6):376.
31. Demir M, Gokturk HS, Ozturk NA, Kulaksizoglu M, Serin E, Yilmaz U. Helicobacter pylori prevalence in diabetes mellitus patients with dyspeptic symptoms and its relationship to glycemic control and late complications. Dig Dis Sci 2008;53(10):2646-9.
32. Türkay C, Erbayrak M, Bavbek N, Yenıdünya S, Eraslan E, Kasapoğlu B. Helicobacter pylori and histopathological findings in patients with dyspepsia. Turk J Gastroenterol 2011;22(2):122-7.
33. Nguyen TL, Uchida T, Tsukamoto Y, Trinh DT, Ta L, Mai BH, Le SH, Thai KD, Ho DD, Hoang HH, Matsuhisa T, Okimoto T, Kodama M, Murakami K, Fujioka T, Yamaoka Y, Moriyama M. Helicobacter pylori infection and gastroduodenal diseases in Vietnam: a cross-sectional, hospital-based study. BMC Gastroenterol 2010;10:114.