

UPPER GASTROINTESTINAL ENDOSCOPIC FINDINGS AND HELICOBACTER PYLORI INFECTION: DIABETIC VERSUS NON-DIABETIC DYSPEPTIC PATIENTS IN SULAIMANI CITY



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ABSTRACT

Background

Diabetes Mellitus is a chronic lifelong condition that widely affects whole body systems. The gastrointestinal tract is one of these systems that is significantly affected by diabetes mellitus. Infection is common and is sometimes severe among diabetic patients.

Objectives

To determine the association between *H. pylori* infection and diabetes mellitus and to show any difference in upper GI endoscopic findings between diabetic and non-diabetic patients who presented with dyspepsia.

Patients and Methods

This study is a retrospective study. Sixty diabetic and 60 non-diabetic patients were enrolled in this study, all presented with dyspeptic symptoms, underwent upper GI endoscopy. *H. pylori* status was evaluated. Glycemic control of diabetic patients was assessed by HbA1c. Comparison between diabetic and non-diabetic patients done.

Results

The prevalence of *H. pylori* infection was 81.66% among diabetic patients, and 68.33% among nondiabetic patients and the difference in the occurrence of *H. pylori* between those 2 groups statistically not significant. The duration of diabetes and level of HbA1c were not related to the prevalence of *H. pylori* infection. The upper GI endoscopic findings were not significantly different between those two groups.

Conclusions

Our study does reveal that although *H. pylori* infection is more common among diabetics than nondiabetic patients such difference was not statistically significant. There was no significant difference in upper GI endoscopic findings between diabetic and non-diabetic groups.

Keywords: *Diabetes, H. pylori, Endoscopy.*

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INTRODUCTION

Diabetes is a group of metabolic diseases characterized by hyperglycemia resulting from defects in insulin secretion, insulin action, or both ⁽¹⁾. People with type 2 diabetes are at elevated risk for a number of serious health problems, including cardiovascular disease, premature death, blindness, kidney failure, amputations, fractures, frailty, depression, and cognitive decline and various gastrointestinal and hepatic manifestations ⁽²⁾.

Dyspepsia is a term describing a symptom complex thought to originate in the upper gastrointestinal (GI) tract. Epigastric pain or discomfort are the cardinal symptoms and may be associated with other symptoms such as postprandial heaviness, early satiety, and nausea. Heartburn and acid regurgitation is now not included in the definition of dyspepsia because they are usually meaning GERD or functional heartburn ⁽³⁾.

Gastrointestinal (GI) symptoms are reportedly common in diabetes mellitus and are usually attributed to autonomic neuropathy ⁽⁴⁾. Although there is evidence that GI symptoms affect the quality of life adversely ⁽⁵⁾ and represent a substantial cause of morbidity in patients with diabetes. Diabetes is associated with increased prevalence of upper and lower GI symptoms related to poor glycemic control, not the duration of the disease, the available epidemiological data relating to the prevalence of GI symptoms in diabetes are conflicting and can be challenged on methodological grounds. Gastroparesis is the prototype of gastric manifestation of DM related to the autonomic neuropathy ⁽⁶⁾.

Diabetes mellitus (DM) often results in motility disorders in the gastrointestinal tract ⁽⁷⁻⁹⁾. Gastrointestinal and hepatic involvement can present with esophageal dysmotility, gastro-esophageal reflux disease (GERD), gastroparesis, enteropathy, nonalcoholic fatty liver disease (NAFLD) and glycogenic hepatopathy ⁽¹⁰⁾. Furthermore, some patients with DM, particularly those with type 2 DM, are severely obese, which increases intra-abdominal pressure.

Helicobacter pylori infection affects approximately 50% of the world's population ^(11,12) and is recognized as the major acquired factor in the pathogenesis of chronic antral gastritis, peptic ulcer disease, and gastric cancer. Identification of risk groups is very important in this respect. Some reports suggested that *H. pylori* might have a high prevalence among patients with diabetes ⁽¹³⁻¹⁵⁾. Impairment of immune system is thought to be responsible for more frequent and more severe infections in diabetic patients ⁽¹⁶⁾.

PATIENTS AND METHODS

In a retrospective study 120 patients were enrolled, 60 diabetic patients and 60 non-diabetic patients. All patients presented with dyspeptic symptoms (epigastric pain, early satiety, postprandial fullness, bloating, etc.). *H. pylori* status was evaluated by the urea breath test, stool *H. pylori* Ag or serology (IgA and IgG titer by ELISA). The patients didn't receive *H. pylori* eradication before, and those underwent a urea breath test and stool *H. pylori* Ag stopped proton pump inhibitors for 2 weeks and antibiotics for 4 weeks before such testing. All patients underwent upper GI endoscopy under conscious sedation by a gastroenterology specialist, and they were fasting for 8 hours. Diabetic control was assessed by HbA1c. Exclusion criteria in this study are an age less than 20 years, pregnancy, history of GIT malignancy, history of Gallstones or ultrasound of abdomen showing gallstones, acute gastroenteritis, history of a chronic liver disease, Aspirin, and NSAID users, and Type 1 diabetes mellitus. Data were collected and entered into SPSS program version 23 and analysis were done.

RESULTS

One hundred and twenty patients were enrolled in this study, 60 patients were diabetic, the mean age was 50.2 ± 9.56 , and 37 (61.6%) were male, while 23 (38.33%) were female. The remaining patients were non-diabetic, their mean age was 46.07 ± 10.19 , and 31 (51.6%) were male, while 29 (48.33%) were female.

H. pylori infection was more common among diabetic patients and the result was 49/60 patients (81.66%), while in non-diabetics the prevalence was 41/60 (68.33%), but the difference was statistically not significant and the *P* value was 0.72.

The infection with *H. pylori* among diabetic patients was not related to the duration of the disease and the level of control of diabetes status (HbA1c) and the *P* values were 0.095, 0.52, respectively (Table 1). The age and gender did not increase the risk of *H. pylori* infection among diabetic patients and their *P* values were 0.099 and 0.125, respectively. In non-diabetic patients, the age and gender did not increase the risk of *H. pylori* infection and their *P* values were 0.69 and 0.51 respectively.

The upper GI endoscopic findings (GERD, esophageal candidiasis, gastropathy, gastric and duodenal ulcers, gastric polyps, gastric stasis and malignant looking masses) were not significantly different between diabetic and non-diabetic groups (Table 2).

Table 1. The average level of HbA1c and the duration of diabetes in patients with or without *H. pylori* infection.

Diabetics	<i>H.pylori</i> positive	<i>H.pylori</i> negative	p. value
HbA1c	8.27±1.33%	7.68±1.54%	0.095
Duration	6.67±4.29 yrs	5.3±3.26 yrs	0.520

Table 2. The upper GI endoscopic findings between diabetic and non-diabetic patients.

OGD findings	Diabetic	Non-diabetic
Normal	3 (5%)	4 (6.66%)
Antral Gastropathy	18 (30%)	20 (33.33%)
GERD	18 (30%)	20 (33.33%)
DU	15 (25%)	11 (18.33%)
Gastric polyp	1 (1.66%)	1 (1.6%)
Malignant GOO	2 (3.33%)	0
Gastric ulcer	0	2(3.33%)
Gastric stasis	2 (3.33%)	0
Serrated duodenal mucosa	0	1(1.66%)
Esophageal candidiasis	1 (1.66%)	0

DISCUSSION

In reviewing the studies that have been done till now there is a clear contradictory in the prevalence of *H. pylori* infection and upper GI endoscopy findings between diabetic patients and non-diabetic patients with dyspepsia. In some reports there is an increased risk of *H. pylori* infection in diabetic patients, these studies depending on serology for the detection of *H. pylori* (17, 13, 18). Other recent studies are showing that there is no significant relation between *H. pylori* infection and diabetes (19-22), and upper GI endoscopic findings are not different significantly between diabetic patients and non-diabetics.

The prevalence of *H.pylori* infection among diabetic patients is between (30%-80%) (23), in our study the prevalence of *H. pylori* infection among diabetics is high because our area is endemic for *H. pylori* infection, and there is a difference between these two groups but statistically not significant.

It is well known that diabetic patients are liable to get an infection, because of suppressed immunity, but there is another mechanism in which the secretion of hydrochloric acid decreased by the effect of diabetes on gastric mucosa that is harmful to the *H. pylori* bacteria (24). In this study, the duration of diabetes and the level of HbA1c increase the risk of *H. pylori* infection but statistically not significant.

In conclusion, although *H. pylori* infection is more common among diabetics than nondiabetic patients such difference was not statistically significant. There was no significant difference in upper GI endoscopic findings between diabetic and non-diabetic groups.

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