THE PREVALENCE OF HELICOBACTER PYLORI INFECTION IN DIABETIC PATIENTS AND ITS RELATION TO THE PRESENCE OF GASTROINTESTINAL TRACT COMPLICATIONS

Osama Mohamady¹, Nagwa Ramadan², Heba Arnaout³

¹,²Department of internal medicine Faculty of medicine, Cairo University,
³Department of microbiology and immunology. Faculty of medicine, Cairo University (EGYPT)
E-mails: os7631092@hotmail.com, dr_nagwa2001@yahoo.com, hananzaghla@hotmail.com

ABSTRACT

Background: it’s well known that diabetic patients are more prone to infection. In these patients, chronic infections are frequent and severe due to impairment of their immune system. The relationship between H. pylori infection and diabetes mellitus have shown in some studies but the relationships remain controversial.

Aim of The study: was to determine the prevalence of Helicobacter pylori infection (H. pylori) among diabetic patients (type1 and type 2 diabetes mellitus) and the relation of H. pylori infection to gastrointestinal (GI) complications in diabetics.

Subjects and methods: the study included 100 subjects were divided into 2 groups. Group I 50 patients with diabetes mellitus and have dyspeptic symptoms, group II 50 non-diabetic with dyspeptic symptoms. This is a case and control study comparison of diabetic and non-diabetic groups. The study was conducted at Police Hospital Cairo during the period from (January-December 2012).

Methods: H. pylori were assessed by H. pylori stool antigen (HpSAg) test among diabetic and non-diabetic group.

Results: a positive cases for H. pylori infection by (HpSAg) test was 61.1% in type 1 diabetic patients and 65.6% in type 2 diabetic patients compared to 50% of the non-diabetic group (p 0.36) non significant (N.S). The prevalence of gastrointestinal symptoms in H. pylori positive diabetic patients as regarding dyspepsia (62.5%), early satiety (56.25%), heart burn (62.5%), bloating (25%), diarrhea (15.63%), constipation (25%), nausea (43.75%), vomitng (9.38%) and abdominal pain (53.13%), but by comparison with negative diabetic group these was statistically insignificant.

Glycosylated Hb (HBA1c) was higher among positive cases, but yet not significant, (p 0.07). Also, FBS (p 0.08) and PPBS (p 0.1) were not significant. The Presence of H. pylori not associated with increase duration of diabetes. The mean age of diabetic patients positive for H. pylori was 48.750±11.09 years compared to 40.167±12.76 years in diabetic patients negative for H. pylori infection (p 0.017) significant (s).

Conclusion: the study reported no significant association between H. pylori infection and the prevalence of diabetes, but there is a borderline increased risk for H. pylori infection in diabetic among participants with a BMI greater than 25 kg/m².

Key words: H. pylori, diabetes mellitus, gastrointestinal tract (GIT) complications

1. INTRODUCTION

Diabetes mellitus is a disease known for production of chronic complications in diabetic patients (1). Type 2 diabetes is an emerging pandemic, with 3.8 million deaths in adult attributed to the disease (2). Infection with H. pylori is acquired in early childhood and if left untreated it becomes a chronic infection (3). The majority of infected people remain asymptomatic while only a small proportion develops illness and this is usually in adulthood (4).

Infection with H. pylori causes gastric and duodenal ulcers and it increase risk of gastric cancer (5), also it increase the likelihood of gastric lymphoma (6). Moreover positive association between H. pylori and extragastric manifestations as iron deficiency anaemia (7) and idiopathic thrombocytopenic purpura (8) have been reported.

Strains of H. pylori play role in the homeostasis of leptin and ghrelin, these 2 hormones are critical to energy homeostasis and metabolism. Infection with H. pylori is associated with chronic inflammation particularly among strains of H. pylori which contain the cag antigen. However, there is evidence that this inflammation may extend beyond the (GIT) to affect insulin and glucose metabolism (9).

In recent years there is a significant association has been reported between H. pylori infection and cardiovascular disease, diabetes and dyslipidemia (10). However there are conflicting reports (11). Reports concerning association between H. pylori infection and various clinical manifestations of the metabolic or insulin-resistant syndrome are conflicting (12). The roles of infection with H. pylori in CVD, and its risk factor (13) and in type 2 diabetes mellitus are not clear.
Although there is no association was founded between H. pylori sero-positive immunoglobulin G and diabetes mellitus (14).

The relationship between H. pylori infection and diabetes mellitus have shown in some studies but the relationships remain controversial (15).

The aim of this study was to determine the prevalence of Helicobacter pylori infection among diabetic patients (type1 and type 2 diabetes mellitus) and the relation of H. pylori infection to gastrointestinal complications in diabetics.

2. SUBJECTS AND METHODS

Patients were conducted from out patient’s clinic and inpatient ward of internal medicine department of Police Hospital Cairo, over 12-month period (January – December 2012). All patients had to sign a consent which was approved by our institutional ethical committee. They were classified into the following groups:

- **Group I**: include 50 subjects’ (18 patients of type 1 diabetes mellitus and 32 patients of type 2 diabetes mellitus) with dyspeptic symptoms.
- **Group II**: include 50 subjects’ non diabetic control group with dyspeptic symptoms.

**Selection of Diabetic Subjects**

Persons were classified as diabetic according to the American Diabetic Association (ADA) criteria (16), or if they were on diabetic medication.

**Selection of Non-Diabetic Subjects**

Non-diabetic (control) subjects aged 18-60 years were identified from the community if their venous blood glucose values were <6.1 mmol/L and if they had never taken any diabetic medication. Control subjects were proven as non-diabetic by 75-g oral glucose tolerance test (OGTT) which was performed according to the World Health organization (WHO) criteria (17).

All subjects were selected upon patients with dyspeptic symptoms and all patients have been subjected to the following:

- **Questionnaire**
  
  We assessed the GI symptoms of patients using "The GI symptom Questionnaire," which was designed based on The (GSRS) GIT symptom rating scale and the Rome III criteria for functional GI disorders (18). The questionnaire is including questions about GI symptoms (frequency, strength, and patterns of GI symptoms), lifestyle, social activities, history of disease, visits to a hospital or clinic, and medications.
  
  For diabetic patients the questionnaire had more questions related to diabetes and specifically disease duration, glucose monitoring, treatment, and diabetic complication.
  
  On other hand the health status was assessed by recording their pulse, blood pressure, lower limb edema, height and weight with measurement of body mass index (BMI). Chest and heart examination were done for exclusion of other causes of gastritis and for detection of any abnormalities in heart. Abdomen examination to detect the presence of any organomegaly and free intraperitoneal fluid.

- **Data Collection Procedure**

  1. For the assessment of diabetes mellitus we had taken venous blood sample and send to laboratory for fasting blood sugar (FBS) level, 2 hour-post prandial blood sugar (2h-PPBS), random blood sugar (RBS) level, glycosylated hemoglobin (HBA1c) and 75-g oral glucose tolerance test.
  
  2. For detection of *Helicobacter pylori* infection we advised the participants for collection of stool sample and send to laboratory for *Helicobacter pylori* stool antigen (HpSA) by Enzyme immunoassay (EIA).

- **H.pylori stool antigen (HpSAg) test:**
  
  The new enzyme linked immunooassay Helicobacter pylori stools antigen (HpSA) is a highly sensitive and specific, non-invasive diagnostic tool for the detection of H. pylori, even for monitoring the efficacy of treatment. It is not time consuming and cheap (19).

  Choi et al (2011) (20) concluded that HpSA showed good diagnostic performance for H.pylori infection with over all sensitivity, specificity, positive and negative predictive value, and accuracy were 93.1%, 94.6%, 95.1%, 92.3% and 93.8% respectively.

**Exclusion criteria**

1. Patients on proto pump inhibitor (PPI), bismuth, H2 antagonists or antimicrobial.
2. Patients already on steroid, immunosuppressive therapy or H. pylori eradication therapy.
3. Patients with cholecystitis or GI major surgery.

**STATISTICAL ANALYSIS**

The collected data was organized, tabulated and statistically analyzed using spss software version for quantitative data, the range, mean and standard deviation were calculated. For qualitative data comparison
between two groups was done using chi -square test ($\chi^2$). For comparison between mean of two groups student t-test was used. For comparison between more than two means the F value of analysis of variance and schafee test was calculated.

3. RESULTS

In group I we had 18 patients with type 1 DM (male=10, female= 8), age of patients ranged from 18 to 40 years, mean of height was (167.33±7.30), mean of weight was (75.67±15.48) and mean of BMI was (26.13±4.82). Also in group I we had 32 patients with type 2 DM (male=16, female=16), age of patients with Type 2 DM ranged from 41 to 60 years, mean of height was (165.16±5.46), mean of weight was (79.88±11.09) and mean of BMI was (29.19±3.50)

In group II we had 50 non-diabetic control group with dyspeptic symptoms (male=26, female =24), age ranged from 22 to 60 years, mean of height was (167.70±7.05), mean of weight was (75.14±9.20) and mean of BMI was (27.86± 2.55).

The H. pylori infection was higher in diabetics than control but it was insignificant (p value=0.36 N.S). So, there is no significant association between H. pylori and diabetes mellitus as shown in table (1).

### Table 1. Frequency of detected H. pylori infection among diabetic and control patients by HPSA test

<table>
<thead>
<tr>
<th>H. pylori infection by HPSA test</th>
<th>Type 1 DM N=18</th>
<th>Type 2 DM N=32</th>
<th>CONTROL N=50</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N</td>
<td>%</td>
<td>N</td>
</tr>
<tr>
<td>+ve</td>
<td>11</td>
<td>61.1%</td>
<td>21</td>
</tr>
<tr>
<td>-ve</td>
<td>7</td>
<td>38.9%</td>
<td>11</td>
</tr>
<tr>
<td><strong>$\chi^2$</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>P</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

By comparison among diabetic patients in relation to detection of H. pylori infection as regards gastrointestinal symptoms, there was insignificant difference of GIT symptoms between H.pylori +ve /–ve diabetic patients. As shown in table (2).

### Table 2. Gastrointestinal symptoms among diabetic patients according to detection of H. pylori infection

<table>
<thead>
<tr>
<th>Symptoms of diabetic patients</th>
<th>+ve HP/DM N=32</th>
<th>-ve HP/DM N=18</th>
<th><strong>P value</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>Dyspepsia</td>
<td>20</td>
<td>11</td>
<td>0.95</td>
</tr>
<tr>
<td>Early satiety</td>
<td>18</td>
<td>13</td>
<td>1.0</td>
</tr>
<tr>
<td>Heart burn</td>
<td>20</td>
<td>10</td>
<td>0.85</td>
</tr>
<tr>
<td>Bloating</td>
<td>8</td>
<td>5</td>
<td>0.85</td>
</tr>
<tr>
<td>Diarrhea</td>
<td>6</td>
<td>3</td>
<td>1.0</td>
</tr>
<tr>
<td>Constipation</td>
<td>8</td>
<td>6</td>
<td>0.8</td>
</tr>
<tr>
<td>Nausea</td>
<td>14</td>
<td>8</td>
<td>0.37</td>
</tr>
<tr>
<td>Vomiting</td>
<td>3</td>
<td>1</td>
<td>1.0</td>
</tr>
<tr>
<td>Abdominal pain</td>
<td>17</td>
<td>10</td>
<td>0.93</td>
</tr>
</tbody>
</table>

Glycosylated Hb was higher among positive cases, but yet not significant, p 0.07. Also, FBS (p 0.08) and 2h-PPBS (p 0.1) were not significant. As shown in table (3).
Table 3. FBS, 2h-PPBS and HBA1c according to H. pylori infection in diabetic patients

<table>
<thead>
<tr>
<th></th>
<th>HP</th>
<th>N</th>
<th>Mean</th>
<th>Std. Deviation</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Positive</td>
<td>32</td>
<td>165</td>
<td>18.0919</td>
<td>0.06</td>
</tr>
<tr>
<td></td>
<td>Negative</td>
<td>18</td>
<td>157.05</td>
<td>13.5189</td>
<td></td>
</tr>
<tr>
<td>2h-PPBS</td>
<td>Positive</td>
<td>32</td>
<td>270.82</td>
<td>39.2354</td>
<td>0.1</td>
</tr>
<tr>
<td></td>
<td>Negative</td>
<td>18</td>
<td>250.0</td>
<td>43.3861</td>
<td></td>
</tr>
<tr>
<td>HBA1c</td>
<td>Positive</td>
<td>32</td>
<td>9.844</td>
<td>1.6531</td>
<td>0.07</td>
</tr>
<tr>
<td></td>
<td>Negative</td>
<td>18</td>
<td>9.000</td>
<td>1.3612</td>
<td></td>
</tr>
</tbody>
</table>

There was no positive association between H. pylori and duration of diabetes of studied cases i.e. Presence of H. pylori not associated with increase duration of diabetes. As shown in table (4).

Table 4. Mean value of duration of DM of diabetic patients in relation to detection of H. pylori infection by HPSA test

<table>
<thead>
<tr>
<th>Diabetic patients n=50</th>
<th>Type 1 DM N=18</th>
<th>Type 2 DM N=32</th>
</tr>
</thead>
<tbody>
<tr>
<td>Duration of DM(years)</td>
<td>4-10</td>
<td>5-15</td>
</tr>
<tr>
<td>H.pylori +ve cases</td>
<td>11</td>
<td>21</td>
</tr>
<tr>
<td>Mean SD</td>
<td>7.7272</td>
<td>8.47</td>
</tr>
<tr>
<td>H.pylori –ve cases</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>Mean SD</td>
<td>1.154</td>
<td>1.549</td>
</tr>
<tr>
<td>T test</td>
<td>1.671</td>
<td></td>
</tr>
<tr>
<td>P Value</td>
<td>0.100 (N.S)</td>
<td></td>
</tr>
</tbody>
</table>

We found statistically significant increase in H. pylori infection with increase age (p < 0.001), so increase in age is related to increase H. pylori infection. As shown in table (5).

Table 5. Mean value of age of diabetic patients in relation to detection of H. pylori infection by HPSA test

<table>
<thead>
<tr>
<th>Diabetic patients HP</th>
<th>N</th>
<th>Mean</th>
<th>Std. Deviation</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>AGE</td>
<td></td>
<td></td>
<td></td>
<td>0.017</td>
</tr>
<tr>
<td>Positive</td>
<td>32</td>
<td>48.750</td>
<td>11.0949</td>
<td></td>
</tr>
<tr>
<td>Negative</td>
<td>18</td>
<td>40.167</td>
<td>12.7660</td>
<td></td>
</tr>
<tr>
<td>T test</td>
<td></td>
<td>2.485</td>
<td></td>
<td></td>
</tr>
<tr>
<td>P value</td>
<td></td>
<td>0.017 *</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

In our study we demonstrate that increase in H. pylori infection with increase of BMI and this was statistically significant (p < 0.001). So, there is positive association between H. pylori and BMI. As shown in table (6).

Table 6. Mean value of BMI of diabetic patients in relation to detection of H. pylori infection by HPSA test

<table>
<thead>
<tr>
<th>HP</th>
<th>N</th>
<th>Mean</th>
<th>Std. Deviation</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>BMI</td>
<td>Positive</td>
<td>32</td>
<td>30.763</td>
<td>2.8103</td>
</tr>
<tr>
<td></td>
<td>Negative</td>
<td>18</td>
<td>24.344</td>
<td>2.4879</td>
</tr>
<tr>
<td>T test</td>
<td></td>
<td>8.4</td>
<td></td>
<td></td>
</tr>
<tr>
<td>P value</td>
<td></td>
<td>&lt; 0.001</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Age and BMI was higher among those positive for H. pylori among studied cases. As shown in table (7).

Table 7. Mean value of age and BMI of all patients in relation to detection of H. pylori infection by HPSA test

<table>
<thead>
<tr>
<th></th>
<th>HP</th>
<th>N</th>
<th>Mean</th>
<th>Std. Deviation</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>AGE</td>
<td>Positive</td>
<td>57</td>
<td>50.000</td>
<td>10.620</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td></td>
<td>Negative</td>
<td>43</td>
<td>45.74</td>
<td>10.43</td>
<td></td>
</tr>
<tr>
<td>T test</td>
<td></td>
<td></td>
<td>5.560</td>
<td></td>
<td></td>
</tr>
<tr>
<td>P value</td>
<td></td>
<td></td>
<td>&lt; 0.001</td>
<td></td>
<td></td>
</tr>
<tr>
<td>BMI</td>
<td>Positive</td>
<td>57</td>
<td>30.340</td>
<td>2.820</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td></td>
<td>Negative</td>
<td>43</td>
<td>25</td>
<td>4.239</td>
<td></td>
</tr>
<tr>
<td>T test</td>
<td></td>
<td></td>
<td>7.153</td>
<td></td>
<td></td>
</tr>
<tr>
<td>P value</td>
<td></td>
<td></td>
<td>&lt; 0.001</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

By comparison between H.pylori (+ve) cases among the studied groups as regards sex, there was statically insignificant difference. (P 1.0) (NS). As shown in figure (1).

4. DISCUSSION

It’s well known that diabetic patients are more prone to infection, in these patients chronic infections are frequent and severe due to impaired their immune status (21). Its now broadly accepted that H. pylori infection is one of the most common chronic infections worldwide (22). Infections with H. pylori affect approximately 50% of the world population (23).

There is a controversy about the relationship between diabetes mellitus (DM) and H. pylori infection (24). In some studies there is a high prevalence of H. pylori infection in diabetic patients with either type 1 (25) or type 2 DM (26). In contrast other studies showed there is no association between H. pylori infection and DM, as there is no difference in prevalence of H. pylori infection between diabetic and non-diabetic (27), regardless of the type (28), and duration of diabetes (29) and/or severity of dyspeptic symptoms in diabetic patients (28).


Also there is controversy about the relationship between gastrointestinal symptoms in diabetes and H. pylori infection. According to some studies, concerning the prevalence of H. pylori related gastro-duodenal disorders; there is no difference between diabetic and non-diabetics (27). Moreover infection with H. pylori was not associated with upper GI symptoms in diabetics (31).
Therefore the prediction of precise prevalence of H. pylori infection in diabetic patients (type 1 and type 2) is required to be investigated, and to determine the relationship between H. pylori infection and GI symptoms among diabetic patients.

Our study was conducted on 100 Participants. Participants were divided into two groups, group I: include 50 diabetic patients (18 subjects of type 1 DM and 32 subjects of type 2 DM) with dyspeptic symptoms, group II: include 50 subjects non diabetic control group with dyspeptic symptoms.

The present study aimed to determine the prevalence of H. pylori infection in patients with type 1 & type 2 diabetes mellitus and to detect the relation of H. pylori infection and diabetes. (HpSA) test was used to detect Helicobacter pylori infection.

The specific advantages of HpSA assay include:

1. A more simple sampling method (only one stool specimen is required) and relatively cheap (32).
2. The lack of a requirement for trained personnel at the testing site.
3. There is no need for expensive equipment as well as indicating the presence of a current active infection; the stool antigen test is very helpful in the diagnosis of H. pylori in children (33).

It was found that the prevalence of H. pylori infection between control (50%) and diabetic patients (64%) are statistically insignificant (p 0.36), although diabetics have 14% higher prevalence of H. pylori infection than control group.

A cross-sectional study from Greece by Anastasios et al (2002), study included 172 dyspeptic patients (67 diabetic and 105 non diabetic), upper endoscopy was done for all patients and H. pylori was detected by histopathological examination. Who reported that the difference of H. pylori prevalence between the diabetic and non-diabetic groups with dyspeptic symptoms was not significant (P 0.78) (27).

On the other hand the higher prevalence of H. pylori infection in diabetes mellitus than in non-diabetics was reported in a study in Japan conducted by Kinjaki et al (1999) (34) and another study in Italy by Marrollo et al (2001) (35) that conducted on 74 diabetic patients with dyspepsia and 117 non diabetic dyspeptic patients, with assessment of presence and severity of dyspeptic symptoms for each patient. All patients underwent upper gastrointestinal endoscopy with biopsy and Helicobacter pylori were evaluated by rapid urease test and histology (Giemsa).

Many authors reported higher prevalence of H. pylori infection among patients with diabetes, suggesting that reduced gastric motility and peristalsis activity (24).

However, one study even showed a lower sero-prevalence of H. pylori infection in patients with diabetes mellitus, in comparison with the healthy population Zelenkova et al (2002). This study conducted on 95 diabetic patients (type I and II) and 216 blood donors, levels of IgG antibodies were determined by ELISA method, who found significant differences in sero-prevalence among the group of diabetic patients (27%) and non diabetic blood donors (5 %) p< 0.001(36). The presence of micro-angiopathy in patients with DM may be a negative factor for colonization by H. pylori because micro-vascular changes in the gastric mucosa may create an unfavorable environment for the establishment or survival of H. pylori (36).

As regard to the relationship between gastrointestinal symptoms in DM and H. pylori infection. Our findings confirmed that the prevalence of GI symptoms in HP +ve and -ve diabetes patients was statistically insignificant. Although, there is increased severity of dyspepsia, heartburn and bloating in diabetic patients more than control. This would be attributed to poor glycemic control and diabetic autonomic neuropathy.

This agreed with a study by Anastasios et al (2002) that reported there is no difference between diabetics and non-diabetics concerning the prevalence of H. pylori-related gastro-duodenal disorders (27).

Also, Harry et al (2001) confirmed that by a study conducted on 429 patients with type 1 (n = 49) or type 2 (n = 380) diabetes mellitus (48.6% women, mean age 60.7 yr) and 170 non-diabetic controls (34.7% women, mean age 60.4 yr). Blood sample was tested for H. pylori infection using a validated ELISA kit. The Sero-prevalence of H. pylori was 33% and 32%, respectively, in patients with diabetes and controls; the difference was (NS). Upper GI symptoms were present in 49% H. pylori -positive and 53% of H. pylori -negative patients with diabetes (p 0.56) (NS) (37).

On the contrary, a study by Marrollo et al., (2001) reported that H. pylori infection significantly associated with the presence of endoscopic gastritis and chronic gastritis in diabetic patients (35).

Moreover, Papamichael et al (2009) reported that high prevalence of esophagitis and peptic ulcer was found in H. pylori -positive patients with DM, especially those with cardiovascular autonomic neuropathy (24).

Also, Gucelik et al (2005) reported that there is a high prevalence of H.pylori infection in diabetic patients and it is correlated with dyspeptic symptoms in patients with diabetic autonomic neuropathy (26).

Charles and Anthonia (2007) reported that in general gastrointestinal (GI) symptoms and GI complications of DM often involve the esophagus, stomach, gallbladder, intestines, and the pancreas. The symptoms include constipation, faecal incontinence, dysphagia, heartburn, abdominal pain, diarrhea and nausea/vomiting. These have been attributed to altered intestinal motility and augmented visceral sensitivity in different regions of the GIT resulting from autoimmune neuropathy, neuroendocrine transmitter imbalance and microangiopathy (38).

In our study, there was statistically significant difference in mean value of age in relation to prevalence of H. pylori infection. The majority of the patients with H. pylori infection in both groups- diabetic and non diabetic were more than 50 years of age.

Similarly, in a study by devrajani et al (2010) who reported that the prevalence of H. pylori infection in both groups, diabetic and non diabetic, were male more than 50 years old (39).
Also, another study performed by Ugwu et al (2008) has shown that the majority of H. pylori infected patients were more than 60 years of age (40).

On other hand lower prevalence rate of H. pylori in elderly has been reported in Pilotto et al (2006) and two hypothesis have been advanced to explain these finding, first: As H. pylori could be present in small number which may not detected by histological examination contribute to decrease of sensitivity to many techniques used for detection of H. pylori including histology. The reason for in old age was explained as with advancing age of patients, H. pylori shows progressive proximal gastric migration which initially appears to colonize the antral region, later the body region, while remaining in elderly patients only in the body and fundus of the stomach. So in this condition reduce the sensitivity not only the rapid urease test but also other histological examination when done only on antral biopsy. Second H. pylori could present in past but eliminated through development of unfavorable gastric environment (41).

This disagreed with our study that reported that the infection by helicobacter pylori increase in elderly with increase of age due to decrease immune status of the body and increase susceptibility for infection.

Our study showed no statistically significant difference between males and females in H.pylori infection. This agreed with shahana et al (1996) (42), but disagreed with a study by Kanbay et al (2005) (43), that reported the Helicobacter pylori infected females were predominant as compared to males, while in another study by devrajani et al (2010) (39) confirmed that Helicobacter pylori infection were predominant in males.

There were no statistically significant difference between the rate of H. pylori infection and the increased duration of diabetes. This agrees with a study by Ko et al (2001) (44) but disagree with a study from Italy by Marrollo et al (2001) (35) reported that the rate of H. pylori infection increased with increase duration of diabetes 1 year was 23% ; 1-3 years was 32% and more than 3 years was 40%.

Our data reported that the prevalence of H.pylori infection increased in obese patients. This is agreed with Bytzer et al (2002) (45) who reported that the prevalence of H. pylori infection might be increase in type 2 DM obese patients as compared to normal.

Furthermore, in a study by Perdichizzi et al (1996 ) , there was a higher prevalence of H. pylori infection both in obese and in diabetic patients with respect to control subjects. Both obesity and type II diabetes may be associated with an increased incidence of H. pylori-colonization. This could be related to the reduced gastric motility and chemical changes in gastric mucosa following non-enzymatic glycosylation processes (46). Also Papamichael et al (2009) (24) and Arslan et al (2009) (47) considered that obesity can be a risk factor for H. pylori infection.

In contrast, in an American study by Ioannou et al (2005) showed that H. pylori seropositivity had no statistically significant relation to body mass index or fasting serum leptin level (48). A study by Fujiwara et al (2002) Found that eradication of Helicobacter pylori reduce ulcer recurrence in patients with peptic ulcer disease, and improve the quality of life but also associated with increase of body mass index (49).

Another study reported that H. pylori causes changes in leptin levels and plasma levels of ghrelin and eradication of H. pylori infection can increase appetite leading to rise in body mass index due to a higher caloric intake (50).

In our study fasting blood glucose level, 2h postprandial blood glucose concentration and HbA1c in relation to H. pylori infection were statistically insignificant. This is disagreed with Arslan et al (2009) who had shown that patients infected with H. pylori had higher mean fasting and 2h postprandial blood glucose concentration than non-infected diabetics (47).

On the other hand, the Chinese study by Ko et al (2004 ) that shown the women infected with H. pylori had lower mean fasting and 2h postprandial blood glucose concentration than non infected diabetics, may be partly attributed to alteration of gastric mucosa as high prevalence of severe acute gastric inflammation. H pylori gastritis has been found to increase glucose and meal stimulated insulin release probably by increasing gastrin secretion (44).

Chen and Blaser believe that H. pylori directly or indirectly Increases HbA1c levels in adults, particularly those who are obese, as H. pylori regulates leptin and ghrelin, which play a key role in energy homeostasis and metabolism (51).

5. CONCLUSION

The study reported no significant association between H. pylori infection and the prevalence of diabetes, but H. pylori infection was associated with a borderline increased risk for diabetes among participants with a BMI greater than 25kg/m².

REFERENCES


