Biochemical and hematological parameters in relation to Helicobacter pylori infection among type 2 diabetic patients in Gaza Strip

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ABSTRACT

The present study is aimed to assess biochemical and hematological parameters in relation to Helicobacter pylori (H. pylori) infection among type 2 diabetic patients in Gaza Strip. This case-control study comprised 90 type 2 diabetic patients (45 males and 45 females) and 90 healthy controls (45 males and 45 females). Blood samples were collected, processed and analyzed. Serum H. pylori IgG was determined by enzyme-linked immunosorbent assay (ELISA). Biochemical tests and complete blood count were performed. Data were analyzed using SPSS version 18.0. Blood glycated hemoglobin (HbA1c), serum insulin, cholesterol, triglycerides, aspartate aminotransferase (AST), alanine aminotransferase (ALT) as well as white blood cell (WBC) and platelet (PLT) were found to be significantly higher in diabetic patients compared to controls (P<0.05). The prevalence of H. pylori among diabetic patients 65 (72.2%) was significantly higher than controls 33 (36.7%) with P=0.000. When related to H. pylori, HbA1c was significantly higher in positive than in negative cases (8.4±1.8 vs 7.6±1.5 %, P=0.042). Cholesterol and triglycerides were significantly increased in H. pylori positive cases (216.4±42.5 and 190.1±91.9 mg/dl, vs 195.6±42.6 and 164.5±61.2 mg/dl, P=0.041 and P=0.033, respectively). The activity of ALT was also increased in H. pylori positive cases (43.1±4.9 vs 40.8±4.8 U/L, P=0.049). The WBC was significantly elevated in H. pylori positive cases (8.1±1.8 vs 7.2±1.5 x10⁹/L, P=0.038). In conclusions, H. pylori infection was significantly higher in type 2 diabetic patients compared to controls. H. pylori infection was associated with HbA1c, cholesterol, triglycerides, ALT and WBC.

Keywords: Helicobacter pylori, biochemical and hematological parameters, Type 2 diabetes, Gaza Strip.

1. Introduction

Diabetes mellitus is a metabolic disorder characterized by the presence of chronic hyperglycemia accompanied by greater or lesser impairment in the metabolism of carbohydrates, lipids and proteins. The origin and etiology of diabetes mellitus can vary
greatly but always include defects in either insulin secretion or response or in both at some point in the course of the disease (Conguet, 2002). Type 2 diabetes usually begins as insulin resistance, a disorder in which the cells do not use insulin properly. As the need for insulin rises, the pancreas gradually loses its ability to produce it (Cohen, 2006). Lack of insulin action and/or secretion in type 2 diabetes induces hepatic glucose output by inhibiting glycogen synthesis and stimulating glycogenolysis and gluconeogenesis then increased rates of hepatic glucose production result in the development of overt hyperglycemia, especially fasting hyperglycemia (Guyton and Hall, 2006 and Holt and Hanley, 2012). In such conditions, lipolysis in adipose tissue is promoted leading to elevated circulating levels of free fatty acids. In addition, excess fatty acids in serum of diabetics are converted into phospholipids and cholesterol in liver. These two substances along with excess triglycerides formed at the same time in liver may be discharged into blood in the form of lipoproteins (Jaworski et al., 2007). In addition, disturbance in liver function was also reported in type 2 diabetes (Atiba et al., 2013). *Helicobacter pylori* is a gram negative spiral shaped bacterium that is found in the gastric mucous layer or adherent to the epithelial lining of the stomach. The presence of *H. pylori* confers a six fold increased risk of gastric adenocarcinoma, account for half of all gastric cancers and strongly implicated in the development of gastric B cell mucosa associated lymphoid tissue (MALT) lymphomas as well as it causes peptic ulcer disease (Lehours and Yilmaz, 2007; Mehmood et al., 2010 and Kate et al., 2013). Recent reports suggested that *H. pylori* might have high prevalence among patients with diabetes. An increased prevalence of *H. pylori* infection among diabetes mellitus patients was first suggested by a report from Hungary (Simon et al., 1989). It was further supported by other studies from developing and developed countries (Gentile et al., 1998; Devrajani et al., 2010 and Taher et al., 2012). In Gaza strip, only one study focused on *H. pylori* infection and malnutrition among type 2 diabetic medical services patients (Abu Jabal, 2012). The present study is the first to assess biochemical and hematological parameters in relation to *H. pylori* infection among type 2 diabetic patients in Gaza Strip.

2. Materials and Methods

2.1 Subjects: This investigation was a case control study. The Sample size calculations were based on the formula for case-control studies. EPI-INFO statistical package version 3.5.1 was used with 95% CI, 80% power, 50% proportion as conservative and OR > 2. The sample size in case of 1:1 ratio of case control was found to be 73:73. Based on our response expectation of 90%, we multiply 73 by 1.11 (100/90). Therefore, the required sample size was found to be 81. For a no-response expectation, the sample size was increased to 90 type diabetic patients (45 males and 45 females) who attending diabetic clinics at Al-Shifa hospital, Gaza Strip. Controls were also 90 apparently healthy non diabetic individuals (45 males and 45 females). Patients and controls were age matched (38-62 years). Exclusion criteria included type 1 diabetic patients and cancer patients. For ethical consideration, the necessary approval to conduct this study was obtained from Helsinki committee in the Gaza Strip.

2.2 Specimen collection and processing: Twelve hours fasting overnight venous blood samples were collected from both patients and controls. Blood samples (8 ml each) were drawn by a well-trained nurse into vacutainer and plastic tubes. About 3 ml blood was placed
into ethylene diamine tetra acetic acid (EDTA) vacutainer tube to perform HbA1c and complete blood count (CBC). The remainder quantity of blood (5 ml) was placed in plastic tube and was left for a while without anticoagulant to allow blood to clot. Serum samples were obtained by centrifugation at 3000 rpm for 10 minutes.

2.3 Biochemical analysis: Serum *H. pylori* IgG was determined by ELISA kit. Insulin was measured by microparticle enzyme immunoassay (MEIA), using Abbott IMx Insulin assay. Cholesterol, triglycerides were determined by enzymatic colorimetric method, using Diasys Diagnostic Systems. AST and ALT were measured by using optimized ultraviolet-test according to International Federation of Clinical Chemistry and Laboratory Medicine, using DiaSys reagent kits. Blood HbA1c was determined calorimetrically by using Stanbio kit.

2.4 Hematological analysis: A complete system of reagents of control and calibrator, Cell-Dyne 1700 was used to determine hematological parameters including WBC, RBC, Hb and PLT.

2.5 Data analysis: Data were computer analyzed using SPSS/PC (Statistical Package for the Social Science Inc. Chicago, Illinois USA, version 18.0) statistical package. Simple distribution of the study variables and the cross tabulation were applied. The independent sample t-test procedure was used to compare means of quantitative variables by the separated cases into two qualitative groups such as the relationship between patients and controls insulin levels. Chi-square test was applied. The results were accepted as statistical significant when the p-value was less than 5% (p<0.05). The percentage difference was calculated according to the formula: Percentage difference equals the absolute value of the change in value, divided by the average of the 2 numbers, all multiplied by 100. Percent difference = (| (V1 - V2) | / ((V1 + V2)/2)) X 100. Microsoft Excel program version 11.0 was used for graph plotting.

3. Results

3.1 Biochemical parameters: Table 1 illustrates biochemical parameters of type 2 diabetic patients compared to controls. The mean levels of blood HbA1c and serum insulin in patients were significantly higher than that in controls (8.2±1.7% and 11.6±9.6 □ IU/ml vs 5.2±0.7% and 6.8±5.1 □ IU/ml, with % differences= 44.8 and 52.2%, respectively, P=0.000). Serum cholesterol and triglycerides were found to be higher in patients (201.4±43.3 and 203.8±97.7 mg/dl, respectively) compared to controls (189.0±37.9 and 153.1±45.7 mg/dl, respectively) with % differences of 6.3 and 28.4%, respectively). This increment was statistically significant for cholesterol and triglycerides (P=0.042 and P=0.000, respectively). Similarly, there were significant elevations in AST and ALT activities in patients compared to controls (36.3±4.7 and 42.4±5.0 U/L vs 16.9±6.0 and 17.8±8.7 U/L, % difference=72.9 and 81.7% respectively, P=0.000).
Table 1. Biochemical parameters of type 2 diabetic patients compared to controls.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Patients (n=90) mean±SD</th>
<th>Controls (n=90) mean±SD</th>
<th>% difference</th>
<th>t</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>HbA1c (%)* (min-max)</td>
<td>8.2±1.7 (5.3-12.0)</td>
<td>5.2±0.7 (3.5-6.5)</td>
<td>44.8</td>
<td>15.174</td>
<td>0.000</td>
</tr>
<tr>
<td>Insulin (µIU/ml)</td>
<td>11.6 ± 9.6 (0.30-50.0)</td>
<td>6.8±5.1 (0.5-18.9)</td>
<td>52.2</td>
<td>4.127</td>
<td>0.000</td>
</tr>
<tr>
<td>Cholesterol (mg/dl)</td>
<td>201.4±43.3 (113-281)</td>
<td>189.0±37.9 (120-250)</td>
<td>6.3</td>
<td>2.046</td>
<td>0.042</td>
</tr>
<tr>
<td>Triglycerides (mg/dl)</td>
<td>203.8±97.7 (57-600)</td>
<td>153.1±45.7 (65-215)</td>
<td>28.4</td>
<td>4.454</td>
<td>0.000</td>
</tr>
<tr>
<td>AST (U/L) (min-max)</td>
<td>36.3± 4.7 (30.0-46.0)</td>
<td>16.9±6.0 (10.0-30.0)</td>
<td>72.9</td>
<td>24.056</td>
<td>0.000</td>
</tr>
<tr>
<td>ALT (U/L) (min-max)</td>
<td>42.4±5.0 (30.0-50.0)</td>
<td>17.8±8.7 (10.0-38.0)</td>
<td>81.7</td>
<td>23.243</td>
<td>0.000</td>
</tr>
</tbody>
</table>

*HbA1c: Glycated hemoglobin. AST: Aspartate aminotransferase. ALT: Alanine aminotransferase. P<0.05: Significant.

3.2 Hematological parameters: As indicated in Table 2, WBC and PLT were significantly increased in patients compared to controls (8.0±1.9 and 262.3±61.3 ×10^9/L vs 7.0±1.4 and 224.8±43.4 ×10^9/L, % differences= 13.3 and 15.4%, P=0.000, respectively). On the other hand, no significant differences were found in RBC and hemoglobin between patients and controls.

Table 2. Hematological parameters of type 2 diabetic patients compared to controls.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Patients (n=90) mean±SD</th>
<th>Controls (n=90) mean±SD</th>
<th>% Difference</th>
<th>t</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>WBC ×10^9/L (min-max)</td>
<td>8.0± 1.9 (3.9-12.9)</td>
<td>7.0±1.4 (5.4-11.4)</td>
<td>13.3</td>
<td>3.973</td>
<td>0.000</td>
</tr>
<tr>
<td>RBCs ×10^12/L (min-max)</td>
<td>4.5±0.49 (3.4-6.7)</td>
<td>4.6±0.47 (4.0-5.8)</td>
<td>2.2</td>
<td>0.966</td>
<td>0.335</td>
</tr>
<tr>
<td>Hb (g/dl) (min-max)</td>
<td>12.6±1.4 (8.8-16.3)</td>
<td>12.7±1.3 (10.6-15.3)</td>
<td>0.79</td>
<td>0.388</td>
<td>0.698</td>
</tr>
<tr>
<td>PLT ×10^9/L (min-max)</td>
<td>262.3± 61.3 (152-500)</td>
<td>224.8±43.4 (147-320)</td>
<td>15.4</td>
<td>4.734</td>
<td>0.000</td>
</tr>
</tbody>
</table>

WBC: White blood cell, RBC: Red blood cell, Hb: Hemoglobin, PLT: Platelet. P>0.05: Not significant, P<0.05: Significant.

3.3 Distribution of Helicobacter pylori: As illustrated in Table 3 and Figure 1, 65 (72.2%) diabetic patients were positive for H. pylori compared to 33 (36.7%) controls. The difference between the two groups was significant (χ²=22.937, P=0.000) with higher distribution of H. pylori among patients.
Table 3. Distribution of Helicobacter pylori among type 2 diabetic patients compared to controls.

<table>
<thead>
<tr>
<th>Helicobacter pylori</th>
<th>patients (n=90)</th>
<th>Controls (n=90)</th>
<th>$\chi^2$</th>
<th>P- value</th>
</tr>
</thead>
<tbody>
<tr>
<td>n (%)</td>
<td>n (%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Positive</td>
<td>65 (72.2)</td>
<td>33 (36.7)</td>
<td>22.937</td>
<td>0.000</td>
</tr>
<tr>
<td>Negative</td>
<td>25 (27.8)</td>
<td>57 (63.3)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

P<0.05: Significant.

3.4 Helicobacter pylori in relation to biochemical parameters of diabetic patients: The relationship between H. pylori and biochemical parameters of patients is illustrated in Table 4. The mean level of blood HbA1c in positive cases was significantly higher than that in negative cases (8.4±1.8 vs 7.6±1.5%, P=0.042). However, serum insulin levels did not show significant relations with H. pylori (P=0.094). Serum cholesterol and triglycerides in positive cases were significantly higher than that in negative cases (216.4±42.5 and 190.1±91.9 mg/dl vs 195.6±42.6 and 164.5±61.2 mg/dl, P=0.041 and P=0.033, respectively). The activity of ALT was also higher in positive cases (43.1±4.9 vs 40.8±4.8 U/L, P=0.049), whereas AST showed no significant difference between positive and negative cases (36.1±5.0 vs 35.2±2.3 U/L, P=0.252).

Table 4. Helicobacter pylori in relation to biochemical parameters of type 2 diabetic patients.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Helicobacter pylori</th>
<th>n</th>
<th>mean±SD</th>
<th>t</th>
<th>P- value</th>
</tr>
</thead>
<tbody>
<tr>
<td>HbA1c (%)</td>
<td>Positive</td>
<td>65</td>
<td>8.4±1.8</td>
<td>2.081</td>
<td>0.042</td>
</tr>
<tr>
<td></td>
<td>Negative</td>
<td>25</td>
<td>7.6±1.5</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Insulin (µU/ml)</td>
<td>Positive</td>
<td>65</td>
<td>12.6±9.6</td>
<td>1.711</td>
<td>0.094</td>
</tr>
<tr>
<td></td>
<td>Negative</td>
<td>25</td>
<td>8.9±9.1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cholesterol (mg/dl)</td>
<td>Positive</td>
<td>65</td>
<td>216.4±42.5</td>
<td>2.078</td>
<td>0.041</td>
</tr>
<tr>
<td></td>
<td>Negative</td>
<td>25</td>
<td>195.6±42.6</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
### Table 5

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Helicobacter pylori</th>
<th>n</th>
<th>mean±SD</th>
<th>t</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>WBCs ×10^9/L</td>
<td>Positive</td>
<td>65</td>
<td>8.1±1.8</td>
<td>2.146</td>
<td>0.038</td>
</tr>
<tr>
<td></td>
<td>Negative</td>
<td>25</td>
<td>7.2±1.5</td>
<td></td>
<td></td>
</tr>
<tr>
<td>RBCs ×10^{12}/L</td>
<td>Positive</td>
<td>65</td>
<td>4.5±0.44</td>
<td>0.432</td>
<td>0.667</td>
</tr>
<tr>
<td></td>
<td>Negative</td>
<td>25</td>
<td>4.6±0.61</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hb (g/dl)</td>
<td>Positive</td>
<td>65</td>
<td>12.6±1.5</td>
<td>0.481</td>
<td>0.633</td>
</tr>
<tr>
<td></td>
<td>Negative</td>
<td>25</td>
<td>12.5±1.1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PLT ×10^{12}/L</td>
<td>Positive</td>
<td>65</td>
<td>282.9±73.4</td>
<td>1.763</td>
<td>0.087</td>
</tr>
<tr>
<td></td>
<td>Negative</td>
<td>25</td>
<td>254.4±54.6</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

WBC: White blood cell, RBC: Red blood cell, Hb: Hemoglobin, PLT: Platelet. P>0.05: Not significant, P<0.05: Significant.

### 3.5 Helicobacter pylori in relation to hematological parameters of diabetic patients:

Table 5 presented *H. Pylori* in relation to hematological parameters. The WBC count was significantly higher in positive compared to negative cases (8.1±1.8 vs 7.2±1.5 ×10^9/L, P=0.038). However, there were no significant differences in RBC, Hb and PLT between positive and negative cases (P>0.05).

Table 5. Helicobacter pylori in relation to hematological parameters of type 2 diabetic patients.

Despite high prevalence of type 2 diabetes and its subsequent health problems, there are under-diagnosis and under-reporting of the disease in the Gaza Strip. Recently, only one study have been focused on *H. pylori* infection and malnutrition among type 2 diabetic medical services patients in Gaza Strip (Abu Jabal, 2012). The present study is the first to assess *H. pylori* infection in type 2 diabetic patients and its relation to biochemical and hematological parameters. Understanding the role of *H. pylori* in diabetes mellitus could be useful in terms of prognosis and management of the disease.

Results of this study showed two-fold increment in *H. pylori* infection among diabetic patients compared to controls, implying that *H. pylori* is associated with type 2 diabetes. Higher prevalence of *H. pylori* was found among diabetics patients compared to non diabetics (Devrajani et al., 2010; Abu Jabal, 2012 and Taher et al., 2012). Blood HbA1c and serum insulin were significantly higher in patients than that controls. Similar results were obtained (Qi et al., 2007 and Nicholas et al., 2013). It is reported that the levels of HbA1c in the blood reflect the glucose levels to which the erythrocyte has been exposed during its lifespan (Goldstein et al., 2004). Therefore, the HbA1c test is attractive as it measures chronic glycaemia, rather than instantaneous blood glucose levels. HbA1c has been used as an
objective marker of average glycaemic control for many years, has an accepted place in the monitoring of patients with diabetes, and is relied on for significant management decisions, such as initiation of insulin therapy (d’Emden et al., 2012). Hyperinsulinemia recorded in the present study indicated the development of insulin resistance in diabetic patient, which minimize the utilization of glucose by the cell leading to hyperglycemia. Insulin resistance was a well established feature in type 2 diabetes (Meier and Bonadonna, 2013). In diabetic patient *H. pylori* infection was found to be associated with HbA1c. This result is in agreement with that obtained by Hsieh et al. (2013). One potential biological mechanism that might explain the link between *H. pylori* infection and HbA1c levels is related to the role of *H. pylori* in the host metabolic homeostasis by affecting the production of gastric hormones such as ghrelin and leptin (Pacifico et al., 2008 and Francois et al., 2011). These hormones are involved in the regulation of appetite and energy expenditure. The other suggested mechanism is that cytotoxin-associated gene A (cagA) protein produced by *H. pylori* could be an important contributor to the inflammatory disorders involved in the metabolic syndrome (Atherton, 2006). Serum cholesterol and triglyceride were found to be significantly increased in diabetic patients compared to controls. This increase was documented by several authors (Yassin et al., 2011 and Al-Hakeim and Ali, 2012). The general increase of serum lipids in diabetic patients may be mainly attributed to increase in the mobilization of free fatty acids from fat depots, since elevation of insulin inhibits the hormone sensitive lipase. Then, excess fatty acids in serum are converted into triglycerides, phospholipids and cholesterol in liver (Scheen, 2003 and Robciuc et al., 2013). When related to *H. pylori*, cholesterol and triglyceride levels were significantly higher in positive than in negative cases. Similar results were previously reported (Ugwu et al., 2008; El Hadidy et al., 2009 and Tanriverd, 2011). The mechanism of how *H. pylori* infection modifies the serum lipid profiles is still not clear, but a plausible explanation is that systemic inflammatory response to the bacterium induces changes in lipid and lipoprotein metabolism (Khovidhunkit et al., 2000). That is, chronic *H. pylori* infection has been postulated to shift the lipid profile toward an atherogenic direction via the action of proinflammatory cytokines, such as interleukins 1 and 6, interferon-alpha, and tumor necrosis factor alpha (TNF-α). These cytokines are capable of affecting lipid metabolism in various ways, including activation of adipose tissue lipoprotein lipase, stimulation of hepatic fatty acid synthesis, influencing lipolysis and the increasing hepatic hydroxyl methylglutary-CoA (HMG-CoA) reductase activity (Khovidhunkit et al., 2004). Thus, *H. pylori* infection could play a role in the atherosclerotic process and may be a reliable indicator for the assessment of cardiovascular disease risk (Lim et al., 2013).

The liver enzymes AST and ALT were markedly elevated in diabetic patients. Such finding is in agreement with the previous studies (Forlani et al., 2008 and Saligram et al., 2012). Elevation of AST and ALT could be due to direct hepatotoxic effect of fatty acid on the liver when it is produced in excess as observed in the present study. Mechanisms for this may include cell membrane disruption at high concentration, mitochondrial dysfunction, toxin formation, and activation and inhibition of key steps in the regulation of metabolism (Cho et al., 2007 and Atiba et al., 2013). Other potential explanations for elevated transaminases in insulin-resistant states include oxidantive stress from reactive lipid peroxidation, peroxisomal
beta-oxidation, and recruited inflammatory cells. The insulin resistant state is also characterized by an increase in pro-inflammatory cytokines such as TNF-α, which may also contribute to hepatocyte injury (Good et al., 2006). It is also hypothesized that the elevated ALT, a gluconeogenic enzyme whose gene transcription is suppressed by insulin, could indicate impairment in insulin signaling rather than purely hepatocyte injury (Villegas et al., 2011 and Atiba et al., 2013). When related to *H. pylori* infection, ALT was found to be significantly higher in positive compared to negative cases. Takuma (2011) demonstrated that *H. pylori* infection was one of the independent risk factors for the development of liver disease. In this context, it is known that the level of ALT activity reflects damage to hepatocytes and is considered to be a highly sensitive and fairly specific preclinical and clinical biomarker of hepatotoxicity (Ozer et al., 2008). White blood cell and platelets were significantly increased in patients compared to controls, whereas no significant change was found in RBC and hemoglobin content. Leukocytosis and thrombocytosis were reported in diabetic patients (Charles et al., 2007 and Farhangi et al., 2013). In addition, Chen et al. (2006) concluded that elevated WBC count but not RBC count was significantly associated with insulin resistance and glycaemic metabolism. The insulin resistant state is characterized by an increase in pro-inflammatory cytokines, which may contribute to leukocytosis (Good et al., 2006 and Farhangi et al., 2013). Platelets hyperactivity may stand behind the detected thrombocytosis in diabetic patients (Demirtunc et al., 2009 and Kodiatte et al., 2012). When related to *H. pylori*, WBC was significantly higher in positive compared to negative cases. Leukocytosis was reported in *H. pylori* infected patients (Tanriverd, 2011 and Jafarzadeh et al., 2013). The elevation of WBC in *H. pylori* observed in infected cases may be attributed to increase production of inflammatory cytokines such as interleukin-8, interleukin-6, and TNF-α from epithelial cells in the gastric mucosa (Iida et al., 2012).

5. Conclusions

The prevalence of *H. pylori* among diabetic patients was significantly higher than controls. When related to *H. pylori*, blood HbA1c, serum cholesterol, triglycerides, ALT and WBC were significantly higher in positive than in negative cases.

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