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Relation Between Severity of *Helicobacter Pylori* Induced Gastritis and Glycemic Control in Egyptian Male Patients with Type-2 Diabetes Mellitus

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ABSTRACT

Background: *Helicobacter pylori (HP) infection is the most common infection in the world. It was associated with many gastrointestinal and extra-gastrointestinal diseases. There was evidence of strong association between H.pylori infection and Diabetes Mellitus, especially Type 2 DM.*

Objectives: *The aim of our study was to investigate the relationship between severity of Helicobacter Pylori induced gastritis and glycemic control in patients with type 2 diabetis.*

Patients and methods: *Fifty type 2 diabetic male patients (mean age 47.50 ± 6.01years) were admitted to gastroenterology unit of the Alexandria Main University Hospital for upper gastrointestinal endoscopy. Thirty of them are H.pylori positive (cases) and twenty were H.pylori negative (controls). Following data were collected from all patients: BMI, waist circumference, CBC, liver enzymes, urea, creatinine, Hemoglobin A1c level, fasting blood glucose. Also biopsy specimens were obtained to Diagnose H.pylori by rapid urease test and to assess severity of gastritis histopathologically by Updated Sydney Score.*

Result: *comparison between the two studied groups according to age, BMI, ESR, liver enzymes and creatinine showed no significant difference while hemoglobin, platete count, blood urea showed significant difference between the two groups. Although fasting blood glucose showed no statistically significant difference, Hemoglobin A1c showed significant difference between the two groups ($p<0.001$). There was a statistically significant relationship between hemoglobin A1C and the severity of H.pylori inflammation in cases group ($p<0.001$). Levels of Hb A1C were positively correlated with neutrophil infiltration, chronic inflammation, glandular atrophy and intestinal metaplasia in histopathology samples from cases group.*

Conclusions: *Helicobacter Pylori infection and severity of H.pylori induced gastritis correlate with higher levels of Hb A1C in type 2 diabetic patients.*

Keywords

Helicobacter Pylori, Diabetes, Gastritis.

Introduction

Helicobacter pylori prevalence is worldwide, but it differs among countries and among populations in the same country [1]. Type 2 diabetes has a complex pathogenesis. Environmental factors are critical in its pathogenesis. It would be very valuable medically and economically to detect any treatable causes of this disease to delay or prevent its onset or delay its progression and complications. Glycated hemoglobin (HbA1c) Levels, which result from the non-enzymatic glycosylation of hemoglobin and reflect the integrated blood glucose levels during the previous 3-4 months, can be used to diagnose diabetes and to estimate diabetes prevalence and incidence [2]. Diabetic patients are more liable to infections and bacterial overgrowth in upper GIT. The interrelation between *H.*

pylori infection and diabetes mellitus have been shown in some studies but the relationship remains debatable [3].

Some biological mechanisms may explicate this relation. First, altered metabolism of glucose may generate chemical changes in the gastric mucosa that help to anchor *H. pylori* infection [4]. Second, gastric infection with H.pylori increases secretion of pro inflammatory cytokines, leading to changes in the structure of insulin receptors, disrupting the interaction between insulin and its receptors [5].

Dyspepsia is a very common symptom in both diabetic patients and *H. Pylori* infected persons, despite the endoscopic examination for *H. pylori* diagnosis is invasive, expensive, and time consuming, it is very valuable in determining clinical prognosis on the basis of the localization of a lesion [6]. It is considered to be the "gold standard" diagnostic tool for diagnosing *H. pylori* infection [7]. We conducted this study to focus on the relation between H.pylori and the glycemic control in type 2 diabetic patients, and to link it to the degree of severity of H.pylori induced gastritis as studied on histopathological basis.

Patients and methods

The study included fifty male patients with type 2 DM divided into 2 groups; group A of 20 controls with negative results for *helicobacter Pylori* infection and group B with 30 cases with positive results of *H. pylori*. We excluded age< 40 years, those who received previous anti-helicobacter treatment, Patients with uncontrolled hypertension, Patients with metabolic syndrome (waist circumference is more than 102 cm), those with liver or renal disease, and smokers.

Patients were selected from those admitted to the gastro-enterology & endoscopy unit of internal medicine department, Alexandria University for gastro-duodenoscopy after approval of the medical ethical committee and an informed written consent from all the patients enrolled in the study was taken.

In the present study all patients were evaluated for BMI and waist circumference to exclude those with metabolic syndrome. Routine lab tests (CBC, urea, creatinine, liver enzymes, ESR,) were done., Lipid profile including total cholesterol, triglycerides, LDL cholesterol and HDL cholesterol were compared in both groups [8]. Hemoglobin A1c levels were measured by using high-performance liquid chromatography method in group A and B [9].

Endoscopic gastric mucosal biopsies were obtained by skilled endoscopist. Gastric mucosal biopsies were taken from the antrum, corpus and incisura angularis (five specimens) to make proper diagnosis of *Helicobacter Pylori* infection in case of patchy distribution. Histo-pathological grading according to Updated Sydney Score was done using both hematoxylin and Gimsa stain [10].

And one more antral specimen for identification of *H. pylori* by rapid urease test. Rapid urease test was considered positive when the colour of gel converts to Pink or red within 24 hours [11].

Statistical analyses used were; student t-test, F-test (ANOVA), Mann Whitney test, Kruskal Wallis test and Spearman coefficient [12].

Result

In our study, age of the study groups and body mass index (BMI) showed no statistically significant difference with P value 0.074 and 0.163 respectively.

Our results showed that there was no statistically significant difference between patients' group and the control group as regard fasting blood glucose (P value=0.070). We found that Mean HbA1c among diabetics with *H. pylori* infection was significantly greater than *H. pylori*-negative diabetics (9.32 ± 1.15% and 7.50 ± 0.63% respectively with P value <0.001).

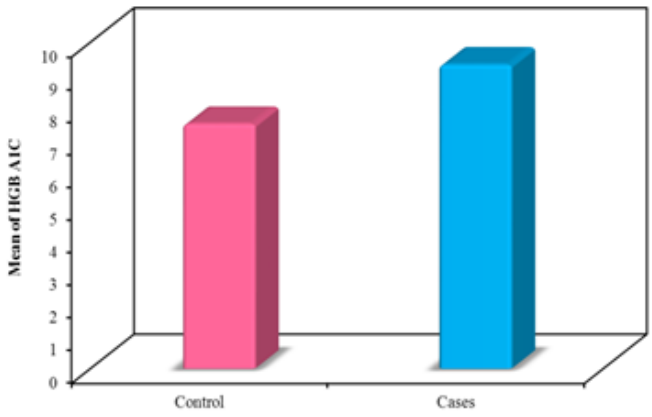

Figure 1: Comparison between the two studied Figure 1: Comparison between the two studied.

The present study showed statistically significant difference between HP (+) and HP(-) patients as regard lipid profile; Mean value of serum cholesterol was significantly higher in cases group as compared with control group(233.07 ± 30.49 and174.20 ± 18.96 respectively with P value<0.001). Triglyceride levels also showed significant difference between cases and control groups (181.27 ± 64.49 and 143.70 ± 22.67 respectively with P value=0.004). Also Low density lipoproteins (LDL) showed statistically significant difference between HP (+) and HP (-) groups (158.17 ± 18.20 and 106.15 ± 20.02 with P value<0.001). However, High density lipoproteins (HDL) showed no significant difference between two groups (P=0.234).

In the present study, There was statistically significant positive correlation between hemoglobin A1c and the severity of H.pylori inflammation (as graded by Updated Sydney System) in cases group (p<0.001). Triglycerides also showed positive correlation with severity of H.Pylori gastritis in cases group with P value=0.041. While LDL, HDL and total cholesterol showed no significant correlation with degree of H.Pylori gastritis as graded by Updated Sydney System (P value equals 0.442, 0.217 and 0.156 respectively).

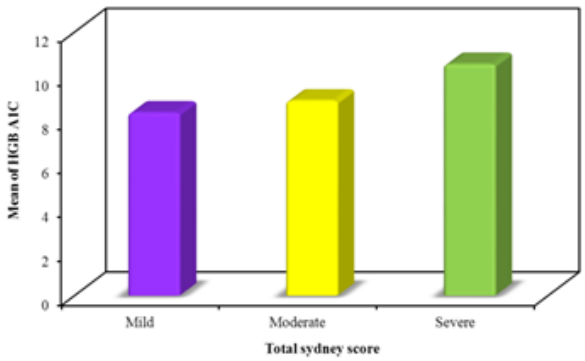

Figure 2: Relation between total sydney score Figure 2: Relation between total sydney score.

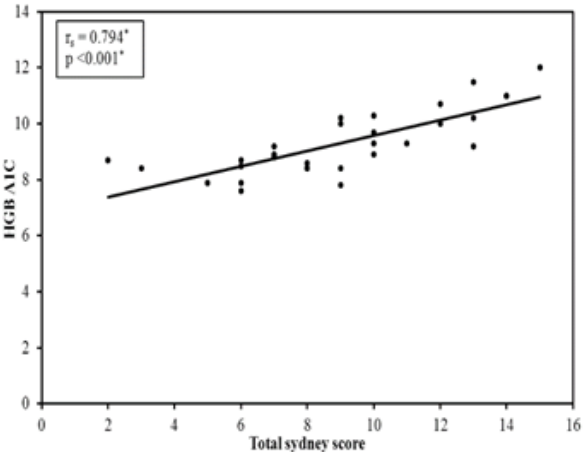

Figure 3: Correlation between Sydney score and Hb A1C in cases group.

Discussion

Patients with diabetes mellitus are often affected by chronic infections. Many studies have evaluated the prevalence of *H. pylori* infection in diabetic patients and the possible role of this condition in their metabolic control. Some studies found a higher prevalence of infection in diabetic patients and reduced glycemic control while others did not support any correlation between glycemic control and *H. pylori* infection [14].

All patients enrolled in our study were male patients. Most of the *Helicobacter pylori* related diseases are associated with male gender, the role of gender as a risk factor for *H. pylori* infection is still debated. Another study conducted by Catherine confirms the male predominance of *H. pylori* infection in adults as a global and homogeneous phenomenon [15].

In the present study, Comparison between the studied groups according to body mass index showed no significant difference. However, Perdichizzi et al. [16]. Determined higher BMI values and waist circumference measurements in HP (+) patients than in HP (−) patientsThe present study is concordant with most of the world literature, where no significant correlation of fasting blood glucose with *Helicobacter pylori* status was reported [17].

We obtained similar results toYu Chen et al as they found that Hb A1c was higher in HP (+) diabetic patients than HP (-) ones. They also observed decrease in level of HB A1c and better glycemic control after HP eradication [18]. The present study provides evidences that *H. pylori* infection is associated with atherogenic lipid profile among patients with type 2 diabetes. *H. Pylori* infected group had statistically significant higher levels of total cholesterol, triglyceride and LDL than non-infected group.

Similarly, Bajaj S.et al reported that diabetic patients infected with *H. pylori* showed an atherogenic lipid profile characterized by an increase in LDL cholesterol or decreased HDL cholesterol compared to uninfected diabetic patients [19]. In the present study, we compared hemoglobin A1c with the degree of h.pylori chronic inflammation in studied groups of diabetic patients, and there was a statistically significant correlation between hemoglobin A1c and the severity of HP chronic inflammation in infected diabetic patients. Similarly, Hsieh et al found a significant association between chronic *H. pylori* infection and high levels of HbA1c levels as well as decreased insulin secretion in a Chinese population. They also found that the prevalence of type 2 diabetes was higher in subjects with chronic *H. pylori* infection than those without chronic *H. pylori* infection [20].

We also found that there was a statistically significant positive correlation between Hb A1c and the degree of intestinal metaplasia in our biopsy specimens of cases group. Ikeda et al obtained similar results as they found evidence that those with higher levels of HB A1C were more susciptable for intestinal metaplasia and gastric cancer [21]. Concerning the relationship between degree of gastritis as graded by updated Sydney score and the lipid profile, updated Sydney System score showed a statistically significant positive correlation with triglycerides levels. These results support the theory that *H. pylori* infection affects lipid metabolism and can increase the risk of atherosclerosis and cardiovascular events, while total cholesterol, LDL-C and HDL-C had no significant relation with the degree of gastritis. However, in a study by Kucukazman et al [22] they found moderate correlation between LDL-C levels and updated system score. This shows that LDL-C levels increase as *H. pylori* infection becomes more severe, and they demonstrated no difference in HDL-C and triglyceride levels between groupswhich contradicts with our study results.

Conclusion

Results of this work have shown that *Helicobacter Pylori* infection affects glycemic control in T2 DM patients as it is correlated to elevated levels of Hb A1c and atherogenic lipid profile so it plays a role in inducing atherosclerosis by elevating LDL cholesterol and triglycerides levels in diabetic patients. Severity of chronic inflammation, intestinal metaplasia, glandular atrophy, neutrophil infiltration and H.pylori density induced by HP infection are positively correlated to higher levels of Hb A1c and atherogenic lipid profile in diabetic patients.

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