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The possible role of eradication of Helicobacter pylori infection on glycemic control in type 2 diabetes mellitus patients

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Abstract
Background: One of the most prevalent chronic infections in the world, Helicobacter pylori (H. pylori), is the primary contributing factor to gastritis, peptic ulcers, and stomach cancer. Chronic infections frequently have an impact on diabetic patients. Numerous studies have assessed the incidence of H. pylori infection in diabetes individuals and its potential impact on the way their metabolism is regulated. While some studies found no association between glycemic control and H. pylori infection, some identified a greater incidence of the infection in diabetes individuals and worse glycaemic control.
Aim and objectives: To investigate the impact treating Helicobacter pylori infection may have on individuals with type 2 diabetes mellitus' ability to maintain glycemic control.
Subjects and Methods: A 50-person type 2 diabetes mellitus randomised controlled trial was conducted at Tanta University Hospital's outpatient diabetes and endocrinology clinic. Each patient underwent a thorough clinical examination, extensive history taking, and investigations.
Result: Before and after, there was a high significant difference in FBG and a significant difference in HbA1C.
Conclusion: Finally, we found that the rate of H. pylori eradication with OCA or OMAB treatment is lower in patients with type 2 diabetes than in non-diabetics, and that H. pylori medication had no effect on glycemic control in people with type 2 DM.

Keywords: Glucose control, Type 2 diabetes, Helicobacter pylori infection, and HbA1C

Introduction
H. pylori, a host-specific bacterial pathogen, may infect the human stomach mucosa for an extended period of time. Chronic infection is a major risk factor for several gastroduodenal illnesses, including mucosa-associated lymphoid tissue lymphoma, gastric cancer, and superficial gastritis [1, 2]. Over the last few decades, a number of epidemiological investigations have demonstrated that H. pylori infections may have effects outside of the digestive tract. These infections may be related to extra-digestive illnesses, especially those that have persistent, low-grade, systemic inflammation [3].
According to a study by Cohen *et al*., those with HP had higher BMI levels even if they had no symptoms. The research also suggested that HP treatment could lead to weight reduction and improve diabetes control [4].
We hypothesised that research on biomarkers of diabetes mellitus, including as fasting blood sugar (FBS), glycated haemoglobin levels (HbA1c), and insulin resistance, may further knowledge of the etiologic significance of HP infection in people with type 2 diabetes.
In order to assess the potential effects of therapy for helicobacter pylori infection on HBA1C and FBS levels in type 2 diabetic patients, we developed this study.

Subjects and Methods
From January 2021 to June 2021, the Tanta University Hospital's intemal medicine department's outpatient clinic conducted this clinical trial investigation.
Fifty people with type 2 diabetes mellitus are present in Tanta University Hospital's outpatient diabetes and endocrinology clinic.

The inclusion criteria: patients with type 2 diabetes and a known infection with Helicobacter pylori

Exclusion criteria: Patients who have undergone a prior gastroscopy or gastrectomy, have type 1 diabetes, or who have used proton pump inhibitors, H2 receptor antagonists, or antibiotics recently.

Sample size: 50 participants with type 2 diabetes mellitus participated in the trial.

Sampling technique: This research on systematic random sampling was carried out.

Methods

Clinical assessment: Complete history taking includes age, sex, place of residence, employment, current complaint being dyspepsia, reflux, stomach discomfort, and presence of comorbidities. past history of gastrointestinal haemorrhage, Age at diabetes diagnosis, plasma glucose level at diagnosis, length of illness, usage of oral hypoglycemics, and insulin dosage (unit/kg/day), treatment history for H. pylori, Comorbidities were assessed, including hypertension. clinical assessment both a general and abdominal examination.

Tests used in laboratories for routine and general evaluation include: With the use of a Positive Stool Antigens test, Helicobacter Pylori infection was identified. Blood samples were obtained, and estimates of HbA1c and fasting blood glucose were made before to eradication treatment. All patients received omeprazole 20 mg B/D treatment**,** Clarithromycin 1gm B/D and amoxicillin 1gm for 14 days**.** One month after eradication, H. Pylori stool Ag was done to assess H. pylori eradication. One month and three months following eradication without modifications to the patients' medication for diabetes mellitus were the first times that gastrointestinal symptoms and glycemic control were assessed. Any patient with persistant H. pylori infection was excluded from study.

Intervention: Before beginning eradication treatment, fasting blood sugar levels and glycated haemoglobin levels (HbA1c) were assessed in blood samples. For 14 days, eradication treatment of omeprazole 20 mg bid, clarythromycin 500 BID, and amoxicillin 1 gm BID mg was administered to all patients. Throughout the course of therapy, every patient was monitored, and those who stopped taking their medications were not included. After receiving therapy for Helicobacter pylori, gastrointestinal symptoms and glycemic control were assessed at the beginning and 1, 2, and 3 months afterwards.

Statistical analysis: The Statistical Programme for Social Science version 20 (SPSS Inc., Chicago, IL, USA) was used to analyse the data. Mean and standard deviation were used to characterise quantitative variables. Number and percentage were used to describe qualitative factors. Student t test was used to compare parametric quantitative variables between two groups. When frequencies fell below five, the chi-square (X2) test or Fisher's exact test was used to compare qualitative variables. In order to evaluate the relationship between two normally distributed variables, Pearson correlation coefficients were utilised. A P value <0.05 is regarded as significant when a variable was not normally distributed.

Operational design: All participants in this study were given an introduction by the researcher, who then requested their participation after outlining the study's objectives. All of the chosen volunteers were given thorough explanations of the study's goals and anticipated advantages. Throughout the whole project, all ethical issues were taken into account.All participants verbally consented after being fully informed, and information confidentiality was guaranteed.

Ethical Considerations: The Tanta School of Medicine's ethics committee granted permission.

Results

Table 1: Distribution of the cases under study in terms of their demographics

	Cases (n = 50)	
Age (Years)		
Min. – Max.	34 – 58	
Mean ± SD.	45.22 ± 7.8	
Sex	No.	%
Female	29	58.0
Male	21	42.0
Residence		
Rural	25	50.0
Urban	25	50.0
Occupation		
Not working	15	30.0
Working	35	70.0
Smoking	8	16.0
Diabetes	50	100.0
Hypertension	7	14.0

This table shows that The examined patients ranged in age from 34-58, with a mean age of 45.22 (±7.8 SD) and a sex breakdown of 29 (58.0%) female and 21 (42.0%) male. there were 25 (50%) urban residents and 25 *50%) rural residents, there were 35 (70%) working, 8 (16%) smoking, 50 (100%) with diabetes and 7 (14%) with hypertension.

Table 2: Distribution of the analysed cases according to lab investigation

	Cases (n = 50)	
	No.	%
Positive H. pylori	50	100.0
Hb		
Min. – Max.	11 – 13.7	
Mean ± SD.	12.34 ± 0.75	
WBCs		
Min. – Max.	5.7 – 11	
Mean ± SD.	8.4 ± 1.56	
PLTs		
Min. – Max.	158 – 350	
Mean ± SD.	248 ± 58.09	
HbA1C		
Min. – Max.	6.7 – 10.8	
Mean ± SD.	8.86 ± 1.11	
FBG		
Min. – Max.	109 – 180	
Mean ± SD.	147.56 ± 23.05	

The following table illustrates that all the studied cases had positive H. pylori, the mean Hb was 12.34 (±0.75 SD) with range (11-13.7), the mean WBCs was 8.4 (±1.56 SD) with range (5.7-11), the mean PLTs was 248 (±58.09 SD) with range (158-350), the mean HbA1C was 8.86 (±1.11 SD) with range (6.6-10.8) and the mean FBG was 147.56 (±23.05 SD) with range (109-180).

Table 3: Division of the examined cases based on treatment response

	Cases (n = 50)	
Response to treatment	No.	%
No respond	17	34.0
Responded	33	66.0

This table displays that 33 (66%) of the investigated cases responded to medical treatment.

Table 4: Cases investigated and their distribution in terms of lab follow-up

	Cases (n = 50)
HbA1C	
Min. – Max.	6.8 – 10.4
Mean ± SD.	8.34 ± 1.01
FBG	
Min. – Max.	108 – 177
Mean ± SD.	132.28 ± 19.34

This table shows that according to follow up lab results the mean HbA1C was 8.34 (±1.01 SD) with range (6.8-10.4) and the mean FBG was 132.28 (±19.34 SD) with range (108-177).

Table 5: Relation between follow up Lab and response to treatment

	Not responded (n = 17)	Responded (1 m after) (n = 33)	Responded (3 m after)	T	p
HbA1C					
Min. - Max.	6.9 – 10.4	6.8 – 9.6	6.5 – 9.0	1.958	0.056
Mean ± SD.	8.72 ± 1.17	8.15 ± 0.87	8.05 ± 0.80		
FBG					
Min. – Max.	108 – 177	109 – 159	115.40±9.80	2.617	0.012*
Mean ± SD.	141.71 ± 23.65	127.42 ± 14.89			

This table displays a statistically significant association between follow-up FBG and the response to treatment.

Table 6: Review of laboratory outcomes in cases that were treated before and after therapy

	Before (n = 33)	After 1 m (n = 33)	t	P
HbA1C				
Min. – Max.	6.7 – 10.8	6.8 – 9.6	2.863	0.007*
Mean ± SD.	8.92 ± 1.09	8.15 ± 0.87		
FBG				
Min. – Max.	110 – 180	109 – 159	6.984	<0.001*
Mean ± SD.	150.24 ± 22.64	127.42 ± 14.89		

This table illustrates that there were major variations in both the HbA1C and FBG before and after.

Discussion

In their investigation, Xia *et al*. found no correlation between HP seropositivity and diabetes; however, Guvener *et al*. found that individuals with type 2 diabetes had a greater incidence of HP gastritis than age-matched healthy controls. Additionally, El Hadidy *et al*. found that people with type 2 diabetes who had HP infection had considerably higher triglyceride levels [5].

To investigate the potential impact of treating Helicobacter pylori infection on individuals with type 2 diabetes mellitus' ability to regulate their blood sugar levels.

According to this study, there were 29 (58.0%) female patients and 21 (42.0%) male cases, with a range of age between (34-58) and 45.22 (7.8 SD).

there were 25 (50%) urban residents and 25 *50%) rural residents, there were 35 (70%) working, 8 (16%) smoking, 50 (100%) with diabetes and 7 (14%) with hypertension.

Nam *et al*., [6] demonstrated that the recommended triple therapy for eradication included amoxicillin (1.0 g b.i.d.), clarithromycin (500 mg b.i.d.), and proton pump inhibitors (b.i.d.) at the normal dose for seven days. Four weeks following therapy, a urea breath test was conducted. To find the parameters linked to a successful eradication, various clinical and analytical data were gathered. 85 of the 119 participants were not diabetic, and 34 had the disease. Diabetes was present on average for 10.16 years. Patients with diabetes were older and had more coexisting conditions.

Vafaeimanesh *et al*., [7] found that among 100 patients in each group, 93 type 2 DM (non-insulin users) and 98 non-diabetic patients who were age and sex matched and executed 13C-urea breath testing were able to keep the treatment courses. Four groups of patients totaling 191 individuals-53.9% of them were men-between the ages of 45 and 81-were scrutinised.

Cheng *et al*., [8] reported that a total of 549 type 2 diabetes patients were included in the current study, comprising 269 women and 280 men. Anti-H. pylori IgG prevalence rates and UBT prevalence rates for H. pylori infection were 58.1% and 37.9%, respectively.

Zojaji *et al*. [9] disclosed that those involved had been selected from the endocrinology clinic at the Shahid Beheshti University of Tehran, Iran, who had type 2 diabetes and had been identified as having Helicobacter pylori infection. 85 patients with a mean age of 52 4.7 years (27 males, 31.8%, and 58 females, 68.2%) were recruited.

This study cleared that all the studied cases had positive H. pylori, the mean Hb was 12.34 (±0.75 SD) with range (11-13.7), the mean WBCs was 8.4 (±1.56 SD) with range (5.7-11), the mean PLTs was 248 (±58.09 SD) with range (158-350), the mean HbA1C was 8.86 (±1.11 SD) with range (6.6-10.8) and the mean FBG was 147.56 (±23.05 SD) with range (109-180).

Cheng *et al*. [8] showed that Between the "active" and "non-active" infection groups, there were no changes in A1C (7.68 ± 1.38 vs. 7.65 ± 1.49%, P = 0.829) or fasting plasma glucose (7.8 ± 2.5 vs. 7.8 ± 2.6 mmol/L, P = 0.935).

Zojaji *et al*., [9] exhibited a mean FBG of 56±37.

Vafaeimanesh *et al*., [7] revealed that the mean FBG was 135.6 (±37.5 SD), while the mean A1C was 9.5 (±1.6 SD).

This study demonstrated that 33 (66%) of the examined patients responded to therapy.

Zojaji *et al*., [9] revealed that 52 (62%) of the 85 individuals enrolled had their HP completely eradicated; these patients were the responders (16 men and 36 women).

This study demonstrated that according to follow up lab results the mean HbA1C was 8.34 (±1.01 SD) with range (6.8-10.4) and the mean FBG was 132.28 (±19.34 SD) with range (108-177).

Zojaji *et al*., [9] revealed that among those who responded to therapy, the mean HbA1C before and after treatment was 8.7±1.1 and 8.30.9, respectively. Analysis of these four variables (before to and during therapy for responders and non-responders) using repeated measure revealed that the mean HbA1C in responders and non-responders to treatment was 8.7 ±1.1 mg/dl and 8.8 ±1.3, respectively. ANOVA test results in two groups and the responders group were both significant.

This study demonstrated a strong relationship between follow-up FBG and therapeutic response. Before and after, there was a high significant difference in FBG and a significant difference in HbA1C.

Vafaeimanesh *et al*., [7] indicated that three and six months after therapy, eradicated instances of type 2 diabetes mellitus had HbA1c and fasting plasma glucose levels equivalent to noneradicated people.

Horikawa *et al*., [10] H. pylori carriers did not have higher HbA1C levels than the noncarriers, according to a recent meta-analysis by another team that examined the relationship between H. pylori and glycemic control in diabetics. The scientists came to the conclusion that H. pylori infection did not make diabetic patients' glycemic control worse. However, they did not assess the calibre of each included study in their meta-analysis. In addition, the authors only looked at one parameter (HbA1C level) to calculate the participants' glycemic control.

Cheng *et al*., [8] The interventional investigation provided additional evidence in favour of the hypothesis that active H. pylori infection was related to a greater level of glycemic therapy. The "H. pylori eradication" group's A1C level fell without any modifications to the anti-diabetic regimens. These findings supported the idea that eliminating H. pylori might enhance the effects of existing anti-diabetic medication on glycemic control.

Furthermore, the amount of time of diabetes, obesity, glycemia, taking oral hypoglycemics or insulin, socioeconomic status, and use of these medications may all have an impact on the H. pylori eradication rate. Patients with type 2 diabetes and insulin users had a noticeably greater rate of H. pylori eradication, based on Tseng's [11] study. Due to the existence of extra co-morbidities, the odds ratios declined as the duration of diabetes rose. Moreover, medication for diabetes taken orally are not consistently associated with a higher rate of H. pylori eradication, even though lower socioeconomic level and the use of calcium channel blockers are. Further research needs to be conducted to find out whether there is any causal relationship between insulin resistance and H. pylori infection, even though data appear to link the two. The genesis and therapy of the insulin resistance syndrome, encompassing type 2 diabetes and non-alcoholic fatty liver disease, may be seriously impacted if a link to cause exists.

All patients with active H. pylori infection were encouraged to have an esophagogastroduodenoscopy examination, followed by H. pylori eradication therapy, in accordance with medical ethics. The baseline features of the "H. pylori eradication" and "non-eradication" groups were equivalent despite non-randomization, which strengthens the findings.

In summary, we found that H. pylori therapy in individuals with type 2 DM has no influence on glycemic control and that the rate of H. pylori eradication with OCA or OMAB treatment is lower in patients with type 2 diabetes than in non-diabetics. Due to H. pylori's poor susceptibility to drugs like metronidazole in the second regimen as well as the low resistance of utilised antibiotics like clarithromycin in triple therapies in the Iranian population, 2-week triple therapy may be superior to 2-week quadruple protocol.

Conclusion

In conclusion, we came across that H. pylori therapies in individuals with type 2 DM has no influence on glycemic control and that the rate of H. pylori eradication with OCA or OMAB treatment is lower in patients with type 2 diabetes than in non-diabetics. Due to H. pylori's poor susceptibility to drugs like metronidazole in the second regimen as well as the low resistance of utilised antibiotics like clarithromycin in triple therapy in the Iranian population, 2-week triple therapy may be favourable to 2-week quadruple protocol. To come up with more successful approaches to get rid of H. pylori among those with type 2 diabetes, more research utilising novel antibiotic combinations needs to be conducted.

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Author's Contribution

Not available

Conflict of Interest

Not available

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