Original Article

Study of the Prevalence of Helicobacter Pylori Infection Among a Cohort of Diabetic Individuals in Alexandria

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Abstract

The aim of our study was to estimate the prevalence of H. pylori infection in diabetics and to elucidate the effect of infection on HbA1C% control; Also, to investigate the correlation between H. pylori infection and duration & complications of DM and other parameters. **Patients & methods:** In this case control study, 100 type 1 diabetic patients, 100 type 2 diabetic patients and 100 control subjects were included. H. pylori were tested by stool antigen test. Also, we estimated HbA1c level. **Results:** We found no association between H.pylori infection and diabetes mellitus; The adjusted odds ratio estimates of H. pylori infection among type 2 diabetics and type1 diabetics compared to non-diabetic subjects were 0.89 (95 % CI: 0.43- 1.85) and 1.33 (95 % CI: 0.24- 1.05) respectively; i.e. non -significant, also H. pylori had no effect on the control of HbA1c level. No correlation was found between H. pylori infection and duration of DM& the incidence of diabetic complications. No association was found between H. pylori infection and the incidence of upper gastrointestinal symptoms among the three groups. No correlation was found between H. pylori infection and age, sex, BMI and social standard in the three groups.

Key words: Helicobacter pylori; Type 2 diabetes; Type 1 diabetes; Insulin resistance; Inflammation; Cytokines; BMI (body mass index); HbA1c (glycosylated haemoglobin).

Introduction

Helicobacter pylori is a gram-negative, spiral shaped pathogenic bacterium that specifically colonizes the gastric epithelium causing chronic gastritis, peptic ulcer disease, and/or gastric malignancy.(1) H. pylori is mainly acquired in childhood by the fecal-oral, oral- oral or gastro-oral route, and has been recognized as a worldwide public health problem that is more prevalent in developing countries.(2) Recently, there is a rapidly growing body of literature concerning a possible association between H. pylori infection and extra-gastric disorders. The extra-digestive diseases have been reported in those with evidence of H. Pylori infections in recent years include ischemic heart disease (3), autoimmune thyroid diseases (4), sideropenic anemia,(5) idiopathic thrombocytopenic purpura,(6) neurologic diseases,(7) and hepatobiliary diseases.(8) Indeed, The postulated role of H. pylori in the pathogenesis of extra gastric disorders is based on the facts; this bacterium produces a chronic low grade inflammatory state that lasts for several decades which is able to induce lesions both locally and remote to the primary site of infection, induces molecular mimicry mechanisms, and interferes with the absorbance of nutrients and drugs, possibly influencing the occurrence and/or evolution of many diseases.(9) Emerging data now indicate a strong relationship between H. pylori infection and the incidence of DM especially type2;(10) Nevertheless, there are other studies reported lack of such associations. (11).

Methods

This study was conducted from May 2015 to May 2017. We enrolled 300 subjects; diabetic patients were from the endocrinology clinic and non-diabetic control patients were from the general internal medicine clinic; the young subjects were from Students health insurance hospital (Sporting) and the older ones were from Alexandria main University hospital (AMUH); they were classified as group I: 100 patients with type 2 diabetes, group II: 100 patients with type 1 diabetes, group III: 100 non diabetics of matched age, sex and socioeconomic standard. We excluded patients who received proton pump inhibitors or antibiotics for the last three months. H. pylori were tested by stool antigen test "On Site H. Pylori Ag Rapid Test-cassette". HbA1c values were determined with High performance liquid chromatography (HPLC) and HbA1c below 6.54 % was considered normal. [12] All subjects gave informed consent to participate in the study. The study was approved by the Ethics Committee of Alexandria University; faculty of Medicine. Chi square and t-test were used for assessing the differences between the nominal and numerical variables respectively. Furthermore, logistic regression analysis was performed to measure the association between diabetes mellitus and H. Pylori infection. All analyses were performed at the 95 % significance level ($P < 0.05$) using statistical software IBM SPSS software package version 20.0 (Armonk, NY: IBM Corp).

Results

There was no statistically significant difference between type 1 DM and type 2. Also there was no statistically significant difference between type 1 DM and the control group. The only statistically significant difference ($p<0.05$) was between type 2DM and the control group where infection was more in the control group than in type 2 DM; Table (I); as the prevalence of H .Pylori is highest in the control group (45%), then 38% in type 1 DM and lowest in type 2DM (31%) but after adjustment for age; Table (II), there was no significant correlation between the prevalence of H. pylori infection and diabetes. As regards the HbA1c control among the H .Pylori infected and the non-infected patients, no statistically significant association was found between the increase in HbA1c level and the presence of H .Pylori infection in both type 1 and type 2 DM; Tables (III, IV). The correlation between H. pylori positivity and the duration of DM in type 1 and type 2 showed that there was no statistically significant association between H. pylori infection and duration of DM; Tables (III,IV). In addition, the correlation between H. pylori positivity and the complications of DM in type 1 and type 2 showed no significant association between H. pylori infection and the incidence of diabetic complications; Tables (III,IV). As regards the correlation between the occurrence of upper GIT symptoms and H. pylori infection in the three groups, there was no statistically significant association between H. pylori infection and upper GIT symptoms in type1 DM& the controls, But there was statistically significant association between H. pylori infection and upper GIT symptoms in type 2 DM; as the percentage of occurrence of upper GIT symptoms was higher (69.6%) in H. Pylori negative than in the H. Pylori positive (32.3%); Tables (III,IV&V). No statistically significant correlation was found between H. pylori infection and age, sex, BMI and social standard in the 3 groups; Tables (III,IV&V). No statistically significant correlation was found between the increase in HbA1c level (≥ 6.5) and H. pylori infection in patients with high BMI (≥ 25 kg/m²) in group I and group II ;Table (VI). We noticed a higher prevalence of the infection in Hadara, Asafra, Sidi bishr and Fectoria.

Discussion

Although there is no concrete evidence demonstrating that H. pylori plays a role in diabetes, the possibility for a causal relationship is an intriguing issue deserving discussion. There to infection in diabetic patients. Firstly, a diabetes-induced impairment of cellular and humoral immunity may enhance an individual's sensitivity to H. pylori infection. [14] Secondly, diabetes-induced reduction of gastrointestinal motility and acid secretion may promote pathogen colonization and infection rate in the gut. [15] Thirdly, altered glucose metabolism may produce chemical changes in the gastric mucosa that promote H. pylori colonization. [16] Finally, individuals with diabetes are more frequently exposed to pathogens than their healthy counterparts as they regularly attend hospital settings. [17] However, there are also indications that H. pylori infection may contribute to the development of diabetes. DM especially type 2 and its complications have a complex pathophysiology including insulin resistance (IR), glucotoxicity, lipotoxicity, B-cell dysfunction, chronic inflammation, and genetic and epigenetic factors; [18] with risk factors related to lifestyle (e.g., diet, obesity& physical activity) and socioeconomic factors; [12] One from its risk factors are the gastrointestinal infections and the

composition of intestinal microbiota. [19] Recent evidence implicates the pathological involvement of inflammation in T2DM, which is an important process induced by H. pylori infection. [18] Recently, there is a growing evidence concerning an association between H. pylori infection and insulin resistance, [20] chronic inflammation,[21] the secretion of gastric-related hormones such as leptin and ghrelin, as well as gastrin and somatostatin which may influence a predisposition to diabetes [22]and insulin secretion deficiency.[23]Furthermore, the presence of Gram-negative bacteria, such as H. pylori, in the gut microbiota leads to increased production of lipopolysaccharide, which also activates innate inflammatory processes. [24] An alternative hypothesis is that gastro duodenal conditions resulting from H. pylori infection could delay gastric emptying [25] which has been postulated to cause poor glucose control in insulin-dependent children with diabetes.[26].The relationship between H. pylori and diabetes mellitus was first explored in 1989 by Simon, et al. [27] who found that the prevalence of H. pylori infection in patients with diabetes mellitus was significantly higher than in asymptomatic controls (62% vs. 21%).%). However, the test used for detecting H. pylori was only a rapid urease test, and their comparison did not adjust for age, which is a major confounding factor. In the present study, there was no statistically significant difference in the prevalence of H. pylori fecal positivity between diabetic patients either type 1(38%) or type 2 (31%) and the controls (45%); it even was higher in the controls than diabetics. i.e. we found no relation between H. pylori infection and diabetes mellitus. The study of Keramat, et al. was from the studies supporting our results where 79 diabetic patients and 84 control subjects with upper gastrointestinal symptoms; the serology test for H. pylori was positive in 54.4 % of diabetics and 61.4 % of non-diabetic patients (P = 0.689, non-significant); the prevalence of H. pylori increased with HbA1c level but there was no statistically significant association (P =0.565). No association was found between H. pylori infection and upper gastrointestinal symptoms among diabetics, prediabetics and controls. [28] Similar findings were observed by Sotuneh, et al. who revealed that in a large population of elderly in northern part of Iran, HP infection is not associated with BMI, serum glucose and lipid profile as well as blood pressure even after adjustment for other risk factors. [29] There are other studies which have not found a higher prevalence of H. pylori in diabetic patients and have not supported any correlation between metabolic control and infection. [30- 32] Therefore, DM might not be a risk factor for acquisition of H. pylori infection; the presence of microangiopathy in patients with DM may be a negative factor for colonization by H. pylori, because microvascular changes in the gastric mucosa may create an unfavorable environment for the establishment or survival of H. pylori. [33] In these cases the results may be also explained by the higher number of antibiotics taken by diabetics and, thus, a more frequent occasional clearance of the infection. [19] Contrary to our findings, Han, et al. [34] suggested that H. pylori infection was associated with the risk of type 2 diabetes in a middle-age and old-age Chinese population; as individuals with H. pylori infection" diagnosed by urea breath test" had a higher prevalence of type 2 diabetes (21.3% versus 20.2%, p = 0.026) . H. pylori infection was associated with higher risk of type 2 diabetes (odds ratio, 1.08 (95% confidence interval: 1.02–1.14); p = 0.008) after adjustment for other confounders. The association was significant among women, those who were above 65 years old, not overweight or obese, and those who did not smoke, did not consume alcohol and without family history of diabetes. Subjects with H. pylori infection had higher levels of HbA1c and fasting blood glucose (p < 0.0001) than those who did not. Similarly, other studies [10, 35- 38] reported a higher prevalence of infection in diabetic patients. Despite the evidence implicating a link between H. pylori infection and inflammation that predisposes individuals to T2DM, there are some contradictory data. Park, et al. [39] reported that metabolic and inflammatory parameters, including blood sugar, lipid profiles, IR, white blood cell count, and C-reactive protein (CRP) levels, were not changed after H. pylori eradication. A study by Jeon, et al. [15] failed to find any significant association between levels of inflammatory mediators (CRP and IL-6) and H. pylori infection or T2DM. A study by Brown, et al. [40] indicated that leptin has a protective role on pancreatic B cell function, showing that leptin (which increases in H. Pylori infection) could prevent apoptosis of pancreatic B cells through modulation of the Bcl protein (anti apoptotic protein) family. Several meta-analyses were conducted to estimate diabetes risk with H. pylori infection; Jun-Zhen Li, et al. [41] conducted a meta-analysis which included 79studies involved 57,397 individuals. The prevalence of H. pylori infection in DM group (54.9%) was significantly higher than that (47.5%) in non-DM group (OR = 1.69, P < 0 001). The difference was significant in

comparison between type 2 DM group and non-DM group (OR = 2.05), but not in that between type 1 DM group and non-DM group (OR = 1.23, 95% CI: 0.77–1.96, P = 0 .38). Their meta-analysis suggested that there is significantly higher prevalence of H. pylori infection in DM patients as compared to non-DM individuals. And the difference is associated with type 2 DM but not type 1 DM. The above conflicting results may be explained by considering that most previous studies attempting to clarify the association between H. pylori infection and DM were limited by cross-sectional analyses. Variables like age, sex, race, economic status, DM prevalence, and strains of H. pylori infection in the included studies varied. For the lack of enough detailed data, subgroup analysis stratified by age, sex, different stages of DM, and strains of H. pylori, which might bring up heterogeneity, could not be carried out. More well-designed and prospective cohort studies are needed for clarifying the association between H. pylori infection and DM to overcome methodological limitations of previous cross-sectional studies. To date, there are only two prospective studies to examine the impact of H. pylori infection on development of diabetes. Jeon, et al. [15] conducted a study in Community dwelling elderly Latinos followed up for 10 years and showed that individuals who were seropositive for H. pylori at enrollment were 2.7 times more likely at any given time to develop DM than seronegative individuals (HR = 2.69; 95%CI: 1.10-6.60), after adjustment for multiple factors, including age, gender, ethnicity, education, and cardio- metabolic risk factors. Thus, that study was able to establish the relative timing of seropositivity and development of DM, giving more credence to a potential causal relationship. However, several issues must be considered in interpreting these results. First, similar studies need to be repeated in other populations to ensure that the findings are related to the presence of infection itself and are not a peculiarity of the H. pylori-infected subjects in their community (i.e., due to particular dietary or living habits that may be linked to vulnerability to infection and diabetes). Second, findings in elderly individuals may not be generalizable to younger individuals considering that a younger population has a shorter history of infection. Third, only a small percentage of the population was seronegative for H. pylori (7%), which limited the power of the study. Finally, evaluation of the H. pylori infection status depended solely on the detection of H. pylori IgG antibody without further laboratory assessment such as urease breath testing. The presence of the H. pylori antibody does not distinguish recent vs. historic H. pylori infection. Candelli et al [42] evaluated the reinfection rate of H. pylori three years after a standard eradicating treatment and the late effect of eradication upon metabolic control in young diabetic patients. 75 diabetic patients and 99 controls re-evaluated for the presence of H. pylori by means of 13C-Urea Breath Test, metabolic control and the prevalence of gastrointestinal symptoms. The prevalence of H. pylori infection was higher in diabetic patients than in dyspeptic controls of similar age, gender and socioeconomical status after three years of follow-up; i.e. young patients with diabetes present a higher risk of H. pylori gastric reinfection than controls. Our study showed there was no statistically significant association between the increase in HbA1c level and the presence of H .Pylori infection in both type 1 and type 2 DM& no statistically significant correlation between the increase in Hb A1C level (≥ 6.5) and H. pylori infection in patients with high BMI (≥ 25 kg/m²). Keramat, et al, [28] Chabot, et al. [31] and others [44] reported no association. Vafaeimanesh, et al. [46] found that in patients with T2DM, the mean decrease in HbA1c and fasting plasma glucose levels in eradicated cases was similar to non-eradicated subjects three and six months after treatment. There are similar reports showing no effect of H. pylori eradication on HbA1c levels. [43, 45] On the other hand, Some authors have found an influence of H .Pylori infection on metabolic control like Han, et al. [34] and Bajaj, et al. [47] .Chen and Blaser [48] in two large national surveys: the National Health and Nutrition Examination Survey (NHANES) III and the NHANES 1999-2000. Their report showed that H. pylori seropositivity, and H. pylori cag A (cytotoxin associated gene A) positivity in particular, was associated with higher mean HbA1c levels, an association that persisted after excluding individuals with a history of diabetes mellitus and controlling for potential confounders. The association was evident mainly in adults over 18 years of age. They also showed a synergistic effect of H. pylori and BMI on increased levels of HbA1c, indicating a role of H. pylori in impaired glucose tolerance in adults that may be potentiated by a higher BMI level. Zojaji, et al. [49] showed that H. pylori treatment can improve the mean HbA1c and the metabolic abnormalities in patients with T2 DM. The data of present study didn't support any significant correlation between H. pylori infection and BMI. Sotuneh, et al. [29] supported our results in this correlation; they

reported that H. pylori infection is not associated with BMI. Furthermore, there was an inverse relationship between morbid obesity and H. pylori seropositivity in the study of Wu MS, et al. [50]Also, there are data demonstrating that H. pylori eradication significantly increases the incidence of obesity in patients with peptic ulcer disease, as it increases BMI [51], and/or enhances the appetite of asymptomatic patients by elevating plasma ghrelin and reducing leptin levels. [52] Han, et al. [34] reported a significant association among those who were not overweight or obese. Contrary to our findings, a study by Cohen, et al. [53] demonstrated that adults infected with H. pylori had higher BMI levels, even if asymptomatic, and further suggested that H. pylori therapy may lead to weight loss and improve diabetic control. In the present study, there was no statistically significant association between H. pylori infection and duration of DM. Similar findings were observed by a study carried out by Chabot, et al., [31] Zikri [36]and others. [44,45,55] In contrast, Arslan, et al. [37] stated that the rate of infection increases with IDDM duration. Sohair B, et al. [54] concluded that the infection by virulent strain is associated with older age of patients, larger BMI, Higher HbA1c and lower age of onset of diabetes; i.e. it is correlated with longer duration. Our study showed no significant association between H. pylori infection and the incidence of diabetic complications; This agree with Zafar, et al.; [55] who found that prevalence of Helicobacter pylori infection had no significant correlation with duration of diabetes, type of diabetes, glycaemia levels of diabetics and complications of diabetics Similarly, Zikry [36] found no significant relation between H. pylori seropositivity and presence of micro albuminuria. On the other hand, Zhou, et al. [32] supported an association between H. pylori infection and diabetic nephropathy. Kayar, et al. [56] reported a significant relationship between H. pylori infections and diabetic complications. In the present study, there was no statistically significant association between H. pylori infection and upper GIT symptoms in T1DM patients and in the controls. But there was statistically significant association in T2DM patients; symptoms were more frequent in H. pylori negative patients (69.6%) than H. pylori positive patients (32.3%). This is consistent with the results of Keramat, et al. [28] and others. [57] Others maintain that H. pylori infection does not affect the rate of gastric emptying in diabetic patients. [58] These findings are inconsistent with those of Arslan [37] and Senturk. [38] Gastroduodenal conditions resulting from H. pylori infection could delay gastric emptying, which has been postulated to cause mismatch between the onset of insulin action and the absorption of carbohydrates in insulin-dependent children with diabetes. [25, 26] However, it has also been suggested that delayed gastric emptying is a potential advantage, rather than a disadvantage, in relation to glycemic control in T2DM patients not treated with insulin. [59] We didn't found any correlation between H. pylori infection and age; Although there have been indications that T2DM may predispose an individual to H. pylori infection, [14-17] this seems unlikely considering the age at which the disease is typically acquired.[29] On the other hand, Han, et al. [34] reported a significant association with those who were above 65 years old. Our study didn't found any correlation between H. pylori infection and sex; a result consistent with Keramat, et al. [28] In contrast; Quadri [33] found higher prevalence of H. Pylori infection in diabetic than in control women (80% vs 37.5%; $p < 0.05$), whereas there was no difference between males. Han, et al. [60] reported a significant association among women. We didn't found any correlation between H. pylori infection and social standard; A strange finding that differs from a settled fact or concept that H. pylori is a poor man's gut pathogen;[60] a fact supported by a study, conducted over 10 years, showed that improved standards of living in Russia have substantially reduced H. pylori transmission. [61] But our finding is probably due to Changing climate, changing demography and changing economy which consequently leads to redrawing the global map of H. pylori epidemiology. Obviously, socioeconomic status is not restricted to income and social class but takes in consideration other factors, including living standards, sanitation, urbanization, and educational level.[62] Educational level, in particular, has been used as a marker of socioeconomic status and has been considered as one of the important determinants of H. pylori prevalence in both developed [63] and developing countries. [64] Rosenstock, et al. found that the short duration of schooling beside low socioeconomic status increases the likelihood of H. pylori infection in Denmark. [63] We found few cases of intra-familial H. pylori infections in the controls and T1DM patients; like two sisters and a boy and his mother; a finding supporting the concept of intra-familial clustering of H. pylori infection [65, 66]. As regards the geographical distribution of the studied cases of the three groups, We noticed a higher prevalence in Hadara, Asafra,Sidi bishrand Fectoria; for future

more accurate epidemiological surveys to verify such distributions, to quantify the risk ratio between diabetics and non- diabetics in these places and to discover contributing factors like hygiene ,clean water availability, living standards, sanitation, urbanization, and educational level if any is present.

Conclusion:

This study showed that there was no statistical association between H. pylori infection and diabetes mellitus. In addition, there was no association between H. pylori infection and HbA1c control, duration of DM, diabetic complications, upper GI symptoms frequency, age, sex, BMI& social standard. Though there is an important literature on some extra-gastric disorders of H. pylori infection, additional studies are needed to examine the strength of the evidence linking these disorders to H. pylori, and to better understand mechanisms on how H. pylori affects them. Although no current data provide concrete evidence that H. pylori plays a role in diabetes mellitus, the possibility cannot be ruled out. Larger prospective studies investigating the impact of H. pylori infection on diabetes and corresponding mediating factors are warranted. Meanwhile, large interventional studies are urgently needed to evaluate the long-term benefit of H. pylori eradication for prevention and progression of diabetes. Evidence supporting an etiological role of H. pylori in the development of T2DM would indicate that preventive measures, such as increased hygiene and treatments using antibiotics and proton pump inhibitor combinations, should be explored as targets of intervention in high-risk communities.

Table (I): Prevalence of H. Pylori Infection in the 3 studied groups

	Group I (n = 100)		**Group II (n = 100)**		**Group III (n = 100)**	
	No.	**%**	**No.**	**%**	**No.**	**%**
H.pylori antigen in stools						
Negative	69	69.0	62	62.0	55	55.0
Positive	31	31.0	38	38.0	45	45.0
Sig. bet. Grps	$p_1 = 0.298$, $\mathbf{p_2 = 0.041^*}$, $p_3 = 0.315$					

p1: p value for comparing between groups I and groups II
p2: p value for comparing between groups I and groups III
p3: p value for comparing between groups II and groups III
*: Statistically significant at p ≤ 0.05

Table (II): Univariate and multivariate analysis for the parameters affecting H. pylori fecal positivity; after adjustment for age

	H.pylori antigen in stool		Unadjusted OR			Adjusted OR		
	Negative	Positive	OR	C.I	p	OR	C.I	P
Group I	69	31	0.55	0.31 – 0.98	0.042*	0.89	0.43 – 1.85	0.756
Group II	62	38	0.75	0.43 – 1.32	0.316	0.50	0.24 – 1.05	0.066
Group III	55	45	1.00	-	-	1.00	-	-

Table (III): Correlation between Pylori fecal positivity and different parameters in group I

	H.pylori antigen in stool				Test of Sig.	P
	Negative (n = 69)		Positive (n = 31)			
	No.	%	No.	%		
Sex						
Male	45	65.2	15	48.4	χ^2= 2.525	0.112
Female	24	34.8	16	51.6		
Age (years)						
31 – 40	6	8.7	5	16.1	χ^2= 4.769	0.190
41 – 50	33	47.8	10	32.3		
51 – 60	16	23.2	12	38.7		
> 60	14	20.3	4	12.9		
BMI (kg/m²)						
Min. – Max.	19.77 – 34.89		22.03 – 34.46		t= 0.886	0.378
Mean ± SD.	27.17 ± 3.30		27.78 ± 2.84			
Median	27.44		27.70			
Social standard						
Low	1	1.4	4	12.9	χ^2= 6.864	^{MC}p= 0.064
Moderate	24	34.8	13	41.9		
Good	37	53.6	13	41.9		
High	7	10.1	1	3.2		
Complications of DM						
No	51	73.9	28	90.3	χ^2= 3.472	0.062
Nephropathty	4	5.8	1	3.2	χ^2= 0.298	^{FE}p= 1.000
Retinopathy	10	14.5	1	3.2	χ^2= 2.774	^{FE}p= 0.165
Diabetic foot ulcer	5	7.2	1	3.2	χ^2= 0.613	^{FE}p= 0.663
Duration of DM (years)						
< 5	20	29.0	9	29.0	χ^2= 2.043	^{MC}p= 0.870
5 – 9	23	33.3	11	35.5		
10 – 14	12	17.4	8	25.8		
15 – 19	8	11.6	2	6.5		
20 – 24	4	5.8	1	3.2		
25 – 30	2	2.9	0	0.0		
Min. – Max.	0.08 – 30.0		1.0 – 20.0		U= 1020.000	0.711
Mean ± SD.	8.51 ± 6.39		7.45 ± 4.54			
Median	7.0		6.0			
HbA1c %						
< 6.5	11	15.9	4	12.9	χ^2= 0.155	^{FE}p= 0.772
≥ 6.5	58	84.1	27	87.1		
GIT symptoms						
Absent	21	30.4	21	67.7	χ^2=12.221*	**<0.001***
Present	48	69.6	10	32.3		

χ^2: **Chi square test** FE: **Fisher Exact** MC**: Monte Carlo**

U**: Mann Whitney test** t**: Student t-test**

p: p value for comparing between the two categories *: Statistically significant at $p \leq 0.05$

Table (IV): Correlation between H.pylori fecal positivity and different parameters in group II

	H.pylori antigen in stool				Test of Sig.	P
	Negative (n = 62)		Positive (n = 38)			
	No.	%	No.	%		
Sex						
Male	36	58.1	25	65.8	χ^2= 0.591	0.442
Female	26	41.9	13	34.2		
Age (years)						
< 10	17	27.4	8	21.1		
10 – 20	45	72.6	29	76.3	χ^2= 1.936	^{MC}p= 0.397
21 – 30	0	0.0	1	2.6		
BMI (kg/m²)						
Min. – Max.	10.58 – 31.50		12.48 – 33.29			
Mean ± SD.	18.90 ± 4.60		19.80 ± 6.46		τ= 0.745	0.459
Median	18.73		18.36			
Social standard						
Low	3	4.8	4	10.5		
Moderate	16	25.8	13	34.2	χ^2= 2.569	^{MC}p= 0.471
Good	35	56.5	18	47.4		
High	8	12.9	3	7.9		
Complications of DM						
No	58	93.5	38	100.0	χ^2= 2.554	^{FE}p= 0.294
Nephropathty	3	4.8	0	0.0	χ^2= 1.896	^{FE}p= 0.286
Retinopathy	1	1.6	0	0.0	χ^2= 0.619	^{FE}p= 1.000
Diabetic foot ulcer	0	0.0	0	0.0	-	-
Duration of DM (years)						
< 5	39	62.9	19	50.0		
5 – 9	15	24.2	17	44.7		
10 – 14	7	11.3	2	5.3	5.071	^{MC}p= 0.116
15 – 19	1	1.6	0	0.0		
20 – 24	0	0.0	0	0.0		
25 – 30	0	0.0	0	0.0		
Min. – Max.	1.0 – 15.0		1.0 – 12.0			
Mean ± SD.	5.09 ± 3.40		4.53 ± 2.26		U= 1165.000	0.925
Median	4.0		4.50			
Hb A1C %						
< 6.5	2	3.2	0	0.0	χ^2= 1.251	^{FE}p= 0.524
≥ 6.5	60	96.8	38	100.0		
GIT symptoms						
Absent	32	51.6	27	71.1	χ^2= 3.681	0.055
Present	30	48.4	11	28.9		

χ^2: **Chi square test** FE: **Fisher Exact** MC: **Monte Carlo**
U: **Mann Whitney test** t: **Student t-test**p: p value for comparing between the two categories

Table (V): Correlation between H.pylori fecal positivity and different parameters in group III

	H.pylori antigen in stool				Test of Sig.	P
	Negative (n = 55)		Positive (n = 45)			
	No.	%	No.	%		
Sex						
Male	26	47.3	23	51.1	χ^2= 0.146	0.702
Female	29	52.7	22	48.9		
Age (years)						
< 10	4	7.3	4	8.9	χ^2= 8.575	^{MC}p= 0.195
10 – 20	7	12.7	8	17.8		
21 – 30	12	21.8	15	33.3		
31 – 40	8	14.5	10	22.2		
41 – 50	17	30.9	7	15.6		
51 – 60	4	7.3	0	0.0		
> 60	3	5.5	1	2.2		
BMI (kg/m²)						
Min. – Max.	10.74 – 36.73		12.62 – 33.20		t= 1.058	0.293
Mean ± SD.	24.64 ± 5.37		23.53 ± 4.99			
Median	25.71		23.04			
Social standard						
Low	3	5.5	2	4.4	χ^2= 1.406	^{MC}p= 0.755
Moderate	17	30.9	15	33.3		
Good	32	58.2	23	51.1		
High	3	5.5	5	11.1		
GIT symptoms						
Absent	29	52.7	15	33.3	χ^2= 3.778	0.052
Present	26	47.3	30	66.7		

χ^2: **Chi square test** MC: **Monte Carlo**
t: **Student t-test**
p: p value for comparing between the two categories

Table (VI):Correlation between H.pylori fecal positivity and HbA1c % in high body mass index (BMI) (≥ 25 kg/m²)

Hb A1C %	H.pylori antigen in stool				Test of Sig.	P
	Negative		Positive			
	No.	%	No.	%		
Group I(77)	(n = 51)		(n = 26)			
< 6.5	9	17.6	4	15.4	χ^2= 0.063	^{FE}p= 1.000
≥ 6.5	42	82.4	22	84.6		
Min. – Max.	5.0 – 15.80		4.90 – 10.90		t= 1.717	0.090
Mean ± SD.	8.69 ± 2.37		7.80 ± 1.65			
Median	**8.40**		**7.45**			
Group II(14)	(n = 6)		(n = 8)			
< 6.5	2	33.3	0	0.0	χ^2= 3.111	^{FE}p= 0.165
≥ 6.5	4	66.7	8	100.0		
Min. – Max.	6.40 – 9.50		7.50 – 10.0		t= 0.076	0.942
Mean ± SD.	8.15 ± 1.46		8.10 ± 0.80			
Median	**8.65**		**8.0**			
Group I & Group II	(n = 57)		(n = 34)			
< 6.5	11	19.3	4	11.8	χ^2= 0.878	0.349
≥ 6.5	46	80.7	30	88.2		
Min. – Max.	5.0 – 15.80		4.90 – 10.90		t= 1.928	0.057
Mean ± SD.	8.63 ± 2.29		7.87 ± 1.49			
Median	**8.40**		**7.70**			

χ^2: **Chi square test** FE: **Fisher Exact**
t: **Student t-test**
p:p value for comparing between the two categories

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