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Original Article

Study of Helicobacter Pylori Gastritis in Diabetic versus Non-Diabetic Patients

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ABSTRACT

Background: Helicobacter pylori is one of the most prevalent infections in humans, it can cause gastrointestinal (GI) problems such as simple gastritis, gastric ulcers, and gastric cancer. It may pose an issue as opportunistic infections in specific situations, such as immunodeficiency and underlying illnesses. One of the underlying disorders caused by H. pylori is diabetes mellitus. Treating the H. pylori infection is essential since diabetic people have GI issues. There are conflicting and erratic reports on Helicobacter pylori infection in diabetics. We aimed to reveal the severity of complications of H. pylori among diabetic and non-diabetic patients and to identify the potential risk factors of H. pylori among diabetic. **Methods:** This case control study was carried out at Internal Medicine Department and Pathology Department, Faculty of Medicine, Zagazig University. Subjects were divided into group (1): 27 patients with DM and group (2): 27 non-diabetic individual's age and sex matched as control. Results: The severity of H. pylori gastritis was insignificantly different between the studied groups. Conclusion: Our results do not support an association between severity of H. pylori infection and diabetes mellitus. This is confirmed by the lack of difference between diabetics and nondiabetics regarding the severity of H.pylori infection.

Keywords: H. pylori; Gastritis; DM.

INTRODUCTION

With 3.8 millions of adult deaths globally as a result of diabetes mellitus, the condition is becoming pandemic and is considered a major risk to public health [1].

The stomach contains Helicobacter pylori, a spiral bacteria that is gram-negative (H. pylori). The H. pylori infection is one of the most common chronic disorders in the world, affecting over 50% of people globally [2].

Roughly 50% of the global populace is infected with Helicobacter pylori, a condition that is more common in underdeveloped nations. The majority of the infection is picked up in childhood, and even with an immunological and humoral response, H. pylori may found in the stomach environment for the duration of the host's life. Adult H. pylori infection is prevalent in Egypt, ranging from 52% to 57% [3].

H. pylori infection is acommon cause of chronic gastritis, It is linked to cancer, lymphoma, and duodenal and stomach ulcers. Moreover, Extragastric diseases including diabetes have been linked to H. pylori [4].Higher concentrations of inflammatory markers, such as TNF-a and IL-6, which are all connected to the onset of insulin resistance and diabetic mellitus, may be the mechanism via which H. pylori infection affects other organs [5].

Numerous research endeavors have examined the part .The role that H. pylori plays in the onset and consequences of diabetes; nonetheless, the findings exhibit inconsistencies. In diabetic dyspepsia, H. pylori infection is primarily linked decreases the production of acid, increases the release of pro-inflammatory cytokines, and raises blood glucose levels. Antral dysmotility and delayed stomach emptying are important causes of dyspepsia in diabetes mellitus, and they may

also contribute to the elevated incidence of H. pylori in DM [2].

It has been noted that even though diabetes mellitus and H. pylori infection are two distinct illnesses, there is a link between higher prevalence of H. pylori infection and poor glycemic control in diabetics. Among diabetics experiencing stomach problems, Infection with H. pylori has been found to be one of the most common outcomes [6].

There are conflicting and erratic reports on Helicobacter pylori infection in diabetics. This study is the first at Zagazig University, as far as I know., so we aimed to reveal the severity of complications of H. pylori among diabetic and non- diabetic patients and to identify the potential risk factors of H. pylori among diabetic.

METHODS

This case control study was conducted at Internal Medicine Department and Pathology Department, Faculty of Medicine, Zagazig University. Subjects were divided into group (1): 27 patients with DM and group (2): 27non-diabetic individual's age and sex matched as control. Informed consent were obtained from participants. Ethical consideration: Patients provided written informed consent along with an explanation of the procedure, possible hazards& IRB approval were attained (number 10603).The study was conducted according to Declaration of Helsinki.

Patients with DM diagnosed by HbA1c using the Medical Care Criteria for Diabetes established by the American Diabetes Association**[7], a**ge 18 years and above of both men and women were involved in the research.

Individuals with a history of H. pylori who were either on proton pump inhibitors or had undergone eradication therapy, patients who have previously used antacids, H2 receptor blockers, Proton pump inhibitors, antibiotics (within the last four weeks), hemoglobinopathies, individuals on immunosuppressive medications, patients with a history of renal failure, chronic liver disease, or cancer, and pregnant women were not allowed to participate in the trial.Every patient had a thorough history taken, a general and local examination, and a laboratory test investigations (CBC, kidney function test, liver function tests, FBG, PPBG, HbA1c, lipid profile, H. pylori Ag in stool and upper GIT endoscopy and gastric biopsy for histopathology.

According to the American Diabetes Association's standards, diabetes was defined in this study as satisfying any of the following criteria **[7]:** (i) FPG ≥ 126 mg/ dL, (ii) 2-h PG ≥ 200mg / dL, (iii) HbA1c ≥ 6.5% and (iv) Self-reported physician diagnosis of diabetes or use of Antidiabetic medication.

Statistical Analysis

Statistical analysis was done by SPSS v26 (IBM Inc., Armonk, NY, USA). Shapiro-Wilks test and histograms were used to evaluate the normality of the distribution of data. Unpaired student t-test, Mann Whitney-test, chi-square test and Fisher's exact test were used. A two tailed P value ≤ 0.05 was considered statistically significant.

RESULTS

HTN were significantly increased in group (1) (DM group) than group (2) (control group) (P value <0.001 and =0.005 respectively). Age, sex, dyslipidemia, COPD, and asthma were insignificantly different between the studied groups. Complain was insignificantly different between the studied groups (Table 1).

Platelets was significantly increased in group (1) (DM group) than group (2) (control group) (P value <0.05). Hb was significantly decreased in group (1) (DM group) than group (2) (control group) (P value =0.047). WBCs were insignificantly different between the studied groups. FBS, PPBS and HbA1C were significantly increased in group (1) (DM group) than group (2) (control group) (P value <0.05). Urea, creatinine, ALT, AST, total bilirubin, and PT/INR were significantly increased in group (1) (DM group) than group (2) (control group) (P value <0.05) (Table 2).

Helicobacter Pylori antigen in stool was insignificantly different between the studied groups (Table 3).Upper GIT endoscopy findings were insignificantly different between the studied groups (Table 4).

Regarding ultrasound findings, normal findings of group (1) (DM group) were significantly decreased than those of group (2) (control group) (P value =0.006). Fatty liver was significantly increased in group (1) (DM group) than group (2) (control group) (P value =0.010). Bulky spleen, congested liver, gall bladder stone, hydronephrosis, pleural effusion, and ovarian cyst were insignificantly different between the studied groups (Table 5).

Eosinophils were significantly decreased in group (1) (DM group) than group (2) (control group) (P value =0.018). Activity, mucosa, lamina propria, intestinal metaplasia, dysplasia, neutrophils, germinal centers and H. pylori were insignificantly different between the studied groups (Table 6, figure 1).

Table 1: Baseline data of the studied groups

		Group (1) (n=27)	Group (2) (n=27)	P value
Age (years)	**Mean ± SD**	55.7 ± 10.25	53.9 ± 12.47	0.565
	Range	41 - 81	37 - 70	
Sex	**Male**	12 (44.44%)	14 (51.85%)	0.586
	Female	15 (55.56%)	13 (48.15%)	
Comorbidities	**HTN**	10 (37.04%)	1 (3.7%)	**0.005***
	Dyslipidemia	2 (7.41%)	0 (0%)	0.491
	COPD	3 (11.11%)	0 (0%)	0.236
	Asthma	3 (11.11%)	1 (3.7%)	0.610
Epigastric pain		12 (44.44%)	13 (48.15%)	0.838
Dyspepsia		4 (14.81%)	1 (3.7%)	
Heart burn		7 (25.93%)	5 (18.52%)	
Vomiting		10 (37.04%)	9 (33.33%)	
Hematemesis		3 (11.11%)	1 (3.7%)	
Diarrhea		2 (7.41%)	1 (3.7%)	
Melena		1 (3.7%)	1 (3.7%)	
Constipation		1 (3.7%)	0 (0%)	

HTN: hypertension, COPD: chronic obstructive pulmonary disease, *: significant as P value ≤ 0.05

Table 2: Laboratory investigations of the studied groups

		Group (1) (n=27)	Group (2) (n=27)	P value
Hb (g/dl)	**Mean ± SD**	12.64 ± 2.22	13.67 ± 1.44	**0.047***
	Range	8.2 - 17.5	9.7 - 16	
WBCs (cells/µl)	**Mean ± SD**	7707.7±3096.76	6666.67±1653.9	0.129
	Range	7.8 - 14000	3500 - 9500	
Platelets (*10^3/ µl)	**Mean ± SD**	359.48±79.23	283.48±65.41	**<0.001***
	Range	209 - 530	202 - 403	
FBS (mg/dl)	**Mean ± SD**	159.22 ± 18.34	84.48 ± 7.19	**<0.001***
	Range	132 - 197	70 - 99	

		Group (1) (n=27)	Group (2) (n=27)	P value
PPBS (mg/dl)	**Mean ± SD**	212.04 ± 35.05	108.15 ± 5.04	**<0.001***
	Range	154 - 290	100 - 119	
HbA1C (%)	**Mean ± SD**	8.4 ± 0.92	4.64 ± 0.37	**<0.001***
	Range	7 - 10	4 - 5.2	
Urea (mg/dl)	**Mean ± SD**	35.81 ± 8.55	27.48 ± 6.77	**<0.001***
	Range	20 - 51	8 - 39	
Creatinine (mg/dl)	**Mean ± SD**	1.04 ± 0.15	0.9 ± 0.12	**<0.001***
	Range	0.7 - 1.3	0.7 - 1.1	
ALT (U/L)	**Mean ± SD**	27.19 ± 10.96	17.11 ± 6.07	**<0.001***
	Range	11 - 57	8 - 31	
AST (U/L)	**Mean ± SD**	34.3 ± 12.74	23.26 ± 8.07	**<0.001***
	Range	15 - 61	9.5 - 45	
Total bilirubin (mg/dL)	**Mean ± SD**	1.07 ± 0.16	0.95 ± 0.09	**0.001***
	Range	0.8 - 1.6	0.7 - 1.1	
PT/INR	**Mean ± SD**	1.1 ± 0.15	0.98 ± 0.1	**<0.001***
	Range	0.7 - 1.3	0.8 - 1.2	

Hb: hemoglobin, WBCs: white blood cells, FBS: fasting blood sugar, PPBS: post-prandial blood sugar, ALT: alanine transaminase, AST: aspartateaminotransferase, PT: prothrombin time, INR: International normalized ratio, *:significant as P value ≤ 0.05

Table 3: Helicobacter Pylori antigen in stool of the studied groups

		Group (1) (n=27)	Group (2) (n=27)	P value
Helicobacter Pylori antigen in stool (µg/ml.)	**Mean ± SD**	13.61 ± 3.13	12.37 ± 2.74	0.128
	Range	0.02 – 29.5	0.01 – 28.7	
Helicobacter Pylori antigen in stool	**Positive**	27 (100%)	27 (100%)	1

Table 4: Upper GIT endoscopy findings of the studied groups

	Group (1) (n=27)	Group (2) (n=27)	P value
Esophagitis	3 (11.11%)	4 (14.81%)	0.540
Esophageal ulcer	4 (14.81%)	0 (0%)	
Axial hernia	1 (3.7%)	0 (0%)	
Pangastritis	10 (37.04%)	10 (37.04%)	
Antral gastritis	12 (44.44%)	11 (40.74%)	
Hypokinetic pylorus	2 (7.41%)	1 (3.7%)	
Duodenitis	4 (14.81%)	5 (18.52%)	
Duodenal ulcer	3 (11.11%)	1 (3.7%)	

Table 5: Ultrasound findings of the studied groups

	Group (1) (n=27)	Group (2) (n=27)	P value
Normal	11 (40.74%)	21 (77.78%)	**0.006***
Bulky spleen	4 (14.81%)	1 (3.7%)	0.351
Fatty liver	7 (25.93%)	0 (0%)	**0.010***
Congested liver	2 (7.41%)	0 (0%)	0.491
Gall bladder stone	1 (3.7%)	1 (3.7%)	1.000
Hydronephrosis	1 (3.7%)	0 (0%)	1.000
Pleural effusion	1 (3.7%)	0 (0%)	1.000
Ovarian cyst	0 (0%)	2 (7.41%)	0.491

Table 6: Histopathological finding of the studied groups

		Group (1) (n=27)	Group (2) (n=27)	P value
Specimen Type	**Endoscope**	27 (100%)	27 (100%)	---
Activity	**Active**	1 (3.7%)	3 (11.11%)	0.134
	Superficial	23 (85.19%)	24 (88.89%)	
	Deep	3 (11.11%)	0 (0%)	
Mucosa	**Normal**	26 (96.3%)	26 (96.3%)	---
	Focal ulceration	1 (3.7%)	1 (3.7%)	
Lamina propria	**Minimal inflammation**	23 (85.19%)	16 (59.26%)	0.081

		Group (1) (n=27)	Group (2) (n=27)	
	Moderate inflammation	3 (11.11%)	10 (37.04%)	
	Dense inflammation	1 (3.7%)	1 (3.7%)	
Intestinal metaplasia	**Present**	4 (14.81%)	6 (22.22%)	0.728
	Absent	23 (85.19%)	21 (77.78%)	
Dysplasia	**Present**	0 (0%)	0 (0%)	---
	Absent	27 (100%)	27 (100%)	
Eosinophils	**Median**	3	4	**0.018***
	IQR	2 - 5	3 - 10	
Neutrophils	**Median**	0	0	0.622
	IQR	0 - 0	0 - 0	
Germinal centers	**Present**	5 (18.52%)	8 (29.63%)	0.340
	Absent	22 (81.48%)	19 (70.37%)	
H. pylori	**Positive**	16 (59.26%)	22 (81.48%)	0.074
	Negative	11 (40.74%)	5 (18.52%)	

FBS: fasting blood sugar, PPBS: post-prandial blood sugar, Hb: hemoglobin, WBCs: white blood cells, ALT: alanine transaminase, AST: aspartate aminotransferase, PT: prothrombin time, INR: International normalized ratio, *: significant as P value ≤ 0.05

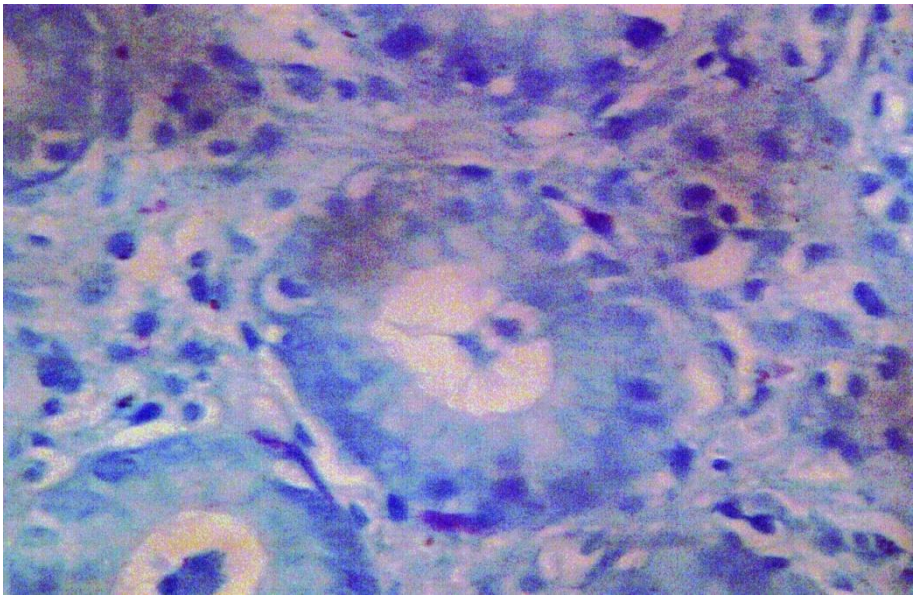

Figure 1: Photomicrograph of endoscopic gastric biopsy of chronic gastritis showing comma shaped H. pylori at the luminal surface, Giemsa stain x 100.

DISCUSSION

We found that HTN was significantly increased in group (1) (DM group) than group (2) (control group) (P value <0.001 and =0.005 respectively). Age, sex, dyslipidemia, COPD, and asthma were not statistically different among the groups under study.

In agreement with our study, Anastasioset al. [8]enrolled 172 patients aged 59.2 years who were eligible for analysis, 99 were men (age 58.8 years) and 73 were women (age 59.76 years). A total of 67 patients (39 men and 28 women aged 61.46 years) were diabetics. Of these patients, 64 (95.5%) had reported a history of diabetes and three patients (4.5%) were newly diagnosed. The remaining 105 subjects (60 men and 45 women aged 58.8613.3 years) did not have diabetes. The mean age or gender difference between the two groups was insignificant (diabetics vs. nondiabetics).

Alzahraniet al. [6] performed a cross sectional study on a 209 age-matched non-diabetic participants and 212 diabetes patients aged 40 or older were covered by the research. It was found that among type 2 diabetics, there was no statistically significant variation in the prevalence of H. pylori according to age groups, gender, smoking status, body mass index, or chronic diseases.

In the present study, epigastric pain was the most common complain, There was no discernible difference in complaints between the groups under study. In agreement with our study,Kutluk et al. [9] studied 339 patients (183 females) and reported that Epigastric pain was the most common complaint in 61.9% of the cases. Dyspeptic symptoms like bloating, early satiety, abdominal distension, and belching were seen in 45.1% of the individuals receiving care. Forty-two percent of the patients had both complaints. Other frequently observed symptoms included anemia in 20.4% of the patients, failure to thrive in 34.5%, and reflux symptoms such as nausea, vomiting, regurgitation, retrosternal pain, and difficulty swallowing in 21.2% of the patients. In addition, 14% of the patients reported additional issues such diarrhea and appetite loss.

Also, Anastasios et al. [8] demonstrated that there was no discernible variation in the frequency of gastric lesions. (gastritis and/or) between the two groups (P50.71) Peptic ulcer prevalence was higher in people who were not diabetics, although the difference was not statistically significant (P<0.08).

One of the most frequent reasons diabetic patients visit the hospital is with dyspeptic problems, which lowers the patients' quality of life. Studies have indicated that individuals with diabetes mellitus experience dyspeptic issues more frequently than those without the disease[10].

In the current investigation, group (1) (the DM group) had considerably more platelets than group (2) (control group) (P value <0.05).In agreement with our study, Mortensen et al. [11], Christensen et al. [12] have shown that diabetes patients frequently experience increases in immature platelet counts, platelet aggregation, and platelet activation—even when using aspirin to prevent vascular damage.

Urea and creatinine levels in the group under investigation were substantially higher (1) (DM group) than group (2) (control group) (P value <0.05).In agreement with our study ,Qais [13]45 individuals in all were enrolled (30 diabetics and 15 controls). Ten of the fifteen control individuals were female and five were male, while sixteen of the fourteen diabetic participants were female. Serum creatinine levels were elevated in 10 persons with diabetes and 0 subjects without the disease. Additionally, just one participant who was not diabetic showed elevated serum urea readings, compared to five who were diabetics. They stated that there was a small rise. There is a statistically significant difference in serum creatinine levels between diabetes patients and controls, but not in blood urea levels between the two groups.

Under real-world clinical conditions, conducting research on how diabetes develops into DKD is difficult and time-consuming [14].

We showed that ALT, AST total bilirubin, and PT/INR were significantly increased in group (1) (DM group) than group (2) (control group) (P value <0.05).

In agreement with our study, Shibabawet al. [15] performed Blood samples were taken from 192 patients with diabetes and 192 volunteers of a similar age and sex who participated in a cross-sectional study. They demonstrated that the mean values of ALT and AST in people with type 2 diabetes were significantly greater than those in the control group (P < 0.001).

Also, a recent study on Sudanese diabetic sufferers and 50 seemingly healthy control persons. The study's findings demonstrated that the diabetic group's mean ALT and AST readings were substantially higher than those of the control group [15]. Salmela et al. [16] revealed that 17.0% of the DM patients in their research had aberrant ALT.

We found that Hb was significantly decreased in group (1) (DM group) than group (2)

(control group) (P value =0.047). In agreement with our study, Arkew et al. [17] demonstrated that the Hb, a measure of anemia, was considerably lower in diabetes patients than in controls.

Other investigations conducted in Bangladesh also corroborate this conclusion [18], India [19], Libya [20], and Nigeria [21] the diabetes patients' Hgbhas been reported to be considerably less than that of the group under control. Anemia in diabetics has a complex etiology that involves, in addition to renal disease, AGEs, oxidative stress, inflammation, chronic hyperglycemia, nutritional inadequacies, medicines, and aberrant hormone levels [22].

We observed that WBCs were not statistically different among the groups under study.In agreement with our study, Oda et al. [23]There was no correlation between WBC count and diabetes during a six-year follow-up.Although there is disagreement, a number of studies have linked White Blood Cells (WBC), a sign of subclinical inflammation, to type 2 diabetes and insulin resistance[24].Our results found that Helicobacter Pylori in stool and in histopathology was not statistically different among the groups under study.

The association between H. pylori infection and diabetes is controversial because certain studies indicate that the infection is more common in those with the disease, while no difference has been observed in the other groups [25].

Stanciuet al. [26]examined whether H. Pylori levels varied between people with diabetes and those without the disease, however it was concluded that the two groups did not differ noticeably from one another.

The results of Alzahrani et al. [6]study found that the prevalence of H. pylori infection was somewhat higher in diabetics than in non-diabetics. (26.9% and 26.3%, respectively). This finding accords with those of several studies. The prevalence of H. pylori infection in type 2 diabetics ranges from 30% to 78%. There was no discernible variation in the frequency of H. pylori infection between individuals with diabetes and those without it. This result is consistent with recent studies that found no discernible variation in the prevalence of H. pylori infection between individuals with and without diabetes [6].

Bener et al. [27]claimed significant variations in the prevalence of H. pylori infection could be attributed to the epidemiological dispersion of H. pylori.

In Hong Kong, Malecki et al. [28]discovered that Chinese people with type 2 diabetes, similar to the controls, had an approximately 50% frequency of H. pylori infection.

In Athens, Greece, Anastasios et al. [8] said that compared to people without diabetes, there was a little rise in H. pylori infection among diabetics (37.3% and 35.2%, respectively), However, there was no statistically significant change.

Xia et al. [29] discovered that the seroprevalence of H. pylori infection in China did not differ statistically significantly between diabetics and non-diabetic controls.

Our results came in line with Oluyemi et al. [30] additionally found that there was no appreciable difference between controls and type 2 diabetics in Nigeria regarding the prevalence of H. pylori infection. Similar outcomes have been observed in other countries, such Italy [31], China [32], Turkey [33], and Romania [26].

Ricci et al. [34] Recent research revealed that patients with diabetes experience upper gastrointestinal symptoms more frequently than those without diabetes (controls), symptoms that are linked to Helicobacter pylori (H. pylori) infection.

Kouitcheu et al. [35] revealed that, in comparison to Patients in the non-diabetic group and those with diabetes showed significantly greater H. pylori infection rates. It was found that H. pylori seropositivity was present in patients with dyspeptic DM to be 73.11% and for the dyspeptic control group it was 58.05% (p = 0.0279), suggesting a positive relationship between H. pylori seropositivity and diabetes.

Gentiles et al. [36] reported H. pylori infection rate of 74.4% against 50% in their diabetic and control population respectively.

Candelli et al. [37] Their research revealed that individuals with diabetes had a higher prevalence of H. pylori infection than the control group, and that the diabetes patients experienced a higher rate of reinfection three years after a normal eradication treatment.

Indeed, a study conducted on the Japanese population found that the prevalence of the metabolic syndrome is elevated by H. pylori infection [38].

He et al. [39] noted that the differences in the reported associations between H. pylori infection and diabetes may be caused by differences in the criteria used to determine H. pylori positivity and diabetic status, small sample sizes, and adjustments for potential confounders like age and socioeconomic status.We demonstrated that the findings from upper GIT

endoscopy did not differ statistically across the groups under study.

Patients with diabetes mellitus frequently complain of dyspepsia, however it's unclear if endoscopic results differ from those of non-diabetic individuals. In research done by Ramazan et al. [40] wherein the When dyspeptic symptom frequency was evaluated across groups with and without diabetes, individuals with DM were shown to have significantly higher dyspeptic complaints. In the same study, patients with diabetes mellitus had a higher incidence of stomach ulcers, but there was no statistically significant difference in terms of other endoscopic findings when the endoscopic results from the two groups were compared.

Vasihnav et al. [41] examined the endoscopic findings between people with and without diabetes. They found that, in comparison to non-diabetics, patients with diabetes had higher rates of pangastritis, bulbitis, and HP.

Ullah et al. [42] demonstrated that, of the endoscopic findings, nodular and congestive gastritis were the most prevalent, occurring in 47.8% of patients. 26.7% of patients had ulcerative gastritis, whereas 12.2% and 13.3% of patients had esophagitis and hiatal hernia, respectively. Their findings are consistent with the study, which found that ulcerative gastritis was the most common condition, followed by congestive and nodular gastritis.

In relation to ultrasonography results, normal findings of group (1) (DM group) were significantly decreased than those of group (2) (control group) (P value =0.006). Fatty liver was significantly increased in group (1) (DM group) than group (2) (control group) (P value =0.010). Bulky spleen, congested liver, gall bladder stone, hydronephrosis, pleural effusion, and ovarian cyst were insignificantly different between the studied groups.

In agreement with our study, Agarwal et al. [43] also discovered that diabetics have a significant prevalence of fatty liver disease as 69.5%, 87% respectively.

Regarding liver abnormalities in DM, The presence of glycogen hepatopathy must be acknowledged. Hepatomegaly caused by excessive liver glycogen deposition was first reported in the early years following the introduction of insulin for the treatment of diabetes [44].

Limitations

Our study limitations are that the study was conducted at a single center with small sample size. We recommend further multi center studies to validate of results. Additional studies are needed on a larger scale for assessment of association between the complications of diabetes and H. pylori infection in relation to gastrointestinal disease in diabetics. Additional studies are required to evaluate the relation between H. pylori and level of HbA1c, duration of diabetes. Multiple studies for assessment of H. pylori infection and the risk of diabetes are required.

Declaration of interest

The authors report no conflicts of interest. The authors along are responsible for the content and writing of the paper.

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CONCLUSIONS

Our findings did not find any link between diabetes mellitus as well as the degree of H. pylori infection. This is supported by the observation that people with and without diabetes had the same level of H. pylori infection.

REFERENCES

1. **Nodoushan H, Nabavi A.** The interaction of Helicobacter pylori infection and type 2 diabetes mellitus. *Adv. Biomed. Res.*, 2019; *8*.
2. **Agrawal V, Agrawal S, Sharma K, Tandia K.** Study of the Association between Type 2 Diabetes Mellitus and Helicobacter Pylori Infection in Indian Population. *Eur. Heart J*, 2023; *13*(1).
3. **Miller K, Williams M.** Helicobacter pylori infection causes both protective and deleterious effects in human health and disease. Genes Immun., 2021; 22(4), 218-226.
4. **Radionova O, Skrypnyk M, Maslova S.** Do H. Pylori status and small intestinal bacterial overgrowth determine the clinical course of chronic active gastritis in patients with type 2 diabetes mellitus.WiadLek, 2020;73(6), 1223-8.

5. **Maluf S, Salgado V, Cysne N, Camelo F, Nascimento R, Maluf T, et al.** Increased glycated hemoglobin levels in patients with Helicobacter pylori infection are associated with the grading of chronic gastritis. *Front. immunol.*, 2020; *11*, 2121.
6. **Alzahrani M, Al Zaidi A, Alzahrani M, Binmahfouz A, Farahat M.** Association between type 2 diabetes mellitus and Helicobacter pylori infection among Saudi patients attending National Guard Primary Health Care Centers in the Western Region, 2018. *J. Fam. Community Med.*, 2020; *27*(1), 8.
7. **American Diabetes Association Professional Practice Committee, American Diabetes Association Professional Practice Committee.** 2. Classification and diagnosis of diabetes: Standards of Medical Care in Diabetes—2022. Diabetes Care, 2022; 45(Supplement_1), S17-S38.
8. Anastasios R, Goritsas C, Papamihail C, Trigidou R, Garzonis P, Ferti A. Helicobacter pylori infection in diabetic patients: prevalence and endoscopic findings. *Eur. J. Intern. Med.*, 2002; *13*(6), 376-379.
9. Kutluk G, Polat E, Çiçek M, Oral K, Karatekin M, Gündüz N. Complaints, Endoscopic and Histopathological Findings in Children with Helicobacter pylori Infection: Are There Any Correlations with Each Other?. *J BehcetUz Child Hosp*, 2023; *13*(2).
10. **Bilgili A.** Endoscopic and Histopathological Findings, And The Presence of Helicobacter Pylori in Diabetic and Non-Diabetic Patients Admitted to the Emergency Department with Dyspeptic Complaints. East. J. Med., 2021; 26(3), 371-375.
11. **Mortensen B, Larsen B, Grove L, Kristensen D, Hvas M.** Reduced platelet response to aspirin in patients with coronary artery disease and type 2 diabetes mellitus. *Thromb Res*, 2010; *126*(4), e318-e322.
12. Christensen H, Grove L, Würtz M, Kristensen D, Hvas M. Reduced antiplatelet effect of aspirin during 24 hours in patients with coronary artery disease and type 2 diabetes. *Platelets*, 2015; *26*(3), 230-235.
13. **Qais A.** Estimation of Urea and Creatinine in Type 2 Diabetes Mellitus Patients. Scand J Clin Lab Invest, 2020; 175-88.
14. **Chen Y, Lee K, Ni Z, He C.** Diabetic kidney disease: challenges, advances, and opportunities. *KIDNEY DIS-BASEL*, 2020; *6*(4), 215-225.
15. **Shibabaw T, Dessie G, Molla D, Zerihun F, Ayelign B.** Assessment of liver marker enzymes and its association with type 2 diabetes mellitus in Northwest Ethiopia. *BMC Res Notes*, 2019; *12*, 1-5.
16. Salmela I, Sotaniemi A, Niemi M, Mäentausta O. Liver function tests in diabetic patients. *Diabetes care*, *7*(3), 1984; 248-254.
17. **Arkew M, Yemane T, Mengistu Y, Gemechu K, Tesfaye G.** Hematological parameters of type 2 diabetic adult patients at DebreBerhan Referral Hospital, Northeast Ethiopia: A

comparative cross-sectional study. *PloS one*, 2021; *16*(6), e0253286.

18. **Alam J, Chandra M, Mokarrama N, Hoque M, Hasan M, Islam S.** A comparative analysis of biochemical and hematological parameters in diabetic and non-diabetic adults. *Adv Med Sci*, 2015; *2*(1), 1-9.
19. Umeji L, Paul A, Felix S, Umeji K, Folake A, Chrisitian N. Haematological profile of diabetes and non-diabetes patients in Abuja, Nigeria. *IJRSI*, 2019; *6*(5), 2321-2705.
20. **Al Salhen K, Mahmoud A.** Hematological profile of patients with type 2 diabetic mellitus in El-Beida, Libya. Sci Rep, 2017; 9, 76-80.
21. **Awofisoye I, Adeleye O, Olaniyi A, Esan A.** Prevalence and correlates of anemia in type 2 diabetes mellitus: a study of a Nigerian outpatient diabetic population. *Sahel Med J, 2019; 22*(2), 55.
22. **Sahay M, Kalra S, Badani R, Bantwal G, Bhoraskar A, Das K, et al.** Diabetes and Anemia: International Diabetes Federation (IDF)–Southeast Asian Region (SEAR) position statement. *Diabetes MetabSyndrObes.: Targets Ther*, 2017; *11*, S685-S695.
23. **Oda E.** High-sensitivity C-reactive protein, but not white blood cell count, independently predicted incident diabetes in a Japanese health screening population. ActaDiabetol, 2015; 52, 983-90.
24. **Kheradmand M, Ranjbaran H, Alizadeh-Navaei R, Yakhkeshi R, Moosazadeh M.** Association between white blood cells count and diabetes mellitus in Tabari cohort study: A case-control study. *Int J Prev Med*, 2021; *12*.
25. **PáezPino P.** Presencia de helicobacter pylori asociado al tratamiento de 14 días con inhibidor de bomba de protones, claritromicina y amoxicilinaenpacientes del servicio de gastroenterología de la consulta externa del Hospital Pablo Arturo Suárez desdeenero a diciembre de 2019. ActaDiabetol, 2022; 12-23.
26. **Stanciu G, Trifan A, Sfarti C, Cojocariu C, Stanciu C.** Helicobacter pylori infection in patients with diabetes mellitus. *Rev Med ChirSoc Med Nat Iasi, 2003; 107, 59-65.*
27. **Bener A, Micallef R, Afifi M, Derbala M, Al-Mulla M, Usmani A.** Association between type 2 diabetes mellitus and Helicobacter pylori infection. *Turk J Gastroenterol*, 2007; *18*(4), 225-229.
28. **Małlecki M, Bień I, Galicka-Latałla D, Stachura J, Sieradzki J.** The prevalence of Helicobacter pylori infection and types of gastritis in diabetic patients. The Kraków study. ExpClinEndocrinol Diabetes. 1996;104:365–9.
29. **Xia X, Talley J, Kam P, Young J, Hammer J, Horowitz M.** Helicobacter pylori infection is not associated with diabetes mellitus, nor with upper gastrointestinal symptoms in diabetes mellitus. *Am. J. Gastroenterol.*, 2001; *96*(4), 1039-1046.
30. **Oluyemi A, Anomneze E, Smith S, Fasanmade O.** Prevalence of a marker of active Helicobacter pylori infection among patients with type 2 diabetes

mellitus in Lagos, Nigeria. *BMC Res. Notes*, 2012; *5*, 1-4.

31. **Dore P, Bilotta M, Malaty M, Pacifico A, Maioli M, Graham Y, et al.** Diabetes mellitus and Helicobacter pylori infection. *J Nutr*, 2000; *16*(6), 407-410.
32. **Ko T, Chan K, Chan B, Sung J, Tsoi L, To F, et al.** Helicobacter pylori infection in Chinese subjects with type 2 diabetes. *Endocr Res*, 2001; *27*(1-2), 171-177.
33. **Demir M, Gokturk S, Ozturk A, Kulaksizoglu M, Serin E, Yilmaz U.** Helicobacter pylori prevalence in diabetes mellitus patients with dyspeptic symptoms and its relationship to glycemic control and late complications. *Dig Dis Sci*, 2008; *53*, 2646-2649.
34. **Ricci A, Siddique R, Stewart F, Sandler S, Sloan S, Farup E.** Upper gastrointestinal symptoms in a US national sample of adults with diabetes. *Scand J Gastroenterol*, 2000; *35*(2), 152-159.
35. **KouitcheuMabeku B, NoundjeuNgamga L, Leundji H.** Helicobacter pylori infection, a risk factor for Type 2 diabetes mellitus: a hospital-based cross-sectional study among dyspeptic patients in Douala-Cameroon. *Sci. Rep.*, 2020; *10*(1), 1-11.
36. **Gentile S, Turco S, Oliviero B, Torella R.** The role of autonomic neuropathy as a risk factor of Helicobacter pylori infection in dyspeptic patients with type 2 diabetes mellitus. *Diabetes Res ClinPract*, 1998; *42*(1), 41-48.
37. **Candelli M, Rigante D, Schiavino A, Gabrielli M, Crea F, Del Lungo M, et al.** High reinfection rate of Helicobacter pylori in young type 1 diabetic patients: a three-year follow-up study. *Age (years)*, 2012; *19*(4.3), 19-8.
38. **Gunji T, Matsuhashi N, Sato H, Fujibayashi K, Okumura M, Sasabe N, et al.** Helicobacter pylori infection significantly increases insulin resistance in the asymptomatic Japanese population. *Helicobacter*, 2009; *14*(5), 496-502.
39. **He C, Yang Z, Lu H.** Helicobacter pylori infection and diabetes: is it a myth or fact? World J Gastroenterol, 2014; 20, 4607-12.
40. **Ramazan O, Karincaoglu M.** Correlation of dyspeptic symptoms and endoscopic findings in diabetic patient. Med SciDiscov, 2018; 5, 130-6.
41. **Vaishnav T, Shaikh R, Bamanikar A, Kakrani L, Tambile R.** Diagnostic upper gastrointestinal endoscopy and prevalence of Helicobacter Pylori infection in dyspeptic type 2 diabetes mellitus patients. *J Dig Endosc*, 2018; *9*(02), 053-060.
42. **Ullah R, Raja K, Zaidi A, Razzak A, Butt S, Kanwal K.** Histological Findings Of Helicobacter Pylori In Diabetic Patients. *Pak J Med Sci*, 2023; *17*(05), 300-300.
43. **Agarwal K, Jain V, Singla S, Baruah P, Arya V, Yadav R, et al.** Prevalence of non-alcoholic fatty liver disease and its correlation with coronary risk factors in

patients with type 2 diabetes. *J Assoc Physicians India*, 2011; *59*, 351-354.

44. **Torbenson M, Chen Y, Brunt E, Cummings W, Gottfried M, Jakate S, et al.** Glycogenic hepatopathy: an underrecognized hepatic complication of diabetes mellitus. *Am J SurgPathol, 2006; 30*(4), 508-513.

citation

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