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CORRELATION BETWEEN HELICOBACTER PYLORI AND TYPE 2 DIABETES IN KIRKUK-IRAQ

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Article history:	Abstract:
Received: Accepted: Published:January 28th 2022 February 28th 2022 April 7th 2022	 Background: Helicobacter pylori is a common bacterial pathogen of human beings, causes different gastric problems as ulcers, tumors and inflammation as gastritis. The chronic inflammation may have impact on glucose and lipid absorption. Diabetes mellitus is characterized by a high level of blood glucose Glycated hemoglobin (HBA1C) The objective of the study is to show the relationship between H. pylori and diabetes mellitus. Methods: The study was conducted on 116 patients with type 2 diabetes mellitus who attended K1 hospital in North Oil Company, Kirkuk, and 80 healthy individuals. <i>H. pylori</i> was detected by using <i>H. pylori</i> antigen rapid test in feces. Estimation of sugar was done by a high level of blood glucose and glycated hemoglobin (HBAIC) using a fully smart automated clinical chemistry analyzer. Results: This study showed the frequency of <i>H. pylori</i> in diabetic patients was 28.4%, the highest rate was among 30-39 years, employees, and illiterates. The lowest was among university-educated level and no cases seen among students. Conclusions: Higher rate of H. pylori among patients with diabetes may indicate a strong link between H. pylori and Type 2 diabetes mellitus.
Keywords: Helicobacter pylori, Type	e 2 diabetes, K1 hospital, Kirkuk.

INTRODUCTION:

Helicobacter pylori is gram-negative, flagellates, microaerophilic bacteria (1). Its main route of transmission is not yet recognized, it principally appear within family members. It is transmitted from person to person through fecal-oral or oral to oral route. Several studies reported several factors level of increase the prevalence of H. pylori, such as low socioeconomic status and overcrowding (2).

It has been reported that has role in several gastrointestinal diseases such as gastritis, peptic ulcer, lymphoma associated lymphoid tissue lymphoma (3,4). It is also shown that has effect on the occurrence of masny extra-gastric diseases such as allergic diseases, cardiovascular, neurodegenerative and hematological disorders and atherosclerosis(5 & 6).

There are controversy about insulin resistance in clinical conditions. Some researchers suggested that H. pylori in chronic state might prevent insulin sensitivity (7 & 8). while others showed that H. pylori may be related to progression and complication of type 2

diabetes mellitus (9). Several studies did not show that H. pylori infection has an association with type 2DM development or worsening (10 & 11).

There are several risk factors has relation with pathogenesis of T2DM of life styles, diet, obesity and socioeconomic, physical activity and genetic background (12). It is well known that T2DM there will be accumulation of sugar in blood stream which is due to pancrease can not produce enough insulin to overcome the cellular loss of sensitivity (13).

Several publications reported that there is significant association between H. pylori and T2 DM, reporting that the prevalence of infection in DM patients is higher than non diabetic ones (14).

The most recent evidence indicates that the initial step in development of T2DM is inflammation which may be induced by H. pylori infection (15 & 16).

The International Diabetes Federation (IDF) estimated that the prevalence of people with diabetes may be doubled in Middle East and North Africa by 20930, it mat reach to 51.7 million, while the total number of



diabetic cases will be increased to 366 million in 2030 (17). Globally, the total number of people with diabetes is projected to rise from 171 million in 2000 to 366 million in 2030. With the greatest increase occurring in developing compared with developed countries. It is expected that by 2030, the number of individuals with diabetes worldwide is expected to rise to 472 million. Diabetes in developed countries, it's a disease of the elderly while in Arab countries, it's a disease of younger age <60 years, which is the most productive age, making the problem of diabetes even worse (17).

Rafat et al. (18) showed a higher frequency of *H. pylori* in diabetic patients, diabetic patients with proteinuria had a higher frequency of *H. pylori* infection. Also, H. pylori-positive patients had significantly higher HbA1c, FBG, and 2H PPBG. Furthermore, Significantly higher cholesterol, triglyceride, LDL, and microalbuminuria was observed in *H. pylori*-positive cases than H. pylori negatives.

AIM

The objective of the study is to investigate the association between *H. pylori* infection and the risk of DM among patients who attended K1 outpatients Clinic, Kirkuk-Iraq.

METHODS

The study was carried out from- beginning of October 2020- to the end of August 2020. A blood sample was taken from 116 diabetic patients who attended K! hospital Kirkuk and 80 healthy controls. Then the

sample was placed into two tubes, the first tube containing sodium citrate as the anticoagulant was shaken to prevent hemolysis; was placed to auto analyser (COBAS) following centrifugation in order to measure HbA1c. and fasting blood sugar using a fully smart automated clinical chemistry analyzer. For detecting *H. pylori*, some drop of the sample was placed on a kit manufactured for this test. The diagnosis was performed using a serological test for H. pylori-specific immunoglobulin; H. pylori antigen was detected by using a rapid test kit in feces (Chromatographic immunoassay).

Statistical analysis: Chi-square was used to show significant differences between two groups in the study. P-value < 0.05 was used to show stat significance (19). Strata version 12 was used to compare the groups.

RESULTS

In the present cross-sectional study all 116 individuals with or without diabetes mellitus

Table 1, shows the distribution of *H. pylori* related to age groups. The overall positive rate of *H. pylori* was (28.4%). No positive cases was detected among 10-19 and 20-29 years age groups. The highest rate of positive cases was among 30-39 years old (50%) followed by 40-49 (33.3%), 50-59 (30.9%), and 60 above (21.6%) respectively. The rate of positive cases among males was lower in males (28.2%) than in females (29.0%).

Table 1: Distribution of H. Pylori according to age and gender.

	No. examined		No. Positive			Positive %			
Age / Year	Male	Female	Total	Male	Female	Total	Male	Female	Total
10-19	2	0	2	0	0	0	0	0	0
20-29	1	1	2	0	0	0	0	0	0
30-39	5	1	6	3	0	3	3.5%	0	50%
40-49	17	10	27	7	2	9	8.2	6.5	33.3
50-59	32	10	42	9	4	13	10.6	12.9	30.9
60 and>	28	9	37	5	3	8	5.9	9.7	21.6
Total	85	31	116	24	9	33	28.2	29.0	28.4

X²=2.530

D.F.=5



Table 2 shows the distribution of H. pylori according to gender. It was shown that the rate of H. pylori-positive cases in females (29.0%) was slightly higher than in males (28.2%).

Table 2	Table 2. Distribution of <i>H. pylori</i> among patients and healthy controls according to gender.					
Gender	Patients attended hospitals			Controls (Healthy)		
Centuer	No. examined	No. positive	Positive %	No. examined	No. Positive	Positive %
Male	85	24	28.2	70	6	8.6
Female	31	9	29.0	10	2	20
Total	116	33	28.4	80	8	10
X ² =9.741				D.F.=1	(P<0.05)

Table 3 shows the distribution of *H. pylori* according to occupations, the rate of positive cases among employees was greatest (31.6%), followed by housewives (30.7%), officials (28.3%), retired (20.0%), no positive case was detected among students.

Table 5. Distribution of <i>Tr. pytoin</i> according to occupation.					
Occupation	No. examined	No. positive	Positive %		
Students	1	0	0		
Housewives	26	8	30.7		
Official	60	17	28.3		
Employee	19	6	31.6		
Retired	10	2	20.0		
Total	116	33	28.4		
Total	110	55	2011		

X²=0.581 D.F.=4 P>0.05 Table 4 shows the distribution of *H. pylori* according to educational levels, the rate of H. pylori was highest among illiterate levels (35.7.0%) followed by primary (26.7%), secondary (24.1%) and the lowest was among university (20.0%) respectively.

Table 4. Distribution of <i>H. pylon</i> according to educational level.						
Education	No. examined	No. positive	Positive %			
Illiterate	42	15	35.7			
Primary	30	8	26.7			
Secondary	29	7	24.1			
University	15	3	20.0			
Total	116	33	28.4			
X ² =1.067		D.F.=3	P>0.05			

Table 4 Distribution of *H. pylori* according to educational level

DISCUSSION

The rate of *H. pylori* among diabetic patients was 59% while in non-diabetics was 31% (20).

It was observed that HbA1c and H. pylori infection were higher among patients with high body mass index (BMI), indicating that the patients had high blood glucose and perhaps high lipid levels leading to chronic inflammation.

The contribution of *H. pylori*-associated with diabetes may be due to insulin resistance and chronic inflammation, as gastritis caused by H. pylori may be affected by disturbance in secretion of different cytokines and gastric related hormones.

According to Hill'S criteria, till now no direct causal relation between diabetes and H. pylori was proved so further meta-analysis is adviced to confirm such association (15).

Overall the link between diabetes mellitus is controversial as some studies report no difference while others show higher in diabetic patients (21,22).

There is strong evidence based medicine that 5% weight loss may prevent developing T2DM among impaired glucose tolerance patients(23).



As it was mentioned previously there is no obvious evidence linking obesity and *H. pylorus* and or high BMI. (24).

This increment *of H. pylori* infection may be due to reduced gastric motility.

According to Cohen et al, hypothesis; treating H. pylori among patients with high BMI may control or prevent T2DM by weight loss (25).

In contrary to Cohen et al, other researchers are demonstrating that, if H. pylori is eradicated it will significantly lead to increasing BMI by enhancing appetite via elevation of plasma ghrelin and reducing leptin levels (26).

Dyslipidemia with high concentration of triglyceride and bad cholesterol (LDL-C) a long with low concentration of good cholesterol (HDL-C) may be attributed to insulin resistance or insulin deficiency enhancing the development of T2DM (26), but not most studies found this association, so more sophisticated studies are needed to prone this link.

H. pylori may have potential benefits in early childhood, as reduced risk of asthma, reactivation of latent TB, diarrhea peptic ulcer disease, and may cause increased glucose intolerance (27).

Previous cohort studies couldn't show any evidence to prove that DM, IGT, diabetic nephropathy, or poor glycemic control had a direct causal association with past *H. pylori*.

In the future, further prospective or meta-analysis may be required to better explore the relationship between new or past *H. pylori* and the development of DM (28). Feww studies link between plasma level of total cholesterol, triglyceride, and LDL-C with *H. pylori* infection but they are insufficient and wide scale studies are needed to verify causal association (29).

H. pylori has two extremes; in adults, it may cause peptic and gastric cancer, and increased glucose tolerance, while in early childhood it may reduce the risk of gastrointestinal diseases and asthma (30).

A cohort study on large scale lacked to find an association between H. pylori and DM, IGT, or diabetic neuropathy, so confirm the exact relationship further studies are needed to prove the hypothesis (31).

CONCLUSIONS

In Diabetic patients, *H. pylori* is common in both genders. The highest rate of infection was among 30-39 and 40-49 years old. H. pylori infection was related to educational

RECOMMENDATION

The current study suggests that, eradication of H. pylori is necessary to control glucose level among T2DM and reduce serious complications.

REFERENCES

- 1. Alatbee AHD. High prevalence of *Helicobacter pylori* in Basra city Southern Iraq. Journal of Physics: L Conference Series, 2019, 1279 012073.
- Thevenot T, Josenhans C, Brown LM. *Helicobacter pylori*: epidemiology and routes of transmission. Epidemiol Rev, 2000, 22: 283-97.
- Personnet J. *Helicobacter pylori* and gastric cancer. Gastroentroenterol Clin. North Am., 1993, 22: 89k-104.
- 4. Wotherpoon AC, Ortiz-Hidalgo C, Falzon MR, Isaacson PG . Helicobacter pylori-associated gastritis and primary B-cell gastric lymphoma. Lancet 1991, 338, 1175-1176.
- YamaoAC, ka Yoshio. *Helicobacter pylori*. Molecular Genetics and cellular biology. Caister Academic Personnet J. *Helicobacter pylori* and gastric cancer. Gastroentroenterol Clin. North Am., 1993, 22: 89-104.
- 6. Gunji T, Matsuhashi N, Sato H. et al. A *Helicobacter pylori* infection significantly increases insulin resistance in the asymptomatic Japanese population. Helicobacter, 2009, 14: 144-150.
- Eshraghian A, Hashemi SA, Hamidian JA, et al. *Helicobacter pylori* infection as a risk factor for insulin resistance. Dig. Dis. Sci. , 2009, 54: 1966-1970.
- 8. Imai J, Yamada T, Saito T. et al. Eradication of insulin resistance. Lancet, 2009, 374: 264.
- 9. Horikawa C, Kodama S, Fujihara K. et al., High risk of failing eradication of *Helicobacter pylori* in patients with diabetes: A meta-analysis. Diabetes Res. Clin Pract., 2014, 106: 81-87.
- 10. Wada Y, Hamamoto Y, Kawasaki Y. et al., The eradication of *Helicobacter pylori* does not affect glycemic control in Japanese subjects with type 2 diabetes. Japn. Clin. Med., 2013, 4: 41-43.
- 11. Agardh E., Allebeck P., Hallqvist J. et al. Type 2 diabetes incidence and socio-economic position: a systematic review and metaanalysis. Int J. Epidemiol., 2011, 40: 804-818.
- 12. Perdichizzi G, Bottari M, Pallio S. et al. Gastric infection by *Helicobacter pylori* and antral gastritis in hyperglycemic obese and in



diabetic subjects. New Microbiol., 1996, 19: 149-154.

- 13. Vafacimanesh J., Rajabzadeh R., Ahmadi A. et al. Effect of *Helicobacter pylori* eradication on glycaemia control in patients with type 2 diabetes mellitus and comparison of two therapeutic regimens. Arab J. Gastroenterol., 2013, 14: 55-8.
- 14. Cong He, Yang Z, LU NH. *Heliciobacter pylori* infection and diabetes: Is it a myth or fact?. World J. Gastroenterology, 2014, 20(16): 4607-4617.
- Devrajani BR, Shah SZ, Soomro AA, Devrajani T. Type 2 diabetes mellitus : A risk factor for *Helicobacter pylori* infection: A hospital based case-control study. Int. J. Diabetes Dev Ctries, 2010, 30: 22-26.
- 16. Stanciu OG, Trifan A, Sfarti C et al. *Helicobacter pylori* infection in patients with diabetes mellitus. Res. Med. Chir Soc Med Nat lasi, 2003, 107: 59-65.
- 17. Mansour AA., Al-Douri F. Diabetes in Iraq: Facing the epidemic. A systematic review. Wulfenia Journal, 2015, 22(3): 258-273. <u>https://www.researchgate.net</u>/publication/280 084146
- Rafat MN, Azeem HA, Antably A. and Al-Sayed MT. Prevalence of *Helicobacter pylori* infection in patients with TYPE 2 Diabetes mellitus. Al-Azhar Assiut Medical Journal, 2015, 13(4) suppl.-1: 93-101.
- 19. Daniel WW. Biostatistics. A foundation for analysis in the Health Science 5 th edition. John Wiley and sons, USA, 2014.
- 20. Simon L, Tornoczky J, Toth M et al. The significance of of Campylobacter pylori infection in gastroenterologic and diabetic practice. Orv Hetil , 1989, 130: 1325-1329.
- 21. Tawfeeq RD, Amin ZA, Nuraddin SM et al. Relationship between type 11 diabetes mellitus and *Helicabacter pylori* infection in Erbil city. Zanco J. Med. Sci., 2019, 23(1): 43-50.
- 22. Jeon CY, Haan MN, Cheng Cetal. Helicobacter pylori infection is associated with an increased rate of diabetes. Diabetes Care, 2012, 35, 520-525. {pub Med}.
- 23. Astrup A., Finer N. Redefining type 2 diabetes "diabesity" or "obesity dependent diabetes mellitus"? Obes Rev 2000, 1: 57-59.
- 24. Isomoto H, Ueno H, Nishi Y. et al. Impact of *Helicobacter pylori* infection on ghrelin and various neuroendocrine hormones in plasma. World J. Gastroenterol, 2005, 11: 1644-1648.

- 25. Cohen D., Muhsen K. Association between *Helicobacter pylori* colonization and glycated hemoglobin levels: is this another reason to eradicate H. pylori in adulthood?. J. Infect Dis., 2012, 205: 1183-1185.
- 26. Nwokulo CU, Freshwater DA, O'Hare P, Randeva HS. Plasma ghrelin following cure of *Helicobacter pylori*. Gut 2003, 52: 637-640.
- 27. Goldberg IJ, Clinical review 124; Diabetic dyslipidemia; causes and consequences . J. Clin Endocrinol Metab, 2001, 86: 965-971.
- Chahil TJ, Ginsberg HN. Diabetic dyslipidemia. Endocrinol Metab Clin North Am, 2006, 35: 491-510.
- 29. Elizalde JL, Pique JM, Moreno V. et al. Changes in biochemical parameters related to atherosclerosis after Helicobacter pylori eradication. Aliment Pharmaco Ther, 2006, 24 Suppl 4: 58-64.
- 30. de Martel C, LIosa AE, Farr SM et al. Helicobacter pylori infection infection and the risk of development of esophageal adenocarcinoma. J. Infect Dis., 2005, 191: 761-767.
- 31. Pyo JH, Lee H, Choi SC et al. Lack of association between past *Helicobacter pylori* infection and diabetes: A two-cohort study. Nutrients, 2019, 11: 1874



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