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ORIGINAL ARTICLE

Helicobacter pylori Infection and Insulin Resistance in Diabetic and Nondiabetic Subjects

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Abstract

Background: ***Recent data found an increased risk of insulin resistance (IR) and Helicobacter pylori (HP) infection in HP-positive individuals regardless of whether they have diabetes.***

Objective: ***To explore the potential role of HP in increasing IR in diabetic and nondiabetic subjects.***

Patients and Methods: ***Of the eighty participants in the trial, 40 had type 2 diabetes (T2DM), and the other 40 were nondiabetics. An upper endoscope diagnosed HP infection, a biopsy was obtained, histopathology was examined, and a stool antigen test was done. Insulin resistance was diagnosed using the Homeostatic Model Assessment for Insulin Resistance (HOMA-IR) method. All patients were collected from Al-Hussien University Hospital – Internal Medicine Department in Cairo. Egypt.***

Results: ***Out of the 40 individuals with (T2DM), 30 had IR, and 10 did not. There were a total of 80 patients in the group. HP infection was substantially more common in IR patients than Non-IR patients (93.3% versus 60%, p-value = 0.011), as verified by stool antigen and histopathology. However, of the remaining 40 nondiabetic individuals, 16 had IR, and 24 did not; moreover, IR patients had a considerably greater rate of HP infection than non-IR patients (100% versus 75%, p-value = 0.030).***

Conclusion: ***This study indicates a significant relationship between HP infection and IR in diabetic and nondiabetic populations.***

Keywords: Helicobacter pylori; Insulin resistance; type 2 diabetes mellitus

1. Introduction

The discovery of Helicobacter pylori (HP) 1982 by researchers Warren and Marshall completely changed how stomach disorders are understood in the hospitality industry and how peptic ulcers are classified as non-infectious diseases. This gram-negative bacterium inhabits the stomach's antrum and body, using colonization factors and acid resistance to shield itself from the hostile environment.[1] HP infection predisposes to autoimmune thyroid illnesses, diabetes, and primary hyperparathyroidism, among other endocrine problems.[2]

HP may, therefore, induce chronic inflammation and modify the hormones in the gastrointestinal tract that regulate insulin, hence promoting insulin resistance. This is because insulin resistance can arise in the presence of inflammation or due to changes in counter-regulatory hormones caused by HP inflammation that impact insulin. [3]

The direct evidence for an association between chronic HP infection and IR showed higher Homeostatic Model Assessment for Insulin Resistance (HOMA-IR) scores in HPpositive individuals. However, this association is contradicted in some studies.[4]

This work aimed to study the potential role of HP in increasing insulin resistance in diabetic and non-diabetic subjects.

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2. Patients and methods

2.1.Study population:

A prospective observational study would involve a sample of 80 participants, 40 of whom were diabetic (T2DM) and 40 of whom were nondiabetic.

Patients were recruited from Al-Hussien University Hospital - Internal Medicine Department in Egypt and were consequently selected according to the following Inclusion criteria:

Age between 15 and 70. Patients who have one or more endoscopic features suggest HP gastritis.

Exclusion criteria: Chronic diseases, such as renal, hepatic, and malignancy; pregnant females; type 1 diabetes mellitus; endocrine disorders, including hypothyroidism and hyperthyroidism; autoimmune disorders that might interfere with laboratory results; and a history of antibiotics and proton pump inhibitors (PPIs) in the previous two weeks before upper endoscopy because of the high incidence of false negativity.

2.2.Subjects:

Participants who met the inclusion criteria were asked about their detailed medical history, history of previous HP medication, and lifestyle habits in order to obtain a full medical history. Also, a General abdominal examination was done. General Examinations, such as measuring blood pressure and pulse, were carried out on every patient.

2.3.Method:

Variables that were measured include age, sex, body mass index (BMI), blood pressure, fasting glucose, postprandial blood glucose, hemoglobin A1c, lipid profile, Renal function (Urea, Creatinine), Liver function (Albumin, Alkaline phosphatase, Alanine transaminase, Aspartate transaminase), and complete blood picture (CBC).

The study measured two main variables: HP infection and IR.

HP infection would also be determined using a stool antigen test, which detects the presence of HP antigens in stool by ELISA automation done by a device named Ultradiagnostic.

Additionally, every patient had an upper digestive tract endoscopy with a FUJIFILM BL-7000 or an Olympus EVIS LUCERA ELITE CV-290 (Olympus Medical System Corporations, Japan Tokyo), and the Sydney system took many stomach mucosal samples: 1, 2, and 3 represent the antrum and the center, 4, the lesser curve and the midway between the cardia and the incisura, and 5, the greater curve and the opposite of 4 respectively.[5]

After that, the samples were examined histopathologically to confirm HP's existence.

Furthermore, the homeostasis model evaluation of insulin resistance (HOMA-IR), a mathematical calculation based on fasting insulin and glucose levels, was used to estimate IR. A blood sample was obtained from every patient 3 ml, and then carried to the laboratory. Consequently, the sample was centrifuged, and the serum was extracted about 500ml. After that, 300ml from the serum was transformed into a device named (Mindray Hormone 960 – China) to get fasting plasma insulin (FPI) level, and the remaining 200ml of the serum was taken to a device called (Mindray Chemical – China) to obtain fasting plasma glucose (FPG) level were measured together with a constant. The product of FPG × FPI is an index of IR. HOMA-IR = (glucose × insulin)/22.5. Insulin concentration is reported in μU/L and glucose in mmol/L. The constant of 22.5 is a normalizing factor.

2.4.Ethical Considerations:

The study complied with the ethical guidelines for using human subjects in research, such as obtaining informed consent, maintaining confidentiality, minimizing risk and discomfort, and ensuring voluntary participation. Data was collected and kept confidential, and all patients were informed about their laboratory results.

The study also adhered to guidelines for handling biological samples and for using statistical methods appropriately. Prior to participant recruiting, the study had institutional review board (IRB) approval.

2.5.Statistical Analysis

Following collection, revision, coding, and entry, the data were loaded into IBM SPSS, a statistical package for social science, version 27. When the quantitative data were determined non-parametrically, they were given as the median and interquartile range (IQR). They were presented as the mean, standard deviations, and ranges when parametric. Quantitative variables were also shown as percentages and numbers. The Chi-square and Fisher exact tests were used to compare the qualitative data between the groups. The independent t-test was used to compare two independent groups with quantitative data and a parametric distribution; the Mann-Whitney test was used for non-parametric distributions. The allowable margin of error was set at 5%, while the confidence interval was set at 95%. Thus, the following p-value was deemed significant: a P-value greater than 0.05 indicates non-significantness (NS). $P < 0.05$ indicates significance (S). HS stands for highly significant (P-value < 0.01).

3. Results

In this study we found that the mean age of our patients who are (T2DM) was 47.20±10.79 years were females 70% and males 30% and for the non-diabetics was 22.70±8.30 were females 80% and males 20%. Table (1)

Additionally, it demonstrated that the mean age of the diabetic group was found to be greater than that of the non-diabetic group, a statistically significant difference. However, there was no statistically significant difference between the two groups with regard to sex, hypertension, or the primary presenting symptoms. Table (1)

Also for (HOMA-IR) the median of (T2DM) patients was 2.85(2.45-3.85) and in non-diabetic patients was 2.2(2.45-3.85) also the percentage of IR in (T2DM) patients was 75% and in non-diabetics was 40%, and they were highly significant statistically. Table (2)

Among patients of (T2DM), 30 patients were IR and 10 patients without IR. HP infection (confirmed by histopathology and stool antigen) was notably greater in IR patients than in non-IR individuals. (93.3% versus 60%, p-value = 0.011). Table (3)

On the other hand, among Non-diabetes mellitus patients were 16 patients with IR and 24 patients without IR, HP infection (confirmed by histopathology and stool antigen) was notably greater in IR patients compared to non-IR individuals. (100% versus 75%,p-value = 0.030). Table (4)

Furthermore, we discovered that there was no statistically significant distinction between individuals with positive and negative HP stool antigen in terms of age, sex, hypertension, diabetes, low density lipoprotein, triglycerides, and body mass index. Patients with HP positive had a substantially higher (HOMA-IR) than patients with HP negative (2.65 versus 2.1, p-value = 0.010). Table (6)

Table 1. Comparison between T2DM and non-diabetic group regarding baseline clinical characteristics

		NON-DIABETIC GROUP No. = 40	T2DM GROUP No. = 40	TEST VALUE	P-VALUE	SIG.
AGE (YEARS)	Mean ± SD	22.70 ± 8.30	47.20 ± 10.79	-11.383•	0.000	HS
	Range	15 – 53	30 – 70			
SEX	Female	32 (80.0%)	28 (70.0%)	1.067*	0.302	NS
	Male	8 (20.0%)	12 (30.0%)			
HYPERTENSION	No	40 (100.0%)	18 (45.0%)	30.345*	0.000	HS
	Yes	0 (0.0%)	22 (55.0%)			
MAIN PRESENTING SYMPTOM	Epigastric pain	20 (50.0%)	20 (50.0%)	0.000*	1.000	NS
	Abdominal pain	8 (20.0%)	12 (30.0%)	1.067*	0.302	NS
	Vomiting	8 (20.0%)	6 (15.0%)	0.346*	0.556	NS
	Weight loss	2 (5.0%)	2 (5.0%)	0.000*	1.000	NS
	Abdominal dyspepsia GERD symptoms	0 (0.0%)	2 (5.0%)	2.051*	0.152	NS
	Chronic diarrhoea (Cohn's disease) + extra ocular manifestation	2 (5.0%)	0 (0.0%)	2.051*	0.152	NS
	Dysphagia	2 (5.0%)	0 (0.0%)	2.051*	0.152	NS
	Melena	0 (0.0%)	2 (5.0%)	2.051*	0.152	NS
SYSTOLIC BLOOD PRESSURE	Mean ± SD	115.00 ± 5.99	132.50 ± 14.98	-6.861•	0.000	HS
	Range	110 – 130	100 – 170			
DIASTOLIC BLOOD PRESSURE	Mean ± SD	74.65 ± 5.48	82.70 ± 4.94	-6.896•	0.000	HS
	Range	65 – 83	70 – 92			
BMI	Mean ± SD	21.90 ± 1.06	26.30 ± 2.94	-8.915•	0.000	HS
	Range	20 – 24	22 – 34			
OBESITY	Normal	40 (100.0%)	16 (40.0%)	34.286*	0.000	HS
	Overweight	0 (0.0%)	20 (50.0%)			
	Obese	0 (0.0%)	4 (10.0%)			

.(BMI = Body mass index)

Table 2. Comparison between T2DM and non-diabetic group regarding laboratory data

		NON-DIABETIC GROUP No. = 40	T2DM GROUP No. = 40	TEST VALUE	P-VALUE	SIG.
HAEMOGLOBIN	Mean ± SD	11.89 ± 1.57	12.45 ± 1.81	-1.480•	0.143	NS
	Range	9.2 – 14.4	8 – 15.3			
PLATELETS	Mean ± SD	268.88 ± 94.67	250.11 ± 71.97	0.953•	0.344	NS
	Range	170 – 547	125 – 377			
WBCS	Mean ± SD	6.06 ± 1.48	6.91 ± 2.12	-1.734•	0.088	NS
	Range	3.9 – 8.2	3.9 – 11.1			
MCV	Median (IQR)	81 (75.5 – 91.2)	39.9 (6 – 74.05)	-2.363≠	0.018	S
	Range	14.3 – 93.7	6 – 74.3			
MCHC	Mean ± SD	29.40 ± 5.23	32.05 ± 1.33	-0.979•	0.347	NS
	Range	21.5 – 34.4	30.9 – 33.2			
ALT	Median (IQR)	15 (11 – 26)	20 (15.5 – 27)	-1.323≠	0.186	NS
	Range	9 – 39	7 – 45			
AST	Median (IQR)	22 (18.5 – 30.5)	29.65 (16.5 – 32.5)	-0.964≠	0.335	NS
	Range	12 – 53	10 – 48			

ALP	Median (IQR)	75 (45.5 – 118.5)	90 (60 – 105)	-0.270≠	0.787	NS
	Range	24 – 195	24 – 278			
SERUM ALBUMIN	Mean ± SD	4.24 ± 0.40	4.26 ± 0.24	-0.217•	0.829	NS
	Range	3.55 – 5.2	3.8 – 4.6			
BLOOD UREA	Median (IQR)	19.5 (14.5 – 22.1)	19.5 (18 – 27.5)	-1.563≠	0.118	NS
	Range	7.3 – 32	12 – 41			
CREATININE	Mean ± SD	0.79 ± 0.12	0.92 ± 0.19	-3.730•	0.000	HS
	Range	0.5 – 1	0.69 – 1.4			
TRIGLYCERIDES	Mean ± SD	116.55 ± 28.25	155.45 ± 38.36	-5.165•	0.000	HS
	Range	80 – 170	83 – 220			
CHOLESTEROL	Mean ± SD	130.65 ± 31.71	178.20 ± 33.90	-6.478•	0.000	HS
	Range	90 – 188	106 – 235			
LDL	Mean ± SD	124.85 ± 34.65	157.45 ± 37.19	-4.057•	0.000	HS
	Range	75 – 194	70 – 210			
HDL	Mean ± SD	38.95 ± 4.14	39.78 ± 5.82	-0.730•	0.468	NS
	Range	29 – 47	32 – 54.5			
FBS	Median (IQR)	95 (90 – 102.5)	165 (144.5 – 180)	-7.711≠	0.000	HS
	Range	70 – 125	130 – 190			
PPBS	Median (IQR)	116 (110 – 131.5)	230 (210 – 254.5)	-7.706≠	0.000	HS
	Range	85 – 154	195 – 302			
HBAIC	Mean ± SD	5.39 ± 0.27	8.81 ± 0.88	-23.599•	0.000	HS
	Range	4.8 – 5.9	7.9 – 11.1			
HOMA-IR	Median (IQR)	2.2 (2.05 – 2.8)	2.85 (2.45 – 3.85)	-3.778≠	0.000	HS
	Range	0.9 – 3.6	0.68 – 5.6			
INSULIN RESISTANCE	No IR	24 (60.0%)	10 (25.0%)	10.026*	0.002	HS
	IR	16 (40.0%)	30 (75.0%)			
H-PYLORI STOOL ANTIGEN	Negative	6 (15.0%)	6 (15.0%)	0.000*	1.000	NS
	Positive	34 (85.0%)	34 (85.0%)			
HISTOPATHOLOGY REPORT	Negative	6 (15.0%)	6 (15.0%)	0.000*	1.000	NS
	Positive	34 (85.0%)	34 (85.0%)			

.(WBCs= White blood cells) .(MCV= Mean corpuscular volume) .(MCHC= Mean corpuscular hemoglobin concentration) .(ALT= Alanine transaminase) .(AST= Aspartate transaminase) .(ALP= Alkaline phosphatase) .(LDL= Low density lipoprotein) .(HDL= High density lipoprotein) .(FBS= Fasting blood sugar) .(PPBS= Postprandial blood sugar) .(HbA1C= Haemoglobin A1c) .(HOMA-IR= Homeostatic Model Assessment for Insulin Resistance)

Table 3. Relation of presence of insulin resistance among T2DM group

		T2DM GROUP		TEST VALUE	P-VALUE	SIG.
		No IR	IR			
		No. = 10	No. = 30			
H-PYLORI STOOL ANTIGEN	Negative	4 (40.0%)	2 (6.7%)	6.536*	0.011	S
	Positive	6 (60.0%)	28 (93.3%)			
HISTOPATHOLOGY REPORT	Negative	4 (40.0%)	2 (6.7%)	6.536*	0.011	S
	Positive	6 (60.0%)	28 (93.3%)			

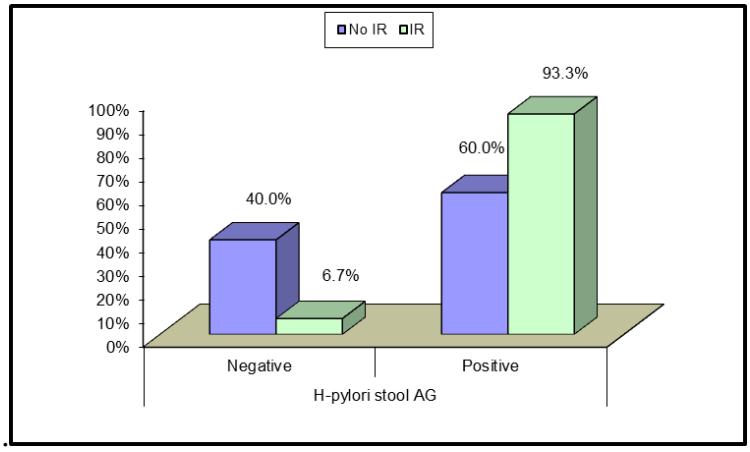

Figure 1. Relation of presence of insulin resistance with H-pylori stool AG among T2DM group

Table 4. Relation of presence of insulin resistance among non-diabetic group

		NON-DIABETIC GROUP		TEST VALUE	P-VALUE	SIG.
		No IR	IR			
		No. = 24	No. = 16			
H-PYLORI STOOL ANTIGEN	Negative	6 (25.0%)	0 (0.0%)	4.706*	0.030	S
	Positive	18 (75.0%)	16 (100.0%)			
HISTOPATHOLOGY REPORT	Negative	6 (25.0%)	0 (0.0%)	4.706*	0.030	S
	Positive	18 (75.0%)	16 (100.0%)			

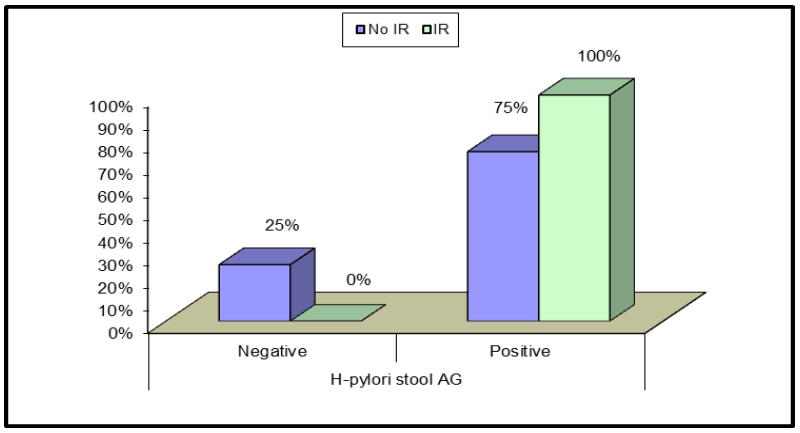

Figure 2. Relation of presence of IR with H.pylori stool AG among non-diabetic group

Table 5. comparison between patients with and without Insulin resistance regarding HP infection

		NO IR	IR	TEST VALUE	P-VALUE	SIG.
		No. = 34	No. = 46			
H-PYLORI STOOL ANTIGEN	Negative	10 (29.4%)	2 (4.3%)	9.632*	0.002	HS
	Positive	24 (70.6%)	44 (95.7%)			
HISTOPATHOLOGY REPORT	Negative	10 (29.4%)	2 (4.3%)	9.632*	0.002	HS
	Positive	24 (70.6%)	44 (95.7%)			

Table 6. Relation between H-pylori infection with multiple variables

		H-PYLORI STOOL ANTIGEN		TEST VALUE	P-VALUE	SIG.
		Positive	Negative			
		No. = 68	No. = 12			
AGE (YEARS)	Mean ± SD	35.06 ± 16.21	34.33 ± 12.13	-0.148•	0.883	NS
	Range	15 – 70	16 – 53			
SEX	Female	52 (76.5%)	8 (66.7%)	0.523*	0.470	NS
	Male	16 (23.5%)	4 (33.3%)			
HYPERTENSION	No	48 (70.6%)	10 (83.3%)	0.831*	0.362	NS
	Yes	20 (29.4%)	2 (16.7%)			
DM	Non-diabetic group	34 (50.0%)	6 (50.0%)	0.000*	1.000	NS
	T2DM group	34 (50.0%)	6 (50.0%)			
BMI	Mean ± SD	24.32 ± 3.25	22.83 ± 1.8	-1.540•	0.128	NS
	Range	20 – 34	21 – 27			
HDL	Mean ± SD	39.63 ± 5.13	37.83 ± 4.37	-1.143•	0.257	NS
	Range	29 – 54.5	29 – 43			
TRIGLYCERIDES	Mean ± SD	138.4 ± 40.68	122.42 ± 21.92	-1.323•	0.190	NS
	Range	80 – 220	83 – 155			
HOMA-IR	Median (IQR)	2.65(2.2 – 3.2)	2.1(2 – 2.4)	-2.578≠	0.010	S
	Range	0.68 – 5.6	0.68 – 3			

.(DM= Diabetes mellitus) .(BMI = Body mass index) .(HDL= High density lipoprotein)
.(HOMA-IR= Homeostatic Model Assessment for Insulin Resistance

4. Discussion

The study explains how HP infection and IR relate to Egyptian populations with or without diabetes.

Our research showed no discernible difference in patients' age, sex, hypertension, diabetes mellitus, LDL, triglycerides, and BMI between those with positive and negative HP. Our findings corroborated those of study[6], which found no evidence of a consistent relationship between HP infection and the incidence of diabetes or other IR syndrome characteristics in American males aged 40–74. Additionally, research number four supported it.

However, we discovered that individuals with positive HP infection had significantly higher (HOMA-IR) than those with negative HP infection, supporting the theory that HP infection and insulin resistance are related. Our findings were consistent with seven studies involving 27 negative HP patients and 63 positive HP patients. There were no differences in age, gender, or BMI between the two groups. In the HP negative group, the HOMA-IR level was 1.73±1.1; in the HP positive group, it was 2.56±1.54, indicating a substantial difference.

This study's key conclusion was that, whether or not T2DM was present, there was a substantial relationship between HP infection and IR.

These results were consistent with study[7], which demonstrated a substantial correlation between HP infection and IR in patients with type 2 diabetes. According to another study, the percentage of diabetic patients with a positive antibody titer for HP infection (IgA > 250) was 63.3%, while the percentage of non-diabetics with the same titer was 48.1%. Similarly, the rate of diabetic patients with a positive antibody titer for HP infection (IgG > 300) was 76.7%, whereas the percentage of non-diabetics with the same titer was 64.8%. [8]

The underlying mechanisms linking HP infection and IR were not fully understood.

Chronic inflammation brought on by an HP infection can influence the release of hormones from the stomach, including somatostatin, gastrin, leptin, and ghrelin, which can cause IR.[9;10]

Moreover, HP infection of the gut microbiota may enhance the synthesis of lipopolysaccharide, a component of bacterial cell walls that triggers innate inflammatory responses.[11]

However, in a Korean population, a study of [12] found no evidence of a significant correlation between HP infection and IR.

Our study's limit was the low number of patients and its conduct in a single canter. Further studies are needed for HP infection and IR.

5. *Conclusion*

This study shows that IR and HP infection are related to diabetes and non-diabetic groups.

Disclosure

The authors have no financial interest to declare in relation to the content of this article.

Authorship

All authors have a substantial contribution to the article

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Conflicts of interest

There are no conflicts of interest.

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