Helicobacter pylori infection as a risk factor for insulin resistance in diabetic patients

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Abstract: **Background:** T2DM is a metabolic disease that is linked to different pathophysiological mechanisms; the role of inflammatory mechanisms in the pathogenesis of this disease is highlighted in recent studies. It is believed that inflammation may increase IR. H. pylori gastritis and its role in mucosal activation of innate immunity and upregulation, IL-1B is also suggested in the pathogenesis of IR. **Objective:** to evaluate the association between H. pylori infection and HOMA-IR in patients with T2DM, who receive appropriate medical treatment other than insulin. **Patients and Methods**: two groups, the first 43 T2DM patients with H. pylori +ve while the second 17 T2DM patients with H. pylori –ve. All participated patients were subjected for full history taking and complete physical examination with special emphasis on age, duration of DM, type of treatment (oral or insulin), smoking habits, history of epigastric pain or H. pylori treatment, Height, weight, BMI, and BP. Laboratory investigations: CBC, ESR, FPG, 2 hours post prandial plasma glucose, ALT, creatinine, total cholesterol(TC), triglycerides (TG), LDL-C and HDL-C, HbA1c, fasting insulin, HOMA-IR were calculated and upper GIT endoscopy and CLO test for detection of H. pylori infection. **Results:** showed that significant increase in fasting insulin, HOMA-IR and HbA1c in +ve CLO group when compared to -ve CLO group while significant decrease in HDL in +ve CLO group when compared to -ve CLO group. Also the study showed that significant positive correlation between fasting insulin and HOMA-IR while significant negative correlation between HDL and HOMA-IR in studied groups. **Conclusion:** H. pylori infection in patients with T2DM might be a risk factor for increased insulin resistance and HbA1c, but decrease HDL.

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Key Words: Diabetes mellitus Helicobacter pylori, insulin resistance, gastritis.

Abbreviations: **T2DM:** Type 2 diabetes mellitus, **IR:** Insulin resistance, **IL:** interleukin, **HOMA-IR:** Homeostatic model assessment- Insulin resistance, **+ve:** Positive, **-ve:** Negative, **CBC:** Complete blood picture, **ESR:** Erythrocyte sedimentation rate, **FPG:** Fasting plasma glucose, **ALT:** Alanine transferase, **LDL-C:** Low density lipoprotein cholesterol, **HDL-C:** High density lipoprotein cholesterol, **HbA1c:** Glycated hemoglobin, **CLO:** Campylobacter like organism.

1. Introduction

Type 2 diabetes mellitus (T2DM) is a metabolic disease that is linked to different pathophysiological mechanisms; the role of inflammatory mechanisms in the pathogenesis of this disease is highlighted in recent studies ***(Akash et al., 2013).*** It is believed that inflammation may increase insulin resistance (IR). The IR is a pathologic state in which normal insulin concentration produce subnormal response in the peripheral tissues ***(Akash et al., 2013). Joen et al. (2012),*** proposed the possible role of altered gut microbiota in the pathogenesis of IR and T2DM. *Also*, concentrations of circulating lipopolysacharide [LPS] (apart of bacterial cell wall = bacterial endotoxin) have been reported to be higher in patients with T2DM than non-diabetic individuals and correlate with IR severity ***(Lassenius et al., 2011).*** *Also*, the sero-positivity for Helicobacter pylori *(H. pylori)* was associated with a higher rate of incidence of DM ***(Joen et al., 2012).*** *Also* a recent meta-analysis study showed that *H. pylori* infection is more frequent in diabetic patients ***(Zhou et al., 2013).*** *H. pylori* gastritis and its role in mucosal activation of innate immunity and upregulation, interleukin-1B (IL-1B) is also suggested in the pathogenesis of IR ***(Basak et al., 2005).*** It seems that *H. pylori* eradication treatment may be helpful in lowering the IR in T2DM patients and reduce homeostatic model assessment -insulin resistance (HOMA-IR) ***(Gen et al., 2010).***

The association of *H. pylori* and IR has not yet been evaluated in diabetic patients and need more clarification.

The aim of our work was to evaluate the association between *H. pylori* infection and HOMA-IR in patients with T2DM, who receive appropriate medical treatment other than insulin.

2. Patients and Methods

The present study was carried out on 60 patients T2DM on oral treatment (43females and 17 males), their ages ranged between 37 and 67 years old (their mean age = 51.43 ± 6.99). Patients were selected from the outpatient clinic and admitted to internal medicine department of Sayed Galal Hospital, Al-Azhar University in the period between Novamber-2015 to March-2016. The study was approved by hospital ethics committee and written consents were obtained from all patients after explaining the nature and the aim of the study. Patients treated with insulin, Smoker's patients, Patients with history of coronary heart diseases, Patients known to have hypertension, Patients have urinary tract infection or any other infectious diseases or septic focus were excluded from this study. All participated patients were subjected to the followings:

1- Full history taking with special emphasis on age, duration of DM, type of treatment (oral or insulin), smoking habits, history of epigastric pain or H. pylori treatment.

2. Clinical examination: complete physical examination with special emphasis on general examination including blood pressure (BP) and body mass index (BMI) calculation.

3. Laboratory investigations including: CBC, fasting plasma glucose (FPG), 2 hours post prandial plasma glucose (2h-PPPG), ALT, creatinine, total cholesterol (TC), triglycerides (TG), LDL-C and HDL-C, HbA1c, erythrocyte sedimentation rate (ESR) and fasting insulin.

4. Insulin resistance by using HOMA index: Homeostatic model assessment has 2 types ***(Matthews et al., 1985)***:

HOMA-1: Which can calculate IR through the following Matthews formula: HOMA1IR = (FPG in mg /dl × fasting insulin inmu/L)/405.

HOMA-2: Which also can calculate IR through calculator, just enter the values of FPG and fasting insulin and through computerized system will give the results according to the choice?

5. Campylobacter like organism (CLO) test for detection of *H. pylori* infection:

Principle of the test:

The Biohit quick test for detecting H. pylori infection in the stomach is based on the activity of the urease enzyme in a biopsy specimen**.** The biopsy specimen taken from the stomach is examined immediately**.** The development of the color in the test gel after 30 minutes indicates whether or not urease enzyme is present in the biopsy specimen. H. pylori produce a large amount of urease**,** which degrades urea to ammonia (NH4+). The ammonia formed is detected by an indicator color present in the gel**.**

Specimen requirements:

Each test plate requires one biopsy specimen**,** which is recommended to be taken with 5 mm forceps either from the greater curvature of the middle antrum or the corpus. In order to assure high sensitivity, it is recommended that the H. pylori quick test is done by using biopsies from antrum and corpus (H. pylori colonization in stomach may sometimes be limited to either antrum or corpus).

On performing the gastroscopy**,** the biopsies for the H. pylori quick test should be taken as early as possible to avoid possible errors caused by neutral and alkaline duodenogastric reflux**.** Before performing the test**,** blood can be removed from the biopsy specimen by placing it briefly on a sterile gauze pad.

Equipment:

H. pylori quick test plate 602015 (50 tests per box).

Biohit positive control 602017.

Method/Procedure:

1) We checked that the box of kits had been of quality control.

2) We allowed the test plate to reach room temperature for at least 30 minutes before use.

3) We opened the label covering the well on the plate and check that the color of the reagent gel was yellow.

4) We remove blood by briefly placing the biopsy on a piece of gauze then we transferred the biopsy specimen from the forceps into the gel of the well, then we push the biopsy into the gel to ensure it is submersed.

5) We labeled the test plate to enable unequivocal identification.

6) We incubated the test plate for 2 minutes at room temperature (20 - 25 °C) then we checked the color of the gel, if the color of the gel turns from yellow-to-red the test is positive if there is no change in color we continued incubation for a full 30 minutes.

7) After 30 minutes we checked the color of the gel again, if it has not turned red the test is negative.

8) We record the results in the patient notes and the record book including who performed the test and the batch number and expiry of the test kit.

Sensitivity/specificity:

The technique is reported as having a sensitivity of 94% and a specificity of 88%. The predictive values of a positive and negative test were 89% and 93%, respectively.

6. Upper GIT endoscopy.

Statistical analysis:

Data were fed to the computer and analyzed using IBM SPSS (Statistical Package for the Social Science) software package version 20 as follows: qualitative data were described using number and

percent, quantitative data were described using range (minimum and maximum), mean ± standard deviation (SD) and median. Comparison between different groups regarding categorical variables was tested using Chi-square test. The distributions of quantitative variables were tested for normality using Kolmogorov-Smirnov test, Shapiro-Wilk test and D'Agstino test, also Histogram and QQ plot were used for vision test. If it reveals normal data distribution, parametric tests were applied. If the data were abnormally distributed, non-parametric tests were used:

For normally distributed data, comparison between 2 independent populations were done using independent t-test *while* > 2 populations were analyzed using F-test (ANOVA) and Post Hoc test (Scheffe), Correlations between 2 quantitative variables were assessed using Pearson coefficient (r).

For abnormally distributed data, comparison between 2 independent populations were done using Mann Whitney test while Kruskal Wallis test was used to compare between different groups. Significance test results are quoted as 2-tailed probabilities. Significance of the obtained results was judged at the 5% level.

All results were considered insignificant: if P-value > 0.05, significant: if P- value ≤ 0.05, and highly significant: if P -value ≤ 0.001.

3. Results

Sixty patients with T2DM were enrolled in this study (43 females and 17 males), their ages ranged between 37 and 67 years old, with mean age 51.43 ± 6.99 years old. Patients were divided into 2 groups according to results of CLO test as follow:

Group I: 43 patients with T2DM and positive (+ve) CLO test for *H. pylori* infection.

Group II: 17 patients with T2DM and negative (-ve) CLO test for *H. pylori* infection (Table 1).

In the present study there was significant increase in fasting insulin, HOMA-IR and HbA1c in +ve CLO group when compared to -ve CLO group (P < 0.05), while there was insignificant difference in 2h-PPPG and FPG in +ve CLO group when compared to -ve CLO group (P > 0.05) (Table 2).

There was significant decrease in HDL in +ve CLO group when compared to -ve CLO group (P < 0.05), while there was insignificant difference in TC, TG and LDL levels in +ve CLO group when compared to -ve CLO group (P > 0.05) (Table 3).

Table (1): Percentage of +ve and -ve H. pylori in the studied T2DM patients:

H. pylori test	Number of patients	%
Negative	17	28.3
Positive	43	71.7
Total	60	100

Table (2): Comparison of HbA1c, 2h-PPPG, FPG, fasting insulin, HOMA-IR in patients:

		-ve H. pylori	+ve H. pylori	Independent t-test	
		No = 17	No = 43	t	P-value
HbA1c (%)	Mean±SD	6.88 ± 0.41	7.12 ± 0.45	1.931	<0.05*
	Range	6.3 - 7.5	6.2 - 8		
2h-PPPG(mg/dl)	Mean±SD	206.29 ± 19.76	202.16 ± 22.10	0.671	0.505
	Range	177 – 246	167 - 271		
FPG(mg/dl)	Mean±SD	128.79 ± 12.24	135.53±17.5	1.500	0.139
	Range	98 – 151	111 - 174		
F. insulin(mIU/ml)	Mean±SD	12.06±3.67	26.84±10.52	5.635	<0.05*
	Range	5.85 - 16.24	16.14 - 65.66		
HOMA-IR (mIU/ml)	Mean±SD	3.97±1.09	8.73±4.15	5.194	<0.05*
	Range	1.82 - 5.36	4 - 24.48		

Table (3): Comparison of lipid profile in the studied patients:

		-ve H. pylori	+ve H. pylori	Independent t-test	
		No = 17	No = 43	t	P-value
TC(mg/dl)	Mean±SD	240.64 ± 42.78	261.44 ± 41.17	1.744	0.087
	Range	168 - 307	159 - 336		
HDL(mg/dl)	Mean±SD	50.76 ± 7.89	43.30 ± 6.34	3.830	<0.05*
	Range	37 - 61	33 - 59		
TG (mg/dl)	Mean±SD	207.59 ± 40.00	193.35 ± 49.83	1.050	0.298
	Range	127 - 276	112 - 288		
LDL (mg/dl)	Mean±SD	152.76 ± 29.40	158.40 ± 34.99	-0.586	0.560
	Range	96 - 195	90 - 203		

Table (4): Correlations between HOMA-IR and parameters of the studied groups:

Parameters of the studied groups	HOMA-IR (mIU/ml)	
	r	P-value
Age (years)	-0.091	0.490
Duration of DM (years)	0.097	0.462
BMI (kg/m^2)	-0.069	0.603
HbA1c (%)	0.216	0.098
2h-PPPG(mg/dl)	-0.066	0.618
FPG (mg/dl)	0.099	0.452
Fasting serum insulin(mIU/ml)	0.958	<0.05
TC (mg/dl)	-0.079	0.550
HDL(mg/dl)	-0.309	<0.05
TG (mg/dl)	-0.169	0.198
LDL(mg/dl)	0.046	0.726
Hb (g/dl)	-0.038	0.774
WBC (/mm^3)	0.091	0.487
PLT (/mm^3)	0.175	0.185
ESR (mm/h)	-0.177	0.177
Creatinine (mg/dl)	-0.103	0.433
ALT (U/l)	-0.141	0.283

There was significant positive correlation between fasting insulin and HOMA-IR (i.e., the greater the degree of HOMA-IR increase, the greater the degree of fasting insulin increase), while there was significant negative correlation between HDL and HOMA-IR (i.e., the greater the degree of HDL decrease, the greater the degree of HOMA-IR increase) in studied groups. There was insignificant correlation between other parameters and HOMA-IR in studied groups (Table 4).

4. Discussion

Life style factors such as obesity, physical inactivity and genetic predisposition are important risk factors for development of T2DM. In addition, chronic infections are well known another risk factor for development of the manifestations of T2DM.

H. pylori infection is present in more than half of the world's population. However, not all infected people exhibit diseases associated with this bacterium. It is the main cause of gastric disorders, such as gastritis in about 20%, peptic ulceration in 10%, gastric adenocarcinoma in 1% - 2%, and gastric mucosa associated lymphoid tissue (MALT) lymphoma in < 0.1% of the people infected ***(Oluwasola, 2014).***

Extra-gastric manifestations include: coronary artery disease (CAD), sideroblastic anemia, Idiopathic thrombocytopenic purpura (ITP), some neurological diseases such as Alzheimer and Parkinson's diseases and biliarysystem diseases ***(Vafaeimanesh et al., 2014).***

A link between H. pylori infection, IR and T2DM had been demonstrated. Many studies have reported a higher prevalence of H. pylori infection ***(Marietti et al., 2013),*** a lower eradication rate and more frequent reinfection prevalence in diabetic patients versus controls. Moreover, H. pylori infection is considered to be associated with metabolic control in diabetics ***(Candelli et al., 2012).***

T2DM is a metabolic disease that is linked to different pathophysiological mechanisms. The role of inflammatory mechanisms in the pathogenesis of this disease is highlighted in recent studies ***(Akash et al., 2013).***

It is stated that *H. pylori* infection gastritis causes susceptibility to DM by affecting gastric hormones such as leptin, ghrelin, gastrin and somatostatin ***(He et al., (2014).***

In a study done by ***Yang et al. (2014),*** they showed that, H. pylori infection was positively associated with DM, but no positive correlation was found between H. pylori infection and pre-diabetes.

The mechanisms linking H. pylori and glycemic control in T2DM are complicated. It is well known that IR is a central pathogenic factor in T2DM. H. pylori play a role in the pathophysiology of IR syndrome by pathologic consequences through chronic inflammation outside the stomach**,** by which the bacterium affects glycemic control in diabetic patients ***(Ram et al., 2013).***

The finding that showed increased IL-1B which causes inflammation in adipose tissue and leads to IR (which plays a key role in developing DM), in addition to the fact that secretion of IL-1B increases in H. pylori +ve patients, helped the researchers in addressing this relationships ***(Zhao et al., (2013).***

However, there are many controversies as regards the relationship between H. pylori infection and DM and its role in developing IR. Also, there are many conflicting points, especially with regard to its role in the development of T2DM.

In order to clarify these conflicting points, it was planned to assess the relation between infection with H. pylori bacterium which confirmed by campylobacter like organism (CLO) test and T2DM patients who received appropriate treatment other than insulin and effect of this infection on HOMA-IR.

In the current study the obtained results showed higher prevalence of H. pylori infection in T2DM patients [71.7% (43/60)], compared to that of those without infection [28.3% (17/60)].

This result was in agreement with that obtained by ***Marrollo et al. (2013),*** who observed that prevalence of H. pylori infection in T2DM patients

was 74.4%, compared to that of those without infection (25.6%). *Similarly*, a study made by ***Małlecki et al. (2006***), reported that prevalence of H. pylori infection was 68%, compared to that of those without infection (32%). Moreover, a study made by ***Vafaeimanesh et al. (2014)*****,** concluded that prevalence of H. pylori infection in T2DM patients was 65.9%, compared to that of those without infection (34.1%). In this context, ***Demir et al. (2008)*****,** noticed that prevalence of H. pylori infection inT2DM patients was 61.7% compared to that of those without infection (38.3%).

Several hypotheses were reported that there was a high prevalence of H. pylori infection among T2DM patients; this might be due to impairment of immune system**,** reduction of both gastrointestinal motility and more acid secretion and higher secretion of pro-inflammatory cytokines ***(Bener et al., 2007).*** The little variability between the results might be attributed to the differences in epidemiological distribution**,** sample size and methods used in the detection of H. pylori infection.

As regards HbA1c, the current work showed that there was significant difference between HbA1c in H.pylori +ve group (7.12 ± 0.45 %), compared to that of H. pylori -ve group (6.88 ±0.41%).

This result was in agreement with the study of ***Fernandini et al. (2008)*****,** who reported significant difference in HbA1c between H. pylori +ve group and H. pylori -ve group. Also, another study of ***Bener et al. (2007)*****,** revealed significant difference in HbA1c between the studied groups, in addition, the study of International journal of medical science reported**,** significant difference between the studied groups in HbA1c. In contrast to our study**,** the study done by ***Vafaeimanesh et al. (2014),*** who reported that there was insignificant difference in the long-term or the short-term glycemic control of patients in the studied groups**,** as there were insignificant differences in both HbA1c and FPG. In this context, the study of ***Jamshid et al. (2014)*****,** also reported insignificant differences in HbA1c and FPG.

As regard FPG, our study showed that there was insignificant difference between H. pylori +ve and H. pylori -ve groups as regards FPG (135.53 ± 17.5 mg/dl and 128.79 ± 12.24 mg/dl, respectively).

This result was similar to that detected by ***Vafaeimanesh et al., (2014),*** as they showed insignificant difference in the long-term or the short-term glycemic control of patients in the studied groups regarding FPG. *Moreover*, the study of ***Jamshid et al., (2014)*****,** reported that there was insignificant difference in the FPG between the studied groups. *However*, ***Fernandini et al. (2008) and Bener et al. (2007)*****,** reported significant difference between the 2 groups with regard to FPG.

As regard fasting serum insulin, H. pylori +ve group had higher fasting insulin level (19.96 ± 13.03 mIU/ml) than H. pylori -ve group (15.60 ± 11.92 mIU/ml) this difference was statistically significant ($P < 0.05$).

This finding was similar to that of ***Vafaeimanesh et al. (2014)*****,** where they reported that fasting insulin was significantly higher in H. pylori +ve group (10.12 ± 7.72 mIU/ml) than H. pylori -ve group (6.97 ± 5.64 mIU/ml) with $P < 0.05$.

Similarly, the study of ***Christie et al. (2012)*****,** revealed significant difference in fasting insulin between the studied groups. But the study of ***Jamshid et al. (2014)*****,** reported that there was insignificant difference in fasting serum insulin between H. pylori +ve and H. pylori -ve groups.

IR is a condition in which greater amount of insulin is required to elicit a quantitatively normal response. IR is postulated to cause disorders of metabolic components involved in metabolic syndrome ***(Weiss et al., 2014).*** IR with a consequent hyperinsulinemia has a central role in pathogenesis of many diseases such as DM, atherosclerosis, hypertension and dyslipidemia ***(Ferrannini, 2008).***

HOMA-IR is the most common method for assessment of IR in clinical practice and epidemiological studies; because it is minimally invasive and needs only fasting blood samples ***(Wallace et al., 2008).***

As regards HOMA-IR, the obtained results showed that H. pylori +ve group had higher HOMA-IR (8.73 ± 4.15 mIU/ml) than H. pylori -ve group (3.97 ±1.09 mIU/ml), this difference was statistically significant ($P < 0.05$).

This is similar to the findings of ***Vafaeimanesh et al. (2014)*****,** that evaluated the association between H. pylori infection and HOMA-IR in 211 patients with T2DM. The main finding of this study was that HOMA-IR is significantly higher in H. pylori +ve group (4.484 ± 3.78 mIU/ml) more than the -ve group (3.160 ± 3.32 mIU/ml) with $P = 0.013$.

Similarly, the study of ***Franceschi et al. (2014)*****,** reported that significant association between H. pylori positivity and HOMA-IR depending on the role in pathophysiology**.**

Another study of ***Polyzos et al. (2009)*****,** suggested a trend toward a +ve association between H. pylori positivity and HOMA-IR was highlighted. Also, the study done by ***Christie et al. (2012)*****,** revealed that there was a significant difference in HOMA-IR between the 2 studied groups.

Also, the study of ***Gen et al. (2010),*** suggested that eradication of H. pylori infection showed an improvement in IR. The study of ***Jamshid et al. (2014),*** reported that HOMA-IR was significantly higher among H. pylori +ve diabetic patients

compared to those with H. pylori -ve findings. *Moreover,* the study made by ***Gunji et al. (2009),*** confirmed this significant difference (39.4% *vs* 28.7%). *In addition*, the study done by ***Aydemir et al. (2005),*** used HOMA-IR to assess IR they reported that there was a significant difference between H. pylori +ve diabetic patients and H. pylori -ve diabetic patients.

The association between the positivity of H. pylori infection and IR could be due to H. pylori infection itself, that might stimulate the release of counter regulatory hormones which induce hyperinsulinemia by decreasing serum concentrations of somatostatin that has inhibitory effect on insulin secretion ***(Aydemir et al., 2005)***.

H. pylori infection might induce dyslipidemia, as it leads to increase plasma levels of TC, LDL and TG concentrations but it decreases that of HDL ***(Chimienti et al, 2003)***. It was postulated that chronic H. pylori infection may promote atherogenic lipid profiles through the action of pro-inflammatory cytokines, such as IL-6, interferon-alpha (IFN-α) and tumor necrosis factor-alpha (TNF-α), which activate adipose tissue lipoprotein lipase, stimulate hepatic fatty acid synthesis and influence lipolysis ***(Chimienti et al, 2003)***.

High concentrations of plasma TG and LDL, along with low concentration of HDL, are attributed mostly to IR and insulin deficiency ***(Chahil and Ginsberg, 2006)***.

As regards lipid profile in the current study, serum HDL showed significant difference between H. pylori +ve and H. pylori -ve groups; they were 43.30 ± 6.34 and 50.76 ± 7.89 mg/dl, respectively ($P < 0.05$). Where HDL was lower in H. pylori +ve group, but TC, TG and LDL showed insignificant difference in the 2groups, however, despite TC showed insignificant difference, but it was higher in H. pylori +ve group compared to H. pylori -ve group; they were 261.44 ± 41.17 and 240.64 ± 42.78 mg/dl, respectively.

These results were similar to those reported in the study done by ***Hoffmeister et al. (2014),*** that reported significant difference in HDL between the 2 studied groups, HDL was lower in H. pylori positive group in comparison to the other group. *However*, the study showed difference in the TG level which was significant statistically but LDL and TC in both groups showed insignificant difference.

Similarly, the study of ***Jamshid et al. (2014),*** reported that only significant difference in HDL, as it was lower in H. pylori positive group. But the study of ***Kim et al. (2011),*** that conducted on 454 Koreans patients; showed that H. pylori infection was associated with increase of LDL, with a correlation to infection severity. *Also*, the study of ***Satoh et al. (2010),*** which conducted on 6289 Japanese subjects, revealed that LDL was significantly higher and HDL was significantly lower in H. pylori positive group. *Moreover,* ***Tanriverdi et al. (2011),*** also showed significant differences in LDL and HDL between the 2 groups. *But* ***Kamada et al. (2005),*** reported that H. pylori positive group showed significant difference in all lipid profile parameters include TC, TG, LDL and HDL. This due that inflammatory cytokines released in the infection alter the metabolism of lipid in different ways include hepatic synthesis of fatty acids and increase lipolysis. In contrast to ***Kamada et al. (2005),*** a meta-analyses study of 18 studies involving 10000 H. pylori positive diabetic patients, found no strong correlation between the infection and serum concentrations of lipid profile parameters ***(Isomoto et al., 2005).***

Also, the obtained results in the current study revealed, significant positive correlation between fasting insulin and HOMA-IR (i.e., the greater the degree of HOMA-IR increase, the greater the degree of fasting insulin increase), while there was significant negative correlation between HDL and HOMA-IR (i.e., the greater the degree of HDL decrease, the greater the degree of HOMA-IR increase) in studied groups. There was insignificant correlation between other parameters and HOMA-IR in studied groups.

This finding was similar to that of ***Vafaeimanesh et al. (2014), and Christie et al. (2012),*** where they reported that significant positive correlation between fasting insulin and HOMA-IR. *Also,* the study of ***Hoffmeister et al. (2014),*** reported that significant negative correlation between HDL and HOMA-IR. in the 2 studied groups *However*, the study showed insignificant correlation between LDL, TC, TG and HOMA-IR in both groups.

5. Conclusion

H. pylori infection in patients with T2DM might be a risk factor for increased insulin resistance and HbA1c, but decrease HDL.

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