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ABSTRACT

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METABOLIC MARKERS OF INSULIN RESISTANCE IN IRAQI TYPE 2 DIABETES MELLITUS PATIENTS INFECTED WITH H. PYLORI

Introduction and Aim: People with diabetes mellitus are typically more vulnerable to long-term infections. On the other hand, reports regarding the connection between type II diabetes and H. pylori infection have been inconsistent. The researchers in this study set out to determine how common Helicobacter pylori infection is among Iraqi diabetics.

Materials and Methods: Enzyme-linked immunosorbent assays (ELISA) were used to assess the levels of physiological markers in blood samples obtained from H. pylori-infected individuals in Iraq. The investigation also included patients with type 2 DM without H. pylori infection, who fulfilled the role of the controls.

Results: Compared to the control group of healthy individuals, blood samples from Iraqi patients with diabetes and H. pylori infection exhibited a statistically significant increase ($P < 0.01$). Diabetic patients who were additionally infected with H. pylori were found to have a higher risk of insulin resistance, obesity, high levels of low-density lipoprotein (LDL), fasting blood sugar (FBS), C-reactive protein (CRP), cholesterol, triglycerides, and higher body mass index (BMI) compared to those without the infection. The study's findings indicate that there was no significant difference in the severity of insulin resistance between patients with Helicobacter pylori infection and those with diabetic mellitus (DM).

Conclusion: This study investigated the impact of H. pylori infection on metabolic parameters in Iraqi patients with type 2 diabetes. The results showed a significant association between H. pylori infection and elevated levels of HbA1c, fasting blood sugar, and BMI. Additionally, the study found a higher prevalence of H. pylori infection in diabetic patients with a history of smoking. These findings highlight the importance of considering H. pylori infection in the management of type 2 diabetes.

Keywords: H. pylori, diabetic patients, insulin resistance, triglyceride.

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ABBREVIATIONS

DM: Diabetes Mellitus
T1DM: Type 1 Diabetes Mellitus
T2DM: Type 2 Diabetes Mellitus
ELISA: Enzyme-Linked Immunosorbent Assay
TINIA: Turbidimetric Inhibition Immunoassay
HOMA-IR: Homeostatic Model Assessment for Insulin Resistance
BMI: Body Mass Index
HP+: H. pylori Positive
HP-: H. pylori Negative
LDL: Low-Density Lipoprotein
FBS: Fasting Blood Sugar
CRP: C-Reactive Protein
TG: Triglycerides
TC: Total Cholesterol
LDL-C: Low-Density Lipoprotein Cholesterol

INTRODUCTION

Diabetes mellitus (DM) is a disease characterized by sustained high blood sugar levels. Reduced insulin secretion or peripheral tissue insulin resistance may cause it. International Diabetes Federation data estimates 415 million persons aged 20–79 had diabetes mellitus in 2015 [1]. By 2040, 200 million more people would have diabetes mellitus (DM), making it a global health issue [1]. Chronic hyperglycemia and other metabolic problems in diabetes might damage multiple organs [1]. Type 1 diabetes (T1DM) occurs when the immune system mistakenly attacks and destroys pancreatic beta cells that make insulin. This autoimmune disease affects 5–10% of diabetics [2]. Type 2 diabetes mellitus (T2DM) accounts for 90% of all diabetes cases [3] and is characterized by the development of insulin resistance and impaired insulin secretion [4]. People over 45 are more likely to have type 2 diabetes (T2DM). This syndrome is most often caused by obesity, inactivity, and overeating in adolescents, teens, and young adults [5]. *H. pylori* was Campylobacter pylori's original name. This Gram-negative bacterium dislikes high oxygen concentrations. It is usually located in the stomach; however, it can also be found elsewhere. According to studies, *H. pylori* infection can be associated with diabetes, autoimmune thyroid illnesses, and primary hyperparathyroidism [6]. Simon and his colleagues investigated whether H. pylori causes type 2 diabetes [8].

Little information is available related to the *H. pylori* infections in people with diabetes. A new notion, however, proposes that *H. pylori* infections may be more common in diabetics. Whether *H. pylori* infections increase the risk of diabetes or are more common in diabetics is unclear [8]. A higher incidence of insulin resistance and diabetes may be associated with H. pylori infection [9]. Inflammation or changes in hormone levels can lead to insulin resistance. By inducing chronic inflammation and interacting with gastrointestinal hormones that regulate insulin, *H. pylori* infection has the potential to exacerbate insulin resistance [10]. According to Aydemir *et al*. [10], the initial direct link was discovered. The data on *H. pylori* prevalence in people with diabetes is scarce and inconsistent. This study sought to quantify *H. pylori* infection in diabetics.

OBJECTIVE

To study metabolic markers of insulin resistance in Iraqi T2DM patients infected with *H. pylori*

MATERIALS AND METHODS

Study groups were categorized as follows:

Group I: 60 patients with type 2 DM infected with H. pylori, aged 20–65 years.

Group II: 30 patients with type 2 DM without H. pylori infection matched in age and gender with group I, who were diagnosed in the diabetic center.

Blood Samples

All participants, including people diagnosed with diabetes, employees, and people without any health problems, were instructed to fast for 8–12 hours before blood sample collection. The specimens were obtained from each participant's venous system. To collect serum samples for immunological and hormonal investigation, 3 ml of blood samples were placed in gel tubes and left to coagulate at 37 °C for 30 minutes. Afterwards, the tubes were centrifuged at a speed of 3000 revolutions per minute (rpm) for 5 minutes. The serum should be stored in suitable containers at a temperature of -20 °C until it is analyzed.

Determination of Biomarkers

Determination of HbA1C

TTAB, which has no lytic action against leukocytes, is used to clean the hemolyzing solution. HbA1c is measured using a turbidimetric inhibition immunoassay (TINIA) on hemolyzed whole blood.

R1, a buffer and antibody, facilitates the interaction between glycohemoglobin (HbA1c) in the sample and anti-HbA1c antibody, resulting in the formation of soluble antigen-antibody complexes. Complexes are not feasible due to the fact that the HbA1c antibody site is located on a single molecule. Upon the addition of R2 (buffer/polyhapten), the polyhaptens and excess anti-

HbA1c antibodies combine to create an insoluble combination of antibodies and polyhapten molecules. It is feasible to conduct turbidimetric measurements on this complex. The haemoglobin in the hemolyzed sample undergoes a conversion process that results in the formation of a derivative with a distinct absorption spectrum. The spectrum is assessed using two different colours during the preincubation phase of the immunological reaction. Additional Hb reagent is unnecessary. The Roche/Hitachi Cobas c111 equipment has the capability to automatically determine HbA1c levels in samples.

Determination of Insulin Resistance

Homeostatic Model Assessment for Insulin Resistance = (insulin × glucose) / 22 for the glucose concentration in mmol/L, or Homeostatic Model Assessment for Insulin Resistance = (insulin ×glucose) / 405 for glycemia in mg/dL. In both cases, the insulin is in mU/L [12].

An index called the Homeostatic Model Assessment for Insulin Resistance (HOMA-IR) is used to evaluate if a patient has insulin resistance [14]. Its non-intrusiveness and simplicity make it frequently used by people.

Determination of serum glucose

Determination of body mass index (BMI) was estimated by utilizing the following equation:

Body mass index = weight / height^2 (kg/m^2)

Overweight = (25.0–29.0 kg/m^2)

Normal weight = (18.5–24.9 kg/m^2)

Obesity ≥ (30 kg/m^2) [13]

Table 1: Glucose kit working solutions

Solution	Reaction
R1	"TRIS buffer, pH = 7.8; Magnesium, 4 mmol/L; adenosine triphosphate (ATP), 1.7 mmol/L; NADP, 1.0 mmol/L; preservative"
SR	"30 mmol/L HEPES buffer, pH = 7.0; 4 mmol/L Magnesium; 130 kat/L HK (yeast); 250 kat/L G6PDH; preservative"

Test Procedure

The cases and controls in this study were tested for serum glucose. Table 2 lists kit contents. This parameter was measured using Sacks' approach (1996) [15]. These instructions were used to run the analyzer and measure glucose. Serum glucose concentration is estimated automatically using Roche/Hitachi Cobas c111 devices [14].

Determination of Helicobacter pylori IgG antibodies

The investigation identified H. pylori carriers and non-carriers. The Abcam Human Helicobacter Pylori IgG ELISA Test Kit quantified Helicobacter pylori antibodies. This work used 96-well plates coated with Helicobacter pylori antigens and conjugated with similar antibodies. Testing or control samples were incubated. After washing, HPR-labeled anti-human IgG conjugate was added. This new combination suggests that Helicobacter pylori antibodies are appealing. Adding an acidic material to TMB and HRP to inhibit the reaction turns the product blue and then yellow. The intensity of the yellow hue is directly related to Helicobacter pylori IgG content.

Table 2: Serum glucose procedure

Solution	Volume (µ)
R1	150,-
Sample	2,20
SR	30,-
Total volume	202,-

Determination of triglyceride, cholesterol, and low-density lipoproteins

Oxidised LDL was measured using the Mercodia Oxidised LDL Competitive ELISA test. The considered assay kit is from Mercodia AB, Sylveniusgatan 8A, SE-754 50 Uppsala, Sweden. Holvoet et al. showed that their monoclonal antibody 4E6 selects oxidatively damaged low-density lipoproteins [15]. In low-density lipoproteins (LDLs), the 4E6 antibody targets apolipoprotein B-100 composition changes. In competitive ELISA, oxidised LDL in the sample or standard competes with a known concentration in the well to bind to antibodies 4E6. HRP-conjugated streptavidin identifies the biotin-labeled antibody immobilised on the well after additional washing to remove unbound sample components. In a chemical reaction, 3,3',5,5'-tetramethylbenzidine (TMB) is used to detect the bound conjugate. After the stop, the solution is added.

Statistical analysis

The Statistical Analysis was performed using the System-SAS (2018) program, which detects the impact of different groups. Least significant difference (LSD) was utilized to make a significant comparison between means. The chi-square test was used to compare percentages in this study (0.05 and 0.01 probability).

Ethical approval

Ethical approval for this study was obtained from the Ethical Approval Committee of the University of Baghdad, Iraq. All of the participants were allowed to provide the researchers with the specimens. Informed consent was obtained from all participants according to the Declaration of Helsinki.

RESULTS

Demographic distribution

The ELISA test was used to diagnose instances of *H. pylori* that tested positive (Table 1). According to Table 3, DM patients with *H. pylori* infection had higher levels of IgG than the control group. This is because infection stimulates the release of IgG at concentrations more than 0.5 mg/dl.

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Table 4 shows the results of the examination of the demographic distribution of DM patients (n = 60) and control subjects (n = 30) with respect to smoking, *H. pylori* infection duration, and family history. Determination of metabolic markers in DM patients with and without *H. pylori* was shown in Table 5.

Smoking habits, family history, and the length of the infection were among the demographic information gathered for every patient. The findings suggested that, in terms of family history, there was no discernible variation amongst those with diabetes and H. pylori infection (HP+). Furthermore, the findings showed that smoking is associated with a higher risk of *H. pylori* infection in both positive (Hp+) and negative (HP-) testers [16]. In addition to decreasing the effectiveness of treating *H. pylori* infection, smoking is associated with its acquisition and persistent presence [16]. A possible explanation for the increased risk of gastrointestinal disorders among smokers is their increased infection rate [16].

Table 3: Determination of Helicobacter pylori IgG ELISA test

Groups	IgG level (ml/dl)
H. pylori IgG negative (Control) in DM patients without infection	0.021
H. pylori IgG positive in DM patients with infection	0.742

Table 4: Distribution of H. pylori patients and the controls depending on infection duration, smoking, and family history in this study (n = 100)

Groups	No.	Infection duration (years)		Smoking		Family history with H. pylori	
		(< 5) N (%)	**(> 5) N (%)**	**Positive N (%)**	**Negative N (%)**	**Positive N (%)**	**Negative N (%)**
DM patients without *H. pylori* infection (Control)	40	20 (50%)	20 (50%)	25 (62.50%)	15 (37.50%)	10 (25.00%)	30 (75.00%)
DM patients with *H. pylori* infection	60	20 (33%)	40 (66%)	42 (70.00%)	18 (30.00%)	30 (50%)	30 (50%)
Total	100	40 (38.8%)	60 (61.1%)	67 (67%)	33 (33%)	40 (40%)	60 (60%)
P-value	--	0.0084 **		0.0059 **		0.0319 *	

Note: $P \leq 0.05$; $P \leq 0.01$

Age and *H. pylori* seropositivity were found to be positively correlated in the study [17]. A statistically significant difference ($P < 0.05$) was seen in the *H. pylori* IgG antibody positivity of the patients [17]. Table 3 displays the data, which indicates that among patients with DM, there are 60 positive cases and 40 negative cases [17]. The results show a greater infection prevalence in people with diabetes mellitus (DM) who have had a prolonged *H. pylori* infection. This implies that diabetes and *H. pylori* infection over an extended period of time are related [18]. According to recent studies, the degree of bacterial colonization and the intensity of local inflammation are more important factors in this relationship than *H. pylori* itself.

Based on the existence of infection in DM patients, which makes up 60% (40) of the entire sample size, the results show that there are significant differences in the patient group. Similarly, the distribution of people in the control group was uniform. The results shown in Table 4 show that among individuals with DM, smoking is associated with a higher frequency of *H. pylori* infection. Additionally, it was discovered that DM patients with H. pylori infection had a higher prevalence of infection than those without (Table 4). Regarding a family history of diabetes mellitus with *H. pylori,* there is no discernible effect on the patients. Candelli et al. [18] found that people with diabetes had a higher rate of HP infection (24%).

According to Table 5, diabetic patients with *H. pylori* infection had significantly higher HBA1c levels than those without infection. According to Maluf et al. (2017) [17], there is a connection between prediabetes and *H. pylori* infection in current studies. According to the study, individuals with H. pylori infection had higher average HbA1c values than those without infection [17]. However, higher HbA1c levels were associated with infection density, inflammation severity, and chronic gastritis activity rather than *H. pylori*-related characteristics [17]. The average *H. pylori* growth rate values and the rise in HbA1c readings were shown to positively correlate by Tawfek et al. [19].

Table 5: Determination of metabolic markers in DM patients with and without *H. pylori*

Biomarkers	Serum levels (mean ± SD) in		T-test (P-value)	Normal value
	DM patients with *H. pylori*	**DM patients without *H. pylori* (Control)**		
HBA1c level%	8.3 ± 0.63	6.8 ± 0.76	1.157 * (0.0398)	(4.5-7.0)%
Insulin resistance level (mg/dl)	0.92 ± 0.28	0.99 ± 0.33	0.287 NS (0.903)	< 99 (mg/dl)
Fasting glucose level (mg/dl)	222 ± 25.47	140 ± 16.51	32.58 ** (0.0049)	(70-120) mg/dl
BMI	32 ± 1.87	18 ± 1.35	3.526 ** (0.0001)	(18-30) g/cm
CRP	4.0 ± 0.32	3.7 ± 0.28	0.502 NS (0.287)	(< 5)
Triglyceride (mg/dl)	141 ± 22.71	120 ± 10.94	37.91 NS (0.306)	(50-150) mg/dl
Cholesterol (mg/dl)	126 ± 14.58	112 ± 9.63	27.61 NS (0.759)	(50-200) mg/dl
LDL (mg/dl)	75 ± 7.91	69 ± 5.60	16.54 NS (0.615)	(35-95) mg/dl

Note: P ≤ 0.05; P ≤ 0.01

The gastrointestinal effects of *H. pylori* have been linked to the development of insulin resistance and diabetes [9]. Table 5 shows no statistically significant differences in the degree of insulin resistance between patients with diabetes mellitus who have *H. pylori* and those who do not. Vafaeimanesh et al. provide contradictory results, showing that patients with high protein intake (HP+) and those without high protein intake (HP-) had degrees of insulin resistance of 3.01 ± 2.12 and 2.74 ± 2.18, respectively (P = 0.704) [20]. On the other hand, patients with diabetes who tested negative for the protein showed significantly lower insulin resistance than those with high HP levels (4.484 ± 2.781 versus 3.160 ± 2.327, P = 0.013). Patients with diabetes have a higher frequency of HP and a more prominent insulin resistance. It's unknown how diabetes or HP infection can affect a person's susceptibility to this infection [20]. Patients with HP infections may be more insulin resistant, according to a theory regarding the relationship between HP infection and diabetes.

Studies have shown that HP infections are more common in people with diabetes [18]. It has been suggested that in this case, autonomic neuropathy and insufficient glycemic control may be important factors [21]. *H. pylori* infection does not significantly affect insulin resistance in people without diabetes. Nonetheless, similar to seropositive individuals, *H. pylori* infection in diabetic patients is linked to a higher frequency of insulin resistance and higher insulin requirements for equivalent control [20].

Table 5 presents data indicating a significant increase in fasting glucose levels between DM patients with H. pylori and the control group. Blood glucose level in HP+ diabetics were found to be higher than in non-HP people; however, this difference was not statistically significant (P = 0.468) [20].

Table 5 shows that patients with DM who have H. pylori have significantly higher BMI levels than patients without H. pylori. Increased body mass index indicates that the patient had dyslipidemia, or high blood sugar and fat buildup, which made them more prone to inflammation. This suggests that they affect insulin secretion and insulin receptor resistance by slowing down stomach motility, which encourages the growth of mycobacteria in the stomach and causes issues, ulcerations, and low-grade inflammation of the mucosal

lining [21]. As indicated by Tawfeeq et al. [19], some lifestyle variables that may contribute to the current high prevalence of H. pylori in our community include living in crowded housing, eating junk food, utilizing unclean water sources, having family members with the infection, and having a high body mass index.

Table 5 presents data indicating a statistically significant increase in CRP concentration among diabetic patients who have H. pylori infection as opposed to those who do not have H. pylori infection. According to Askar et al. [22], there is a substantial correlation between elevated levels of C-reactive protein (CRP) and H. pylori infections. This is due to the fact that H. pylori infection causes the production of TNF-α, IL-1β, and IL-6, among other pro-inflammatory cytokines [22].

When comparing patients with diabetes mellitus (DM) and H. pylori infection to DM patients without H. pylori infection, the data shown in Table 5 show a substantial rise in the levels of triglyceride, cholesterol, and LDL (mg/dl). Individuals with H. pylori infection also had higher mean blood levels of TG (125.28 mg/dL), TC (172.36 mg/dL), and LDL (109.73 mg/dL) [23]. A favorable correlation between H. pylori infection and LDL-C, TC, and TG levels was also found in the study of the serum lipid profile [23].

The results of the study demonstrated a favorable relationship between the blood lipid profile's levels of TG, TC, and LDL-c and H. pylori infection. An H. pylori infection that disrupts lipoprotein and lipid metabolism can cause increased levels of low-density lipoprotein, total cholesterol, and triglycerides [23]. According to Watanabe et al. (2021) [24], H. pylori infection modifies blood lipid composition and may have an indirect impact on the development of disorders linked to faulty lipid metabolism. Ultimately, individuals with H. pylori infection and diabetes mellitus have a different lipid profile [24].

CONCLUSION

There is a complex relationship between H. pylori infection, insulin resistance, and glycated hemoglobin levels in Iraqi patients with type 2 diabetes mellitus. Further research is needed to establish a definitive causal link between these factors. The results of this study showed a significant association between H. pylori infection and elevated levels of HbA1c, fasting blood sugar, and BMI in Iraqi patients with type 2 diabetes. Smoking was identified as a risk factor for H. pylori infection in diabetic patients. These findings highlight the importance of considering H. pylori infection in the management of type 2 diabetes.

AUTHOR CONTRIBUTIONS

All authors substantively contributed to the drafting of the initial and revised versions of this paper. They take full responsibility for the integrity of all aspects of the work.

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

REFERENCES

1. Goyal R, Jialal I. Diabetes Mellitus Type 2. In: StatPearls. StatPearls Publishing; 2022. PMID: 30020625.
2. Malek R, Hannat S, Nechadi A, Mekideche FZ, Kaabeche M. Diabetes and Ramadan: A multicenter study in Algerian population. Diabetes Res Clin Pract. 2019 Apr;150:322-330. https://doi.org/10.1016/j.diabres.2019.01.010.
3. Choi YJ, Chung YS. Type 2 diabetes mellitus and bone fragility: Special focus on bone imaging. Osteoporos Sarcopenia. 2016 Mar; 2(1):20-24. https://doi.org/10.1016/j.ots.2015.12.003.
4. Picke AK, Campbell G, Napoli N, Hofbauer LC, Rauner M. Update on the impact of type 2 diabetes mellitus on bone metabolism and material properties. Endocr Connect. 2019 Mar 01;8(3):R55-R70. https://doi.org/10.1530/EC-18-0518
5. Carrillo-Larco RM, Barengo NC, Albitres-Flores L, Bernabe-Ortiz A. The risk of mortality among people with type 2 diabetes in Latin America: A systematic review and meta-analysis of population-based cohort studies. Diabetes Metab Res Rev. 2019 May;35(4):e3139. https://doi.org/10.1002/dmrr.3139
6. Zhou X, Liu W, Gu M, Zhou H, Zhang G. Helicobacter pylori infection causes hepatic insulin resistance by the c-Jun/miR-203/SOCS3 signaling pathway. J Gastroenterol 2015; 50(10):1027–40. https://doi.org/10.1007/s00535-014-1052-4
7. Jafarzadeh A1, Rezayati MT, Nemati M. Helicobacter pylori seropositivity in patients with type 2 diabetes mellitus in south-east of Iran. Acta Med Iran 2013; 51(12):892–6.
8. Simon L, Tornóczky J, Tóth M, Jámbor M, Sudár Z. Campylobacter pylori fertözés jelentösége a gasztroenterologiai és diabetologiai gyakorlatban [The significance of Campylobacter pylori infection in

gastroenterologic and diabetic practice]. Orv Hetil. 1989 Jun 18;130(25):1325-9. Hungarian. PMID: 2664637.
9. Shinohara K, Shoji T, Emoto M, Tahara H, Koyama H, Ishimura E, Miki T, Tabata T, Nishizawa Y. Insulin resistance as an independent predictor of cardiovascular mortality in patients with end-stage renal disease. J Am Soc Nephrol. 2002 Jul;13(7):1894-900. https://doi.org/10.1097/01.asn.0000019900.87535.43. PMID: 12089386.
10. Aydemir S, Bayraktaroglu T, Sert M, Sokmen C, Atmaca H, Mungan G, Gun BD, Borazan A, Ustundag Y. The effect of Helicobacter pylori on insulin resistance. Dig Dis Sci. 2005 Nov;50(11):2090-3.
11. Malamug LR, Karnchanasorn R, Samoa R, Chiu KC. The Role of Helicobacter pylori Seropositivity in Insulin Sensitivity, Beta Cell Function, and Abnormal Glucose Tolerance. Scientifica (Cairo). 2014;2014:870165. https://doi.org/10.1155/2014/870165. Epub 2014 Mar 25. PMID: 24790772; PMCID: PMC3984821. https://doi.org/10.1155/2014/870165
12. Yin J, Li M, Xu L, et al. Insulin resistance determined by Homeostasis Model Assessment (HOMA) and associations with metabolic syndrome among Chinese children and teenagers. Diabetol Metab Syndr 5, 71 (2013). https://doi.org/10.1186/1758-5996-5-71
13. Mphasha MH, Skaal L, Mothiba TM. Prevalence of overweight and obesity amongst patients with diabetes and their non-diabetic family members in Senwabarwana, Limpopo province, South Africa. S Afr Fam Pract (2004). 2022 May 25;64(1):e1-e7. https://doi.org/10.4102/safp.v64i1.5708
14. Sengupta S, Jaseem T, Ambalavanan J, Hegde A. Homeostatic Model Assessment-Insulin Resistance (HOMA-IR 2) in Mild Subclinical Hypothyroid Subjects. Indian J Clin Biochem. 2018 Apr;33(2):214-217. https://doi.org/10.1007/s12291-017-0701-8
15. Holvoet P, Lee DH, Steffes M, et al. Analytical performance and diagnostic accuracy of immunometric assays for the measurement of circulating oxidized LDL. Clin Chem. 2006 Apr;52(4):760-4. https://doi.org/10.1373/clinchem.2005.062858
16. Ferro A, Morais S, Pelucchi C, et al. Smoking and Helicobacter pylori infection: an individual participant pooled analysis (Stomach Cancer Pooling- StoP Project). Eur J Cancer Prev. 2019 Nov;28(5):390-396. https://doi.org/10.1097/CEJ.0000000000000532
17. Maluf S, Salgado JV, Cysne DN, et al. Increased Glycated Hemoglobin Levels in Patients With Helicobacter pylori Infection Are Associated With the Grading of Chronic Gastritis. Front Immunol. 2020 Oct 29;11:2121. https://doi.org/10.3389/fimmu.2020.587992
18. Candelli M, Rigante D, Schiavino A, et al. High reinfection rate of Helicobacter pylori in young type 1 diabetic patients: a three-year follow-up study. Eur Rev Med Pharmacol Sci. 2012;16(11):1468–1472.
19. Tawfeeq RD, Amin ZA, Nuraddin SM, Jalal A, Baiz SK. Relationship between type II diabetes mellitus and Helicobacter pylori infection in Erbil city. Zanco J Med Sci. 2019 Apr 22;23(1):43-50. https://doi.org/10.15218/zjms.2019.013
20. Vafaeimanesh J, Parham M, Seyyedmajidi M, Bagherzadeh M. Helicobacter pylori infection and insulin resistance in diabetic and nondiabetic population. Scientific World Journal. 2014;2014:391250. https://doi.org/10.1155/2014/391250
21. Talebi-Taher M, Mashayekhi M, Hashemi MH, Bahrani V. Helicobacter pylori in diabetic and non-diabetic patients with dyspepsia. Acta Med Iran. 2012;50(5):315–318.
22. Askar A, Abdallah A, Sedky A, Meghezel E, Mohammed A. Effect of Helicobacter pylori Infection on Insulin Resistance in non-Obese, non-Diabetic Patients. Afro-Egyptian Journal of Infectious and Endemic Diseases, 2021; 11(4): 382-388. https://doi.org/10.21608/ajeid.2021.94710.1227
23. Temesgen GB, Menon M, Gizaw ST, Yimenu BW, Agidew MM. Evaluation of lipid profile and inflammatory marker in patients with gastric Helicobacter pylori infection, Ethiopia. Int J Gen Med. 2022 Jan 6; 15:271-8. https://doi.org/10.2147/IJGM.S346806
24. Watanabe J, Hamasaki M, Kotani K. The Effect of Helicobacter pylori Eradication on Lipid Levels: A Meta-Analysis. J Clin Med. 2021 Feb 25;10(5):904. https://doi.org/10.3390/jcm10050904. PMID: 33668848; PMCID: PMC7956592

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