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# The Influence of Helicobacter Pylori Infection on Cardiovascular Risk Factors in Patients with Gastritis

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**Abstract**

Helicobacter pylori (HP) is the etiopathogenic agent of gastric and duodenal disorders ranging from gastritis to malignancy. It is also associated with many extra-intestinal diseases, including cardiovascular disease and its associated risk factors. To evaluate the link between HP infection and some cardiovascular risk factors by studying the effects of HP infection on body mass index, blood pressure, and serum lipid profile among patients having gastritis with and without HP infection. A cross-sectional study included 1214 patients who had gastritis diagnosed by gastroscopy examination. Those patients were in the age range of 30-65 years and they were divided according to their gender into 725 females and 489 males depending on the 13C urea breath test, they were divided into HP positive ( +) groups (550 female & 300 male ) and HP negative (-) group (175 female & 189 male). The blood pressure and body mass index (BMI) were measured for each patient and following at least 10-h fasting, a lipid profile test was performed. Our study exhibited a significant difference ($p > 0.05$) in Body Mass Index (BMI) between HP (+) and HP (–) participants. HP (+) participants were obese (34.29 Kg/m2) while HP (-) participants were lean. The mean systolic & diastolic blood pressures were non- significantly higher in HP (+) group than those in HP (-) group. The TC, TG, and LDL parameters scored the highest mean value in HP (+) group (212.47±18.35, 117.17±37.14, and 79.30±15.42) respectively. In contrast, HDL scored the lowest mean value in HP (-) group (40.59±2.38). HP infection significantly alters lipid profile test and may be one of the risk factors for obesity, dyslipidemia and hypertension.

**Keywords:** Helicobacter pylori; cardiovascular disease; Body Mass Index; Blood pressure; Lipid Profile Test.

# تأثير الإصابة ببكتريا المعدة على عوامل الخطر القلبية لدى المصابين بالتهاب المعدة

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**الخلاصة**

تعتبر الجرثومة المعوية (HP) العامل المسبب للاضطرابات المعوية التي تتراوح من التهاب المعدة إلى المسرطنة، كما أنه مرتبط أيضًا بالعديد من الأمراض خارج الجهاز الهضمي، بما في ذلك أمراض القلب وعوامل الخطر المرتبطة بها. تقييم الارتباط بين عدوى HP وبعض عوامل خطر الأمراض القلبية من خلال دراسة تأثير عدوى HP على مؤشر كتلة الجسم، وضغط الدم، وملف الدهون في الدم بين المرضى الذين يعانون من التهاب المعدة مع وبدون عدوى HP. دراسة مستعرضة شملت 1214 مريضًا تم تشخيصهم بالتهاب المعدة بواسطة فحص المنظار الهضمي. كان هؤلاء المرضى في الفترة العمرية من 30 إلى 65 عامًا، وتم تقسيمهم حسب جنسهم إلى 725 إناث و 489 ذكور، وتم تقسيمهم بناءً على اختبار النفخة باليوريا C13 إلى مجموعة HP إيجابية (+) (550 إناث و 300 ذكور) ومجموعة HP سلبية (-) (175 إناث و 189 ذكور). تم قياس ضغط الدم ومؤشر كتلة الجسم (BMI) لكل مريض، وبعد صيام لا يقل عن 10 ساعات، تم إجراء اختبار ملف الدهون في الدم. أظهرت دراستنا فرقًا معنويًا ($p > 0.05$) في مؤشر كتلة الجسم (BMI) بين المشاركين HP (+) و HP (-). كان المشاركون HP (+) ذو وزن عالي (34.29 كجم/م2) بينما كان المشاركون HP (-) اقل وزنا. كانت متوسط ضغط الدم الانبساطي والانقباضي أعلى بشكل غير معنوي في مجموعة HP (+) من تلك في مجموعة HP (-). سُجلت أعلى قيمة متوسطية لمعايير TC و TG و LDL في مجموعة HP (+) (212.47±18.35، 117.17±37.14، و 79.30±15.42) على التوالي. بالمقابل، كانت قيمة متوسط ال HDL الأقل في مجموعة HP (-) (40.59±2.38). تؤثر الاصابه بـ HP بشكل ملحوظ على اختبار ملف الدهون في الدم وقد تكون واحدة من عوامل الخطر للسمنة واضطرابات الدهون في الدم وارتفاع ضغط الدم.

## 1. Introduction

Cardiovascular diseases (CVDs) are leading causes of global morbidity and mortality [1,2], taking approximately 17.9 million lives each year. The pathogenic mechanism of CVDs is atherosclerosis process which is multi-factorial and the factors behind this process are diabetes mellitus, hypertension, smoking, and obesity [3]. Additionally, a theory states that immuno-inflammatory mechanisms initiated by viral or bacterial infections are the leading cause of atherosclerosis [4]. Helicobacter pylori (HP) is considered the etiopathogenic agent of gastric and duodenal disorders ranging from gastritis to malignancy [5]. It is also associated with many extra-intestinal diseases, including cardiovascular disease and its associated risk factors [6,7]. The relationship between HP infection and CVDs are controversial. Some studies demonstrated a higher incidence of HP infection in patients with CVDs [8,9]. whereas others claimed that HP infection was not associated with CVDs and their risk factors [10,11]. The American Heart Association states that to prevent heart disease early in life it is important to look for and manage risk factors affecting cardiovascular disease [12]and if HP has a direct or indirect role in atherosclerosis processes, its eradication may be necessary to prevent progression of atherosclerosis, especially in a high-risk population. Hence, the objective of the present study was to evaluate the link between HP infection and some cardiovascular risk factors by studying the effects of HP infection on body mass index, blood pressure, and serum lipid profile among patients having gastritis with and without HP infection.

## 2. Patients and Methods

This cross-sectional study was conducted in gastroenterology clinic of Baqubah Hospital & private hospitals in Baghdad. A total of 1214 patients were recruited in this study from January 2020 to April 2022. Those patients were in the age range of 30-65 years and divided according to their gender into 725 female and 489 male. Patients who had gastritis diagnosed by gastroscopy examination were divided into HP positive (+) group (550 female & 300 male) and HP negative (-) group (175 female & 189 male). HP infection was diagnosed by a 13C urea breath test.

Socio-demographic data were collected from each patient: age, gender, education, marital status and occupation.

The Body Mass Index (BMI, kg/m2) was measured according to World Health Organization (WHO) as overweight defined as BMI 25 – <30 and obesity defined as BMI ≥ 30[13].

After at least 10 min of rest blood pressure was measured and hypertension is defined as an average systolic blood pressure ≥140 mmHg, diastolic blood pressure ≥90 mmHg [14].
Laboratory Investigations: were performed after a 10-h fast, including total cholesterol (TC), low-density lipoprotein cholesterol (LDL), high-density lipoprotein cholesterol (HDL) and triglyceride (TG). The cut-off values for abnormal lipid profile were: TG ≥150 mg/dL, total cholesterol ≥200 mg/dL[15,16].

Statistical analysis was performed by SPSS version 23.0 and Graph pad prism version 6. Parameters, fitting for both normality (Kolmogorov-Smirnov and ShapiroWilk ) tests, were measured as mean ± Standard deviation while the parameters that did not fit for normality tests were measured as median and range, and significant differences between median were assessed by Mann-Whitney (for comparison between two groups). Data were considered significant if p-value ≤ 0.05 [17].

## 3. Results

A total of 1214 participants with gastritis 725 (59.72 %) female and 489 (40.28 %) male were included in this study. With respect to seropositivity for HP; 850 (70.02%) patients with gastritis had (+) HP infection and 364 (29.98 %) had (-) HP infection. The prevalence of HP was higher among males compared with females (64.71% vs. 35.29%). On the other hand, 51.92% of females vs. 48.08 % of males had (-) HP infection. (Table -1) In our study, BMI exhibited a significant difference ($p > 0.05$) between HP (+) and HP (–) participants (Table -2). Based on the definition of WHO for BMI; HP (+) participants were obese ( 34.29 Kg/m2) while HP(-) participants were lean. Furthermore, there is a statistically significant difference ($P<0.01$) in BMI between males and females as the BMI of females (35.6 kg/m2) was higher than males(30.26 kg/m2). The mean systolic & diastolic blood pressures were increased in HP (+) group than those in HP (-) group, but this increment was statistically non-significant ($p>0.05$) (Table-4). Based on gender, the mean systolic & diastolic blood pressures were higher in males than in females and the current study showed that this increment was statistically non-significant ($p>0.05$) (table-5).The mean levels of serum TC, TG, LDL and HDL were compared between HP groups and there were significant differences ($p<0.05$) between the above lipid profile markers.

The TC, TG, and LDL parameters scored the highest mean value in HP (+) group (212.47±18.35, 117.17±37.14, and 79.30±15.42) respectively. In contrast, HDL scored the lowest mean value in HP (-) group (40.59±2.38) (Table -6). Based on gender, the mean levels of serum TC, TG, and LDL were highest in males in HP (+) group (227.37±13.81, 208.53±14.40, and 108.92±14.42) respectively and least in females in HP (-) group (174.72±5.15, 98.44±8.61, and 68.88±7.37) respectively. On the other hand, the mean levels of serum HDL were highest in females in HP (-) group (49.41±3.36) and least in males in HP (+) group (40.47±2.71) (Table-7).

**Table 1-** Gender Distribution of the study population.

| Gender | HP (+) No. (%) | HP (-) No. (%)) | Total |
|---|---|---|---|
| **Male** | 550 (64.71%) | 175 (48.08%) | 725 (59.72%) |
| **Female** | 300(35.29%) | 189 (51.92%) | 489 (40.28%) |
| **Total** | 850(100%) | 364(100%) | 1214 (100%) |

**Table 2-** Body Mass Index (BMI) of participants and HP infection

| | Group | Mean | SD |
|---|---|---|---|
| **BMI(Kg/height m)2 18.5-24.9** | HP(+) | 34.29 | 0.29 |
| | HP (–) | 23.12 | 0.43 |
| **P value** | **P<0.01**** | | |

**Table 3-** Gender Association between HP infection and BMI Measures.

| BMI (Kg/height m)2 18.5-24.9 | Gender | Gastritis HP (+) | | HP ( – ) | | |
|---|---|---|---|---|---|---|
| | **Male** | **Mean** | **SD** | **Mean** | **SD** | **statistics** |
| | | 30.26 | 0.69 | 23.69 | 1.79 | **P<0.05*** |
| | **Female** | 35.6 | 0.63 | 24.49 | 0.73 | **P<0.01**** |
| **P value** | | **P<0.05*** | | **p>0.05** | | |

**Table 4-** Correlation between HP infection and blood pressure measures

| Blood Pressure (mmHg) | Gastritis HP (+) Mean ± SD | HP (-) Mean ± SD | |
|---|---|---|---|
| **Systolic** | 12.17± 2.57 | 11.81± 0.65 | P>0.05 |
| **Diastolic** | 8.59±0.66 | 8.02± 0.32 | P>0.05 |

**Table 5-** Gender association between HP infection and blood pressure measures

| Blood pressure | Gender | Gastritis HP (+) Mean ± SD | HP (-) Mean ± SD | Statistics |
|---|---|---|---|---|
| **Systolic (mmHg)** | Male | 12.41 ± 0.39 | 12.11± 0.60 | P>0.05 |
| | Female | 11.93 ± 3.69 | 11.50 ± 0.56 | |
| **Diastolic (mmHg)** | Male | 8.92± 0.75 | 8.00 ± 0.37 | P>0.05 |
| | **Female** | **8.26 ± 0.35** | **8.03± 0.28** | |

**Table 6-** The Comparison of Serum Lipid Profile levels Between HP (+) and HP (-) patients

| Lipid Profile (mg/dl) | Gastritis HP + | Gastritis HP - | P value |
|---|---|---|---|
| **TC** | 212.47 ±18.35 | 176.36± 5.37 | P<0.001*** LSD=11.12 |
| **TG** | 117.17 ±37.14 | 109.85 ±15.43 | P<0.001*** LSD=21.20 |
| **HDL** | 40.59± 2.38 | 45.84± 4.62 | P<0.001*** LSD=2.49 |
| **LDL** | 79.30± 15.42 | 69.00± 6.37 | P<0.001*** LSD=9.61 |

**Table 7-** Gender Comparison of Serum Lipid Profile between HP (+) and HP (-) Patients

| Lipid Profile (mg/dl) | Gender | Gastritis HP (+) Mean ± SD | Gastritis HP( -) Mean ± SD | P value |
|---|---|---|---|---|
| **TC** | Female | 197.57 ±5.18 | 174.72 ±5.15 | P<0.001*** |
| | Male | 227.37 ±13.81 | 178.00 ± 5.34 | LSD=14.21 |
| **TG** | Female | 147.80 ±25.59 | 98.44 ±8.61 | P<0.001*** |
| | Male | 208.53±14.40 | 100.26 ±11.79 | LSD=28.49 |
| **HDL** | Female | 40.71 ± 2.14 | 49.41 ±3.86 | P<0.001*** |
| | Male | 40.47± 2.71 | 42.27± 1.32 | LSD=3.81 |
| **LDL** | Female | 89.68 ± 9.40 | 68.88± 7.37 | P<0.001*** |
| | Male | 108.92±14.42 | 69.12 ±5.59 | LSD=12.31 |

## 4. Discussion

In this study, the prevalence of HP infection was higher in males than females. These findings were compatible with the results of previous studies [18-21]. This sex variation may be attributed to the protective role of female sex hormones against gastric inflammation and the role of these hormones on the vascularity and thickness of mucous membrane of the stomach. Relation of HP infection and BMI is controversial. In this study, a significant association was found between obesity and HP infection which is in consensus with the results of some studies [22-25]. However, other studies exhibited no association between HP infection and BMI [26,27]. The relationships between HP infection and overweight /obesity are still unclear. It is unclear whether obese people are more liable to HP infection or whether HP infection raises the possibility of obesity. By previous studies, it has been shown that in obesity, there was a reduction of polymorphonuclear bactericidal capacity, monocyte maturation and natural killer cell activity [28,29,30]. Such impairment in the immune system of obese people provides a suitable environment for HP growth. On the other hand, HP infection could lead to increased appetite and weight gain by decreasing the circulating levels of leptin and ghrelin [30,31]. In our previous study, it was shown that fasting insulin and glucose levels in addition to HOMA-IR were higher in HP (+) group than HP (-) group (20). In turn, pro-inflammatory cytokines which were induced by HP infection stimulate many kinases that disrupt signaling of insulin in fat and liver cells leading to insulin resistance [29,32]. Therefore; obesity can be developed secondary to insulin resistance and hyperinsulinemia [33]. In our study, the mean systolic & diastolic blood pressures were non- significantly higher in HP (+) group than HP(-) The

correlation of HP infection and hypertension with both positive [34,35]. and negative findings [36,37] are controversial. The rise in the means of systolic and diastolic measures in HP infected group may be attributed to the activation of IL-6 and tumor necrosis factor-alpha (TNF-a) [38,39]. and these inflammatory cytokines promote insulin resistance which may further increase the total peripheral tension [32].The relationships between HP infection and dyslipidemia are still contradictory. Some studies have suggested that HP infection has a significant role in dyslipidemia and increases the risk of atherosclerosis (40-43). Other studies do not support the idea of HP - dependent dyslipidemia [44]. In the current study, the mean levels of serum TC, TG and LDL were significantly increased in HP (+) group. This was in agreement with a study done in Ethiopia but their results were not statistically significant [45]. On the other hand the mean level of serum HDL in our study was significantly decreased in HP (+) group. Similar results were reported in Japan and Ethiopia [45,46]. Our results indicate that HP infection may be involved in lipid profile alteration and hence HP infection may be a predisposing factor for CVDs. The mechanisms by which HP infection may induce CVDs are dysfunction of endothelium, accumulation of reactive oxygen species (ROS) and autoimmunity. The above three mechanisms lead to inhibition of lipoprotein lipase resulting in increased TG and reduced HDL (44).In our study, the mean levels of serum TC, TG and LDL in HP (+) group were higher in males than females. On the other hand, the mean level of HDL was lower in males than females. This gender difference suggests the effects of sex hormones i.e. the atheroprotective effect of estrogen and the atherogenic effect of testosterone on lipoprotein metabolism. Estrogens increase the serum levels of HDL by suppressing lipase activity in the liver and decrease the serum levels of LDL by enhancing hepatic clearance of LDL [47]. Whereas, androgens decrease the serum levels of HDL and LDL by activating the gene expression of hepatic lipase and reducing LDL receptors and lipoprotein lipase activity respectively [48].

## 5. Conclusion

HP infection significantly alters lipid profile test and may be one of the risk factors for obesity, dyslipidemia and hypertension and hence HP infection may be a predisposing factor for CVDs.

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