Association of Helicobacter pylori infection with cardiovascular risk among patients attending Cairo University outpatient clinics

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Abstract:

Introduction: Cardiovascular disease (CVD) is the primary cause of morbidity and death worldwide. The WHO estimates that CVD accounts for 32% of all deaths worldwide annually. Egypt has one of the highest rates of mortality from CVD regionally. In Egypt, CVD accounted for 46.2% of all fatalities. Research indicates a correlation between Helicobacter pylori (H. pylori) and a higher risk of CVD. This study aims to assess the link between H. pylori and CVD. **Methods:** This case-control study was done on two groups complaining of dyspepsia from December 2022 till June 2023. The participants were 46 with high cardiovascular risk (cases) and the same number with low cardiovascular risk (controls). The Pooled Cohort Risk Estimator Plus assessed the Atherosclerotic CVD (ASCVD) risk. All participants were subjected to a structured interview using a pre-designed questionnaire. A pooled cohort equation using logistic regression evaluated the association between H. pylori infection and cardiovascular risk. Multivariate logistic regression tests adjusted for confounders like gender, age, body mass index (BMI), smoking, lipids, diabetes (D.M.), and hypertension. **Results**: There was an increase in H. pylori infection (p = 0.04) in case group patients (74%) compared to control group patients (63%), which is significant with a statistically significant association with increased CVD risk (OR 1.661, 95%CI 0.682–4.043; P < 0.0001) and remained so after multivariable adjustment of confounders. **Conclusions**: H. pylori infection is a possible independent risk factor for CVD

Keywords: Disease Prevention, Health Promotion, Heart diseases, Risk factors.

Background

WHO estimates that 17.9 million deaths worldwide are attributed to cardiovascular disease (CVD), with 75% of all deaths occurring in low-income countries.(1) One of the U.N. sustainable development goals is to reduce mortalities from non-communicable diseases via prevention and treatment.(2) The management of CVD risk factors can potentially prevent myocardial infarction, stroke, and CVD-related deaths.(3)

Diabetes, dyslipidemia, smoking, and hypertension are the most common risk factors of atherogenesis that lead to coronary artery disease. (4) Former research revealed a link between elevated CVD and H. pylori infection., dyslipidemia,

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hypertension, D.M., insulin resistance, metabolic syndrome, and the development of CVD.[5, 6] The high infection rate with H. pylori and its impact on CVD has attracted public attention in recent years.[7]

Over half of the world's population is infected with H. pylori., with Africa having the highest prevalence at 80%. [8] and Egypt is one of the highest-prevalence countries. [9]

Several possible processes and mechanisms have been proposed to elucidate the relationship between H. pylori infection and CVD. The successful isolation of H. pylori from atherosclerotic plaques and the identification of its DNA supports the theory that endothelial dysfunction, which is caused by direct action on the endothelium's surface, might contribute to CVD.[1]

The most frequent underlying H. Pylori mechanism in the emergence of endothelial dysfunction appears to be oxidative stress and vitamin deficiency, particularly vitamin B12. Another common mechanism is inflammation; oxidative stress and inflammation are causally related. By depositing on the intima, C-reactive protein (CRP), an acute phase inflammatory protein, directly influences nitric oxide bioavailability, which results in oxidative stress and endothelial dysfunction. This directly contributes to the early phase of atherosclerosis. [10]

The activation of pro-inflammatory cytokines like CRP, tumor necrosis factor-α (TNF-α), interleukin (IL-6), and IL-18 has been demonstrated in recent research to be a significant cardiovascular disease risk factor caused by atherosclerosis. Along with causing low-grade systemic inflammation, H. pylori is also responsible for local inflammation in the stomach mucosa. [11]

Atherosclerosis is induced by H. pylori, as evidenced by the correlations found between anti-H. Pylori IgG antibodies, coronary artery calcium, arterial stiffness, and subclinical artery stenosis. [1]

It has been proposed that elevating systolic and diastolic blood pressure (SBP) values is one way H. pylori may raise the risk of CVD. [12]

Numerous studies have connected a persistent H. pylori infection to preventing insulin sensitivity. Even though it's also linked to type 2 Diabetes Mellitus. [13]

Dyslipidemia is one such route. H. pylori infection may lead to CVD. Derangements in the lipid profile, such as low HDL levels, high LDL, total cholesterol, or high triglyceride levels, were noted in individuals with an H. pylori infection.[5]

Considering the association between elevated homocysteine levels and a higher risk of CVD, hyperhomocysteinemia is a novel cardiovascular risk factor.[14] It is commonly known that the caused gastritis decreases the absorption of vitamin B12 and folate, resulting in secondary hyperhomocysteinemia and elevated CRP, potentially signifying the start of atherosclerotic plaque formation.[1]

The hypothesis that *H. pylori* causes CVD remains controversial due to the inconsistency of epidemiological studies.[15] More research is recommended to understand how this infection contributes to the development of CVD. Therefore, this study aims to assess the relationship between an increased risk of CVD and H. pylori infection. to definitively contribute to better treatment, improved health quality of infected patients, and prevention of complications.

Methods:

This is a case-control study involving 92 participants (46 cases and 46 matched controls), patients attending family medicine clinics at Cairo University hospitals over six months from December 2022 to June 2023.

Inclusion criteria: Adult Patients aged 20-79 of both genders complaining of dyspepsia were eligible. They were divided into 46 individuals with high cardiovascular risk (cases) and an equal number with low cardiovascular risk (controls).

Both groups were matched regarding age, residence, social class, chronic diseases, medications, anthropometric data (weight, height, BMI, and waist circumference), and systolic and diastolic levels. Exclusion criteria: Patients known to have D.M., hypertension, cardiac diseases, familial hypercholesterolemia, and inherited lipid disorders were excluded from the study.

The paragraph edited to: According to the evidence from a previous study [16] and using the primary outcome to be the mean difference in LDL between participants who tested positive for H. pylori infection and controls. The sample size was calculated by STATA 16 assuming 80% power, a 0.05 significance level, considering 2.33 mean in the H. pylori infection group, 1.71 mean in the controls with a standard deviation of 1, estimated effect size 0.15, Sample size = 84 participants (42 / group). Considering the 10% drop-out rate, thus the final sample size was 92 participants (46 / group).

The Faculty of Medicine Scientific Research Ethical Committee and the Ethics

Committee at Cairo University approved the study protocol, which has the approval number MS-382-2022. Every participant provided informed written consent.

Personal and socio-demographic data, medical data, anthropometric measurements, physical examination, and requesting laboratory investigations. In a second visit, we calculated cardiovascular risk and determined H. pylori laboratory results.

Personal data: name, age, address, occupation, and marital status. Socio-demographic: information sheet, including the candidate and her husband's education, occupation, social state, residence, home environment & sanitation, family possessions, and economic and health state using the socioeconomic status scale for health research in Egypt. The SES was graded based on the quartiles of the measured score as low (42), middle (43-63), and high levels (64-84).[17]

Medical history: including any chronic disease, medications, previous psychological problems, and life habits (exercise, smoking, substance abuse). Clinical examination: All participants measured their blood pressure, weight, height, and waist circumference, and their BMI was calculated for each participant.

Assessment of ASCVD risk

Assessment of ASCVD risk was done by the Pooled Cohort Risk Estimator Plus (tools.acc.org/ascvd-risk-estimator-plus).[18]

The following variables were used: sex, race, total cholesterol, LDL, HDL, smoking status, history of diabetes, systolic blood pressure (SBP), aspirin, hypertension treatment, and dyslipidemia. [19]

In patients aged over 40 years, the 10-year risk was calculated and sorted into low-risk (<7.5%) and high-risk (≥7.5%). [19]

Assessment of H. pylori infection

To perform the H. pylori stool antigen test, patients and controls were informed to collect stool samples (1-2 ml or 1-2g) in a clean, dry specimen collection container. The stool samples were immediately transported to the Department of Parasitology and processed on the same day they were received.

The test was performed by a rapid immune chromatographic assay using a commercially available kit. The tests were conducted in compliance with the instructions provided by the manufacturer. The reports were read as positive and negative.[20]

Statistical Design

The data were analyzed using (SPSS version 21) the statistical package for Social Sciences. The data was described using appropriate central tendency, dispersion, and percentage measures as indicated. T-tests were employed to test continuous variables. And were expressed as means ±SD. At the same time, chi-square tests were used for categorical variables.

The association of H. pylori infection with cardiovascular risk was assessed using unadjusted (univariate) and adjusted (multivariate) logistic regression analysis. In the multivariate regression analysis, the first model was adjusted for age and sex, while in the second model, age, sex, plus BMI, and lipid were entered; in the final model, age, sex, BMI, lipid, smoking, D.M., and HTN were entered. The statistical significance level was set at p ≤0.05; all tests were two-tailed.

Results

This study involved 92 patients in total. Of these, 46 were patients complaining of dyspepsia with high cardiovascular risk, and the remaining 46 were matched controls with low cardiovascular risk.

Table (1) demonstrates the socio-demographic characteristics, medical and clinical data, and laboratory results of cases and control groups. There was no significant difference between cases and controls regarding age, residence, social class, chronic diseases, medications, anthropometric data (weight, height, BMI, and waist circumference), and systolic and diastolic blood pressure measurements. At the same time, there was a statistically significant difference in gender and level of education.

The mean age among cases was 36.78±10.48, and among controls was 38.9 ± 9.35, with the difference being statistically insignificant(P=0.32).

Table (2) demonstrates the distribution of H. pylori seropositivity in case and control groups. It indicates that 74% of the case and 63% of the control groups had H. pylori positivity, respectively. Additionally, there was a significant difference in the frequencies of H. pylori positivity between case and control (P-Value = 0.04).

There was a difference in the prevalence of smoking among infected (64%) and non-infected (21%) participants, which is significant (P-Value = 0.0001).

Table (3) demonstrates the logistic regression analysis of the association of H. pylori infection with the case and control group. Age, gender, BMI, lipid profile, smoking status, D.M., and hypertension

were entered into the logistic regression model.

As shown, the cases were at significantly higher risk for H. pylori affection than controls. Multivariate logistic regression after adjusting for confounding factors revealed a significant association between H-pylori infection and CVD risk (p <0.0001) Figure (1).

Discussion

Our main findings revealed a significant difference regarding H. pylori infection between case and control groups. Using logistic regression, cases were at significantly more risk for H. pylori affection than controls.

According to our research, 74% of cases had H. pylori infection, and there was a significant difference between the groups (P=0.04). This result agrees with a cohort study conducted in Austria.

The H. pylori-negative and positive patients were 2659 and 625, respectively. H. pylori-positive prevalence was higher in high cardiovascular risk patients (23% vs.18%; p<0 .003).[21]

On the contrary, a cross-sectional study conducted at the Health Examination Center of Busan Metropolitan City Medical Center revealed a higher average value of the Framingham risk score in the negative (6.73%±6.4%) than in the positive group (6.84%±6.41%), with no significant difference between the two groups.

This difference may be due to different places and populations.[22] Moreover, the hypothesis that *H. pylori* causes CVD remains controversial due to the inconsistency of epidemiological studies.[15] More research is recommended to clarify the role of this infection in CVD occurrence.

In our study, in multilevel logistic regression, cases were at significantly more risk of H. pylori affection than controls and remained significant after adjustment for confounders such as sex, age, BMI, lipid, smoking, D.M., and hypertension (P < 0.0001).

Inconsistent with the present study, Wernly *et al.*[21] stated that SCORE2 and H. pylori positivity were significantly and independently correlated. (r = .64; 95% CI 0.31– 0.96; p < 0.001) and associated considerably (r = .33; 95% CI 0.09– 0.57; p = 0.006) after sex, age, and the metabolic syndrome diagnosis adjustment.

Also, consistent with our study, a study was conducted at Isfahan Chamran Heart Hospital. Multivariate logistic regression analysis after confounder factors adjustment showed a significant association between H. pylori infection and CHD (OR 3.18, 95%CI

1.08-9.40).[23] This can be explained by recent studies that inflammation is one of the main risk factors for atherosclerosis-induced CVD through activating pro-inflammatory cytokines like CRP, TNF-α, IL-6, and IL-18. H. pylori is causing local inflammation in gastric mucosa and low-grade systemic inflammation.[11]

On the other hand, Christodoulou *et al.* [24] studied the theory that H. pylori infection is linked to higher documented rates of CVD in patients undergoing elective upper gastrointestinal endoscopy.

After controlling for confounding variables such as sex, age, diabetes mellitus, hypertension, smoking, and serum parameters, multiple regression analyses proved that confirmed H. pylori is not associated with documented CVD. Differentiating between the two studies' results might be caused by factors such as adjusting for possible confounders and determining the H. pylori status.

The current study revealed the presence of a significant difference between H. pylori-negative and positive groups as regards smoking. This is by a survey conducted in Erbil City, Iraq, found that the prevalence of H. pylori-positive infection in male nargileh and cigarette smokers was 64.9% and 45.5% in non-smokers.[25]

On the contrary, Salama *et al.*[26] reported the absence of a significant difference between smokers and non-smokers regarding the positivity rate of H. pylori. A limited number of population samples may be the cause of this discrepancy. And the absence of a matching sex group.

This emphasizes that smoking is a global public health issue and that it plays an essential role in the development of lots of common medical conditions, like cancer and chronic obstructive pulmonary diseases. Additionally, smoking's effects on the microbiome allow oral pathogens to proliferate, which in turn causes several diseases, so many studies were needed to explain the relationship between smoking and H. pylori infection.

Study limitations

Our study relied solely on stool antigen testing to detect H. pylori infection. Furthermore, the small sample size and the poor matching between cases and controls concerning gender and level of education could potentially affect our findings.

Conclusion

Our study indicates an association between H. pylori infection and an increased risk of cardiovascular disease. H. pylori infection is a probable independent

risk factor for CVD. Therefore, screening and management of H. pylori are important from a cardiovascular perspective.

Recommendations

Considering the study's findings, we advise screening for cardiovascular disease using different CVD risk assessment tools for all patients coming for medical assessment who are diagnosed with H. pylori infection.

Large-scale epidemiological research is necessary to fully comprehend the impact of H. pylori on bio-psycho-social aspects and cardiovascular health. Biomarkers for cardiovascular risk prediction may be used to make risk stratification for cardiovascular diseases more evident.

We also recommend educational training programs about H. pylori infection to be conducted at hospitals for patients and their relatives to promote the importance of treatment of H. pylori and provide health education messages about H. pylori infection prevention, complications and its CVD association.

Declarations

- Competing interests: The authors declared no competing interests.
- Funding: no fund was received for this work
- **Authors' contributions:**" M.S. was responsible for collecting, analyzing, and interpreting the patient data and shared it in the writing. I.T. supervised and revised the whole manuscript. S.M. shared in data collection, and E.R. was a significant contributor to writing the manuscript, conceiving and designing the work, and interpreting the data. All authors read and approved the final manuscript.

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Table 1 : Demographic, anthropometric clinical data and laboratory results of cases and controls

	Case Group	Control Group	P value
Age (Mean ± S.D.)	36.78 ± 10.84	38.91 ± 9.35	0.32
Gender			**0.001***
Male	25 (54.3%)	38	
Female	21 (45.6%)	8	
Residence			0.052
Slums	12 (26%)	10 (21.7%)	
Urban	21 (45.7%)	12 (26%)	
Rural	13 (28.3%)	24 (52.17%)	
Level of education			**0.001***
Illiterate	8 (17.4%)	12 (26%)	
primary	9 (19.5%)	10 (21.7%)	
secondary	12 (26%)	9 (19.5%)	
high	12 (26%)	15 (32.6%)	
intermediate	5 (10.8%)	0	
Social class			1
low	34 (74%)	34 (74%)	
moderate	12 (26%)	12 (26%)	
high	0	0	
Chronic disease	8 (17.4%)	6 (13%)	0.9
Medication	13 (28.2%)	13 (28.2%)	1
Smoking	31 (67.4%)	6 (13%)	**0.0001***
Systolic bp (Mean ± S.D.)	123.48 ± 15.95	119.39 ± 13.13	0.19
Diastolic BP (Mean ± SD)	80.22 ± 10.7	79.24 ± 11.35	0.17
Weight (Mean ± SD)	80.15 ± 14.37	76.23 ± 15.2	0.708
Height (Mean ± S.D.)	164.63 ± 8.91	160.11 ± 10.87	0.186
BMI (Mean ± SD)	29.62 ± 5.17	29.49 ± 6.6	0.7
Waist circumference (Mean ± S.D.)	100.72 ± 10.6	102.65 ± 13.19	0.14
*Indicates a statistically significant difference between studied groups			

Table (2): Distribution of H. pylori seropositivity in case group and control group (n=92)

H. pylori	Case Group (n=46)	Control Group (n=46)	Total	P value
Negative	17 (37%)	12 (26%)	29(31.5%)	0.04*
Positive	29 (63%)	34 (74%)	63(68.5%)	
**Significant P value*				

Table 3 : Logistic regression analysis of the association of H. pylori infection with case and control group.

	OR (95% CI)	P. Value
Model 1	1.661 (0.682-4.043)	<0.001
Model 2	2.722 (0.707-6.966)	<0.001
Model 3	2.638 (0.907-7.676)	<0.001
Model 4	2.211 (0.64-7.639)	<0.001
Model 1: Not Adjusted Model 2: Adjusted Age, Sex Model 3: Adjusted Age, Sex, BMI, Lipid Model 4: Adjusted Age, Sex, BMI, Lipid, Smoking, D.M., HTN		

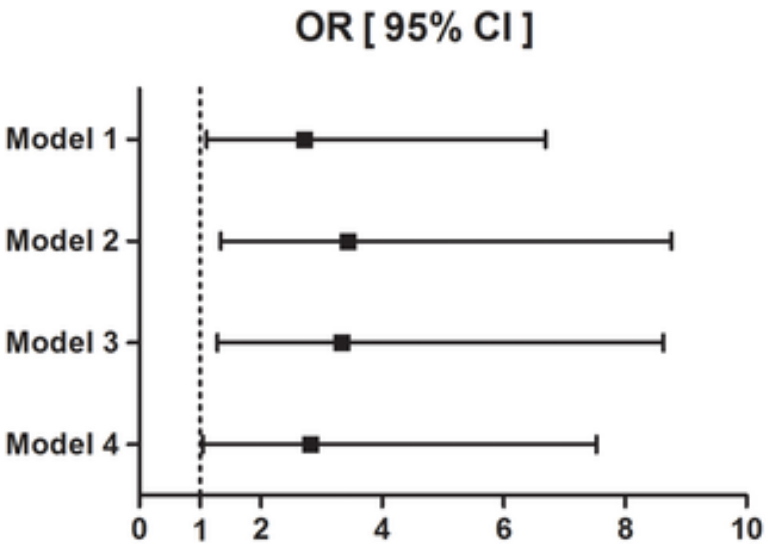

Figure 1 : Logistic regression for significant cardiovascular disease according to the H. pylori infection

الملخص العربي

ارتباط العدوى بالبكتيريا الحلزونية بالإصابة بأمراض القلب والأوعية الدموية بين المرضى المترددين على العيادات الخارجية بجامعة القاهرة

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[١] قسم طب الأسرة، كلية الطب، جامعة القاهرة

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الخلفية: إن الهيليكوباكتر بيلوريهي بكتيريا حلزونية موجودة في المعدة. ويعد أكثر من خمسين في المئة من سكان العالم مصابون بالبكتيريا الحلزونية. يختلف انتشار البكتيريا الحلزونية جغرافياً مع ارتفاع نسبه الانتشار في البلدان النامية وتعتبر مصر من أكثر الدول انتشارًا للإصابة بالبكتيريا الحلزونية. تعتبر الطريقة الأكثر شيوعًا للانتقال هي من شخص لآخر عن طريق الفم الي الفم أوالفم الى البراز. يمكن أن يتأثر معدل الإصابة بالبكتيريا الحلزونية بالحالة الاجتماعية والاقتصادية والعمر والنظافة الشخصية. تُظهر عدوى البكتيريا الحلزونية مجموعة واسعة من العلامات المرضية بما في ذلك أمراض المعدة وأمراض خارج المعدة. وقد كشفت بعض الدراسات أن عدوى البكتيريا الحلزونية قد تكون مرتبطة بزيادة أمراض القلب والأوعية الدموية، وارتفاع ضغط الدم وارتفاع الدهون بالدم ومقاومة الأنسولين ومرض السكري ومتلازمة التمثيل الغذائي. وكما تؤثر العدوى على فسيولوجيا القلب وبطانة الأوعية الدموية وخللها الوظيفي، وتكاثر العضلات الملساء، والالتهابات الموضعية، وارتفاع نسبة التجلط بالدم، وأكسدة البروتين الدهني منخفض الكثافة. **الهدف من هذه الدراسة:** هو تقييم ارتباط عدوى البكتيريا الحلزونية بالمخاطر القلبية الوعائية ومقارنتها مع مجموعة التحكم المطابقة، وتقييم تأثير عدوى البكتيريا الحلزونية على صحة القلب. **طرق البحث** : كانت هذه دراسة تحليلية للحالات والضوابط أجريت على عينتين ٤٦ من المشاركين يشكون من عسر الهضم مع ارتفاع أخطار القلب والأوعية الدموية (الحالات) ويشكو ٤٦ من المشاركين من عسر الهضم مع انخفاض أخطار القلب والأوعية الدموية (الضوابط) بهدف تقييم العلاقة بين عدوى الملوية البوابية بهدف تقييم العلاقة بين عدوى البكتيريا الحلزونية وأمراض القلب والأوعية الدموية وعوامل الخطر الخاصة بها. تم جمع البيانات على شكل استمارة جمع بيانات مكونة من الأقسام التالية **القسم ١:** البيانات الشخصية والخصائص الاجتماعية، والديموغرافية، والطبية. **القسم ٢**: تقييم مخاطرمرض تصلب القلب والأوعية الدموية(ASCVD). **القسم٣**: تقييم عدوى البكتيريا الحلزونية بواسطة اختبار البراز. **النتائج**: كشفت الدراسة عن زيادة ذات دلالة إحصائية في الإصابة بالبكتيريا الحلزونية فمجموعة الحالات المرضية (٧٤٪) مقارنة بمرضى المجموعة الضابطة (٦٣٪).وقد كانت البكتيريا الحلزونية مرتبطة بشكل كبير بأمراض القلب والأوعية الدموية وظلت كذلك حتى بعد تعديل متعدد المتغيرات.كانت هناك فروق ذات دلالة إحصائية بين عدوى البكتيريا الحلزونية والجنس ومستوى التعليم والإقامة والطبقة الاجتماعية والتدخين. **الاستنتاج**: تعتبر عدوى البكتيريا الحلزونية واحدة من عوامل الخطر المحتملة للأمراض القلبية الوعائية بغض النظر عن مؤشر كتلة الجسم ، وخلل فى الدهون، والتدخين ،والسكر وضغط الدم. **التوصيات:** بناءً على نتائج هذا البحث، نوصي بفحص أمراض القلب والأوعية الدموية باستخدام أدوات مختلفة لتقييم أخطار الأمراض القلبية الوعائية لجميع المرضى القادمين للتقييم الطبي والذين تم تشخيص إصابتهم بعدوى البكتيريا الحلزونية.