Relation between Patients with Gastric Helicobacter Pylori Infection and Dyslipidemia

Edson Guzmán1,3,5,6*, Pedro Montes2,3,6, Eduardo Monge2,3,4

Abstract
Objective: To evaluate the relation between gastric H. pylori infection and dyslipidemia.

Methods: Eligible subjects were all adult; we enrolled those patients with dyspepsia who had undergone esophagogastroduodenoscopy. They were divided in 2 groups: Case group: patients who have a histologic diagnosis of infection for H. pylori. Control group: Patients who had previous negative biopsy for H. pylori. All patients had basic serology and lipid profiles, both groups were compared.

Results: 120 patients were evaluate, 63 were female (52.5%). Of 77 patients with dyslipidemia, 40 were positive H. pylori (51.9%), and 20 of 43 non – dyslipidemic patients were positive H. pylori (46.5%). Cholesterol values were 196.6 ± 42.1 and 191.7 ± 29.5; triglyceride 164.4 ± 89.1 and 139.2 ± 69.1; LDL 119.2 ± 33.8 and 115.9 ± 27.4; VLDL 33.1 ± 17.6 and 29.1 ± 15.6; HDL: 44.4 ± 9.1 and 42.5 ± 10.7 for the positive and negative H. pylori groups, respectively. “p” values in all cases were not statistically significant: cholesterol (p = 0.4), triglycerides (p = 0.08), HDL (p = 0.3), LDL (p = 0.5), VLDL (p = 0.1).

Conclusions: Gastric H. pylori infection does not have significant relation with the presence of dyslipidemia. The alterations of the serum lipids profiles (cholesterol, triglycerides, LDL and VLDL) are discreetly higher in the patients infected by H. pylori, but they are not statistically significant.

Key Words: Helicobacter pylori, Dyslipidemia, Relation

What is already known about this subject?
- Little is known about the relation between H. pylori and dyslipidemia.
- It is believed that there is a relation between these two entities but this belief is unsubstantiated

What does this study add?
This study finds there is little evidence that H. pylori infection likely contributes to an alteration in the metabolism of lipids.
have found a strong relationship between increasing levels of low-density lipoprotein (LDL) cholesterol or decreasing levels of high-density lipoprotein (HDL) cholesterol and increasing risk for coronary artery disease (CAD) events[6-8].

In recent years, the relation between *H. pylori* infections and coronary and cardiovascular diseases has been studied. However, most prospective studies have not confirmed the association between chronic infections and coronary artery disease. *H. pylori* and other gram negative microorganisms, have been involved in the etiology of pathologies like vascular coronary disease and atherosclerosis, with controversial results[9].

The objective of this study is to evaluate the relation between gastric *H. pylori* infection and dyslipidemia; given that proving a relation between *H. pylori* and cardiovascular risk factors might be an important finding to reduce the incidence of cardiovascular disease.

### Material and Methods

The subjects of this study were patients admitted to the Gastroenterology Unit of the Daniel Alcides Carrion Hospital in Callao, Perú, over a one-year period. Eligible subjects were all adults (18 years or older). We prospectively enrolled the patients with dyspepsia who had undergone esophagogastroduodenoscopy. All the patients underwent physical examinations. They were divided in 2 groups: The case group included patients who had a histologic diagnosis of gastric infection for *H. pylori*, in a previous biopsy taken in a upper endoscopy and who also took other tests: hemogram, liver function test and serum lipids, as a complementary study. The control group included patients with previous negative biopsy for *H. pylori* and who also took the aforementioned tests.

We evaluated the following as potential confounders: BMI, smoking behavior, recent alcohol use, aspirin or nonsteroidal anti-inflammatory (NSAID) drugs use (including over-the-counter use), and comorbidities. Also excluded from the study were patients whose diagnosis of infection for *H. pylori* was performed with another method (e.g. test of breath, serum test, etc.), and the subjects that did not have a complete serum profile or lack of any of the mentioned tests, as well as those with history of some type of chronic pathology as cardiovascular diseases, acute myocardial infarction (AMI), unstable angina, presence of some type of neoplastic disease or diabetes mellitus. Finally, we also excluded the patients that did not agree to voluntarily take part in the study under informed consent.

Biopsy specimens stained with HE and Giemsa were used to detect *H. pylori*. The histologic degree of activity (neutrophil infiltration), inflammation (mononuclear cell infiltration), glandular atrophy, and intestinal metaplasia were classified as none, mild, moderate, and severe in accordance with the updated Sydney System.

### Statistical Analysis

We performed the followed analyses: we compared the physician-assigned positive *H. pylori* group (case group) with negative *H. pylori* population (control group). We compared the demographic characteristics of every group as well as endoscopic and histological findings and serum lipids profiles.

Statistical analyses were performed using SPSS18 statistical analysis software. Unless otherwise noted, any test of a hypothesis was two-sided and the level of significance was set at 5%. Potential associations between the clinical and biologic parameters were tested for by univariate analysis using Student’s t test or chi-square test. The results were expressed as means±SDs.

### Results

In the present study, a total of 120 patients were evaluated; they were equally distributed in two groups: 60 patients in the case group with *H. pylori* infection and 60 patients in the group without infection (negative *H. pylori*). All the patients admitted into the present study were submitted to an upper endoscopy for the diagnosis of dyspepsia. None of the patients presented warning signs (e.g decrease weight, hemorrhage, or any other sign of organicity).

Of these 120 patients, 57 were male and 63 female, with mean age of 52.5 (range 18-82) years. The demographic characteristics were analyzed; of 63 female patients, 36 (57.1%) were positive *H. pylori*, and of 57 male patients, 24 (42.1%) were positive *H. pylori*, (p = 0.1; OR = 0.5; IC 95% (0.2 –1.1).

Of these 120 patients, 77 showed some type of dyslipidemia (64.1%). Of these 77 patients with dyslipidemia, 54.5% (42 patients) were female, whereas the remaining 45.5% (37 patients) were male. (OR = 0.7 IC 95% (0.3-1.6). Forty of the 77 were *H. pylori* (+) (51.9 %) and 37 *H. pylori* (-) (48.1%). Of the patients without dyslipidemia 20 were *H. pylori* (+) (46.5%) and 23 were *H. pylori* (-) (53.5%). (p = 0.5; OR = 1.2; IC = 95% (0.5 –2.6).

The weight ranges (kg) were 69.7 ± 14.4 and 67.8 ± 11.7 in the case group and the control group respectively (p = 0.4), whereas BMI (kg/m²) was 27.9 ± 4.8 and 26.3 ± 5.1 in each group respectively (p = 0.07). Demographics features are shown in Table 1.

### Lipids Profiles

Results of lipid profiles are shown in Table 2. The values of cholesterol (mg/dL) were discreetly higher in the *H. pylori* (+) group (196.6 ± 42.1 mg/dL) than *H. pylori* (-) group (191.7 ± 29.5 mg/dl) as well as in the values of triglycerides(mg/dL), 164.4 ± 89.1 vs. 139.2 ± 69.1; LDL (mg/dL), 119.2 ± 33.8 vs. 115.9 ± 27.4; and VLDL (mg/dL) 33.1 ± 17.6 vs. 29.1 ± 15.6. On the other hand, the values of HDL were in the (+) group (196.6 ± 42.1 mg/dl) than (-) group (191.7 ± 29.5 mg/dl) as well as in the values of triglycerides(mg/dL), 164.4 ± 89.1 vs. 139.2 ± 69.1; LDL (mg/dL), 119.2 ± 33.8 vs. 115.9 ± 27.4; and VLDL (mg/dL) 33.1 ± 17.6 vs. 29.1 ± 15.6. None of these values showed statistically significant differences.

### Table 1: Comparison of the demographics of the case and control group

<table>
<thead>
<tr>
<th></th>
<th>H. pylori (+) (n = 60)</th>
<th>H. pylori (-) (n = 60)</th>
<th>P</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex (Male / Female)</td>
<td>24 / 36</td>
<td>33 / 27</td>
<td>0.1</td>
<td>NS</td>
</tr>
<tr>
<td>Aged (years) ( ± SD)</td>
<td>50.1 ± 12.0</td>
<td>50.5 ± 14.6</td>
<td>NS</td>
<td></td>
</tr>
<tr>
<td>Hypertension (Yes /No)</td>
<td>4/56</td>
<td>6/54</td>
<td>0.5</td>
<td>NS</td>
</tr>
<tr>
<td>Smokers (Yes /No)</td>
<td>1/59</td>
<td>0/60</td>
<td>NS</td>
<td></td>
</tr>
<tr>
<td>IMC kg/m² ( ± SD)</td>
<td>67.9 ± 14.4</td>
<td>67.8 ± 11.7</td>
<td>0.4</td>
<td>NS</td>
</tr>
<tr>
<td>Dyslipidemia (Yes / No)</td>
<td>27.9 ± 4.8</td>
<td>26.3 ± 5.1</td>
<td>0.07</td>
<td>NS</td>
</tr>
</tbody>
</table>

www.ommegaonline.org
and vascular disease can be associated with atherosclerosis. Reports have suggested that the chronic infections for patients and between the two study groups (p = 0.1). Five cases of gastric ulcer were found in patients with dyslipidemia, whereas it was not present in non-dyslipidemic patients. Chronic and erosive gastritis was proportionally similar in each group. (See Table 3). Five cases of gastric ulcer were found in patients with dyslipidemia, whereas it was not present in non-dyslipidemic patients. Chronic and erosive gastritis was proportionally similar in each group. (See Table 3).

### Table 3: Endoscopical and histological findings between patients with and without dyslipidemia

<table>
<thead>
<tr>
<th>ENDOSCOPY FINDINGS</th>
<th>SYMPTOMS</th>
<th>NO SYMPTOMS</th>
<th>p</th>
<th>SIGNIFICANCE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cronic Gastritis</td>
<td>62</td>
<td>35</td>
<td>0.1</td>
<td>NS</td>
</tr>
<tr>
<td>Nodular Gastritis</td>
<td>2</td>
<td>0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Erosive Gastritis</td>
<td>8</td>
<td>8</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gastric Ulcer</td>
<td>5</td>
<td>0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>HISTOLOGICAL FINDINGS</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mild Gastritis</td>
<td>34</td>
<td>21</td>
<td>0.5</td>
<td>NS</td>
</tr>
<tr>
<td>Moderate Gastritis</td>
<td>41</td>
<td>22</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Severe Gastritis</td>
<td>2</td>
<td>0</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

### Histological Findings

The different histological findings and severity of gastritis were determined. (See Table 3). Most were moderate and severe gastritis in the H. pylori (+) group (70%), although in the H. pylori (-) group, mild gastritis was most frequent (61.6%). Only two patients with dyslipidemia showed severe gastritis; 55.8% of patients with dyslipidemia had moderate gastritis and 48.8% in this group showed mild gastritis (p = 0.5). The coronary risk was 4.7 ± 1.5 in the H. pylori (+) patients and 4.5 ± 1.2 in H. pylori (-) patients.

### Discussion

*Helicobacter pylori* infection is an etiological factor for the development of peptic ulcer and gastric cancer[1,2]. Preliminary reports have suggested that the chronic infections for *H. pylori* and other infections can be associated with atherosclerosis and vascular disease[1,3,4]. One hundred and twenty patients were investigated, which were distributed in two similar groups with the only difference being having gastric infection for *H. pylori* or not.

The demographic characteristics investigated were sex, age and the pathological precedents, all of which were similar in both groups of study. The distribution showed a light predominance in female patients in the positive *H. pylori* group (57.1%), whereas male patients showed higher frequency of being negative *H. pylori* (57.9%). In spite of this difference between both groups, no statistical significance was found.

The dyslipidemia includes a group of disorders in the metabolism of cholesterol and triglycerides. These disorders bring implications inside the cardiovascular system producing pathologies such as vascular coronary disease and atherosclerosis[5]. The alterations of the serum lipidic profile or dyslipidemias are classified principally under those who present hypercholesterolemia, hyperglycemia, a mixed form of both, or the decrease of the cholesterol HDL. In our study of 120 patients, 77 showed some degree of dyslipidemia (64.1%). The most important analysis of our study was the comparison of the risks of developing dyslipidemias among patients who are and are not infected by *H. pylori*.

Of these 77 patients with dyslipidemia, 40 were positive *H. pylori* (51.9%) and 37 were negative *H. pylori* (48.1%). In the group of the patients without dyslipidemia, 20 of 43 patients were positive *H. pylori* (46.5%) and 23 were negative *H. pylori* (-) (53.5%). This information indicates that a statistically significant difference between the studied groups does not exist, in reference to acquire dyslipidemia as a consequence of this infection. Analyzing separately both groups of lipidic affection, we observed that the risk of the patients with dyslipidemia is of 0.9.

In the study of Seung-Won Jin et al[6], held in 2007, that investigated the association between *H. pylori* infection and coronary artery disease, the authors observed that the infection for *H. pylori* was lower in the control patients that in the patients with coronary disease (30.7% and 40.6% respectively), without statistical differences. In our study we found that *H. pylori* gastric infection would not have a major implication in the risk of developing a coronary disease for alteration in the lipids.

When different components of serum lipidic profile were individually analyzed, we observed that patients with gastric infection for *H. pylori* showed a higher risk of hyperglycemia than with other types of dyslipidemia (p = 0.08), that hypercholesterolemia, HDL decrease, or increase of LDL and VLDL. Values of triglycerides in patient’s positive *H. pylori* were 164.4 ± 89.1 mg/dl and negative *H. pylori* were 139.2 ± 69.1 mg/dl. None of the different components of the lipidic profile showed a difference between the patients with positive *H. pylori* and negative *H. pylori*. Nevertheless, it is important to say that the patients *H. pylori* (+) showed higher hypercholesterolemia, hyperglycemia, values higher of cholesterol LDL and VLDL. Values of cholesterol were 196.6 ± 42.1 and 191.7 ± 29.5; triglycerides; 164.4 ± 89.1 and 139.2 ± 69.1; LDL: 119.2 ± 33.8 and 115.9 ± 27.4 and VLDL: 33.1 ± 17.6 and 29.1 ± 15.6; for the groups of positive *H. pylori* and negative *H. pylori* respectively.

Niemelä et al[7] in 1996, showed that 64% of the patients with arterial coronary disease and 53% of the control patients were positive for *H. pylori*. Among the control patients, those that were positive for *H. pylori* had higher concentrations of triglycerides with a statistically significant difference (p = 0.03) and in those who were negative for *H. pylori*, these concentrations were similar and without statistically significant dif-
An interesting piece of information to analyze is that, as it is well-known that HDL values lower than 40 mg/dl are a source of major coronary risk, we found in our study that HDL values are discreetly higher in patients positive for *H. pylori* than patients negative for *H. pylori* (44.4 ± 9.1 and 42.5 ± 10.7 respectively). Nevertheless, this is not a major source of coronary risk. This differs from the study of Yi SJ, in his study dyslipidemia and *H. pylori* in gastric xantomatosis[10]. In this study where prevalence of *H. pylori* infection was similar in the patients with xantomatosis and the controls, was observed that in the first group the level of cholesterol LDL was lower than in the control group (48.8 ± 12.3 vs. 62.9 ± 40.5; p = 0.028), and the values of cholesterol LDL were much higher in the patients with gastric xantomatosis than in the control group (112.9 ± 29.9 vs. 95.9 ± 22.4, p = 0.032). Values of cholesterol in this study did not show significant differences (188.7 ± 32.8 vs. 189.2 ± 30.9).

Niemiälä et al[10], concluded that *H. pylori* was an independent risk factor for arterial coronary disease and that *H. pylori* can modify and increase the serum concentrations of lipids and therefore increase the appearance of a coronary disease. In our study, we can affirm that a not significant increase exists statistically in the patients who are positive *H. pylori*.

According to the study conducted by Gulden et al[11], in 2009, the infection for *H. pylori* can stimulate the atherogénesis for indirect effects, such as a systemic inflammation or autoimmune reactions, due to the fact that the *H. pylori* is rapidly eliminated in the systemic traffic before reaching the wall of blood vessels[11-14].

We concluded that coronary risk is discreetly higher in the patients who present infection for *H. pylori*, nevertheless the difference is not statistically significant (p = 0.3).

Some characteristics of the patients can interfere in the correct interpretation of the relation that can exist between the gastric infection for *H. pylori* and the dyslipidemia, such as the coexistence of diabetes mellitus or a metabolic syndrome. This is the reason why these were excluded as criteria of incorporation in our study because it would influence the result.

Some other factors were studied that did not show implications in the results, for example: 8.3% of the patients (10 of the 120) had the diagnosis of arterial hypertension, the difference between both groups of study (positive and negative *H. pylori*) was not significant; 4 of 60 patients positive for *H. pylori* (6.6%) were hypertensive, whereas 6 of 60 patients negative for *H. pylori* (10 %) had this pathology. Only one of the patients in this study mentioned that he was a smoker; nevertheless this data point did not alter the results of our study. The BMI and the nutritional condition also did not show differences when the frequencies were compared between the groups of study (positive and negative *H. pylori*).

### Conclusion

In conclusion, the gastric *H. pylori* infection does not have a significant relation with the presence of dyslipidemia. The alterations of the serum lipids profiles (cholesterol, triglycerides, LDL and VLDL) are discreetly higher in the patients infected by *H. pylori*, but they are not statistically significant. Future studies are needed to evaluate and find a relation between *H. pylori* and coronary diseases.

### Disclosure Statement: The authors have nothing to disclose.

### References