The relationship between helicobacter pylori infection and myocardial infarction

Abstract

Background: Coronary Artery Disease is known as the main cause of death in industrialized countries. Relation between this disease and some infections such as Helicobacter pylori (H.pylori) has been shown in several studies. The purpose of this study was to demine the relationship between Hypylori and myocardial infarctions.

Methods: Seventy-three myocardial infarction patients and 78 individuals with no history of this disease were compared. Patients and control matched for age and sex person to person by the match method. Levels of serum IgA and IgG antibodies against H. pylori were measured by Elisa method. Also, cholesterol, triglyceride, LDL, HDL measured in both groups and data were compared between two groups in terms of relation with cardiac risk factors.

Results: From 151 participants, 73 were patients and 78 were control subjects. The percentage of IgG positive cases against H. pylori was 57.5% in the case group and 32.1% in the control group (p=0.002, OR: 2.87 CI: 95%; 1.5-5.6). Meanwhile, there was no significant difference in IgA positive cases between the two groups (42.5% and 48.7% in the case and control groups, respectively) (p=0.44; OR: 0.78 95% CI; 0.41-1.48). The study showed 74.2% of cases in the case group and 45.2% in the control group were positive for both IgG and IgA (p=0.01; OR: 3.5 95% CI; 1.3-9.5). No significant differences were found between two groups in terms of relation between H. pylori related antibodies level and heart disease classic risk factors (smoking, hypertension,...), sex, and age, but between dyslipidemia and H. pylori related antibodies was significant differences in case group (p=0.05).

Conclusion: According to the results, it seems there is a relation between H. pylori infection and myocardial infarction. Also, between dislipidemia and H. Pylori antibodies in case group were significant difference. Therefore, H. pylori can be a new risk factor for atherosclerosis or can be exacerbate effect of other risk factors. Proper diagnosis and treatment of these infections can be useful in prone patients. More studies with larger sample groups are needed to review the possible role of this pathogen as a heart disease risk factor.

Key words: Helicobacter pylori, IgG, IgA, Myocardial infarction.

Concomitant condition, like a genetic predisposition in increasing fibrinogen levels, seem to further increase the effect of H. pylori on myocardial infarction risk (4, 5). Helicobacter pylori causes one of the most common chronic bacterial infections. Serological evidences indicate that half of the adult population is infected (6).

This bacteria with organisms such as Chlamydia pneumoniae and viruses such as HSV1 and CMV in the pathogenesis sclerosis have been introduced with specific and nonspecific mechanisms such as increased clotting ability, increased production of adhesion molecules and CRP causes of this phenomenon represented (7). Mendall et al. suggested the relationship between Helicobacter pylori infection and coronary heart disease, for the first time (8). Later, many studies reported conflicting findings about the microorganisms involved in coronary heart disease (9-11).

However, if H. pylori is involved in causing atherosclerosis, treatment of gastrointestinal symptoms in atherosclerosis patients with H. pylori infection could have a preventive effect for myocardial infarction.

Due to high prevalence of arthrosclerosis and importance of considering heart disease risk factors, the current study set out to investigate the possible effect of H. pylori infection on developing acute MI.

Methods

In this case-control study, 78 individuals with no history of heart disease and 73 acute myocardial infarction patients (all between 30 and 80 years old) were compared in two separate groups. There was no significant difference in the sex ratio and mean age of participants in two groups (53 males and 20 females in the case group and 49 males and 29 females in the control group with the mean age of 59.8±11.5 and 56.4±13.9 years, respectively).

The case group admitted in CCU ward was selected by convenient non probability sampling method.

Patients had the history of admission to CCU with confirmed clinical symptoms of MI, positive ECG, and increased levels of cardiac enzymes. For each case among the control patients in surgery ward were selected, who were admitted for minor surgeries and had no history or positive physical examination or ECG results for cardiovascular diseases. Patients and control matched were in age and sex by the match method. History included factors such as heart disease, diabetes, hypertension, smoking, and hyperlipidemia.

Gastrointestinal drugs consumers excluded in both groups. Blood samples (5 cc) were obtained and sent to laboratory at -20 °C for H. pylori IgG and IgA was measured by ELISA method using Monobind kit. Also, we measured cholesterol, triglyceride, LDL, HDL in both groups.

Data were analyzed using Chi-square (X2), T-test and Odds Ratio estimation in SPSS -15 software.

Results

There were 53 males (72.6%) and 20 females (27.4%) in the case group while the control group was made up of 49 (62.8%) men and 29 (37.2%) women (p=0.1). The mean ages in the case group and the control group were 59.8±11.5 and 56.4±13.9 years, respectively (p=0.1).

Also, no significant differences were found between two groups in diabetes, hypertension, smoking, and dislipidemia.

57.5% (42 patient) of those in the case group and 32.1% (25 people) in the control group were IgG positive against H. pylori. The difference was statistically significant (p=0.002). Meanwhile, there was no significant difference between IgA levels of two groups (p=0.44) (Table 1).

Table 1: Comparison of relative frequency of positive IgG and IgA in both groups

<table>
<thead>
<tr>
<th></th>
<th>Negative N(%)</th>
<th>Positive N(%)</th>
<th>Total N(%)</th>
<th>OR (CI)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>IgG</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Case</td>
<td>31 (42.5)</td>
<td>42 (57.5)</td>
<td>73 (100)</td>
<td>2.87</td>
<td></td>
</tr>
<tr>
<td>Control</td>
<td>53 (67.9)</td>
<td>25 (32.1)</td>
<td>78 (100)</td>
<td>(95%; 1.5-5.6)</td>
<td>0.002</td>
</tr>
<tr>
<td><strong>IgA</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Case</td>
<td>42 (57.5)</td>
<td>31 (42.5)</td>
<td>73 (100)</td>
<td>0.78</td>
<td></td>
</tr>
<tr>
<td>Control</td>
<td>40 (51.3)</td>
<td>38 (48.7)</td>
<td>78 (100)</td>
<td>(95%; 0.44)</td>
<td>0.41-1.48</td>
</tr>
</tbody>
</table>

31 (42.5%) cases and 38 (48.7%) controls had positive IgA results against H. pylori. 23 (74.2%) cases and 19 (45.2%) controls were positive for both IgG and IgA with a statistically significant difference (p=0.01) (Table 2).
Table 2: Relative frequency of positive IgG and IgA in case group

<table>
<thead>
<tr>
<th>IgA</th>
<th>Negative</th>
<th>Positive</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N(%)</td>
<td>N (%)</td>
<td>N(%)</td>
</tr>
<tr>
<td>Positive</td>
<td>8 (25.8)</td>
<td>23 (74.2)</td>
<td>31 (100)</td>
</tr>
<tr>
<td>Negative</td>
<td>23 (54.8)</td>
<td>19 (45.2)</td>
<td>42 (100)</td>
</tr>
<tr>
<td>Total</td>
<td>31 (42.5)</td>
<td>42 (57.5)</td>
<td>73 (100)</td>
</tr>
</tbody>
</table>

χ² = 6.120 df= 1 p= 0.01 OR: 3.5 CI: 95%; 1.3-9.5

Except for dislipidemia in the case group, there was no meaningful relationship between H. pylori antibody levels and heart disease classic risk factors (p=0.05) (Table4).

Table 3: Comparison of relative frequency of IgA and dislipidemi in case group

<table>
<thead>
<tr>
<th>IgA</th>
<th>Negative</th>
<th>Positive</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N(%)</td>
<td>N (%)</td>
<td>N(%)</td>
</tr>
<tr>
<td>Dislipidemi</td>
<td>IgA</td>
<td>IgA</td>
<td>N(%)</td>
</tr>
<tr>
<td>Positive</td>
<td>31 (73.8)</td>
<td>16 (51.6)</td>
<td>47 (64.4)</td>
</tr>
<tr>
<td>Negative</td>
<td>11 (26.2)</td>
<td>15 (48.4)</td>
<td>26 (35.6)</td>
</tr>
<tr>
<td>Total</td>
<td>42 (100)</td>
<td>31 (100)</td>
<td>73 (100)</td>
</tr>
</tbody>
</table>

χ² = 3.832 df= 1 p= 0.05 OR: 0.38 CI: 95%; 0.14-1.01

No significant difference was seen between age, sex, smoking, hypertension, and H. pylori antibodies in any of the two groups.

Discussion

In this study, a significant difference in H. pylori IgG levels was shown between cases and controls (p=0.002) but the difference was not significant for IgA (p=0.44).

The case group had a statistically significant difference with the control group in terms of concurrent high levels of IgA and IgG (p=0.013). In different seroepidemiological studies, results have been conflicting (9-11). Some researchers have suggested a significant relationship between Helicobacter pylori and coronary heart disease (12-15). To justify the different results, some believe that the H. pylori CagA gene is effective in generating (16).

In some studies, H. pylori infection was mainly found in those of a lower socioeconomic status. Therefore, the connection between coronary disease and H. pylori infection could be due to close relationship of these two diseases on the social level (17). Cardiovascular risk factors compared with antibodies against H. pylori were statistically significant differences observed. Although hypertension, diabetes, smoking, and hyperlipidemia are concerned as major risk factors, about 20% of all cardiovascular events occur in the absence of such factors (18). H. pylori antibody levels could have a relationship with serum HDL levels. En-zhi-jin reported significant lower HDL levels in the H. pylori seropositive cases (19) the findings which were not observed in the current study. Chronic viral and bacterial infections, in addition to the ground as artherosclerosis, may also play a role in aggravation (20).

Chronic infections increase the production of various metabolites, such as inflammatory cytokines, that affect the blood flow in vessels and cause endothelial dysfunction and further shrinkage of small vessels (21-23). Elevated concentrations of cytokines in the gastric mucosa of H. pylori infected patients could increase serum fibrinogen and leukocytes. It seems that inflammatory response and related reactions in patients with H. pylori infection could justify accompaniment of this infection and acute coronary syndrome (24).

Because of the role of chronic inflammation in developing atherosclerosis, H. pylori infection can be considered a new risk factors. Also, these was a significant difference between dislipidemia and Helicobacter pylori antibodies in case group. Considering the above results, people with impaired fat are more likely to have conflict with Helicobacter. Therefore, proper diagnosis and treatment of these infections can be useful in patients prone to this most studies in this area with a larger volume are suggested.

Acknowledgements

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Conflict of interest: None declared.

References


