کارگاه‌های آموزشی مرکز اطلاعات علمی

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آموزش مهارت های کاربردی در تدوین و چاپ مقاله
**Helicobacter pylori** Infection and Atherosclerosis: a Systematic Review

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**Abstract** - *Helicobacter pylori* (*H. pylori*) is a spiral-shaped gram negative bacterium that naturally colonizes the human gastric epithelium. In recent years, large evidence has come to the literature strongly proposing causal link between *H. pylori* and extra gastric disorders. Cardiovascular system is one of the extra gastric organs that can be affected by *H. pylori* infection. The first evidence suggestive of such an association comes from seroepidemiological evaluations, but histopathological and eradication studies have strongly confirmed existence of a causal association between *H. pylori* infection and cardiovascular events.

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**Keywords**: *Helicobacter pylori*; Infection; Atherosclerosis; Systematic Review

**Introduction**

*Helicobacter pylori* (*H. pylori*) is a spiral-shaped gram negative bacterium that has been found to naturally colonize the human gastric epithelium and numerous reports support a causal relation between this infection and chronic gastritis, peptic ulceration, and gastric carcinoma (1,2).

In recent years, large evidence has come to the literature strongly proposing causal link between *H. pylori* and extra gastric disorders. There are evidences suggestive of a link between *H. pylori* and metabolic disorders including diabetes mellitus (3), neurological disorders, especially stroke events (4), psychiatric complications (5), gynecological disorders, from hyperemesis gravidarum (6) to pre-eclampsia (7) and infertility (8), ophthalmological diseases like glaucoma (9), dermatologic diseases including resistant chronic urticaria (10), alopecia areata (11) and Behçet’s disease (12), ear, nose, and throat (E.N.T.) diseases from benign disorders (13) to malignancies like laryngeal carcinoma (14) and lung cancer (15), hematologic disorders from iron deficiency anemia (16) to idiopathic thrombocytopenic purpura (ITP) (17), several disorders of hepatobiliary system (18,19) and cardiovascular diseases.

Overwhelming evidence suggests that chronic *H. pylori* infection plays a role in the initiation, progression and outcome of vascular diseases (20-22). Although treatment is not always definite (23), seroepidemiological and eradication studies have showed a causal association between *H. pylori* infection and cardiovascular events (24) and mortality (25), but histopathological evaluations of atherosclerotic vascular injuries more strongly confirmed such associations (22).

On the other hand, well-known risk factors of atherosclerosis formation including homocysteinemia (26) and hyperlipidemia (27) have also been shown to have associations with *H. pylori* infection. In this article, we aimed to review the existing literature on associations between *H. pylori* infection and cardiovascular disorders.

**Seroepidemiological evidence for the role of *H. pylori* infection in developing cardiovascular disease**

Several studies have investigated that whether serological evidence for *H. pylori* infection has any predictive value for the development of cardiovascular disorders. The first data on any association between these two conditions comes from seroepidemiological evaluations. Longo-Mbenza et al., (24) prospectively followed 205 individuals with cardiovascular risk factors and evaluated their *H. pylori* IgG. After 10 years of follow-up, *H. pylori* IgG positive patients were independently more likely to develop acute coronary syndromes including incident angina pectoris.
were not a predictor of cardiovascular disease. In dialysis population, Lentine et al., (43) found no significant association between H. pylori seropositivity and cardiovascular events.

To investigate the association between H. pylori infection and early stages of atherosclerosis, Saijo et al., (44) evaluated arterial stiffness defined by brachial-ankle pulse wave velocity (PWV) and correlated it to H. pylori seropositivity. Authors found that in Younger male subjects, H. pylori seropositivity (odds ratio (OR) 1.27 (95% confidence interval, 1.05-1.52) was significantly related to a high value of PWV, while this association was not found for older males or females. Consistent with this study, Ongey et al., (45) reporting from a population-based German study indicated that the sero-prevalence of H. pylori and their combination was not associated with the prevalence of cardiovascular diseases. It has also been suggested that H. pylori seropositivity is associated with enhanced platelet activation in patients with intermittent claudication (46). H. pylori stool antigen has also been related to the intensity of atherosclerotic disease in cardiovascular patients with more severe vasculopathy in the infected patients (Adiloglu, reff188). In a study on young women, Bloemenkamp, et al., (47) showed a significant correlation between H. pylori infection and peripheral arterial disease 1.6 (95% CI 1.1-2.2).

IgG antibodies against H. pylori has also been significantly associated with advanced atherosclerosis (≥2 vascular regions) measured by coronary angiography and/or carotid artery duplex sonography, in a study by Espinola-Klein et al., (48). Kahan et al., (49) in a case-control study of 200 subjects reported a significant association between H. pylori seropositivity and having history of acute myocardial infarction in univariate analysis. Multivariate analysis showed that this relation is independent of conventional risk factors (odds ratio 1.35 with 95% confidence interval 1.01-1.83, P=0.046). On the other hand, Jia et al., (50) provided controversial evidence suggesting no association between H. pylori seropositivity and cardiovascular diseases.

**Endothelial dysfunction as early stage of atherogenesis and H. pylori**

A study in children by Coskun et al., (51) showed that H. pylori IgG seropositivity has no relation to endothelial dysfunction. Khairy et al., (52) in a study on young men showed that anti-H. pylori seropositivity does not predict endothelial dysfunction. Similar findings have been reported on adult individuals as well.
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(53). Prasad et al., (54) although found a significant relation between H. pylori infection and endothelial dysfunction, in multivariate analysis, H. pylori infection has lost its significance. However, a study by Liuba et al., (55) based on a mouse model survey suggested that Co-infection with Chlamydia pneumoniae and Helicobacter pylori results in vascular endothelial dysfunction.

Antibodies to heat shock proteins (HSP)-60 and/or -65 are considered risk factors for atherosclerosis and they have been proposed as diagnostic markers for atherosclerosis and cardiovascular risk (56). Moreover, these antibodies have been reported to mediate endothelial toxicity (57).

Besides, H. pylori is supposed to induce its atherogenic effects through HSPs (58), and this risk enhancement is independent of other inflammatory factors (59). Serum concentrations of anti-(hsp) 60 auto-antibodies are reported to be significantly higher in patients with more severe cardiovascular disease, and less in those who undergone successful coronary artery bypass grafting (60). Although there are studies suggesting that anti-(hsp) 60 antibodies are higher in atherosclerotic patients, even when both case and control groups are anti-H. pylori antibody negative (61).

Effects of H. pylori Eradication and cardiovascular system

Knowing the association between H. pylori infection and atherosclerosis formation, it would be logical to think that treatment of H. pylori infection might be associated with an improvement in the cardiovascular risk. In fact, several studies have suggested such a relation. Wu et al., (62) in a rabbit model showed that the treatment of H. pylori infection decreases factors associated with atherosclerosis. Literature also suggests that eradication treatment of H. pylori favorably affects oxidative stress (63,64), myeloperoxidase activity (63) and fat mass (64), which are important biomarkers in pathogenesis of atherosclerosis.

Aydemir et al., (65) detected significant decrease in serum ADMA levels, another biomarker of atherosclerosis for patients in whom H. pylori has been eradicated. Kanbay et al., (66) treated 78 stool-H. pylori antigen positive patients and observed CRP levels decreased and HDL levels increased in the group that H. pylori had been eradicated, but no significant difference has been found in the patients in whom H. pylori had not been eradicated after treatment.

Eradication of H. pylori infection has also been suggested to improve blood pressure values in hypertensive patients (67). It has also been proposed that H. pylori eradication attenuate the reduction in coronary artery lumen after percutaneous transluminal coronary angioplasty while eradication therapy had no effect on serum lipid profile and homocysteinemia (68). Successful eradication of H. pylori has been reportedly associated with a significant improvement in endothelial dysfunction, and early stages of atherosclerosis formation (58, 69). Despite all the above-mentioned favorable effects reported to be associated with the eradication of H. pylori, there are also controversial reports suggesting no beneficial effect of H. pylori eradication on acute phase reactants (70).

Epidemiology of H. pylori DNA detection within atherosclerotic plaques

Although seroepidemiologic evidence of associations between H. pylori infection and atherosclerosis is overwhelming, they do not provide direct data for the existence of such a relationship. However, in more recent studies, with the detection of H. pylori within the coronary and carotid arterial walls using histopathological evaluations and polymerase chain reaction (PCR), very strong evidence came out corroborating initial data provided by seroepidemiological studies suggesting associations between H. pylori infection and atherosclerosis. In a previous study of ours, we found that a 29.5% of coronary atherosclerotic plaques achieved from patients undergoing coronary artery bypass grafting are colonized by H. pylori infection, detected through PCR tests (22). Jha et al., (71) also reported comparable rates of infection with 33.5% and 27.2% coronary & carotid arterial wall infection rates, respectively, confirmed by PCR test in their series.

Similar methodology in 18 Italian patients represented no individual positive PCR for H. pylori from carotid endarterectomy specimens (72). Hagiwara et al., (73) in his study on 50 carotid endarterectomy patients also found no H. pylori positive specimen. Comparable finding has been reported by Latsios et al., (74) in a study on 83 carotid atherosclerotic plaque specimens in which only 2 (2.4%) were positive for H. pylori. No plaque specimen in a study by Dore et al., (75) was PCR positive for H. pylori. Similar finding is reported by Kaklakkaya et al. in a Turkish study on aorta-iliac atherectomy specimens (76).

On the other hand, Kilic et al., (77) in a study from Turkey found H. pylori in 48.2% atherosclerotic and 19.6% non-atherosclerotic vascular wall specimens with a significantly higher H. pylori infection rate in
atherosclerotic specimens of coronary artery and abdominal aorta, but no significant difference in carotid artery. Putting together, we conclude that *H. pylori* institution within arterial walls of the coronary artery and aorta can serve as a predictor of atherosclerosis formation, but this association cannot be observed for carotid artery; although there are controversial reports as well. Arias et al., in a study from Argentina reported 83% *H. pylori* infection rate in carotid atherosclerotic plaques, but we should consider that the study was not comparative.

Moreover, Kaplan et al., (78) reported that 17.3% of carotid artery plaques were positive for *H. pylori* PCR while no positive specimen was found from healthy aorta specimens, but we should note that the control specimens were not achieved from the same artery (carotid). Moreover, one should not think that in all studies investigating *H. pylori* infection in coronary artery plaques the microorganism has been found. Sulewska et al., (79) in a study from Poland reported no positive case of *H. pylori* infection within coronary arterial walls of patients undergone CABG.

But Russu et al., (80) studying atherosclerotic plaques from different arteries reported that 22.2% of the specimens were positive for *H. pylori* PCR, but similar study in Turkey revealed 37% *H. pylori* positive rate (81). However, in aortic aneurysm plaque specimens, *H. pylori* was found in none of the 51 patients evaluated (82). An important issue in concluding all these controversial data is that PCR test is not performed perfectly all over the world, and several studies have reported low sensitivity and specificity of this test (83).

**Role of CagA, VacA**

The virulence of *H. pylori* infection can be a crucial determinant of its atherogenic ability. Vacuolating cytotoxin gene A is the most virulent *H. pylori* strain that has been associated with an increased local inflammatory response and also it can cause severe damage to the gastric epithelium (84). One of the proteins associated with VacA is the cytotoxin associated gene A (CagA) and seropositivity to CagA has been broadly used to detect infections with virulent *H. pylori* strains (85). In this section, we review studies investigating effects of *H. pylori* strains on three major arteries, separately:

**Coronary artery**

The severity of coronary atherosclerosis has also been associated with CagA positivity. Huang et al., (86) in a cross-sectional study of 159 coronary artery disease patients reported that the severity of the coronary atherosclerosis was significantly increased in CagA+ *H. pylori* group. Niccoli et al., (87) studied 40 coronary artery disease patients and compared their data with 20 normal controls. The anti-CagA antibody titer was not only significantly higher in patients with CAD compared to normal controls, but also a significant correlation was found between anti-CagA antibody titer and extent score (R=0.35).

Multivariate analysis showed an independent correlation between anti-CagA antibody titer and the extent of coronary atherosclerosis (OR 0.051). Acute coronary events have also been associated with seropositivity to CagA; Franceschi et al., (88) in a meta-analysis of 4241 cases reported that seropositivity to CagA was significantly associated with the occurrence of acute coronary events (OR 1.34; 95% CI 1.15-1.58). The same findings are reported by another meta-analysis with OR of 2.11 (95% CI 1.70, 2.62) (89). While Chimienti et al., (90) failed to detect any significant effect of Cag A positive *H. pylori* infection on the lipid profile of 211 healthy volunteer blood donors. Consistent with this study, Chmiela et al., (91) reported no difference in the prevalence of anti-CagA IgG in the coronary heart disease patients compared to controls. In a large cohort study, Kowalski reported that CagA IgG significantly was found more frequently in CAD group than in controls (85% vs. 36%; OR 2.5, 95% CI 1.1-5.6) (68).

Murray et al., (92) conducted a case-control study in 259 patients with myocardial infarction and the same population size of healthy controls and tested them for CagA positivity. Although in univariate analysis, CagA seropositivity was more frequently observed in cases than in control groups (OR 1.41; 95%CI 1.00, 1.99), multivariate analysis after adjustments for conventional risk factors and demographics eliminated the significance level (OR 1.16; 95%CI 0.79, 1.70). Infection with cagA positive *H. pylori* strains has surprisingly been inversely associated with cardiovascular mortality by Schöttker et al., in a cohort study (HR 0.62; CI 0.41-0.94) (21), and in a cross-sectional population based study of 1179 type 2 diabetic patients by Schimke et al., (93).

**Carotid artery**

In a study on 64 patients and 65 controls, CagA antibody titers were significantly higher in symptomatic patients with advanced carotid stenosis (OR 8.8; 95%CI 5.8-32.7) compared to either asymptomatic patients (4.7; 2.1-8.8) or the control
group (5.0; 2.2-7.9) (93).

Immunoreactivity between monoclonal CagA antibodies and antigens nested within the atherosclerotic specimens was significantly higher among symptomatic patients compared to asymptomatic patients (97.0 vs. 74.2%) (94). In a 3 years prospective cohort of 68 CagA positive stroke patients and 102 CagA negative patients, Kaplan-Meier survival analysis, CagA-positive patients showed a significantly higher risk for stroke recurrence than CagA-negative ones (45.6% vs. 17.6%; \( P < .001 \)). Difference in the rate of recurrent stroke between the two groups persisted after Cox regression analysis taking into account possible confounding factors (hazard ratio=3.5; 95% CI 1.9-6.4; \( P <0.001 \)) (95). Zhang et al., (89) in a meta-analysis of 26 studies reported that patients with anti-CagA positive strains of \( H. \) pylori infection had a trend of increasing the risk of ischemic strokes (OR 2.68, 95% CI 2.20, 3.27).

A prospective population-based study of 684 subjects revealed that either incident measures or changes (through 5 years) of carotid artery intima-media thickness were significantly higher in subjects seropositive to CagA than their counterparts infected with CagA-negative \( H. \) pylori strains. As well, there was a direct relation between anti-CagA antibody levels and both intima-media thickness and atherosclerosis risk, which was consistent to CRP levels. Pietroiusti et al., evaluated CagA seropositivity in 138 patients with large-vessel stroke (Group A), in 61 patients with cardiometabolic stroke (Group B), and in 151 healthy control subjects. They reported that after multivariate analysis, the prevalence of CagA-positive strains was higher in group A than in group B (OR 3.04, 95% CI 1.43 to 6.49) and higher in group A than in the control group (OR 4.3, 95% CI 2.12 to 8.64) (97).

In a population-based cross-sectional study of 983 normal individuals in the UK, Markus et al., (98) reported that amongst \( H. \) pylori seropositive individuals, those infected with the CagA strain were more likely to have enhanced carotid intima-media thickness after controlling for age and sex (OR 0.0256, 95%CI: 0.001-0.050), although further adjustments eliminated the significance level. Nevertheless, a recent meta-analysis suggested that the positive anti-Cag A IgG is predictive for ischemic stroke risk (OR 2.33 (95% CI: 1.76-3.09) (4).

**Aortic artery**

A prospective cohort study of 188 subjects by Shmuely et al., (99) revealed that CagA-positive \( H. \) pylori seropositivity remained an independent factor significantly associated with risk of developing aortic atheroma (OR 4.4; 95% CI, 1.4-14.7; \( P=0.01 \)). Nyberg et al., (100) in a case-control study of 119 patients with abdominal aortic aneurysm and 36 controls failed to show any connection between \( H. \) pylori CagA seropositivity and abdominal aortic aneurysm rupture. \( H. \) Pylori VacA has a more important role than CagA in the development of two aneurysms especially in ruptured ascending aortic aneurysm (101).

In this comprehensive review of the literature, we reviewed studies with controversial findings, so having a conclusion that satisfies all the reviewed manuscripts seems hard. However, based on the findings of the majority of the articles either on seropositivity to \( H. \) pylori infection or histopathological evidence to it, literature suggests that \( H. \) pylori infection can promote the process of atherosclerosis both in the general population, and in patients undergoing coronary intervention, and eradication therapy of the pathogen can attenuate the promotion of atherosclerosis in the latter patient population. Moreover, CagA seropositivity has been associated with atherosclerosis and vascular events both in the coronary and/or carotid arteries, although there are controversial data, as well.

On the other hand, surprisingly, CagA positivity was protective against cardiovascular mortality. In aortic lesions, CagA positivity was associated with higher atherosclerosis rate, but no association with aortic rupture has been reported. VacA has been reported to have a stronger association with aortic rupture than cagA strains. Moreover, \( H. \) pylori positivity has been associated with disturbance in lipid profile and elevated CRP - but not with homocysteine levels - as predictors of atherosclerosis formation and progression, nonetheless these findings are in most cases controversial.

Table 1 summarizes data of some of the major studies on this latter issue. Finally, we suggest prevention and treatment of \( H. \) pylori infection, especially CagA positive strain ones, in high-risk individuals to decrease the risk of cardiovascular diseases. Treatment strategies are most important in patients who undergo surgical interventions to repair the atherosclerotic artery.
Table 1. *H. pylori* and its effects on some conventional risk factors of atherosclerosis

<table>
<thead>
<tr>
<th>Risk factors</th>
<th>Findings [study reference]</th>
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<tr>
<td>HDL</td>
<td>- Serum HDL was significantly lower in the HP-seropositive group [24].</td>
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<td></td>
<td>- No association with <em>H. pylori</em> infection [102].</td>
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<td></td>
<td>- HDL was significantly lower in seropositive patients [50].</td>
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<td></td>
<td>- HDL was significantly lower in the atrophic gastric patients [31].</td>
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<td></td>
<td>- Significant increase after <em>H. pylori</em> eradication [66].</td>
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<td>- Lower mean value for <em>H. pylori</em>-seropositive group, after adjusting for age [104]</td>
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<td></td>
<td>- Significantly decreased levels in <em>H. pylori</em> seropositive patients [103].</td>
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<td></td>
<td>- No significant difference in males with positive IgG and IgA antibody titers for <em>H. pylori</em> [105]</td>
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<td></td>
<td>- In multivariate analysis, <em>H. pylori</em> infection was associated with high LDL cholesterol level (&gt;140 mg/dL) [OR 3.11; 95% CI 1.36-7.02] [102].</td>
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<td>- Higher in <em>H. pylori</em> positive patients [106].</td>
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<td>Total cholesterol</td>
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<td></td>
<td>- <em>H. pylori</em> specific IgG was positively correlated with triglyceride level [29]</td>
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<td>- Serum TG levels of <em>H. pylori</em> positive subjects were significantly higher than negatives [107]</td>
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<td>- No significant association with <em>H. pylori</em> positivity [108].</td>
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<td>- They also found that CagA positive patients had significantly higher levels of CRP [97]</td>
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<td></td>
<td>- No significant correlation was observed between CRP levels and HP-LgG level titers [36].</td>
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<td>- The hsCRP levels did not vary with <em>H. pylori</em> IgG status [43].</td>
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<tr>
<td>Apolipoprotein B</td>
<td>- Higher values after multivariate analysis for <em>H. pylori</em> seropositive group [104]</td>
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<td></td>
<td>- Higher but not statistically significant in <em>H. pylori</em> antibody positive cases [109]</td>
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</table>

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