J Investig Biochem. 2013;2(2):132-135 *ISSN: 2146-8338*

GESDAV

Journal of Investigational Biochemistry

available at www.scopemed.org

Original Research

Serum lipid profile in Helicobacter pylori infected patients

Saad Al-Fawaeir[1], Mohammad Abu Zaid[2], Ayman Abu Awad[2], Baker Alabedallat[2]

[1]*Department of Medical Biochemistry, Gulhane Military Medical Academy, Ankara, Turkey*
[2]*Department of Clinical Chemistry, King Hussein Medical Center, Amman, Jordan*

Received: May 06, 2013

Accepted: June 03, 2013

Published Online: June 10, 2013

DOI: 10.5455/jib.20130603084538

Corresponding Author:
Saad Abed Aljalil Al-fawaeir,
Gulhane School of Medicine, Department of Biochemistry, Ankara, TURKEY
alfaouri1@yahoo.com

Key words: Helicobacter pylori; lipid profile; atherosclerosis; immunoglobin G antibody.

Abstract

Background: *Helicobacter pylori (H. pylori)* is a controversial risk factor for atherosclerosis. An association between *H. pylori* infection and lipid concentrations has been reported.
Aim: the aim of this study was to investigate any possible association between *H. pylori* infection and serum lipid levels.
Design and methods: the study population consisted of 350 subjects. *H. pylori* infection status was determined by assaying serum anti-*H. Pylori* immunoglobin G antibody (IgG). Triglyceride, total cholesterol and HDL-cholesterol were measured for each subject; the data were compared among *H. pylori* infected subjects and healthy group.
Results: A total of 77% of the subjects were positive for *H. pylori* IgG antibodies and 23% were antibody-negative. The serum cholesterol concentrations were significantly higher in patients group when compared with healthy group (189.32±45.15 vs. 179.41±36.37 mg/dL ($p < 0.05$), serum triglyceride and total cholesterol/HDL-cholesterol concentrations also were significantly higher in patients group (169.46±68.53 vs. 135.67±94.35 mg/dL ($p < 0.05$) and 3.93±1.23 vs. 3.51±1.62, ($p < 0.05$) respectively).
Conclusion: Collective results support the hypothesis that *H. pylori* infection may modify lipid modulation which will increase the risk of atherosclerosis.

INTRODUCTION

Helicobacter pylori (H. pylori) is a Gram-negative , unipolar, multiflagellate microaerophilic, gently spiral bacterium, which commonly causes chronic infection of the gastric mucosa, *H. pylori* infection is the most chronic bacterial infection in the world, *H. pylori* is probably spread by consuming food or water contaminated with fecal matter. *H. pylori* causes changes to the stomach and duodenum. The bacteria infect the protective tissue that lines the stomach, this leads to the release of certain enzymes and toxins and activation of the immune system. Together these factors may directly or indirectly injure the cells of the stomach or duodenum, this leads to chronic inflammation of walls of stomach or duodenum. *H. pylori is* a causative agent for digestive disorders such as chronic gastritis, duodenal ulcer and stomach cancer, by turn this causing modification of lipid profile levels [1, 2].

Many studies have reported a positive association between *H. pylori* infection and the risk of cardiovascular disease, whereas the others have not confirmed these finding [3-6]. *H. pylori* induces a long standing low-grade persistent inflammation stimulus. Some studies have articulated that *H. pylori* infection can modify the serum lipid concentration [3] and being associated with an atherogenic lipid pattern [7-9] while in others this correlation has not found [10, 11].

H. pylori has also indirect effects as proinflammatory procoagulant, and atherogenic action; these can change risk factors (lipid profile, coagulation, levels of oxidative metabolites), production of cross reactive antibodies, malabsorption of nutrients and vitamins,

and metabolic factors such as overproduction of ammonia [9].

This study designed to investigate if there is any possible association *H. pylori* infection and modifications of serum lipid profile.

SUBJECTS AND METHODS

Subjects

The subjects were 350 (233 male, 117 female) individuals including 268 seropositive *H. pylori* and 82 healthy controls, the subjects of this study were selected from non-smoker patients attending to the outpatient specialty clinics in King Hussein Medical Center during the period from 15 September 2012 to 27 February 2013. The members of both groups were enrolled in the study voluntarily. After obtaining written informed consent for the study, blood samples were collected and centrifuged and biochemical tests done in the same day while serum was frozen at -20^{o} C and then used for serological tests. All tests were done in Princess Iman Center for Research and Laboratory Sciences in King Hussein Medical Center in Amman-Jordan. This study was supported by ethics community King Hussein Medical Center in Jordan.

Exclusion criteria

In the present study the following cases were excluded: history of indigestion, gastrointestinal disease, patients with familial hypercholesterolemia or hypertriglyceridemia, liver disease patients, cardiovascular disease, diabetes mellitus, acute infectious diseases, malignant tumors and smokers.

Measurements of serum lipids

Blood samples were taken after 14 h overnight fasting. The triglyceride, total cholesterol and high- density lipoprotein cholesterol concentrations (HDL-cholesterol) were measured using Hitachi 917 (Roche Diagnostics GmbH, Mannheim, Germany), levels of low-density lipoproteins cholesterol (LDL-cholesterol) were calculated by Friedwald formula.

Serological studies

Serum levels of anti-*H. pylori* IgG were measured by the use of commercial ELISA kit (DIA, PRO. Diagnostic Bioprobes, Via.G.Carduccu n 27, 20099 San Giovanni (Milano-Italy) based on a monoclonal antibody against 64-kD *H. pylori* antigen.

Statistical analysis

Results are reported as mean ± standard deviation (SD), All statistical analysis were performed using SPSS for windows 15.0 (SPSS Inc. Headquarters, Chicago, III., USA) software program and Microsoft Excel 2007 program. $P < 0.05$ was considered to be statistically significant.

RESULTS

The study group consisted of 350 subject (233 male, 117 female), aged between 35 and 57 years (48 ± 11 mean ± SD) , the subjects were divided into 268 (77 %) *H .pylori* seropositive and 82 (23%) *H. pylori* seronegative (Table 1). The H. *pylori* Seropositive subjects were older than the seronegative subjects 45 .9 ± 12.6 years versus 33.7 ± 13.7 years as mean ± SD ($p<0.001$), males were the predominant in seropositive subjects (64 %).

Subjects with *H. pylori* have significantly higher total cholesterol concentrations compared with control group (189.32±45.15 vs. 179.41±36.37 mg/dL, $p< 0.05$); also they have higher triglyceride concentrations (169.46±68.53 vs. 135.67±94.35 mg/dL, $p < 0.05$). The mean ratio of total cholesterol/HDL-cholesterol was significantly higher in the *H.pylori* seropositive than seronegative (3.93±1.23 vs. 3.51±1.62, $p< 0.05$). LDL-cholesterol concentration were lower in healthy groups (117.14±53.19 vs. 105.86±29.73 mg/dL, $p = 0.125$) (Table 2).

Table 1. Characteristics in both groups

	***H. pylori* seropositive**	***H. pylori* seronegative**	***P*-value**
n	268	82	
Age (mean ± SD)	45.9±12.6	33.7±13.7	0.04
Male	96	49	0.013
Female	172	33	0.023

Table 2. Serum lipid concentrations in patients group and healthy group, the results given as mean±SD.

Factor	unit	*H. pylori* seropositive	*H. pylori* seronegative	*P*-value
Total Cholesterol (TC)	mg/dL	189.32±45.15	179.41±36.37	0.02
Triglyceride	mg/dL	169.46±68.53	135.67±94.35	0.014
HDL-C	mg/dL	48.14±13.97	51.72±17.57	0.159
LDL-C	mg/dL	117.14±53.19	105.86±29.73	0.125
TC/HDL-C	mg/dL	3.93±1.23	3.51±1.62	0.02
LDL-C/HDL-C	mg/dL	2.21±1.14	1.37±0.98	0.038

DISCUSSION

In this study we found that *H. pylori* seropositive subjects have higher serum triglyceride, total cholesterol and LDL-C concentrations when compared to healthy subjects. Earlier studies suggested that there is a significant association between *H. pylori* infection and cardiovascular diseases, this association due to the effect of the infection on lipid metabolism [3,4,12]. A study suggests that the association might be indirect and related to the social status [13].The social background of the population in the present study is very homogenous; therefore the effects of social status are not possible. Smoker persons also excluded in order to avoid any probable effects since smoking seems to be effect the lipid metabolism [14, 15].

With reference to serum HDL-C levels in patient group present study showed a decrease in the HDL-C level when compared to healthy group. Reciprocally a statistically significant increase in LDL-C levels in the patients with *H. pylori* was shown. Several studies by Pilotto and Malfertheiner [16], Yudkin [17] on patients with gastritis and cardiovascular diseases showed that there is a significant decrease in HDL-C level. Furthermore, in Laurila study [18] on patients with *H. pylori* a significant decrease in HDL-C level was reported. Studies of Scragg et al. [19] and Kowalski [20], on the relationship between *H. pylori* and cardiovascular diseases showed that LDL-C concentration is increased by *H. pylori* infection. Findings in all these studies are agreed with that in present study.

The serum triglyceride level and the level of serum total cholesterol were found significantly higher in patients group. Although the levels of serum total cholesterol and LDL-C are higher in patients group but not statistically significant. Our results suggest a negative and not statistically significant effect of *H. pylori* positivity on plasma HDL-C concentration. The results in our study are similar to studies results from other countries [4, 9, 21].

In the present study we found that TC/HDL-C and LDL-C/HDL-C ratio was significantly higher in patients subjects compared to control group. TC/HDL-C ratio and LDL-C/HDL-C ratio as known that they are risk factors for developing cardiovascular diseases. Concerning these ratios, our finding showed a statistically significant difference between the patients groups compared to control group. Hoffmesister et al. and Arrabi et al. results confirmed our results [7, 9].

CONCLUSION

On consideration of our study results it is assume that there is an association between *H. pylori* infection with various cardiovascular risk factors, exceptionally increased level of TC, triglyceride and LDL-C, and decreased levels in HDL-C. However, maybe other cofactors are involved in the lipid modulation along with the strain of *H. pylori* including host genetic and environment factors. *H. pylori* infection can cause lipid metabolism disorders leading to increase the risk factors of cardiovascular diseases.

REFERENCES

1. Megraud F. Epidemiology of Helicobacter pylori infection. Gastroenterology Clin North Am. 1993;22:73-88.
2. Antherton JC. The pathogenesis of Helicobacter pylori induced gastro-duodenal diseases. Annu Rev Pathol. 2006;1:63-96.
3. Patel P, Mendall MA, Carrington D, Strachan DP, Leatham E, Molineaux N, Levy J, Blakeston C, Seymour CA, Camm AJ, et al. Association of Helicobacter pylori and Chlamydia pneumoniae infections with coronary heart disease and cardiovascular risk factors. BMJ. 1995;311:711-4.
4. Niemela S, Kartunen T, Korhnonen T, Laaea E, Karttunen R, Ikaheimo M. Could Helicobacter pylori infection increase the risk of coronary heart disease by modifying serum lipid concentration? Heart. 1996;75:573-5.
5. Whincup P, Mendall MA, Perry IJ, Walker M. Prospective relation between Helicobacter pylori

infection, coronary heart disease, and stroke in middle aged men. Heart. 1996;75:568-72.

6. Regnström J, Jovinge S, Båvenholm P, Ericsson CG, De Faire U, Hamsten A, Hellenius ML, Nilsson J, Tornvall P. Helicobacter pylori seropositivity is not associated with inflammatory parameters, lipid concentrations and degree of coronary artery disease. J Intern Med. 1998;243:109-13.
7. Hoffmeister A, Rothenbacher D, Bode G, Persson K, März W, Nauck MA, Brenner H, Hombach V, Koenig W. Current infection with Helicobacter pylori, but not seropositivity to Chlamydia pneumoniae or cytomegalovirus, is associated with an atherogenic, modified lipid profile. Arterioscler Thromb Vasc Biol. 2001;21:427-32.
8. Takashima T, Adachi K, Kawamura A, Yuki M, Fujishiro H, Rumi MA, Ishihara S, Watanabe M, Kinoshita Y. Cardiovascular risk factors in subjects with Helicobacter pylori infection. Helicobacter. 2002;7:86-90.
9. Aarabi MH, Alvani S, Ehteram H. Lipid profile in subjects with *Helicobacter pylori* Infection. Iranian J of Pathol. 2010;5:199-203.
10. Zhu J, Quyyumi AA, Muhlestein JB, Nieto FJ, Horne BD, Zalles-Ganley A, Anderson JL, Epstein SE. Lack of association of Helicobacter pylori infection with coronary artery disease and frequency of acute myocardial infarction or death. Am J Cardiol. 2002;89:155-8.
11. Volanen I, Raitakari OT, Vainionpää R, Arffman M, Aarnisalo J, Anglé S, Kallio K, Simell O. Serum lipid profiles poorly correlate with Chlamydia pneumoniae, Helicobacter pylori, and cytomegalovirus seropositivity in prospectively followed-up healthy children. Arterioscler Thromb Vasc Biol. 2005;25:827-32.
12. Murray LJ, Bamford KB, O'Reilly DP, McCrum EE, Evans AE. Helicobacter pylori infection: relation with cardiovascular risk factors, ischaemic heart disease, and social class. Br Heart J. 1995;74:497-501.
13. Wald NJ, Law MR, Morris JK, Bagnall AM. Helicobacter pylori infection and mortality from ischaemic heart disease: negative result from a large, prospective study. BMJ. 1997;315:1199-201.
14. Venkatesan A, Hemalatha A, Bobby Z, Selvaraj N, Sathiyapriya V. Effect of smoking on lipid profile and lipid peroxidation in normal subjects. Indian J Physiol Pharmacol. 2006;50:273-8.
15. Jeeyar, Hemalatha, Wilma Delphine Siliva CR. Evaluation of effect of smoking and hypertension on serum lipid profile and oxidative stress. Asian Pacific J of Tropical Disease. 2011:289-291.
16. Pilotto A, Malfertheiner P. Review article: an approach to Helicobacter pylori infection in the elderly. Aliment Pharmacol Ther. 2002;16:683-91.
17. Yudkin JS. Lipids, thrombosis and cardiovascular disease in diabetes. Proc Nutr Soc. 1997;56:273-80.
18. Laurila A, Bloigu A, Näyhä S, Hassi J, Leinonen M, Saikku P. Association of Helicobacter pylori infection with elevated serum lipids. Atherosclerosis. 1999;142:207-10.
19. Scragg RK, Fraser A, Metcalf PA. Helicobacter pylori seropositivity and cardiovascular risk factors in a multicultural workforce. J Epidemiol Community Health. 1996;50:578-9.
20. Kowalski M. Helicobacter pylori (H. pylori) infection in coronary artery disease: influence of H. pylori eradication on coronary artery lumen after percutaneous transluminal coronary angioplasty. The detection of H. pylori specific DNA in human coronary atherosclerotic plaque. J Physiol Pharmacol. 2001;52:3-31.
21. Adachi K, Arima N, Takashima T, Miyaoka Y, Yuki M, Ono M, Komazawa Y, Kawamura A, Fujishiro H, Ishihara S, Kinoshita Y. Pulse-wave velocity and cardiovascular risk factors in subjects with Helicobacter pylori infection. J Gastroenterol Hepatol. 2003;18:771-7.