Is there an association between helicobacter pylori infection and dyslipidemia?

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Abstract:

Various studies suggest that H. pylori infection is associated with atherosclerosis, but its mechanism is still unclear. This study was conducted to investigate the association between Helicobacter Pylori (H. pylori) infection and the lipid profile among elderly Afghani population.

Patients and methods: the study was conducted on a total of 300 subjects (mean age 61.2± 7.6yr) from January 2018 up to March 2019. Serum anti-Helicobacter pylori antibody titer and serum lipid profile were assessed in the study population, data were statistically analyzed by SPSS version 16.P value < 0.05 were considered significant.

Results in the current study, 62% of the case were serologically positive for H. pylori. among male cases, the level of low density lipoprotein (LDL) was higher in H. pylori positive patients in comparison with H. pylori negative patients (p = 0.03); although level of triglyceride (TG) was higher and the level of high density lipoprotein (HDL) was lower in H. pylori positive patients; but was not statistically significant difference among TG, HDL in H Pylori positive and negative cases. Among female cases, the negatives (P = 0.001); but here was no significant difference between H. pylori positive and negative cases regarding the level of LDL and HDL.

Conclusion; The results of this study shows that the LDL level was high among male H. pylori positive cases and TG level was low in H. pylori positive female cases.

Key words: Helicobacter pylori, LDL, HDL, TG, Cholesterol.

Introduction:

Coronary artery diseases are the leading cause of death worldwide. Atherosclerosis causes artery obstruction and thus limits blood flow to the related are of the heart. Every factor that contributes in athrerogenic, also causes coronary artery diseases. Hypertension, diabetes, smoking, dyslipidemia and others causes atherogenicity. In this study we try to find an association between H. pylori infection and dyslipidemia [1] However, the results of several other studies failed to confirm the association [2].

A large amount of epidemiologic and clinical data regarding associations with non-gastric systemic diseases, including cardiovascular diseases and their risk factors, and H. pylori infection have been reported [3-4] . A number of epidemiologic studies report a significant correlation of cardiovascular disease or tis risk factors with H. pylori infection [5-6].

Elevated serum level of low density lipoprotein (LDL) and low levels of high density lipoprotein (HDL) are the major risk factors of atherosclerosis [7].

H. pylori causes' chronic inflammation of the gastric mucosa and thus various studies suggested that mild systemic inflammation induced by H. pylori is associated with atherogenesis [8-9]. But in various studies this fact still remained unproved [10-11].

Also, Byrne et al. showed that H. pylori binds to von Will brand factor (vWF), which causes platelet aggregation, and thus leads to the formation of atherosclerotic plaque[12].

Thus, in this study we tried the effect of H. pylori infection on serum lipid profile, which is a risk factor for coronary artery diseases.

Material and method:

The current study was conducted on a total of 300 volunteer afghainanss from January 2018 up to march 2019. The age was in rage of 18 – 68 years. Including criterias: 1. Patients aged more than 18 years are included. 2. Patients that were not on H. pylori eradication therapy. Excluding criterias: 1. pregnant women's are excluded. 2. Patients that were already on lipid lowering drugs were excluded.patientd had their triglyceride of more than 400mg/dl were also excluded from this study. Finally the results of total 300 cases were analyzed. In current study a written consent were obtained from all cases. The case group was H. pylori positive and control group was H. pylori negative.

Data collection: in current study a questionnaire was used which included on medical history, age, sex, smoking habits, marital status, physical activity and education. Blood samples were obtained in the morning from antecubital vein while the patient was on fasting. The blood were then centrifuged and serum were isolated.

HDL-cholesterol was measured by a commercial kit including magnesium chloride and phosphotungstate, after sedimentation of lipoproteins including apo B. TG was measured by an enzymatic method. LDL – cholesterol profile was calculated based on friedewald formula [13, 14].

Friedewald formulas are as following: total cholesterol = HDL –c + VLDL + LDL-c

VLDL-c = TG/5 mg/dl, LDL = total cholesterol – HDL-c-TG/5 mg/dl.

Serum H. pylori IgG titer was measured by an enzyme – linked immune sorbent assay (ELISA) kit (hilgen co.,); IgG titers $\geq$ 22U/ml were reported as positive. Statistical analysis was performed by SPSS version 16; P values < 0.05 were considered significant.

Results:

The current study was conducted on 300 cases from 2018 to 2019. Among the cases, 185 were male and 115 female.

Demographic and clinical data of the cases are shown in table 1.

Table 1.

Variables	Male	Female	P value
Age (year)	41.5$\pm$2	44$\pm$3	0.002

Triglyceride (mg/dl)	163±2	134±5	0.001
Cholesterol (mg/dl)	198±6	193±5	0.9
LDL (mg/dl)	114±7	115±4	0.89
HDL (mg/dl)	37.8±3	44.5±6	0.001
H. pylori (+)	70	63	0.8

LDL, low density lipoprotein; HDL, high density lipoprotein; TG, triglyceride;

According to table 2, the level of LDL was significantly higher among the cases with H. pylori infection, compared to that of the ones without the infection (p value = 0.03); although higher levels of TG and lower levels of HDL were reported among H. pylori seropositive cases, there was no significant difference between the infected and non-infected cases regarding the level of TG and HDL (table 2).

Table 2: results of Helicobacter pylori seropositive and seronegative, Male cases.

Variables	H. pylori seropositive	H. pylori seronegative	p. value
Age (year)	46±2	42.5±4	0.001
Triglyceride (mg/dl)	173±8	165±3	0.7
Cholesterol(mg/dl)	195±11	185±6	0.07
LDL (mg/dl)	110±7	107±7	0.03
HDL(mg/dl)	38.8±3	37.2±4	0.7

LDL, low density lipoprotein; HDL, High density lipoprotein; TG, triglyceride;

Table 3: results of helicobacter pylori in seropositive and seronegative female cases

Variables	H. pylori seropositive	H. pylori seronegative	p. value
Age (year)	48.5±.2	40±3	0.001
Triglyceride (mg/dl)	127±2	155±10	0.001
Cholesterol(mg/dl)	187±12	186±6	0.001
LDL (mg/dl)	110±7	115±8	0.06
HDL(mg/dl)	44.8±3	43.5±3	0.15

LDL, low density lipoprotein; HDL, High density lipoprotein; TG, triglyceride;

According to table 3, in female cases, the level of TG was significantly lower among cases with H. pylori infection, compared with the ones without such infection (p value = 0.001); but the difference between H. pylori seropositive and seronegative cases, regarding the level of LDL and HDL, was insignificant (Table 3).

Discussion:

Helicobacter pylori infection is the most common bacterial infection worldwide, especially in the developing countries; its prevalence varies in different countries, 30% in the developed countries versus 80% in the developing countries [15]. The prevalence of H. pylori among the current study population was 62%.

Helicobacter pylori infection may develop many extra intestinal complications, during the recent years, many studies were conducted on the relationship between H. pylori infection and atherosclerotic diseases such as ischemic Heart disease [3].

Helicobacter pylori may play a role in the development of ischemic heart disease through different methods such as colonization of endothelial cells, changes in lipid profile, hypercoagulation, platelet aggregation, induction of molecular mimicry mechanism,, and progression of low-grade systemic inflammation [16].

The current study aimed at evaluating the relationship between H. pylori serological status and serum lipid profile. According to the results of the current study, 62% of total population was H. pylori seropositive; the prevalence of infection among male and female cases was 70% and 63%, respectively; it was 46, 8% and 39.6% in a similar study [18].

According to the results of the current study,, in male cases, H. pylori seropositivity resulted in significant increase of serum LDL (p.value = 0.03); although higher levels of TG and lower levels of HDL were observed among H. pylori seropositive cases, there was no significant difference between seropositive and seronegative cases regarding the level of HDL and TG. Results of a study conducted in Japan indicated that H. pylori infection in Japanese patients indirectly caused changes in serum lipid profile including an increase in LDL – cholesterol and a decrease in HDL-cholesterol [18].

Also, previously performed studies showed that H. pylori infection is associated with lower levels of HDL-cholesterol in Europeans living in the USA [17].

Results of a large epidemiological survey by Laurilaet et al. showed that the level of TG and total cholesterol was significantly higher and the level of HDL – cholesterol was significantly lower in male patients with H. pylori infection, compared with the ones without such infection [10]. In a similar study conducted in South Korea, the relationship between H. pylori infection and higher levels of LDL – cholesterol was reported [19].

According to the results of the current study, the level of TG was significantly lower in female cases with H. pylori infection than the female cases without the infection (P.value = 0.001); however, the difference between H. pylori seropositive and seronegative female cases regarding the level of LDL and HDL was insignificant; the result were consistent with those of a study in Japan [18].

Results of a study showed that in patients with frequent infection with H. pylori, the level of LDL and HDL increase and decreases from the base level, respectively, compared to those of the healthy case [20]

however, result of the current study and those of some other similar studies to some extent indicated the effect of H. pylori on lipid profile in patient with H. pylori infection, although result of the current study were not to the extent of justifying the atherogenic effect of H. pylori ant it seem that the bacteria induce their possible atherogenic effect through other mechanism.

however, the mechanism in which accordingly H. pylori infection cause the increase in lipid profile is not identified completely . Results of an in vitro study (1992) showed that H. pylori can increase the absorption of cholesterol from serum and egg yolk; hence, it can be concluded that cholesterol binding to H. pylori can reduce absorption of dietary cholesterol [21].

Result of an experimental study showed that interleukin - (IL-8) i produced in the H. pylori infected mucosa more than normal range. production of IL-8 in H. pylori infection results in the stimulation of mucosa by oxidized LDL and monocyte , and then increase the immigration of T lymphocyte to smooth muscle cell, and consequently lead to the production

of plaque thrombosis [22].

Interleukin -10 (IL-10) is produced by mononuclear cell after the incidence of inflammation. HDL - cholesterol can regulate the production of cytokine by itself. Inflammation, by changing the level of HDL- cholesterol, stimulates the production of IL-10 by circulating mononuclear cells [23].

Some studies showed the positive effect of H. pylori eradication therapy on lipid profile; for example, successful eradication of H. pylori can reduce the risk of high LDL and low HDL cholesterol [20].

The H. pylori eradication therapy increases apo A and HDL - cholesterol, while total cholesterol and LDL are not changed [23].

briefly, at least some patient with H. pylori infection showed permanent or long term complication of atherogenic lipid profile, which can accelerate the incidence of atherogenesis and many other complicated clinical diseases, such a coronary heart disease , brain stroke, and peripheral artery occlusive diseases.

Conclusion

according to the result of the current study, H. pylori can increase the level of LDL in seropositive male patients; the bacteria also play a role in the incidence of coronary artery disease through affecting atherogenic lipid profile; but in seropositive female patient, considering the lower level of TG, it seems that the atherogenic effect of H. pylori mostly affect the level of other factors like blood sugar etc. and thus play role in atherogenicity.

Suggestions and recommendations

1: we advise lipid profile for H pylori positive patients

2: People should take care of their hygiene for prevention of H pylori

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