Full Length Research Paper

Effect of *Helicobacter pylori* infection on the lipid, lipoproteins, apolipoprotein-A1, Lipoprotein (a) and Apolipoprotein-B in patients with gastritis

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There are evidences of relationship between chronic infections of *Helicobacter pylori* (*H. pylori*) and gastritis in some previous studies. The gastritis caused by *H. pylori* leads to a change in serum levels of lipids, lipoproteins, apolipoprotein-A1 (Apo-A1), apolipoprotein- B (Apo-B) and lipoprotein- a (Lp(a)) . These changes will lead to thrombogenesis and other cardiovascular diseases. The aim of this study is to find out a correlation between lipids profiles and helicobacter gastritis. In this study 20 males and 20 females in age between 30 - 50 years old with gastritis resulting from *H. pylori* were chosen as patients group. Patients serum levels of Apo- A1, Apo -B and Lp(a) as well as total cholesterol, triglyceride, High density lipoprotein (HDL -C) and Low density lipoprotein (LDL-C) were measured. As control, 26 persons with other types of gastritis (helicobacter negative), without having diseases such as: diabetes, hyperlipidemia, renal failure, liver diseases, with similar age ranges were chosen. Mean levels of total Cholesterol, triglyceride, LDL-C, ApoB lipoprotein and Lp (a), as well as total Cholesterol/HDL ratio and LDL-C/HDL-C ratio in patients with gastritis resulted by *H pylori* were significantly increased compared to control groups. In addition HDL-C and Apo- A1 lipoprotein were decreased significantly. Therefore it was possible to conclude that *H. pylori* infection can cause lipid metabolism disorders that may act as risk factors for cardiovascular diseases.

Key words: Gastritis, *H. Pylori*, lipids, lipoprotein, Apo lipoprotein, Lp (a).

INTRODUCTION

H. pylori are a gram-negative, unipolar, multiflagellate, microaerophilic, gently spiral or curved bacilli. *H. pylori* infection is an important cause of peptic ulcer disease and other gastrointestinal disorders (Khalil, 2004). Gastritis is a nearly high incident disease in whole world and almost 10% of people of the world are suffering from it. Its incidence is a little more in males than females (Yamagata et al., 2000; Kato et al., 2004; Parlak et al., 2001; Pérez-Aisa et al., 2005). There are several hypotheses to des-

cribe the reasons of duodenum ulcers (Axon, 2007) . The idea of correlation between *H. pylori* infection and atherosclerosis come by Mendall (Mendall, 1998). *H. pylori* is a cause of duodenum ulcer (Wang et al., 2005; Loffeld et al., 2002; Leung et al., 2005; Apostolov et al., 2005) and correlate also with chronic gastritis (Kawai et al., 2006) . *H. pylori* are the most important etiologic factor of peptic ulcer disease (Yi, 2007). Chronic gastritis is thought to be involved in the gastric glandular atrophy, which is considered as a precursor of gastric cancer (Chen et al., 2007).

The prevalence of *H. pylori* infection increases with age world-wide (Jin et al., 2007). The prevalence of *H. pylori* among adult population in the Middle East was estimated

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Table 1. Lipid and Lipoproteins means and P-values in the patients with gastritis resulted by *H. pylori* and the patients with gastritis that is not resulted by *H. pylori* but other causes as control.

Factor	Unit	Patients	Controls	P-Value
Total Cholesterol	mg/dl	143.1	115.61	0.004
Triglyceride	mg/dl	135.97	92.23	0.013
HDL-C	mg/dl	44.67	59.11	0
LDL-C	mg/dl	136.45	66.38	0
VLDL-C	mg/dl	32.22	18.26	0.048
Total Cholesterol/HDL-C	----	3.23	2.06	0
LDL-C/HDL-C	----	3.06	1.13	0

to be in the range of 70 to 90% (Sung et al., 2005). Various studies showed that the *H. pylori* presence in digestive system ulcers leads to changes in lipid indices included cholesterol, triglyceride and lipoproteins HDL-C and LDL-C (I can et al., 1998). As substances like Apo-A1 and Apo-B are the fundamental structures of plasma lipoproteins, there must be a relationship between changes of their levels in cardiac diseases and the increase of plasma lipids indices by *H. pylori*.

An increase in Apo-B level will lead to an increase in the risk of cardiovascular disorders (Chimienti et al., 2003). Lipoprotein-A is present only in human and has many properties in common with low density lipoprotein. The mechanisms of metabolism and actions as well as atherogenic specifications of Lp (a) is not known clearly (Berglund and Ramakrishnan, 2004). Its serum levels will be increased by an increase in atherosclerosis risk and cardiovascular disorders (von Eckardstein et al., 2001; Kostner et al., 1999; Sharrett et al., 2001; Jin et al., 2007) as well as in disorders of other veins included brain vessels (Rifai et al., 2004). Nowadays the Lp (a) is considered as an independent risk factor of cardiovascular disorders (Gillum, 2004; Romanic et al., 1998) and its dangerous effects will be increased significantly in the patients with high plasma concentrations of LDL-C (Maher et al., 1995; Suk et al., 2006). Therefore the purpose of this study is further investigation for finding the relationship between *H. pylori* infection and serum levels of lipids, lipoproteins, Apo-A1, Apo-B and Lp (a).

MATERIALS AND METHODS

Serum from 66 patient ages between 30 - 50 years old with gastritis were separated and frozen at -20 till assay day. For all the patients gastric endoscopic procedures were carried out and biopsy performed. On urease positive samples (Berry et al., 2006), PCR test were carried out (Cinnagen, Tehran Iran). In addition ELISA tests on *H. pylori* immunoglobulins IgG, IgA and IgM by IBL (Hamburg, Germany) kits were performed. Using these criteria 40 people were selected as patient groups and those who were negative in urease test and PCR method, cathagorized as control group (26 people). These samples were validated twice.

To minimize officious factors, we oust patients with pre-history of specific disorders like diabetes, renal failures, hepatic diseases, thyroid abnormalities, as well as smokers, alcoholics and people with low activities. Total cholesterol, triglyceride, HDL-C and LDL-C were measured by direct enzymatic assay, using kits made by BioSystems company (BioSystems, Barcelona, Spain). Apo-A1 and Apo-B assay were carried out by nephlometric method using kit by the company of the Binding Site (Birmingham, Uk). Lp (a) level was measured by an ELISA method (Progan Biotechnic GmbH). Collected data were analyzed using t-test method for consumptive analysis.

RESULTS

In the patient groups mean serum levels of total cholesterol, triglyceride and LDL-C were 143.1 mg/dL, 135.97 mg/dL and 136.45 mg/dL respectively and serum levels of Apo-A1, Apo-B and Lp (a) were 56.05, 100.3 and 39.4 mg/Dl, respectively. In the control group mean serum levels of LDL-C, Apo-A1, Apo-B and Lp (a) were 66.38, 151.81, 69.42 and 19.11 mg/dL, respectively.

As shown in Table 1 and 2 the Mean levels of total Cholesterol, triglyceride, LDL-C, ApoB lipoprotein and Lp (a), as well as total Cholesterol/HDL ratio and LDL-C/HDL-C ratio in patients with gastritis resulted by *H. pylori* were significantly increased compared to control groups. In addition HDL-C and Apo- A1 lipoprotein were decreased significantly.

DISCUSSION

There are many studies on the relationship of *H. pylori* with various disorders like cardiovascular diseases (Strachan et al., 1998). The results of Danesh et al. (2000) on the relation of cardiovascular disease and *H. pylori* showed a significant increase and direct relation of cardiovascular disease and *H. pylori* infection. It is very important to diagnosis the type of gastritis before choosing right therapy to this disorder and also to prevent economic and social problems. To understand more about pathogenesis and the variation made by this microorganism on human body, we tested serum levels of lipids, lipoprotein

Table 2. Apolipoproteins and Lipoprotein (a) means and P-values in the patients with gastritis resulted by *H. pylori* and the patients with gastritis that is not resulted by *H. pylori* but other causes as controls.

Factor	Unit	Patients	Controls	P-Value
Apo A1	mg/dl	56.05	151.81	0
Apo B	mg/dl	100.3	69.42	0
Lp (a)	mg/dl	39.4	19.11	0.001
Apo A/Apo B	----	0.56	2.24	0

lipids, lipoproteins and Apo-A1, Apo-B and Lp (a) in patients with gastritis resulted by *H. pylori* compare to patients with gastritis resulted by other causes. Results showed there was statistically significant relationship between serum cholesterol and triglyceride levels in the patients groups as compared with the control group.

The study of Hoffmeistrer et al. (2001) was also showed that the cholesterol level was increased. Same results were shown by Mendall work's too (Mendall, 1998). Also results was obtained by Niemela et al. (1996), Strachan et al. (1998), Maisch et al. (1998) were in agreement with our findings. Concerning serum HDL-C level in the case group in comparison with the control group, present study showed a decrease in the HDL-C level in patients group. Conversely a statistically significant increase in LDL-C levels in the patients with gastritis resulted by the *H. pylori* was shown compare to control group. Various studies by Pilotto and Malfertheiner (2002), Yudkin (1997) on patients with gastritis and cardiovascular diseases showed that there is a significant decrease in HDL-C level. Also in Laurila works (1999) on patients with cardiovascular disease and gastritis resulted by *H. pylori* a significant decrease in HDL-C level was shown. Studies of Scragg et al. (1996) and Kowalski (2001) on the relationship of *H. pylori* and cardiovascular diseases showed that LDL-C level is increased by *H. pylori* infection.

It was known that Cholesterol/HDL ratio and LDL/HDL ratio are risk factors for developing cardiovascular diseases. Concerning these ratios, our findings showed a statistically significant difference in the patients groups compared to control group. Hoffmeister et al. (2001) and Niemel et al. (1996) results confirmed these findings. Mean Apo- A1 changes in the patients with gastritis induced by *H. pylori* showed a decrease in its mean related to the patients with gastritis that was not resulted by *H. pylori* but other causes. This difference was statistically significant and had a reverse and negative relation to the gastritis resulted by *H. pylori.* Other studies like Hoffmeisler et al. (2001) indicated significant decrease in mean Apo-A1 level in the patients with gastritis resulted by *H. pylori* as well as cardiovascular patients. Various studies indicated that Apo-B would increase in the patients with gastritis resulted by *H. pylori*.

The Apo-A1/Apo-B ratio was significantly decreased in

the studied patients with gastritis resulted by *H. pylori* compare to gastritis with other causes. In the study of Hoffmeisler et al. (2001) patients with CHD and chronic inflammation of *H. pylori* showed a significant decrease in this ratio. Lp(a) levels have been significantly correlated with coronary artery disease (Berglund and Ramakrishnan, 2004). Study of Chimienti et al. (2003) showed that *H. pylori* infection, a proposed but still controversial factor of risk for cardiovascular diseases, affects the serum lipid profile in a way that can increase the risk of atherosclerosis, in particular considering the interaction with the atherothrombogenic lipoprotein Lp(a). *H. pylori* infection is significantly associated with acute myocardial infarction (AMI) independent of the classic coronary risk factors (Jin et al., 2007) . The speculation of the exact relationship between *H. pylori* and cardiovascular risk factors might be an important issue in an attempt to reduce the cardiovascular disease incidence (Mendall, 1998; Kostner et al., 1999).

Conclusion

Based on our findings it was possible to assume an association between *H. pylori* infection with various cardiovascular risk factors, especially increased level of total cholesterol, Apo-B, LDL-C, Lp (a) and decreased levels in HDL-C and Apo-A. *H. pylori* infection can cause lipid metabolism disorders that may act as risk factors for cardiovascular diseases.

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