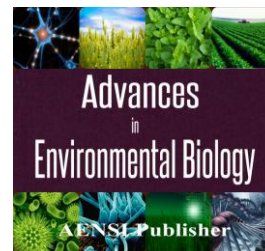




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### Evaluation of Lipid Profiles Serum in Subjects Infected and Non-Infected with Helicobacter Pylori

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#### ABSTRACT

**Background and Aims:** Helicobacter pylori infection is one of the most important factors of peptic ulcer and other gastrointestinal disorders. Gastritis caused by H. pylori infection has a high prevalence and about 10 percent of people in their lifetime will suffer from gastritis. Some studies have shown an association between H. pylori and atherosclerosis that may exist. The results of studies showed that the presence of H. pylori in peptic ulcer gut leads to changes in lipid profile including cholesterol, triglycerides and lipoproteins are HDL-c and LDL-c. The purpose of this study was to evaluate the serum lipid profile and inflammatory factors in individual's infected and non-infected with H. pylori. The measurement levels of lipids profiles and inflammatory factors in patients with H. pylori infection as cases of atherosclerosis and heart disease rate would be very useful vessel. **Materials and Methods:** In this study 58 patients with active peptic ulcer and positive rapid urease test (22 female & 36 male) referring to gastroenterology clinic with mean age of  $45.30 \pm 13.16$  as case group and 58 patients without active peptic ulcer and negative rapid urease test (20 female & 38 male) with mean age of  $42.67 \pm 16.04$  were selected as control group. Two groups of patients were selected we took one blood sample and two gastric biopsy specimens. Biopsy specimens to confirm the absence of infected or non-infected with H. pylori has been sent to the pathology laboratory. Blood samples for measurement of serum lipid profile in use. Total cholesterol, triacylglycerol, HDL- cholesterol, LDL- cholesterol, was measured. **Results:** In the case group rate of serum lipid profiles, including total cholesterol, triacylglycerol, LDL and HDL compared with the control group are statistically significant and increased (In all  $p < 0.0001$ ). **Discussion:** Because heart disease burden on health and economic society entered the building. According to the results of this study and other studies certainly can be said that H. pylori infection have increased levels of lipid profile. The increase of this factors the risk of causing a very high potential in cardiovascular diseases and atherosclerosis.

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#### INTRODUCTION

Helicobacter pylori is a negative-gram, unipolar, multi flagella and microaerophilic bacteria of curved bacilli which can cause acute and chronic gastritis, duodenal ulcer and stomach cancer in human. Helicobacter pylori infection is one of the most important factors in peptic ulcer and other gastrointestinal disorders [1]. Helicobacter pylori infection gastritis is relatively common among people; almost 10% of people suffer gastritis disease during their lifetime. It is relatively more rampant among men compared to women [2]. there are various theories about reasons of duodenal ulcers. Some studies have shown that there might be a relation between Helicobacter pylori and Atherosclerosis. Chronic gastritis and stomach atrophy has been reported as predisposing factor of stomach cancer. As people grow older, the prevalence of Helicobacter pylori infection increases, all over the world. Helicobacter pylori infection prevalence among adult people of Middle East has been estimated in range of 70-90%.

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On one hand coronary artery disease is the most prevalent reason of causalities both in industrial developed countries and developing countries such as Iran. Annually almost 1.5 million American people suffer myocardial infraction, 500000 of whom die, consequently. The classic etiologic factors of coronary artery disease are blood high lipids, higher blood pressure, diabetes, smoking and family history. Moreover, recently infectious factors, also, have been considered as contributing factor of this disease.

Different studies have indicated that the presence of *H. Pylori* in gastrointestinal ulcers results in change in lipid profile of serum including: cholesterol, triglyceride and LDL-c, HDL-c lipoproteins. Various studies have shown that there is a reverse relationship between high density lipoprotein and risk of coronary artery disease, as a result HDL is considered as strong predictor of disease. HDL causes postpone in Atherosclerotic injuries formation through eliminating extra cholesterol of cells, preventing endothelial injuries. Hence the aim of present study is to examine serum lipid profile degree among people infected and uninfected to *H. Pylori* infection in Tabriz City. So measuring total Cholesterol, triglyceride, HDL cholesterol and LDL cholesterol degree among patients with *H. Pylori* infection will be considered as a golden standard to evaluate atherosclerosis symptom developments and cardiovascular disease, since referred lipid profiles give an attitude to the physician to know that to what extent his treated patient is vulnerable to cardiovascular disease.

## MATERIALS AND METHODS

Patients referred to Endoscopy professional clinic of Tabriz Imam Reza Hospital were treated under endoscopy by gastroenterologist. One blood sample and two biopsy samples of gastric anteroom, three centimeters left to pylori, were taken from chosen patients. One of these biopsy samples was taken for rapid test of Urease and the other one to provide direct smear. It is worth mentioning that with providing direct smear of biopsy samples and coloring it in gram method and microscopic investigation of it along with investigation of existence or no existence of *H. Pylori* we were able to confirm Urease test results which would probably had false results (biopsy samples were homogenized by homogenizer and then direct smear was obtained from them). Relying on these criteria patients were divided into two groups of infected and uninfected to *H. Pylori*. From these patient 58 were selected as case group or patients infected to *H. Pylori* and 58 patients, who were negative in Urease test and direct investigation of smear, were selected as uninfected group. In order to minimize interfering factors we excluded patients who had special disorders history including: diabetes, disabilities, renal disorders, hepatic disease, thyroid disorders, also alcoholics, smokers and patients with low activity. It is worth mentioning that taken serum samples were kept at  $-70^{\circ}$  freezers till the time of doing experiment. Total cholesterol, triacylglycerol, HDL cholesterol and LDL cholesterol were measured in calorimetric method. Moreover, before taking sample from patients, some information related to their personal characteristics including age, gender, history of being infected to other disease were obtained and registered at checklist provided for this aim, to balance both groups.

### Results:

#### *Comparing Mean Age of Both Studies Groups:*

In this study a total number of 116 patients were examined, 50% Of whom was infected to *H. Pylori* infection, called case group and 50% was not infected to this bacteria that were called control group. Case group was involved 36 men (67.5%) and 22 women (32.5%) and control group involved 38 men (67.5%) and 20 women (30.2%). Among case group minimum age was 22 and maximum age was 75 and mean age was  $54.30 \pm 13.16$ . Moreover among patients of case group minimum age was 28 and maximum age was 68 and mean age of control group was  $42.67 \pm 16.04$ . Using one-way variance analysis it was identified that mean age of control group and case group had no significant difference, statistically (see table 1). In a better word age equalization was done appropriately in both case and control group.

To analyze underlying parameters of present study first, considered mean factors of each group was determined individually, compared to each other one-way ANOVA using SPSS (version 16). In these tests  $p < 0.05$  was considered significant.

#### *Comparing mean lipid profile of blood serum in the tow studied groups:*

As it can be observed in table 2 and Fig. 1, 2, 3, 4, using one-way ANOVA a significant difference can be seen among means of studied groups. It is worth mentioning that using SPSS of version 16 and one-way ANOVA it was determined that blood serum lipid profile degree has significant difference, statistically, in case group or patient infected with *Helicobacter pylori* with active peptic ulcer, compared to control group. Obtained results have been indicated as standard deviation mean ( $p=0.0001$  in all cases)

### Discussion:

Cardiovascular disease is the main reason of causalities among men and women all over the world. Such diseases take different forms including: high blood pressure, coronary atherosclerosis, heart attack, stroke, heart

defect, atherosclerosis of other arteries such as visceral and organ vessels. As cardiovascular disease bear heavy economical and healthy costs to the society, during recent decades various studies have been done to determine dangerous factors increasing risk of cardiovascular disease. Having knowledge of such risky factors and their controlling way creates an opportunity to prevent incidence of such disease or in the case of incidence to reduce its intensity. Cardiovascular disease (such as heart attack, stroke, environmental artery involvement) is considered as the main factor threatening human developed communities, though, fortunately, through making positive changes in lifestyle, we can prevent incidence of such disease or their exacerbation. Even after their incidence we can reduce their side effects and causalities, considerably. These preventive changes including healthy nutrition, increasing physical activities, eradication of infections using microorganisms, quitting harmful habits such as smoking cigar, pipe, and hubble-bubble and drinking alcohol, is of great importance.

Cardiovascular disease is appeared gradually, having no symptom at initial stages. Factors such as high cholesterol, high Triacylglycerol, high LDL-C and low HDL-C might be prevalent from childhood or initial youth, without being diagnosed. Hence the best solution is early investigation, diagnose and control of risky factors and preventing cardiovascular disease. According to several decades of studies in the area, making positive changes in lifestyle not only prevents suffering from cardiovascular disease, but also reduces the dangerous disease risk including cancer and diabetes.

Understanding atherosclerosis pathophysiology is useful in treating its consequences. Common conditions and exact mechanism of vascular atherosclerosis disease onset is still unknown. Though, still, there are more similarities between inflammation and atherosclerosis processes and there is increasing reason for the role of active inflammation procedure in atherosclerosis pathogen in coronary blood stream and other areas. Specially, monocytes and macrophages were studied as atherosclerosis plaques components. Increased levels of lipid profiles and pre inflammation cytokines is in direct relationship with increased risk of cardiovascular events. It is probable that there would be an unknown chronic contamination behind inflammation symptoms.

H. Pylori infection is in direct relationship with disease out of gastrointestinal especially vascular disorders (for example ischemic heart disease, initial Rinord phenomena and ischemic stroke), self immunity disorders. Most case-control studies have reported that there is a considerable relationship between positive H. Pylori and cardiovascular disease and ischemic electrocardiography changes which are independent of risky factors and socio-economical factors.

The role of inflammation mechanism in pathogenesis and cardiovascular disease development has been discussed increasingly; still it has been remained unrecognized. Epidemiologic studies have proposed a relationship between Atherosclerosis and chronic infection of H. Pylori. Correlation of H. Pylori with Atherosclerosis, especially with cardiovascular disease, is based on serologic findings; still it is a controversial issue.

In present study we have tried to determine the least part of mechanisms and reactions among host cells along with those of H. Pylori to take least steps of clarifying confrontation method of such global microbial contamination. In this study we have examined serum lipid profile amount of patients infected and uninfected to H. Pylori simultaneously and repeatedly. Results obtained from our study indicated that cholesterol amount, triacylglycerols, HDL-cholesterol and LDL-cholesterol of patients infected with H. Pylori is significantly more than patients uninfected to H. Pylori ( $p < 0.0001$  in all cases).

Increase in serum lipid profile including cholesterol, triacylglycerols, increase in cholesterol with Low Density Lipoproteins and reduce in cholesterol with High Density Lipoproteins are risky factors of cardiovascular disease incidence. Though whether it is indicator of involvement extent or not is still under question [10, 11].

Medal *et al* [12], Danesh *et al* [13] and Glynn *et al* indicated a close relation between serologic Helicobacter pylori and heart coronary artery disease. Pellicano *et al* reported significant high prevalence of H. Pylori infection with heart coronary artery disease among patients compared to control group (77% compared to 59%). Results of our study are in balance with their studies indicating that: Helicobacter pylori infection is accompanied with increase in incidence risk of cardiovascular disease and our results in this study indicated that this correlation is extremely strong.

Though, Danesh *et al* found that there is a limited relationship between Helicobacter pylori infection and cardiovascular disease. But these scholars, also, never rejected the relationship between H. Pylori contamination and cardiovascular disease. They suggested that wide epidemiologic studies should be carried out to determine decisively the relationship between these two disorders. Other studies like those of Singh *et al* [17] and Pasceri *et al* [18] examined positive H. Pylori prevalence of Cag-A strains among cardiovascular patients. Their studies indicated higher incidence of H. Pylori of positive Cag-A strain among cardiovascular patients compared to control group (52% compared to 43% or 43% compared to 17%). These results are in balance with our findings.

Bacterial infection with H. Pylori influences the development of atherosclerosis changes in coronary arteries, accepting destructive effects of microorganisms or their products (including cytokines, endotoxins, cytotoxins and other virulence factors) on coronary endothelium as a hypothesis. Niemela *et al* [19] and De Lvis *et al* [20] indicated that infection with H. Pylori result indifferent changes in lipid, protein thrombotic.

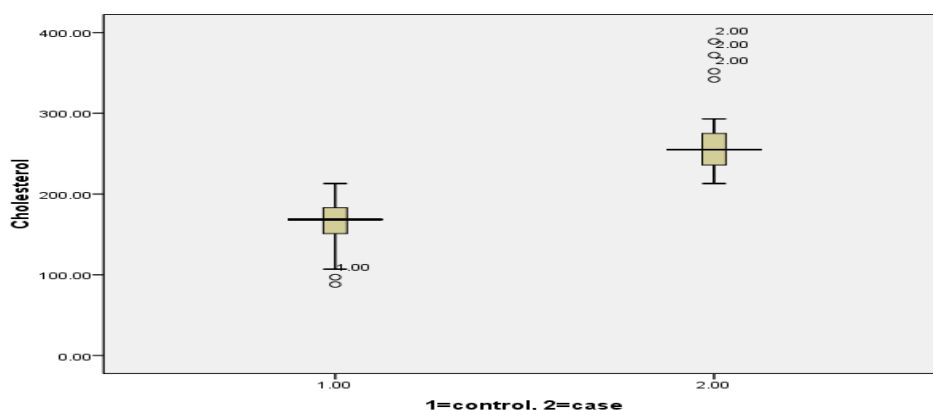
Hoffmeister *et al* indicated a well correlation between *H. Pylori* infection and reduced HDL cholesterol which is in balance with our findings. Pieniasek *et al* examined the dependence between *H. Pylori* infection and CRP levels, cholesterol, lipid pattern and fibrinogen plasma. These scholars indicated higher level of identified parameters among cardiovascular patients compared to control group which was accorded from age and gender point of view. Pellicano *et al* reported that *Helicobacter pylori* infection activates and accumulates blood placates, increasing plasma level of triglycerides, different pro iatrogenic factors including homocysteine which is also in balance with our findings. Recently it might be that *Helicobacter pylori* infection has been accompanied with increased risk of cardiovascular disease through activation of critical phase responses and blood placates factors.

Abdelmoutallebi, Gasbarrini, Stone *et al* reported that immune response of host to cloned bacteria of gastritis plays an important role in vessels disorder pathogen through material performance of different vasoactives (vessel activator) including cytokine, icosanoid and ... gastritis infection by *Helicobacter pylori* can induce acute phase reactive synthesis, activating immune mechanisms with antibodies having intersectional *H. Pylori* reaction, and heat shock proteins with HSP60,65 derivate from endothelial.

Birnie *et al* reported a strong relationship between anti HSP 60 titles and Coronary Arthroscleroses. Moreover eradication therapy results in reducing anti HSP65 titles. Franceschi *et al* indicated that anti-Cag. A antibodies have interaction with cytoplasm, monocytes cores and arthroscleroses vessel wall. Schoars proposed that cross reaction results in arthroscleroses development. Mendall *et al* indicated a strong correlation between increase in serum levels of some of pre inflation cytokines (TNFa, IL-8, IL-1B) and cardiovascular factors risks.

#### Conclusion:

According to results obtained in this investigation and most other studies, it can be definitely claimed that most people infected with *H. Pylori* in Tabriz city have higher amount of increased lipid profiles and this increase cause potential and high risk of cardiovascular disease which should be surely considered by considered by gastroenterologist and cardiovascular specialists, taking needed actions to prevent serious side effects among such patients. Finally it is suggested that patients with *H. Pylori* infections would be referred to cardiovascular disease specialist surely and drug eradication of such contamination along with regimens and purposeful treatment should be done to reduce lipid profile of such patients.



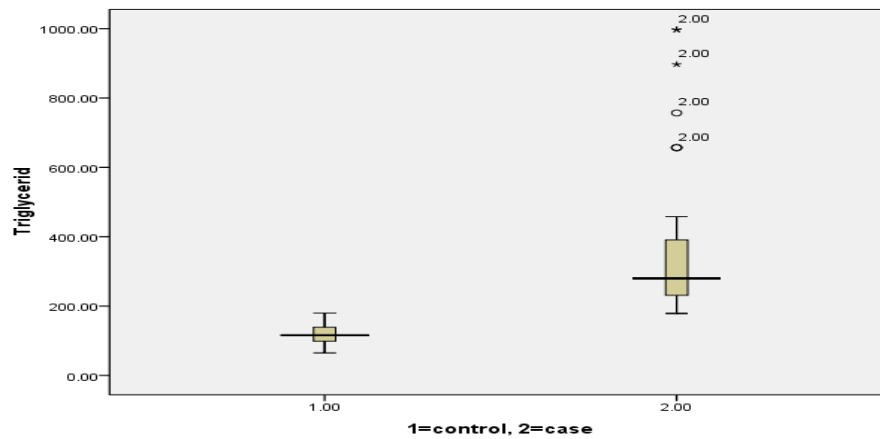
**Fig. 1:** curve of comparing mean cholesterol in the two studied groups.

**Table 1:** Data related to comparing mean age of control group with case group.

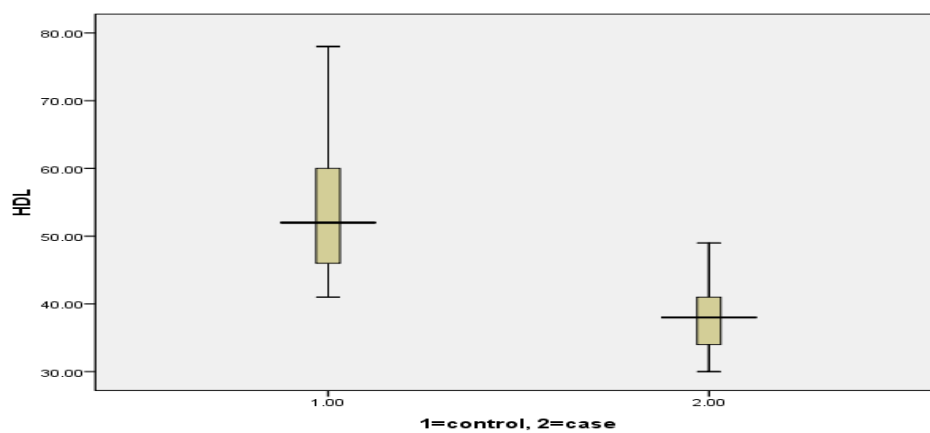
CI <sub>95%</sub>	p-value	F	Mean±SD year(	n	Age
10.32 – 5.06	0.812	0.360	16.04±42.67 13.16±45.30	58 58	Control group Case group

**Table 2:** information related to comparing lipid profile mean in the two studied groups.

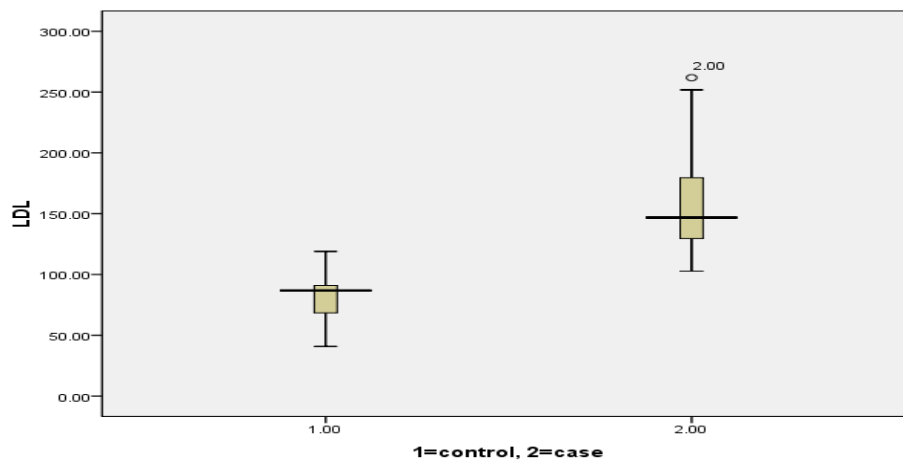
p-value	Mean±SD (case group)	Mean±SD (control group)	n	Lipid profile
0.0001	35.86 ±259.7	26.84 ±164.8	116	Cholesterol )mg/dl (
0.0001	190.68± 349.4	29.24 ±119.8	116	Triacylglycerol )mg/dl (
0.0001	0.64±37.86	1.23 ±58.93	116	)mg/dl (HDL
0.0001	4.41 ±154.18	2.43 ±82.54	116	)mg/dl (LDL



**Fig. 2:** Curve of comparing mean triacylglycerol in the two studied groups.



**Fig. 3:** Curve related to comparing mean lipoprotein with High Density Lipoprotein in the tow studied groups.



**Fig. 4:** curve related to comparing mean lipoprotein with Low Density Lipoprotein in the tow studied groups.

#### REFERENCES

- [1] Dolatkah, H., S. Babae, M. Rahbani-Nobar, 2011. Diabetes Mellitus and Helicobacter Pylori. LAP Lambert Acad. Publ.
- [2] Dolatkah, H., M. Rahbani-Nobar, E. Fattahi, M. Ansari, A. Mirza-Aghazadeh, L. Eftekhari-Vash, A. Fakhrjo and A. Bahrami, 2011. Evaluation of glycemic control, gastric juice nitric oxide and oxidative stress in diabetic patients infected by Helicobacter pylori. Journal of Medical Genetics and Genomics, 3(1): 1-6.

- [3] Axon, ATR, 2007. Relationship between *Helicobacter pylori* gastritis, gastric cancer and gastric acid secretion. *Adv. in Med. Sci.*, 52: 55-60.
- [4] Mendall, MA., 1998. Inflammatory responses and coronary heart disease. *B.M.J.*, 316: 953-4.
- [5] Chen, DF., L. Hu, P. Yi, WW. Liu, DC. Fang, H. Cao, 2007. *H pylori* exist in the gallbladder mucosa of patients with chronic cholecystitis. *World J Gastroenterol*, 13: 1608-11.
- [6] Jin, SW., SH. Her, JM. Lee, HJ. Yoon, SJ. Moon, PJ. Kim, SH. Baek, KB. Seung, JH. Kim, SB. Kang, JH. Kim, KY. Kim, 2007. The association between current *Helicobacter pylori* infection and coronary artery disease. *Korean J. Intern. Med.*, 22: 152-6.
- [7] Sung, KC., EJ. Rhee, SH. Ryu, SH. Beck, 2005. Prevalence of *Helicobacter pylori* infection and its association with cardiovascular risk factors in Korean adults. *Int. J. Cardiol.*, 102: 411-7.
- [8] Ican, A., R. Yi\_ito lu, A. Ona, N. Vurgun, Z. Ari, P. Ertan, AZ. Sengil, 1998. Should children with infection be tested for lipid, lipoprotein and apolipoprotein?. *Acta. Paediatr. Jpn.*, 40: 47-51.
- [9] Ridker, PM., CH. Hennekens, JE. Buring, N. Rifai, 2000. C-reactive protein and other markers of inflammation in the prediction of cardiovascular disease in women. *N Engl J Med.*, 342: 836-843.
- [10] Kullo, IJ., KR. Bailey, JP. McConnell, 2004. Low-density lipoprotein particle size and coronary atherosclerosis in subjects belonging to hypertensive sib ships. *Am J Hypertens*, 17(9): 845-51.
- [11] Sposito, AC, 2001. Triglyceride AND lipoprotein (a) are markers of coronary artery disease severity among postmenopausal women. *Maturitas*, 39(3): 203-8.
- [12] Mendall, M., P. Goggin, J. Levy, 1994. Relation of *Helicobacter pylori* infection and coronary heart disease. *Br Heart J.*, 71: 437-439.
- [13] Danesh, J., R. Collins, R. Peto, 1997. Chronic infections and coronary heart disease: is there a link? *Lancet*, 350: 430-436.
- [14] Danesh, J., Y. Wong, M. Ward, 1999. Chronic infection with *Helicobacter pylori*, *Chlamydia pneumoniae*, or *Cytomegalovirus*: population based study of coronary heart disease. *Heart*, 81: 245-247.
- [15] Pellicano, R., MG. Mazzeo, S. Morelloni, 1999. Acute myocardial infarction and *Helicobacter pylori* seropositivity. *Int J Clin Lab Res.*, 29: 141-144.
- [16] Danesh, J., L. Youngmann, S. Clark, 1999. *Helicobacter pylori* infection and early onset myocardial infarction: case-control and sibling pairs study. *Br Med J.*, 319: 1157-1162.
- [17] Singh, RK., AD. McMahon, H. Patel, 2002. Prospective analysis of the association of infection with CagA bearing strains of *Helicobacter pylori* and coronary heart disease. *Heart*, 88: 43-46.
- [18] Pasceri, V., G. Caramota, G. Patti, 1998. Association of virulent *Helicobacter pylori* strains with ischemic heart disease. *Circulation*, 97: 1675-1679.
- [19] Niemela, S., T. Karttunen, T. Korhonen, 1996. Could *Helicobacter pylori* infection increase the risk of coronary heart disease by modifying serum lipid concentrations? *Heart*, 75: 373-375.
- [20] de Luis, DA., A. Garcia-Avello, MA. Lasuncion, 1999. Improvement in lipid and haemostasis patterns after *Helicobacter pylori* infection eradication in type 1 diabetic patients. *Clin Nutr.*, 18(4): 227-231.
- [21] Hoffmeister, A., D. Rothenbacher, G. Bode, 2001. Current infection with *Helicobacter pylori*, but not seropositivity to *Chlamydia pneumoniae* or *Cytomegalovirus*, is associated with atherogenic, modified lipid profile. *Arterioscler Thromb Vasc Biol.*, 21: 427-432.
- [22] Pieniżek, P., E. Karczewska, A. Duda, 1999. Association of *Helicobacter pylori* infection with coronary heart disease. *J Physiol Pharmacol.*, 50(5): 743-751.
- [23] Pellicano, R., N. Broutet, A. Ponzetto, 1999. *Helicobacter pylori*: from the stomach to the heart. *Eur J Gastroenterol-Hepatol.*, 11(11): 1335-1337.
- [24] Abdelmouttaleb, T., N. Dauchin, C. Ilardo, 1999. C-reactive protein and coronary artery disease additional evidence of the implication of an inflammatory process in acute coronary syndromes. *Am Heart J.*, 137: 346-351.
- [25] Gasbarrini, A., F. Franceschi, G. Cammarota, 1998. Vascular and immunological disorders associated with *Helicobacter pylori* infection. *Ital J Gastroenterol Hepatol.*, 30: 115-118.
- [26] Stone, AF., MA. Mendall, 2000. *Helicobacter pylori* is an ethiological factor for ischemic heart disease: the case in favour. *Digest Liver Dis.*, 32: 62-64.
- [27] Mendall, MA., P. Patel, L. Ballam, 1996. C-reactive protein and its relation to cardiovascular risk factors: a population based cross sectional study. *Br Med J.*, 312: 1061-1065.
- [28] Murray, LJ., KB. Bamford, DP. O'Reilly, 1995. *Helicobacter pylori* infection: relation with cardiovascular risk factors, ischemic heart disease, and social class. *Br Heart J.*, 74: 497-501.
- [29] Birnie, DH., ER. Holme, IC. McKay, 1998. Association between antibodies to heat shock protein 65 and coronary atherosclerosis. Possible mechanism of action of *Helicobacter pylori* and other bacterial infections in increasing cardiovascular risk. *Eur Heart J.*, 19: 387-394.
- [30] Franceschi, F., AR. Sepulveda, A. Gasbarrini, 2002. Cross-reactivity of anti-CagA antibodies with vascular wall antigens: possible pathogenic link between *Helicobacter pylori* infection and atherosclerosis. *Circulation*, 106: 430-434.

- [31] Mendall, MA., P. Patel, M. Asante, 1997. Relation of serum cytokine concentrations to cardiovascular risk factors and coronary heart disease. *Heart*, 78: 273-277.