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Original Article

Evaluation of Lipid Profile in H. Pylori Infected Coronary Artery Disease Patients

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ABSTRACT

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INTRODUCTION

H. pylori is a gram-negative bacterium, initially sequestered in gastric mucosa by Marshall and Warren in 1983 [1]. *H. pylori* had known to be the cause liable of maximum of the cases of gastric mucosal damage. Mostly *H. pylori* infection does not harm but most of times they are responsible for stomach ulcer and also ulcer of small intestine [2]. Coronary artery disease progresses once the main blood vessels come to be damaged or diseased. Cholesterol comprising deposits in arteries and inflammation leads to coronary artery disease [3]. The relation of coronary artery disease and *H. pylori* infection is based on 3 main evidences. Microbial, pathological and epidemiological mechanism of postulation [4]. There are many ways in which infection organism introduce and enhances atherosclerosis. This goes through protruding invasion into vessels wall causing response of inflammation which enhances macrophages, lymphocytes, cytokines production and factors of tissue growth [5, 6].

Increase in low density lipoprotein level and decrease in high density lipoprotein level result to

coronary artery disease. Metabolism of lipids regulated during host response to H. pylori

infection. **Objective:** To analyze the serum levels of lipid profile in *H. pylori* infected coronary

artery disease patients. **Methods:** It was a comparative Cross-Sectional study. This study was done at the Department of Biochemistry, Peoples University of Medical Health Sciences for

Women (PUMHSW) from 1st July 2022 to 15th December 2022. A sample of 60 subjects was divided

into 2 groups. Group A (Control) comprised of 30 subjects and group B (cases) of 30 subjects. 5

mL of blood from each participant was collected under aseptic conditions. For the Lipid profile,

2 mL of the blood was collected in the Gel test tubes. A Spectrophotometer was used to perform

the lipid profile. For the data analyzes SPSS Version-22.0 was used. Results: In this study we

found that Helicobacter pylori positive subjects have higher levels of serum LDL.C, Triglycerides

and total cholesterol. The outcomes of present research showed that H. pylori is associated with

low level of HDL-C. The present study results shown an association among H. pylori infection and

coronary artery possibility influence. **Conclusions:** We concluded in this study that serum levels

of lipid profile become worse in positive H. pylori infected patients as compared to the control

group participants which were negative H. pylori with coronary artery disease.

Lipopolysaccharides releases endotoxins due to systemic effect of infection cause release of lipopolysaccharides which ultimately damage to epithelium increase in cytokines with enhancing inflammatory parameters and stimulate procoagulants which leads to ischemia and thrombosis which all predisposes towards coronary artery disease [7]. Increase in low density lipoprotein level and decrease in high density lipoprotein level result to coronary artery disease. Metabolism of lipids regulated during host response to *H. pylori* infection[8].Lipids show host defense with lipoproteins for infectious particles like endotoxins. This is mediated through cytokines like TNF a, interleukin 6, interleukin1 and interferon. Cytokines also decreases lipoprotein lipase activity and clearance of triglycerides and also increase very low-density lipoprotein levels[9].

METHODS

This study was done at the Department of Biochemistry Peoples University of Medical Health Sciences for Women Nawabshah Shaheed Benazirabad (PUMHSW) from 1st July 2022 to 15th December 2022 along the cooperation with Medicine OPD/Ward PMCH. The analysis of sample had been done at diagnostic and research laboratory PUMHSW, Shaheed Benazirabad (SBA). Study design was comparative cross sectional. Sample technique was non probability purposive sampling. Both male and female subjects were included in the study which were diagnosed cases of Myocardial infarction from age 35 to 65 years with H. pylori infection and coronary artery disease. The patients with hepatic carcinoma, renal or hepatic failure and using drugs that affect the H. pylori were excluded. The sample size of study was based upon 60 subjects divided into two groups. Group A (Control) comprised of 30 participants with H. pylori negative coronary artery disease and group B (Cases) comprised of 30 participants with H. pylori positive coronary artery disease. We collected complete medical data and pertinent information from every subject through filling out a proforma. All participants gave verbal and written agreement after being informed about the study's purpose. Blood sample collection was done by venipuncture of the participants. 5 mL of blood from each participant was taken under aseptic conditions. For the Lipid profile, 2 mL of the blood was collected in the Gel test tubes. The blood was centrifuged for 10 minutes at 3500 rpm, fractionated, and conveyed to eppendorf cups before being stored at -20°c until analysis. The material was allowed to reach room temperature before being utilized. A Spectrophotometer was used to perform the lipid profile. For the data analyzes SPSS Version 22.0 was used. Results were shown as mean and standard deviation. Total cholesterol, HDL, LDL, TAG was performed using spectrophotometer. Lipoproteins are fractioned,

after centrifugation, the supernatant contains chylomicron, which may be detected using the CHOD-PAP assay and lipid clearing factor (LCF) while LDL and VLDL were fractionated and precipitated by addition of polyethylene glycol (PEG). This study had been approved by the Review Committee of PUMHSW Nawabshah.

RESULTS

Total 60 cases of coronary artery disease were analyzed and they were equally divided in to two groups. Group A, *H. pylori* negative (n=30) and group B, *H. pylori* positive (n=30) subjects. Table 1 shows the age distribution of the study participants. In group A the mean age of study subjects was 46.7 ± 5.7 years. In group B the mean age of study subjects was 55.7 ± 9.6 years. The other main finding of the study was that the *H. pylori* positive subjects were older than the negative subjects. There was statistically significant difference of age between group A and group B subjects shown in Table 1.

Table 1: Distribution of Subjects According to Age in Years n=60

Group A H. Pylori -Ve	Group B H. Pylori +Ve	t-value	p-value
46.7±5.7	55.7±9.6	16.51	0.03

The mean and standard deviation of lipid profile of study groups is shown in Table 2. The mean triglycerides of group A subjects was 177.5±77.25 while in group B the mean triglycerides were 232.01±65.53. Subjects with H. pylori positive have significant higher triglyceride concentrations. In group A subjects the mean low-density lipoproteins were 110.3±21.6 while in group B was 126.94±49.8. Low density lipoprotein concentration was significantly lower in subjects with H. pylori negative groups. The mean high-density lipoprotein level in group A subjects was 43.4±10.11 while in group B the mean highdensity lipoprotein level was 40.3±11.76. Subjects with H. pylori positive have insignificantly decreased high density lipoproteins concentrations compared with control group. The mean total cholesterol level in group A subjects was 165.35±31.40 while in group B total cholesterol level was 179.47±46.31. Subjects with H. pylori positive have significantly higher total cholesterol concentrations compared with control group subjects.

Table 2: Comparison of Lipid Profile Variation in H. Pylori - Ve AndH. Pylori + Ve

Variables	Group A H. Pylori – Ve	Group B H. Pylori +Ve	p-value
Triacylglycerol (mg/dl)	177.52±77.25	232.01±65.53	0.01
HDL-C (mg/dl)	43.4±10.11	40.3±11.76	0.69
LDL-C (mg/dl)	110.3±21.6	126.94±49.8	0.001
Total Cholesterol (mg/dl)	165.35±31.40	179.47±46.31	0.02

DISCUSSION

In this study we found that *Helicobacter pylori* positive subjects have higher levels of serum LDL.C, Triglycerides

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developingatherosclerosis.

Authors Contribution

Conceptualization: MM¹, NA, WA Methodology: GQ, MM², MM³ Formal analysis: GQ, MM², MM³ Writing-review and editing: MM¹, NA, WA

All authors have read and agreed to the published version of the manuscript.

Conflicts of Interest

The authors declare no conflict of interest.

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Kucukazman et al., who found total cholesterol and LDL concentrations increased in H. pylori positive patients than in negative H. pylori patients [10]. Similarly Sung et al., also reported the increased levels of TG, TC and LDL but decreased levels of HDL-C in H. pylori infected patients [11]. The results of present study showed that H. pylori is associated with low level of HDL-C. Hoffmesister et al., and Takashima et al., demonstrated that H. pylori causes low HDL-C level [12, 13]. The results of present study consistent to the study done by Malekiet al., who found in his study that there is a relationship in H. pylori infection and cardiovascular diseases [14]. Higher occurrence of H. pylori was found among CAD patients. When related to H. pylori triglyceride levels were increased in positive than that in negative cases, on the other hand HDL-C levels were in positive cases. Shimamoto et al., estimated the association between H. pylori infection and the serum lipid profile revealed that *H. pylori* infection is positively associated with LDL-C, TC, and TG and negatively associated with HDL-C [15]. Findings of the current study showed similar results. Guzman et al., reported that gastric H. pylori infection does not have significant relation with the presence of dyslipidemia [16]. The alteration of the serum lipid profile was discreetly higher in the patients infected by *H. pylori* but they were not statistically significant. Hissun et al., reported that the serum of level of total cholesterol were significantly increased in group which had H. pylori positive and coronary artery disease, while in other group which had H. pylori positive without coronary heart disease the serum levels of LDL was significantly increased. These results are inconsistent with the present study [17]. Nam et al., estimated increased lowdensity lipoprotein (LDL) and decreased high-density lipoprotein (HDL) than H. pylori-negative group which was comparative to the present study [18]. Abdu et al., reported that serum LDL levels were high in H. pylori positive coronary artery disease patients as compared to the H. pylori negative patients which was similar to the present study findings [19]. The results of present study were consistent to the study of Longo-Mbenza et al., who found in his study that there is a relationship in H. pylori infection and cardiovascular diseases [20].

and total cholesterol. These results are supported by

CONCLUSIONS

The present study results shown a relation between *H. pylori* infection and coronary artery risk factors. *H. pylori* infection affects the development of cardiovascular disease as it introduces the chronic long-term infection in epithelium, which leads to locally and systematically inflammation. *H. pylori* infection enhances the risk of cardiovascular disease by decreasing the level of HDL concentration and it may be understood as a risk factor of

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