RELATION BETWEEN LIPID PROFILE CHANGES AND *HELICOBACTER PYLORI* INFECTION

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**ABSTRACT**

*Helicobacter pylori* infection is one of the most common diseases among the developing countries. The present study was undertaken to investigate the changes in lipid profile in patients with a *H. pylori* infection. We analyzed the Total cholesterol, LDL-cholesterol, Triglyceride and Lipoprotein (a) of 24 male patients suffering with *H. pylori* infection confirmed by urease breath test (UBT). The results were compared with 24 healthy controls included in the study. In the present study, we found a significant increase in lipid profile in patient with *H. pylori* infection. The finding supports the hypothesis that, there is a relation between the lipid profile change and *H. pylori* infection.

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INTRODUCTION
Helicobacter pylorus is a Gram-negative bacterium, responsible for infection of the gastric mucosa [1]. Recent studies have suggested that, the H. pylori infection associated with the risk of coronary heart disease [2, 3]. Lipid metabolism is derailed by so many acute infections especially by Gram-negative bacteria [4]. Several studies have been shown that there is a decline in high density lipoprotein in microbial infections [5]. The effect of H. pylori infection on the serum lipid profile is still a matter of debate. The purpose of this study was to investigate the relation between H. pylori infection and the serum lipid profile.

MATERIALS AND METHODS
The study was done at Shridevi Institute of Medical Sciences & Research Hospital. The Institutional Ethical Committee clearance was obtained and utmost care was taken during experimental procedure according to the Declaration of Helsinki 1975.

The study was carried out in 24 male patients with H. pylori infection confirmed by urease breath test (UBT). The results were compared with 24 healthy controls included in the study. The patients having history of H. pylori eradication or gastric surgery, who had already received anti-hyperlipidemic therapy, severe liver or renal dysfunction or malignancy, were excluded from the study.

After obtaining a written consent from all the participants, total 3ml blood was withdrawn aseptically from the antecubital vein from each subject after 12 hrs overnight fast. The samples were centrifuged at 3000 rpm for 10 min to separate. The separated serum was used for analysis of all the parameters. Fasting levels of Total cholesterol, LDL-cholesterol, Triglycerides and Lipoprotein (a) were measured by enzymatic methods using Kits from Erba chem. Limited on spectrophotometer techniques (Model Chemiline CL/310 UV/VIS) in the Biochemistry lab, Shridevi Institute of Medical Sciences & Research Hospital.

Urease breath test (UBT):
The urea breath test (UBT) is one of the most important non-invasive and cheaper methods for detecting Helicobacter pylori infection. Urea with 13C was used, which was given orally at the dose of 100 mg and if H. pylori are present in the stomach its urease enzyme hydrolyses the urea producing isotopically labelled carbon dioxide. This diffuses into blood, is excreted by the lungs, and can be detected in breath. Breath samples were collected at 0 and 20 min after administration of a UBT tablet and UBT value was measured by infrared spectrometry. The cut-off value for the UBT was 2.5% at 20 min [6]. When UBT values were > 2.5% indicated as positive H. pylori.

Statistics:
The statistical analysis was carried out by using the SPSS- 16 (Statistical Package for Social Sciences) software. The Paired ‘t’ test was applied for the statistical analysis and P values <0.05 were considered as significant.

RESULTS

<table>
<thead>
<tr>
<th>Parameter (mg/dL)</th>
<th>Control Group (n= 24)</th>
<th>H. pylori infected patients (n= 24)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total cholesterol</td>
<td>169.32 ± 1.14</td>
<td>193.2 ± 2.69*</td>
</tr>
<tr>
<td>Triglycerides (TG)</td>
<td>106.2 ± 2.23</td>
<td>139.32 ± 1.73*</td>
</tr>
<tr>
<td>LDL-cholesterol</td>
<td>111.16 ± 2.54</td>
<td>121.08 ± 1.09*</td>
</tr>
<tr>
<td>Lipoprotein (a)</td>
<td>19.52 ± 1.07</td>
<td>24.048 ± 2.96*</td>
</tr>
</tbody>
</table>

The results are expressed as mean ± S.E.M
* P < 0.05 compared to the control group (Paired ‘t’ test)

DISCUSSION
H pylori, potentially involved in the pathogenesis of various extra-gastric diseases. The relation between H pylori infection and obesity, diabetes and on lipid profile remains controversial. Therefore, these associations should be interpreted cautiously.

In the present study we showed that, H. pylori positive males have higher serum triglycerides, total cholesterol, LDL, Lipoproteins when compared to the control subjects. The change in the lipid profile level may be due to derailment of lipid metabolism by Helicobacter pylori [7] or the production of pro-inflammatory cytokines, such as interleukins 1 and 6 (IL-1 and IL-6),
interferon-α and tumour necrosis factor-α (TNF-α). These cytokines are capable of affecting lipid metabolism in different ways, including activation of adipose tissue lipoprotein lipase, stimulation of hepatic fatty acid synthesis and influencing lipolysis [8].

Patients infected with H. pylori showed an atherogenic lipid profile characterized by an increase in LDL cholesterol or decreased HDL cholesterol compared to uninfected patients [9]. Our result also provided additional evidence supporting the hypothesis that H. pylori played a role in elevating the Total cholesterol, Triglycerides (TG), LDL-cholesterol, Lipoprotein (a). Based on these results, it is conceivable that H. pylori infection is a predisposing factor for the atherosclerotic process and can be a reliable indicator for the assessment of cardiovascular disease risk. Our study did not demonstrate the lipid profile changes after H pylori eradication treatment, this relationship needs to be confirmed in further prospective studies.

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REFERENCES