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## RESEARCH ARTICLE

### The biochemical changes associated with *Helicobacter pylori* infection

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#### Abstract

This study was conducted in laboratory of AL-Hussein Hospital , it was carried out on 40 patients infected with *H. pylori* ( 28 male and 12 female with mean age  $42.5 \pm 34.8$  years) and 23 healthy individuals with matched age mean , between December 2012 and March 2013. It aims to demonstrate the biochemical changes associated with *H. pylori* infection .The results reveal that the level of LDL was significantly ( $P < 0.05$  ) increased in those with *H. pylori* in comparison with control. However VLDL and blood sugar were significantly ( $P < 0.05$ ) decreased , while triglycerides , total cholesterol and blood urea were not significantly changed.

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## INTRODUCTION

*Helicobacter pylori* is a gram-negative bacterium that inhabits the gastrointestinal tract of humans (stomach and small intestine). It is aerophilic in nature, it is able to tolerate gastric acids due to its ability to produce an enzyme called urease. it is probably spread by consuming food or water contaminated with fecal matter (Wichelhaus A, et al., 2001). Urease can neutralize stomach acid, making it easy for the bacteria to survive in its own acid free zone. Because of their corkscrew shape, the bacteria can easily penetrate the stomach's protective mucous lining. *H. pylori* is commonly causes chronic infection of the gastric mucosa, and it is the most chronic bacterial infection, it persists for decades ( NYU. 2012). *H. pylori* releases of certain enzymes and toxins and activates the immune system, together these factors may directly or indirectly injure the cells of the stomach or duodenum. *H. pylori* is a causative agent for digestive disorders such as chronic gastritis, stomach and duodenal ulcer, and stomach cancer. The other suggested mechanism is that cytotoxin-associated gene A (*cagA*) protein produced by *H. pylori* could be an important contributor to the inflammatory disorders involved in the metabolic syndrome (Al-Fawaeir, et al., 2013)(Antherton J., 2006). *H. pylori* causes a chronic gastric infection, which has been associated with coronary heart disease (Laurila A, et al., 1999). To evaluate the mechanisms of this association. A number of previous studies have suggested that mild systemic inflammation provoked by *H. pylori* infection is associated with metabolic syndrome and atherosclerotic cardiovascular disease( Kim, H. et al., 2011). Some studies have demonstrated a relationship between *H. pylori* infection and extra digestive disease ( Aarabi M. H., et al., 2010). Cholesterol ,triglycerides and lipids, are insoluble in plasma, the later transport and circulating in plasma by lipoproteins to various tissues for energy utilization, steroid hormone production, lipid deposition and bile acid formation. Plasma lipoproteins are divided into 5 major classes based on their relative density: chylomicrons, very low-density lipoprotein (VLDL), intermediate-density lipoprotein (IDL), low-density lipoprotein (LDL), and high-density lipoprotein (HDL). The bacterium persistent inflammation or the expression of the cytotoxin-associated gene A (*CagA*) affect serum lipids as well as Lipoprotein a ( Chimienti G. et al., 2003). Besides their role in lipid transport, lipoproteins participate in immunity, which is the first line of host defense against invading microorganisms. In addition, anti-inflammatory effects of lipoproteins, especially HDL, have been demonstrated in both in vitro and in vivo studies. Circulating lipoproteins bind and detoxify lipopolysaccharides and toxins of gram-negative (Nassaji M. and Ghorbani R. 2012). Infection and inflammation produce a variety of profound changes in plasma lipid and lipoprotein concentration,

composition, and function. Many of the changes are induced by cytokines that are released during infections and inflammations (Carpentier Y.A. and Scruel O.2002). A new study by researchers at NYU Langone Medical Center reveals that the presence of *Helicobacter pylori* is associated with elevated levels of glycosylated hemoglobin (HbA1c), an important biomarker for blood glucose levels and diabetes .( NYU.2012)

This study was designed to investigate any possible association between *H. pylori* infection and serum lipid levels, blood sugar and blood urea.

### Materials and Methods:

The study was carried out on 40 *H. pylori* infected patients, between December 2012 and March 2013. Patients were included 28 males and 12 females with mean age  $42.5 \pm 34.8$  years and 23 healthy control with matched age mean . 5 ml of blood were collected from each patient or control. Blood samples were left for 1-2 hour at room temperature to clot, and then centrifuged at 3000 rpm for 10 minutes, sera were stored at  $-20^{\circ}\text{C}$  until used. *H. pylori* infection, was diagnosis by assaying serum anti- *H.Pylori* which determined by measurement of the serum anti- IgG antibody using an ELISA (IgG, EIA, Trinity Biotech, USA). According to the manufacturer's instructions. Triglyceride, total cholesterol, HDL, LDL ,VLDL, blood urea and glucose were measured for patients and healthy control with routine enzymatic methods using diagnostic kits( Biolabo SA.,Maizy ,France ). Normal levels based on the kit used were: cholesterol 130-200, TG <150, HDL 40-70, LDL<130.While B. sugar B. urea. were estimated by use of enzyme colorimetric kit (Randox Laboratories ,Ltd , Admore Antrim ,United Kingdom ). The data were compared between *H. pylori* infected subjects and healthy group. All testes done in the laboratory of AL-Hussein Hospital .

### Statistical analysis:

Values were expressed as mean  $\pm$ SD, differences between the mean values were analyzed by chi- square test ,the criterion for significance was ( $p \leq 0.05$ ) .

### Result:

The association between blood biochemical changes and the presence of *H.pylori* infection was summarized in (Table 1).

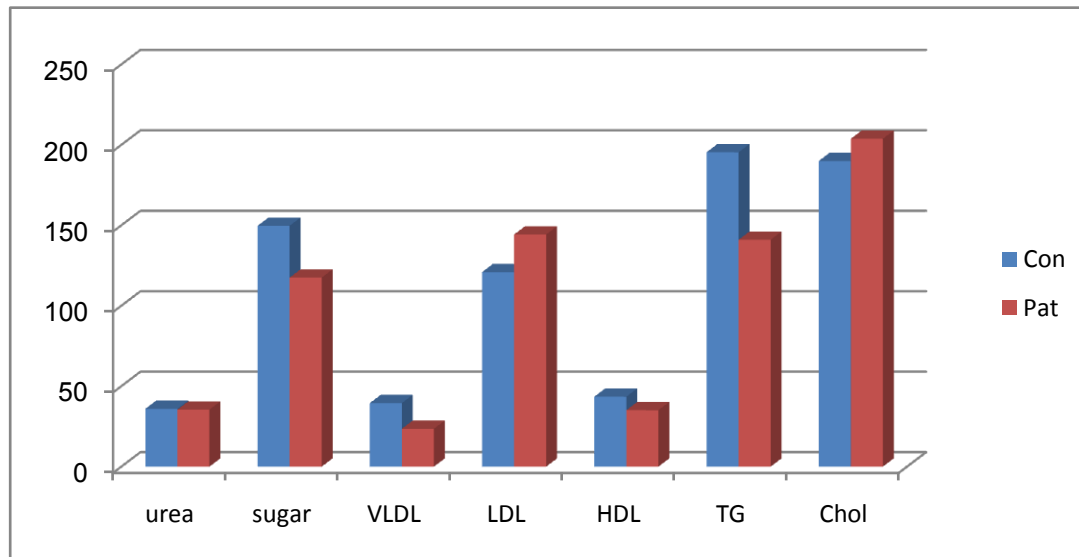
The level of LDL was significantly ( $P < 0.05$ ) increased in those with *H pylori* in comparison with control. However VLDL and blood sugar were significantly ( $P < 0.05$ ) decreased , while triglycerides , total cholesterol and blood urea were not significantly changed.

**Table- 1: Comparison of some biochemical parameters in *H. pylori* patients and healthy controls**

Test	Patients (N 40) mean $\pm$ SD	Control (N 23) mean $\pm$ SD	p-value
VLDL	23.55 $\pm$ 7.62	39.5 $\pm$ 20.60	P<0.05
LDL	144.9 $\pm$ 23.00	120.9 $\pm$ 16.40	P<0.05
HDL	35.08 $\pm$ 6.85	43.46 $\pm$ 7.51	P<0.05
Triglyceride	141 $\pm$ 145.00	195.4 $\pm$ 94.50	NS
Cholesterol	203.8 $\pm$ 31.80	190 $\pm$ 40.90	NS
Blood urea	35.45 $\pm$ 7.18	35.96 $\pm$ 7.36	NS
Blood sugar	117.5 $\pm$ 35.20	149.7 $\pm$ 29.60	P<0.05

NS: Not significant

**Figure -1: The changes in the blood biochemical parameters in *H. pylori* infected patients in comparison with healthy control.**



### Discussion:

The present results demonstrate significant increase in the LDL levels of patients compared to control. On the other hand, a significant decline was recorded in HDL and VLDL between patients and control. Elevation of LDL in patients was documented by several studies (Aarabi M. H, et al., 2010) (Viswanath V., et al., 2013). Our study showed that total cholesterol was also elevated, but it didn't reach the significant limit, however, this study also showed that HDL was significantly decreased. Laurila et al 1999 (Laurila A, et al., 1999) (Chimienti G, et al., 2003) found that *H. pylori*-seropositive patients have higher serum cholesterol and lower HDL than sero-negative patients. These results, regarding cholesterol level, were in agreement with our results. However regarding HDL, similar results were previously reported (Tanriverdi Ö 2011) (Ugwu N C, et al., 2008). The authors suggested that *H. pylori* affected the lipid metabolism.

However, our results were in agreement with that recorded by Aarabi et al 2010 (Aarabi M H, et al., 2010) who found that the triglyceride level was higher in *H. pylori* seropositives than in negatives. However Volanen et al 2005 (Volanen I, et al., 2005) found that serum triglyceride and HDL-cholesterol levels can change during the acute phase of bacterial infection. The mechanism of how *H. pylori* infection modifies the serum lipid profiles could be attributed to the systemic inflammatory response to the bacterium which induces changes in lipid and lipoprotein metabolism (Khovidhunkit W., et al., 2010). The exact pathophysiology underlying the change in the level of serum lipids in severe illness and infection was not fully understood. Different mechanisms were suggested, including imbalance between synthesis and utilization of plasma lipids, usage of lipids to restore damaged cell membranes, and interaction of cytokines and bacterial toxins with lipids (Nassaji M and Ghorbani R. 2012). Alterations in the composition and function of lipoproteins, due to decreased reverse cholesterol transport, and increased oxidation of lipids occurs by bacterial infection (Khovidhunkit W., et al., 2010). Accordingly, it was suggested that increasing the low plasma lipoprotein concentrations that are common during acute illness was a therapeutic option for preventing and treating the clinical syndromes associated with infection or immune response produce changes in plasma lipid. *H. pylori*, is one of the infections caused metabolic disorders leading to change of lipid profile and increase the risk factors of cardiovascular diseases (Al-Fawaeir S., et al., 2013).

There was significant decrease in blood glucose level in association with *H. pylori* in our study, this could be attributed to the nature of epidemiology of *H. pylori* infection which showed significant increase in hypoglycemic subjects especially type 2 diabetes (Manco M. et al., 2010; Assal A H. et al., 2013). However, El-Hadidy, et al., 2009 found no significant variations in the prevalence of *H. pylori* infection among diabetics and non diabetics.

Many authors mentioned that, serum urea nitrogen level was significantly associated with *H. pylori* prevalence but there is no significant evidence that *H. pylori* infection is directly associated with progression of renal dysfunction (Shousha S, et al., 1990; Eiyas J, et al., 2009; Taher M, et al., 2012) these finding could be related to the age of patients

and not to the infection, so Jaspersen D, et al., 1995 found no relationship between *H. pylori* colonization and serum urea. (as recorded in our results.

### Conclusion:

Our results assume that there is an association between *H. pylori* infection with various lipid profile changes and. Increased serum level of LDL and could be considered as metabolic disorder which may led to increasing the risk factors of cardiovascular diseases.

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