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Original Research Article

Levels of Interleukin - 40 and Lipid Profile in patients with *Helicobacter pylori* Infection

Khalil Ismail Abid Mohammed[1*], Ameen Abdul Hasan AL-Alwany[2], Saad Hasan Mohammed Ali[1], Wifaq M. Ali[1], Suha A. AL-Fakhar[1], Jinan M. Mousa[1]

[1]Clinical Communicable Disease Research Unit /College of Medicine, University of Baghdad, Baghdad, Iraq
[2]Department of Internal Medicine, College of Medicine, University of Baghdad, Baghdad, Iraq

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***Corresponding author:** Khalil Ismail Abid Mohammed
Clinical Communicable Disease Research Unit /College of Medicine, University of Baghdad, Baghdad, Iraq
Email: dr.khalilmohammed1967@gmail.com

Abstract

The study was carried out to detection of *H.pylori* in (200) patients who attended two teaching hospitals in Baghdad. The diagnosis done by Immunochromatography methods. Stools and blood samples was taken from each patient as well as other (30) healthy control matching in their age. The study included detection the Levels of Interleukin-40 and CRP in sera of patients and control. The result indicated presence of H pylori antigen in 115 cases 59 cases of males and 51 of females, Also, the result indicated increasing levels of IL-40, cholesterol, Triglycerides, Low density lipoprotein, Very Low density lipoprotein increased significantly while the level of High density lipoprotein decreased in patients sera in comparison with healthy control.

Keywords: *Helicobacter pylori*, IL-40, cholesterol, Triglycerides, Low density lipoprotein, Very Low density lipoprotein, very Low density lipoprotein.

INTRODUCTION

H. pylori are a spiral-shaped, gram-negative, microaerophilic rod with 4–7 flagella. The flagella help in the settlement of the bacterium to the gastric mucosa layer [1]. The virulence factors of *H. pylori* can cause gastric inflammation, disruption of the gastric mucosal barrier, and extra gastric effect [2]. Chronic inflammations are the critical component of the disease process of H. pylori, which is the initial step starting from superficial gastritis to chronic gastritis, intestinal metaplasia to dysplasia, and until invasive adenocarcinoma [3, 4]. H.pylori promote gastric inflammation by stimulating interleukin (IL-8) secretion from the gastric cell, inducing neutrophil-endothelial cell interactions, activating platelet-activating factor, injecting the lipopolysaccharide of *H. pylori* to the gastric mucus coat, and secreting urease enzyme [5-7].

H. pylori also disrupt vacuolating cytotoxin, reactive oxygen species, and induce programmed cell death [4, 5]. Infected with *H. pylori* leads to an increase in inflammatory adhesion molecules such as intracellular adhesion molecule-1 and inflammatory mediator cytokines such as hsCRP, IL-1, IL-6, and tumor necrosis factor-α (TNF- α). *H. pylori* infection causes malabsorption of nutrition such as vitamin B12 and folic acid which leads to an increase in serum homocysteine levels and results in endothelial dysfunction [8, 9]. *H. pylori* infection resulting not only in inflammation but also causes the accumulation of reactive oxygen species (ROS) and oxidative DNA damage in the gastric mucosal layer. Therefore, the accumulation of ROS has been resulting in the initiation of multiple disease processes the gastric mucosal barrier because by expressing which have a contribution to the pathogenesis of cardiovascular disorders through the expression of adhesion molecules, stimulation of vascular smooth muscle proliferation and migration, apoptosis in the endothelium, and oxidation of lipids [10, 11].

The research aimed to detect the presence of Helicobacter pylori (H. pylori) in 200 patients who sought medical attention at two prominent teaching hospitals in Baghdad.

MATERIALS AND METHODS

During a period of eight months from December 2022 to April 2023, a study was conducted at two teaching hospitals in Baghdad on freshly collected stool samples from a total number of 200 cases of gastroenteritis among adult patients. Stool samples were collected from each patient in sterile disposable screw cap containers. These were labeled with number, date, and name of each subject. A questionnaire containing demographic, clinical, and environmental data was obtained from each case. The existence of *H. pylori* in fresh stool samples was investigated at the microbiology laboratory of the using an immuneochromatographic test.

H. pylori Antigen Detection

Immunochromatographic assay (Weifang Kanghua Biotech china) for antigenic detection of *H.pylori* and were done according to instructions of the manufacturers. Allowing the card –device, test reagents and stool samples to reach to room temperature prior to testing. A separate stool collection tube and device were used for each sample and the assay was done right after collection. To detect *H.pylori*, approximately 100mg or 100 microliter of stool sample was put and shaken in collection tube containing the diluents. Four drops or 100µl was dispensed in the circular window of the card. The results (appearance of the colored bands) were read after 10 minutes. This *H.pylori* KIT is qualitative Immunochromatographic assay for determination of *H.pylori* in fecal samples. The membrane on the test band region is pre coated with mouse monoclonal antibodies against *H.pylori* antigens. During testing, the sample is allowed to react with the colored conjugates (anti-H.pylori mouse monoclonal antibodies-red microspheres) which were pre-dried on the test. The mixture then moves upward on the membrane by capillary action. As the sample flows through the test membrane, the colored particles migrate. In the case of positive result, the specific antibodies present on the membrane will capture the colored particles and a red colored line becomes visible.

The mixture captures the colored particles and a red colored line becomes visible. The mixture continues to move across the membrane to the immobilized antibody placed in the control band region, a red- colored band always appear. The presence of this red band serves as 1-verification that sufficient volume is added, 2-that proper flow is obtained and 3-as an internal control for the reagents. Insufficient specimen volume, incorrect procedural or deterioration of the reagents are the most likely reasons for control line failure. Negative results were indicated by only one green band (control line). For positive result, in addition to the Red control band, a red band also appear on the site of result line. A total absence of the control colored band (Red) regardless the appearance or not of the result line (red) was evaluated as an invalid result.

Blood samples

Three mL of Venous blood was obtained from each patients and collected in sterilized screw cap plastic tube, blood samples were left for 30 min. at room temperature, then centrifuge at 3000 rpm for five minute, then the serum for each sample was collected in eppendorf tubes and stored in deep freeze at - 20c°until the time for using. The current study included Immunological & Clinical biochemical aspects. The level of interleukin -40 (IL-40) estimated by ELISA according to manual procedure of Sunlong Biotech, China). Serum HDL, Triglyceride, total cholesterol, LDL-cholesterol were analyzed by BS-230 Full automated biochemical analysis (Mindray). The reference range of HDL (40–60 mg/dL), TG (<130mg/dL), TC (<200 mg/dL), LDL (<130 mg/dL).

Statistical Analysis

The results were analyzed using statistical system SPSS version -18 (T-testing).

RESULTS

Gender

Distribution of *H. pylori* patients according to their gender, were studied, among them 59 were males out of 110 and 56 were females out of 108. In a general *H. pylori* antigen was revealed in 115 of fecal samples out 200 (Table-1).

Table-1: Distribution of *H. pylori* patients according to their gender

H pylori Antigen	Total	Positive		Negative	
		No.	%	No.	%
Male	110	59	53.63	51	46.37
Female	90	56	51.85	52	48.15
Total	200	115	52.75	103	47.25

Immunological parameters

The levels of the IL-40 increased significantly ($p \leq 0.05$) in patients suffering from *H. pylori* in comparison the healthy individual (Table-2).

Table-2: Levels of IL-40 (ng/L) in patients sera and healthy control

Groups	IL-40
patients(HP+)	9.88 ± 4.20
control(HP-)	5.18 ± 1.1
*** (P<0.05) significant**	

Biochemical parameters

The levels of cholesterol, Triglycerides, Low density lipoprotein, Very Low density lipoprotein, very Low density lipoprotein increased significantly while the level of High density lipoprotein in patients suffering from *H. pylori* in comparison the healthy individual (Table-3).

Table 3: Lipid profile in patients serum with *H. pylori* and Healthy control

Variables	H. pylori Positive	H.pylori Negative
Serum T C	178.25 ± 38.12	144.50 ± 20.04
Serum T G	155.18 ± 41.77	127.34 ± 53.76
Serum HDL	35.34 ± 8.43	44.32 ± 6.46
Serum LDL	110.53 ± 12.18	100.13 ± 17.24
Serum VLDL	31.22 ± 1.60	26.43 ±2.32
*** (P<0.05) significant**		

DISCUSSION

Helicobacter pylori was identified in 115 stool samples of patient out of 200 samples (Table - 1). The infections may be due to lack of sanitary facilities and poor living condition among the major causes of infection. The result was consistent with that reported in Diyala by Hasan *et al*., [12], in Basrah by Al-Hamdi and Khashan [13]. But the variation in the rate of infection between different studies may be due to the type of the sample (blood, stool and tissue), size of the sample, place and period of the study and techniques used for detection of the bacteria. The rate of infection in males was higher than females. the results in line with other results were reported in Diyala by Al-Ezzy [14]. The Result indicated an increased level of IL-40 in *H. pylori* patients (Table-2) in comparison with healthy control probably contributes to inflammatory response in patients.

H. pylori induce the secretion of TGF- ß and IL-10 which play a vital role in the prevention of infection-induced immunopathology or prolongation of persistence, by suppressing Th1 responses [15, 16]. TGF- ß potentiates the expression of IL-40 by activated B cells [17]. The ability of cytokines like IL-10 and TGF-ß to potentiate IL-40 production by B cells suggests that optimalIL-40 expression by B cells may require a specific differentiation mechanism to achieve optimal production [18].

The increasing level of cholesterol, Triglyceride and Low density Lipoprotein may be due to ability of *H. pylori* infection to linked with modified lipid profile. These alterations are may be mediated by cytokines, particularly TNF-α, which inhibits lipoprotein lipase and thus results in the mobilization of lipids from tissues as well as increased serum TG and reductions in HDL. The induction of TGF- ß altered lipid profile, including an increased LDL level and decreased HDL level, is one of the most important risk factors for atherosclerotic disease [19, 20].

CONCLUSION

The result indicated presence of H pylori antigen in 115 cases 59 cases of males and 56 of females, Also, the result indicated increasing levels of IL-40, cholesterol, Triglycerides, Low density lipoprotein, Very Low density lipoprotein increased significantly while the level of High density lipoprotein decreased in patients sera in comparison with healthy control.

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