Original Article

Investigation of Serum Interleukin-1β and Interleukin-10 Levels in *Helicobacter pylori* Infected Patients

Nadia Saad Ali, Mohammed Yawoz Noraldeen[1], Najdat Ali Al-Kadhi[1]

Kirkuk Health Department, Ministry of Health, Altakhi Health Care Center, Kirkuk II Sector, [1]Department of Medical Laboratory Techniques, College of Health and Medical Techniques, Northern Technical University, Kirkuk, Iraq

Abstract

Background: *Helicobacter pylori* is a human pathogen colonizes epithelial stomach. In the development of diseases, cytokines play crucial functions and may have an impact on the disease process. **Objectives:** To estimate the levels of interleukin-1β (IL-1β), interleukin-10 (IL-10), and cytotoxin associated gene (CagA) IgG in individuals infected with *H. pylori*. **Materials and Methods:** The study, performed from November 2022 to April 2023, included 66 individuals divided into two groups: 40 patients (60% male, 40% female patients) with a mean age of 45.25 who had positive *H. pylori* stool antigen tests, and 26 healthy individuals as a control (50% males, 50% females) mean age 45.26. Two types of samples had been collected: stool samples for the quick identification of *H. pylori* antigen by the immunochromatographic cassette, and 5 mL of venous blood to quantitatively estimate serum *H. pylori* IgG, *H. pylori* CagA IgG, IL-1β, and IL-10 using sandwich enzyme linked immunosorbent assay technology. **Results:** There was a highly significant increase ($P = 0.01$) in *H. pylori* IgG titers in the patients (male 89.49 ± 24.03, female 93.35 ± 25.18) compared to the control (male 47.92 ± 20.13, female 62.26 ± 19.03), and *H. pylori* CagA IgG (male 354.7 ± 29.4, female 356.2 ± 23.0) compared to control (male 229.5 ± 25.4, female 219.3 ± 27.1). Also, there was a highly significant increase ($P = 0.01$) in the levels of IL-1β and IL-10 in patients compared to control. **Conclusion:** The individuals infected with *H. pylori* had elevated levels of IL-1β and IL-10.

Keywords: CagA, *Helicobacter pylori*, interleukin-10, interleukin-1β

Introduction

Helicobacter pylori, a gram-negative microaerophilic human pathogen bacterium, is one of the major risk factors for gastrointestinal diseases in humans. It has been associated to a number of gastro-intestinal conditions, such as stomach cancer, peptic ulcer, and chronic gastritis.[1-4] It is estimated that more than 50% of people worldwide are infected with this bacteria. *Helicobacter pylori* primarily affects children. Because of socioeconomic factors, the annual rate of infection rises in developing countries among adults and decreases in developed nations. Fecal-oral, oral-oral (dental), gastro-oral, and sexual conduct are the routes through which the disease is spread.[5-7] During the progression of gastrointestinal disorders, *H. pylori* was discovered to have a strong relationship with the host immune system, where it causes the production of several proinflammatory cytokines such interleukin-1β (IL-1β), and tumor necrosis factor-alpha and anti-inflammatory cytokine interleukin-10 (IL-10).[1,8] It has a number of virulence factor including Cytotoxin associated gene (CagA), vacuolating cytotoxin A, urase enzyme, flagella, and lipopolysaccharides, which play a role in the onset of this inflammatory process.[9-11]

Four steps are essential for *H. pylori* colonization and pathogenesis after entering the stomach of the host: (1) resisting an acidic stomach environment by secreting urease that hydrolyzes urea into ammonia and carbon dioxide, which neutralize acidity and form a buffer layer around *H. pylori*.[12] (2) moving toward epithelial cells by flagella-mediated motility from areas of high pH to areas

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Address for correspondence: Prof. Nadia Saad Ali, Kirkuk Health Department, Ministry of Health, Altakhi Health Care Center, Kirkuk II Sector, Kiruk, Iraq.
E-mail: nadia.saad@ntu.edu.iq

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of low pH.[13] (3) adhesin-mediated attachment to host receptors: *H. pylori* has a number of adhesion proteins that are all important because they enable bacterial adherence to gastric epithelial cells, induce many inflammatory cytokines secretion, and stimulate immune cell activation and infiltration.[12,13] (4) By releasing toxins that cause tissue injury, like vacuolating cytotoxin A (VacA), that produce vacuoles (pores) on a host cell and make it vulnerable to the activities of the urease enzyme of *H. pylori*. It also stimulates the autophagy pathways in cells.[14] CagA is involved in the damage of host tissue and intracellular replication. It causes cellular change and cytoskeleton alteration that impair motility, proliferation, and apoptosis, as well as inducing gastric epithelial cells to secrete inflammatory cytokines.[15] Also considered a main mediator of carcinogenesis because it affects epithelial cell junctions, prevents H+/K+-ATPase, and influences intracellular pathways that will determine cell proliferation and transformation.[16]

Both innate and adaptive immunity activate during *H. pylori* infection led to stimulation of immune cells and subsequent generation and release of a wide spectrum of pro-inflammatory cytokines.[17] Duodenal ulcer disease and atrophic gastritis are both highly influenced by the level of stomach acid secreted and the production the response of a pro-inflammatory.[18] IL-1β is a strong pro-inflammatory cytokine.[19-21] The polymorphic IL-1β and IL-RN genes are found in a gene cluster that also encodes the IL-1β cytokine, the most effective known blocker of acid secretion.[19,22] IL-10 is believed to play a role in regulating human immunological responses. This cytokine is one of the most significant regulatory interleukins that affects antigen-presenting cells by suppressing cytokines and human leukocyte antigen class II. Furthermore, the proliferation and differentiation of T cells are directly influenced by them.[23]

Therefore, the study aimed to estimate the levels of pro and anti-inflammatory cytokines in individuals infected with *H. pylori* in addition to evaluate the concentration of CagA virulence factor.

Materials and Methods

In this study, performed from November 2022 to April 2023, 66 individuals (males and females) visited at Azadi Teaching Hospital in Kirkuk City. In that, 40 of them (24 males and 16 females patients) had positive *H. pylori* stool antigen tests, and their ages ranged from 15 to 73 considered a patient group, and 26 as control groups, which included healthy (13 males and 13 females) persons with negative *H. pylori* stool antigen test, ranging in age from 21 to 69 years. They answered the questioner's requests about their age, sex, body mass index, type of medication, duration of diabetic history, other chronic diseases, etc.

Samples

Two types of samples included in this study: stool samples for the quick identification of *H. pylori* antigen utilizing the immunochromatographic approach (Neonostics cassette, Suzhou, China). A distinctive pink band is clearly visible in the test line regions as a positive outcome, in addition to a pink line in the control line regions. Although negative, there are no lines in the test line area. 5mL of venous blood samples were obtained and placed in plain tubes, and then spun at 3000rpm for 10min in centrifuge to separate the serum samples. The serum samples were then transferred to Eppendorf tubes and kept at –20ºC until all samples had been collected. Using sandwich enzyme linked immunosorbent assay (ELISA) (BioTek, USA), to quantitatively estimate serum *H. pylori* IgG, *H. pylori* CagA IgG, IL-1β, and IL-10 (Sunlong Kits, China).

Statistical analysis

Statistical analysis was done by the statistical programs statistical package for the social sciences IBM version 26 to measure the mean and standard deviation (mean ± SD) between groups using the one-way one-way analysis of variance. Probability value > 0.05 is considered non-statistically significant; P value < 0.05 is considered statistically significant; and P value < 0.01 is highly statistically significant. Correlation was done by using Pearson's bivariate test, IBM version 26 (Chicago, IL, USA).

Exclusion criteria

Patients with a prior *H. pylori* infection who had been receiving treatment for it, patients who had a history of *H. pylori* antibiotic treatment, and patients who were taking long-term, persistent non-steroidal anti-inflammatory drug therapy.

Ethical approval

The study protocol, subject data, and consent forms were reviewed and approved by a local ethics committee in accordance with the document number (1-11-2022/48204).

Results

The participants in this study amounted to 66 participants who were divided into two groups according to the result of the *H. pylori* antigen test in the faces, and 40 of them were positive for this test and were classified as patients, whereas 26 were negative for this test and were classified as the control group.

Table 1 shows the age, sex, body mass index (weight/height^2), and smoking behavior between the two groups of *H. pylori*-positive patients and the control group.

Concentration of serum *H. pylori* IgG between groups

Table 2 shows a highly significant increase (P = 0.01) in *H. pylori* IgG titer in the patient group male (89.49 ± 24.03), female (93.35 ± 25.18) compared to the control (male 47.92 ± 20.13, female 62.26 ± 19.03).

Concentration of serum *H. pylori* CagA IgG between groups

Concentration of *H. pylori* CagA IgG in Table 2 shows a highly significant increase between the patient (male

354.7 ± 29.4, female 356.2 ± 23.0) and control (male 229.5 ± 25.4, female 219.3 ± 27.1) groups ($P = 0.01$).

Concentration of serum IL-1β between groups

Table 3 illustrates the concentration of IL-1β was highly significant increase ($P = 0.01$) in the patients group compared to the control. Where the concentration of IL-1β was (male 93.92 ± 11.46, female 100.11 ± 13.74) in the patients group and (male 45.83 ± 10.49, female 49.90 ± 10.26) in the control group.

Concentration of serum IL-10 between groups

The concentration of IL-10 showed a highly significant increase (male 75.65 ± 7.350, female 76.36 ± 8.080) in the patients group compared to (male 46.00 ± 8.060, female 47.97 ± 8.920) in the control group ($P = 0.01$) as shown in Table 3.

Table 1: Age, gender, body mass index, and smoking behavior between study groups

Groups parameters	*H. pylori* positive patients	Control
H. pylori stool antigen test	(40) positive	(26) negative
Age		
Range	21–73	21–69
Mean	45.25	45.26
Gender		
Male	(24) 60%	(13) 50%
Female	(16) 40%	(13) 50%
BMI		
18–23	7	8
23.5–28.5	22	14
29–34	11	4
Smoking		
Yes	9	0
No	31	26

BMI, body mass index

In Table 4, we noticed a positive correlation between *H. pylori* IgG with CagA IgG and IL-1β, while there was a negative correlation between *H. pylori* IgG with IL-10.

Discussion

In this study, we determined the level of serum *H. pylori* IgG and *H. pylori* CagA IgG titers in patients whose stool antigen tests for *H. pylori* gave positive results in addition to a control group with negative results. We found both titers were higher in the patient group compared to the control group. *H. pylori* triggers a systemic antibody response.[24] A study suggests that IgG antibody detection in serum is used to determine whether *H. pylori* colonization has occurred. The degree of gastritis is correlated with the height of the antibody titer.[25] The stomach epithelial damage and persistent inflammation induced by *H. pylori*, especially with its virulence factors CagA was associated with chronic infection and the response of the immune system.[26] Therefore, we saw an increase in the level of virulence factor in people infected with *H. pylori*. Moreover, during the progression of gastrointestinal diseases, *H. pylori* was found to have a strong relationship with the host immune system. *H. pylori* causes the production of numerous proinflammatory and anti-inflammatory cytokines, including IL-1β, IL-10, and tumor necrosis factor-alpha.[27] Pathogen-associated

Table 4: Correlation between serum *H. pylori* IgG with CagA IgG, IL-1β and IL-10 in patients infected with *H. pylori*

Markers	Serum *H. pylori* IgG	
	R-value	*P*-value
H. pylori CagA IgG	0.610	0.000
IL-1β	0.533	0.000
IL-10	–0.308	0.053

R: correlation coefficient

Table 2: Concentrations of *H. pylori* IgG and *H. pylori* CagA IgG in patients and control groups

Groups parameters	*H. pylori* positive patients		Control group		P-value
	Mean ± SD		Mean ± SD		
	Male (24)	Female (16)	Male (13)	Female (13)	
H. pylori IgG (pg/mL)	89.49 ± 24.03	93.35 ± 25.18	47.92 ± 20.13	62.26 ± 19.03	0.01**
H. pylori CagA IgG (pg/mL)	354.7 ± 29.4	356.2 ± 23.0	229.5 ± 25.4	219.3 ± 27.1	

0.01**: highly significant difference, SD: standard deviation

Table 3: Concentrations of serum IL-1β and IL-10 in patients and control groups

Groups parameters	*H. pylori* positive patients		Control group		*P*-value
	Mean ± SD		Mean ± SD		
	Male (24)	Female (16)	Male (13)	Female (13)	
IL-1β (pg/mL)	93.92 ± 11.46	100.11 ± 13.74	45.83 ± 10.49	49.90 ± 10.26	0.01**
IL-10 (pg/mL)	75.65 ± 7.350	76.36 ± 8.080	46.00 ± 8.060	47.97 ± 8.920	

0.01**: highly significant difference, SD: standard deviation, IL: interleukin

molecular patterns from *H. pylori* as well as damage-associated molecular patterns from destroyed epithelial cells are what stimulate inflammation, which react to pattern recognition receptors in immune and nonimmune cells and initiate subsequent signaling cascades, resulting in the formation and release of pro- and anti-inflammatory cytokines to modify the immune response.[28]

In our study, we found that IL-1β and IL-10 were increased in *H. pylori*-infected patients. The scientific explanation for these results is that when *H. pylori* reaches the submucosa, the dendritic cells serve as the primary antigen-presenting cells. Gastric epithelial cells express certain toll-like receptors (TLR) that recognize many *H. pylori* factors; for example, TLR2, TLR3, TLR4, TLR5, TLR9, and TLR10 can recognize lipopolysaccharide, peptidoglycan, flagellin, RNA, and DNA for *H. pylori* and stimulate intracellular signaling pathways through the activation of nuclear factor kappa B (NF-kB), a transcription factor that provokes the release of pro-inflammatory cytokines (IL-1β) and anti-inflammatory cytokine (IL-10).[29] The results of our study are in agreement with many other studies; for example, Jang demonstrated that *H. pylori* T4SS and flagellin are necessary for IL-1β secretion by neutrophils. When flagellin is detected by TLR5, NF-kB is activated, and then pro-inflammatory cytokines are produced.[30] Furthermore, Kim shows that cagPAI, a key virulence factor in *H. pylori*, stimulates IL-1β production by enhancing the transcriptional induction of IL-1β.[31] Pérez-Figueroa *et al.*[32] recently revealed that *H. pylori* stimulates human neutrophils to produce IL-1β in a manner that is independent of TLR2 and TLR4.

IL-1β produced by a variety of cells, including immune cells, epithelial cells, and fibroblasts, IL-1β can also be characterized as an important inducer and activator of inflammation.[33,34] Because of IL-1β has been shown to enhance the host's inflammatory response in response to both endogenous and exogenous stimuli, as well as to other cytokines like IL-2, IL-6, IL-12, and TNF-α, as well as other pro-inflammatory mediators (like inducible nitric oxide synthetase and cyclooxygenase 2), it has been concluded that IL-1β is a key regulator of the host's inflammatory response.[35] Furthermore, it has recently been demonstrated that genetic variations in the IL-1β gene's promoter regions change cytokine expression, resulting in a hypoacidity environment that supports the long-term survival and colonization of *H. pylori*.[36] Thus, the hyperproliferation of gastric epithelial cells and subsequent oncogenesis may be attributed in large part to the overexpression and increased release of IL-1β.[37]

We have also noticed an increase in the level of IL-10 in people with *H. pylori*. There are many studies that mention this association.[38,39] A study achieved by Jalal that is compatible with our results recorded a high level of IL-10 among the children patients infected with *H. pylori* compared to healthy controls.[40] Depending on the early stimulation of tyrosine phosphorylation of Jak1 and tyk2, which subsequently phosphorylate the intracellular domain of IL-10 receptor, IL-10 works in a similar way to IL-6 via the STAT3 motivation.[41] Its effects on immune system responses are included in its ability to control the activity of antigen presenting cells and macrophages, as well as the expression and production of pro-inflammatory cytokines by T-lymphocyte cells.[42] *Helicobacter pylori* commonly utilizes IL-10's immunosuppressive function to escape the defense mechanisms of the host, survive, and remain for a long time in the gastric milieu.[1]

Therefore, we conclude from the results obtained in this study that *H. pylori* with its virulence factor CagA can stimulate the production of inflammatory interleukins such as IL-1β and IL-10 in people infected with it.

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Conflicts of interest
There are no conflicts of interest.

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