Original Article

Correlation between *H. pylori* Infection and IL-8 and IL-17 in Chronic Gastric Patients in Iraq

Israa Hamza Jasim Al-Mamoory[1,2], Alaa Hani Al-Charrakh[1], Hadi F. Al-Yasari[1]

[1]Department of Microbiology, College of Medicine, University of Babylon, Hilla, Iraq, [2]Department of Medical Laboratory, College of Medical and Health Technologies, University of Alkafeel, Najaf, Iraq

Abstract

Background: Several studies revealed that Helicobacter pylori can stimulate innate immunity by activating recurrent macrophages and neutrophils to initiate inflammation and also stimulate adaptive immunity mediated by the naïve T cell variation into Th 1, Th 17, and Th 2 immune cells. **Objectives:** This investigation examined the serum levels of two interleukins (IL-8 and IL-17) and the likely associations between them in *H. pylori* infection patients and healthy individuals at various stages. **Materials and Methods:** This study involved 145 participants, 80 of whom had *H. pylori* infection and 65 were healthy individuals. Serum levels of the ILs were measured using the enzyme-linked immunosorbent assay technique. The data were statistically analyzed to assess the socio-demographic characteristics and the correlation between ILs in both groups. **Results:** The results showed that women had more early-stage *H. pylori* infection diagnoses, while men had more advanced-stage cancer diagnoses. Younger people, men, and those with early *H. pylori* infection had higher levels of ILs. The levels of IL-8 and IL-17 were higher in the *H. pylori* infection groups. A strong correlation was found between IL-8 and IL-17 levels in both groups ($P = 0.0001$). **Conclusion:** This study suggested that cytokine variation profiles could be useful for detecting *H. pylori* infection and predicting its outcome.

Keywords: Gastric patients, *H. pylori*, IL-17, IL-8

Introduction

Helicobacter pylori is co-existing with individuals for more than 110,000 years. *H. pylori* is generally transferred in childhood and can persist in the tissues of the gastrointestinal tract, especially the stomach, for a long period to persist years or decades or even a whole lifetime. *H. pylori* affected approximately 255 million people in 2020, with a prevalence of more than (50%) of the worldwide population, according to the Global Burden of Disease. In Iraq, the prevalence of *H. pylori* infection was 50% in older individuals and 56% in younger children.[1-3]

H. pylori infection is a condition that is mainly caused by complex bacteriological virulence factors and their relationship with the human immune response and environmental influences, such as genetic causes. Atypical bacterial infections play a role in both gastritis and the stimulation and exacerbation in children and adults. In addition, numerous genetic polymorphisms and immune factors have been related to susceptibility to *H. pylori* infection and antibiotic resistance.[4-6]

H. pylori is microaerophilic, Gram-negative, spiral-shaped (S or curved-shaped bacteria), and motile by unipolar flagella. The bacterial cell has a unique characteristic that allows adaptation to the normal habitat of *H. pylori* in the human stomach. *H. pylori* is involved in the pathogenesis of several inflammatory degrees, including peptic ulcers.[7,8]

Based on several studies, *H. pylori* can stimulate innate immunity in the stomach by activating recurrent macrophages and neutrophils to initiate inflammation

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Address for correspondnece: Dr. Alaa Hani Al-Charrakh, Department of Microbiology, College of Medicine, University of Babylon, Hilla, Iraq.
E-mail: ahani67@gmail.com

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and also stimulate adaptive immunity mediated by the naïve T cells' differentiation into Th 1, Th 17, and Th 2 immune cells.[9]

Interleukin (IL)-8 intermediates important pro-inflammatory functions in gastritis, involving the differentiation of T-helper 2 cells to release different cytokines, the promotion of macrophages, neutrophil movement across the endothelium, the expression that sustains the inflammatory cascade, and the gastric epithelium's reaction with the bacterium when macrophages are activated by Toll-like receptors with bacterial *VacA*, *CagA,* and other virulence factors.[10]

IL-17 is an important pro-inflammatory cytokine produced by intestinal Th17 cells after differentiation due to bacterial infection. IL-17 is the most vital pro-inflammatory cytokine and can generate significant inflammatory responses even when existing in small amounts. IL-17 plays an essential role in chronic inflammation, gastric precancerous lesions, and gastric adenocarcinoma.[11,12]

Materials and Methods

Subjects and study design

This case–control study included individuals with gastric disorders of different ages who were attended to during the period extending from December 2023 to April 2024; these samples were collected from Al-Sader Hospital, a special digestive system center in AL-Najaf City, Al-Sadiq Hospital in Babylon Province, and from private laboratories in AL-Najaf City. This study indicated that their ages ranged from <15 years to 70 years, and their genders of them were included males and females for approximately 80 samples. Samples were taken using a sterile container and then transported to the laboratory (stool and blood). Samples were divided for serological (stool) and immunological (blood) analyses. The sample size: Prevalence 34% = 80 samples.

Data collection

Demographic and clinical data were collected by obtaining information from patients and monitoring them. Sociodemographic and observation of the data: sex, age, weight, marital status, student status, diabetes mellitus, address, and family history of other diseases such as cancer and exposure to smoking.

Gastric severity and control

The degree of *H. pylori* infection and controls were identified depending on the international standards for diagnosis mentioned in the National Library of Medicine (National –Center For Biotechnology Information and WHO.

The degrees of *H. pylori* infections:

1. Mild or moderate *H. pylori* infection.
2. Severe *H. pylori* infection (ulcers and exudate): bacteria infect the stomach during childhood and adulthood, leading to peptic ulcers.

Rapid test

Rapid testing is an immunoassay for the rapid detection of *H. pylori* antigens in stool samples. Rapid test cassette: This test has antibodies that react with bacterial antigens to detect infections by the High-top kit according to the instructions from Shandong, China. The mixture travels and aggregates on the membrane by duct action to react with antibodies (anti-*H. pylori*) and produces a colored line. The presence or absence of this colored line shows positive or negative results, respectively.

Determination of interleukin-8 and interleukin-17 by the enzyme-linked immunosorbent assay technique

The enzyme-linked immunosorbent assay (ELISA) method for quantitative determination of human IL-8 and IL-17 concentrations in serum from patients was performed according to the manufacturer's instructions (Beijing Solar Bio Science and Technology Co.Ltd, China). This assay employs the quantitative sandwich enzyme immunoassay technique. A monoclonal antibody specific for IL-8 and IL-17 has been pre-coated on top of a microplate. The standards and samples are added into the wells of the microplate, and any presenting cytokines (IL-8 or 17) are captured by the coated antibody after incubation.

The ELISA results were calculated based on the optical density readings for each standard and sample's optical density. Then, the standard curves were plotted by the mean OD value for each standard on the *X*-axis against the concentration on the *Y*-axis and a best-fit curve through the points on the graph.

Statistical analysis

Data were introduced into IBM SPSS Statistics Version 25 (San Diego, California, USA) for statistical analysis, while the figures were constructed using the EXCEL program of Microsoft Office 2010 (GraphPad prism Microsoft). The results were expressed as average, percentage, median, and p value. The Kruskal–Wallis test is a nonparametric test used to compare the median ± interquartile range when the p value of Levene's test is less than 0.05. *P* value of <0.05 indicates statistical significance and is highly significant if the *P* value is <0.001.

Ethical approval

All subjects involved in this work were informed, and consent was obtained verbally from each one before the collection of the samples. This study was approved by a local committee on publication ethics at the Al-Najaf Health Directorate, according to document No. 581 on October 13, 2023.

Results

Clinical findings of *Helicobacter pylori*

In this study, several clinical manifestations were recognized as being reasonably associated with mild, acute, and chronic upper gastrointestinal complaints and dyspepsia (defined as epigastric discomfort in the upper abdomen) for all patients in this study. These clinical findings can be caused by *H. pylori* and other idiopathic etiologies. All patients who underwent many tests to determine the reasons for dyspepsia found that 81.25% of all patients (31% and 50%, moderate and severe gastritis caused by *H. pylori*, respectively) and 18.74% of chronic dyspepsia cases were caused by other idiopathic etiologies such as gastric cancer and infections by other microbes.

Antigen stool assay (rapid test) for *H. pylori*-infected participates

We show in [Figure 1] significant results in *H. pylori* infection. Out of a total of 80 patients, 65 (81.3%) were found to be positive for *H. pylori* infection and 15 (18.8%) were found negative for infection. The noninvasive analytical technique depends on immuno-chromate-graphic test (I.C.A) for the presence of *H. pylori* infection with high-top kits. Sensitivity and specificity of (HP-AS) for *H. pylori* (95%) were evaluated.

Correlation between *Helicobacter pylori* and total serum interleukin-8 assay

Table 1 showed a highly significant difference in total IL-8 between *H. pylori*-positive patients and healthy controls. Out of a total of 80 *H. pylori*-infected patients, 65 (81.25%) had found to have positive *H. pylori* infection, as shown in [Figure 2], compared with 65 (100%) healthy controls with 17.9(29.3) and 1.3(1.1) for IL-8, respectively, with *P* value < 0.0001.

Correlation of interleukin-17 between *Helicobacter pylori*-infected patients and healthy control

Table 2 shows a highly significant difference in total IL-17 between *H. pylori*-positive patients and healthy controls. Out of a total of 80 *H. pylori*-infected patients, 65 (81.25%) were found with positive *H. pylori* infection, as shown in [Figure 2], compared with 65 (100%) healthy controls with 3.3 (2.6) and 1.3 (0.6) for IL-17, respectively, with *P* value < 0.0001.

Discussion

In this study, a noninvasive diagnostic test (rapid test or antigen stool test) was used for the detection of *H. pylori*. This assay had advantages and disadvantages depending on the clinical findings and cannot be used as the gold standard in clinical diagnosis.

The results were consistent with those of previous international studies conducted by Haider *et al.*[13] who mentioned that *H. pylori* infections were more common in Pakistan. The detection of *H. pylori* infection using a quick immuno-chromate-graphic antigen test indicates that *H. pylori* increases the risk of severe clinical issues caused by the presence of distinct virulence factors.

On the other hand, Krausse *et al.*[14] suggested that the highly significant differences in performance between

Table 1: Comparison of interleukin-8 between positive *Helicobacter pylori* infection and healthy control

	Cases Median (IQR)*	Controls Median (IQR)	*P* value
Interleukin-8	17.9 (29.3)	1.3 (1.1)	0.0001

*IQR: Interquartile range

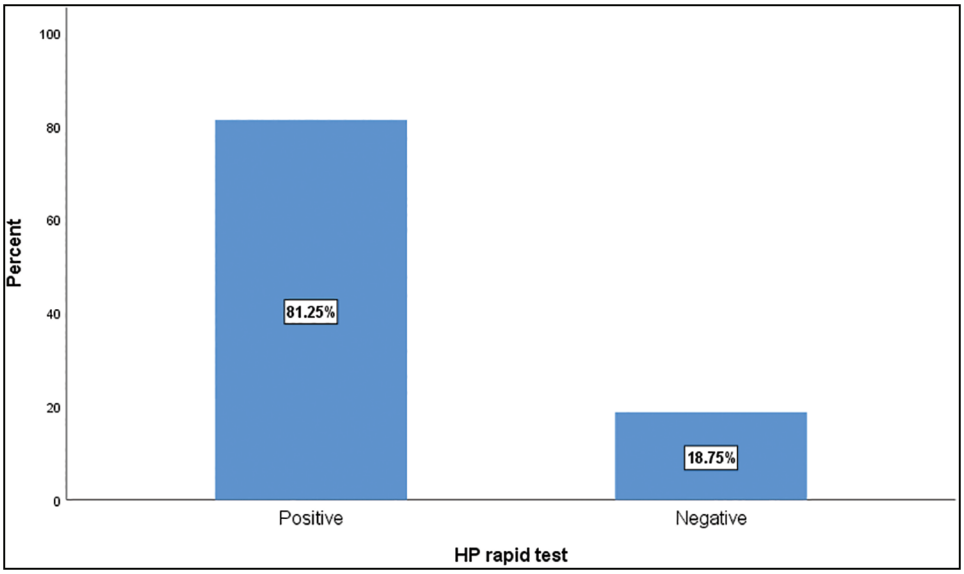

Figure 1: Distribution of *Helicobacter pylori*-infected patients by rapid testing

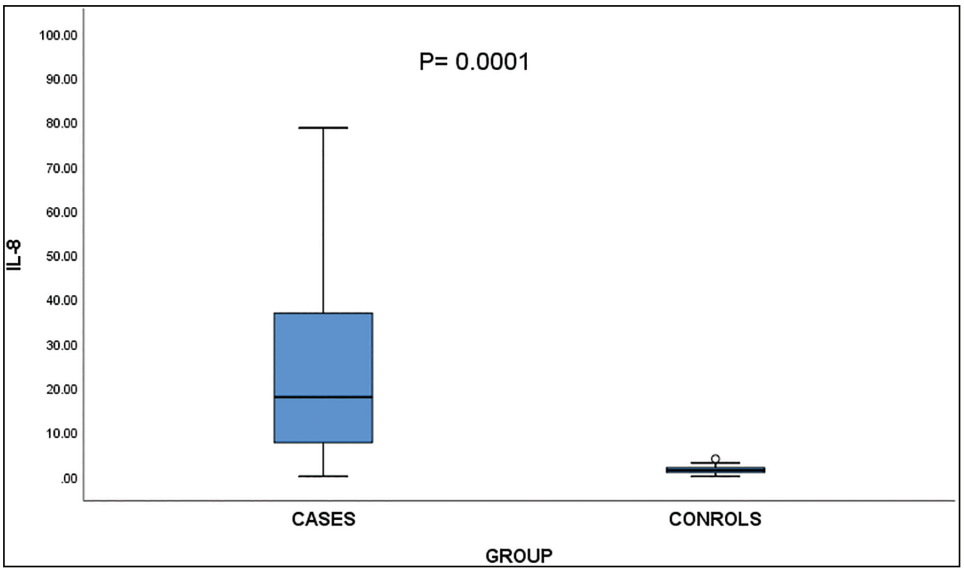

Figure 2: Correlation between *Helicobacter pylori* and immune response

Table 2: Comparison of interleukin-17 between positive *Helicobacter pylori* infection and healthy control

	Cases Median (IQR)*	Controls Median (IQR)	*P* value
Interleukin-17	3.3(2.6)	1.3(0.6)	0.0001

*Correlation of high significance with *P* value < 0.0001. IQR: Interquartile range

sexes and ages were observed, with higher efficiency in female patients and young adults (18–23 years old). Other studies conducted by Monteiro[15] and Hassan, 2016[16] found that the diagnosis of *H. pylori* by rapid tests can be useful for initial screening tests, but not definitive.

Other findings suggested by Attumi and Graham[17] revealed that HP-SA was noninvasive or minimally invasive, highly accurate, affordable, and easily accessible, and it distinguishes between current and previous infections with the organism. Other studies conducted by Thung *et al.*[18] and Kishore *et al.*[19] found that invasive tests for *H. pylori* infection were more reliable than noninvasive tests.

The results were consistent with those of previous studies conducted by Hibaoui *et al.,*[20] and a high sensitivity of Rapid Antigen Test for *H. pylori* (100%) was observed, with specificity (90.6%), positive predictive value 89.6, and the negative predictive value 94.8%.

The results of this study were consistent with those of a previous study conducted by Metwally *et al.*[21] who found that the stool antigen test had high sensitivity (94%) and specificity (97%).

Regarding the correlation between *H. pylori* and total serum IL-8 assay, the result was consistent with that of a previous study conducted by Yang *et al.,*[22] and it was found that *H. pylori*-positive patients have higher IL-8 secretion, a chemoattractant that drives neutrophil recruitment, promoting inflammation and the recruitment of T cells, with P <0.001. Another previous study conducted by Dincă *et al.*[23] had found that the elevation of IL-8 levels in *H. pylori*-associated gastritis through one of the first primary ILs secreted by the infested gastric epithelium shows a highly significant effect on the inflammatory reaction. In a previous study conducted by Dixon *et al.,*[24] it was found that IL-8 can inhibit H. *pylori* growth in gastric epithelial cells with $P < 0.04$.

Regarding the correlation of IL-17 between *H. pylori*-infected patients and healthy control, the results were consistent with those of the previous study conducted by Mohammed *et al.*,[25] who investigated of the total IL-17 levels in *H. pylori*-positive infections when compared with healthy controls, and they showed a significant difference $P \leq 0.05$. On the other hand, another study conducted by Hasan *et al.*,[26] was consistent with other study in Basrah conducted by Al-Hamdi and Khashan in 2017.[27] Al-Ezzy[28] found a significant effect of IL-17 secretion in *H. pylori* infection with $P < 0.0001$ in the human gastric mucosa, which induces the secretion of neutrophil-attracting chemokines.

Kabir[29] found that T regulatory cells and Th-17 changing secretion levels induce IL-17 inflammatory response can lead to bacterial presence with $P < 0.0003$. Another study conducted by Shehab *et al.*,[30] showed that levels of IL-17 in gastritis patients can be elevated, with a significant $P < 0.0062$ when compared with the healthy control group.

Francesco *et al.*[31] found that *H. pylori* infection with chronic gastric inflammation may increase the levels of IL-17 with $P < 0.0005$. Shehab *et al.*,[30] revealed significant

increases in IL-17 with $P < 0.0001$ when compared with healthy control. Compatible with a previous study conducted by Dixon *et al.,*[24] it was found that *H. pylori* stimulates Th-17 to secrete pro-inflammatory IL-17A, which has antibacterial reaction and controls bacterial colonization.

Bagheri *et al.*[32] revealed that chronic gastritis caused by *H. pylori* infection leads to gastric cancer. Patients with progressive cancer exhibited a higher percentage of Th-17 cells in peripheral blood and in tumor-draining lymph nodes compared to non-cancerous subjects. Kako *et al.*[33] found that the IL-17 cytokine family consists of six structurally associated molecules, from IL-17A to F. IL-17A and IL-17F were produced by immune cells, particularly Th2 and Th17 cells, respectively. Th17-derived IL-17 contributes to collagen production, fibroblast multiplication, and inflammatory cell recruitment to vascular endothelial cells, which increases during bacterial infection with $P < 0.0003$.

This study was compatible with a previous study conducted by Dixon *et al.*[24] who found significant differences for IL-17 with $P < 0.001$ causing chemokine stimulation due to *H. pylori infection,* which stimulated T-helper cells (CD4+) and Th 17 to secrete pro-inflammatory IL17A and IL-17F that control bacterial colonization. Della Bella *et al.*[34] found that *H. pylori* infection stimulates secretion of IL-17 in the stomach mucosa with $P < 0.001$. Gastric inflammation is caused by *H. pylori* which affects IL-17 secretion through vacA and cagA. Shehab *et al.*[30] found that IL-17 levels in patients with *H. pylori* gastritis significantly increased $P < 0.0062$ when compared with healthy controls.

Conclusion

The findings of this study suggested that cytokine variation profiles could be useful for detecting *H. pylori* infection and predicting its outcome. The significant differences for IL-17 may cause chemokine stimulation due to *H. pylori*, which stimulated T-helper cells (CD4+) and Th 17 to secrete pro-inflammatory IL17A and IL-17F that control bacterial colonization.

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Conflicts of interest

There are no conflicts of interest.

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