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Research Article

Molecular Detection of *Helicobacter pylori* Infection and Evaluation of Interleukin-4 as an Immune Marker

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Abstract

Background: A type 2 cytokine called interleukin (IL-4) donates negatively to immunoregulatory responses in humans. Amplified synthesis of IL-10 is facilitated by IL-4, which also constrains interferon-γ production. Doubt surroundings IL-4's impact on lymphocytes' proliferative response, though. In individuals with *Helicobacter pylori (H. pylori)* infection, we have previously found an increase in IL-4 manufacture. This study aims to enhance the molecular detection of *Helicobacter pylori* infection by incorporating a focused evaluation of Interleukin-4 (IL-4) expression. Through this combined approach, we seek to improve diagnostic accuracy while gaining insight into the immune response dynamics associated with the infection.
Material and method: Between September 1, 2022 and June 30, 2023 at the community hospital and numerous private clinics in Al-Najaf province. The study included 100 samples collected from patients presenting with gastrointestinal symptoms, 50 men and 50 women spanning 15-85 years of age (50 patients and 50 healthy controls).
Results: The current study includes 100 samples, gastric patient= 50 (50%), and Control = 50 (50%). The research found that gastric patients were 34 (68%) gave positive result while the 16 (32%) were gave negative result for detecting *H. pylori* by Rapid urease test among all 50 patients using one step test. Following 16SrRNA detection by PCR, the study discovered that 40 (80%) of the positive gastric patients and 10 (20%) of the negative patients were found. In addition, this research found that the concentration of Interleukin (IL)-4 in *H. pylori* infected patients increase significantly($P< 0.05$) than healthy control.

1. Introduction

It has been demonstrated that *Helicobacter pylori* is the infectious agent responsible for the development of gastric cancer and other gastric disorders. There is a well-established pathogenic cascade associated with *H. pylori*'s prolonged settlement of the epitheliah gastric [1]; yet, the precise molecular pathways through *Helicobacter pylori* causes gastric cancer are still unclear [2].

Among the primary causes of death worldwide, gastric cancer accounted for 1,089,103 (5.6%) of newer detection of cancer cases and 768,793 (7.7%) of newfangled cancer demises in 2020 [3].

A major contributor to the development of stomach cancer is the complex interplay between the virulence factors of the infecting organism and the host's biological response. The pathogenic components of *Helicobacter pylori* cause chronic inflammation, which can lead to lesions, tissue narcosis, and in certain cases, stomach cancer [4]. *H. pylori* infections are more frequently linked to several known disease-causing factors. While not all of these factors are expected to enhance the risk of infection, a considerable number of them are thought to do so, leading to an increase in the production of pro-inflammatory cytokines [5].

The chromosome of Human 5 (5q31.3) has a long arm wherein the IL-4 gene is accompanied by a group of T help 2 (Th2) cytokine genes [6]. The cytokine IL4, which plays a role in immune regulation, stimulates Th2 cells and inhibits the Th1 response [7]. Two SNPs within the regulatory areas of IL4, IL4 rs2243250 (also known as 590T>C) and IL4 rs34142320, are thought to have an effect on the expression of IL4 [6].

Many of the previous studies [8, 9] that looked at the relationship between the IL4 gene polymorphism and stomach disorders also looked at *Helicobacter pylori* infection at the similar period. The IL4 rs2243250 polymorphism may reduce the risk of stomach cancer in Caucasians, according research by [10]. A meta-analysis by [11] found no significant association between stomach cancer and the IL4 rs2243250 polymorphism. While some previous study addressed the association between IL4 gene polymorphism and *H. pylori* -related gastroduodenal diseases, few investigations explicitly evaluated the link between these gene polymorphisms and *Helicobacter pylori* contagion. The stomach colonization of *Helicobacter pylori* encourages humoral and cellular immune responses [12]. They often do not lead to the removal of microorganisms. *H. pylori* may live in the human stomach for decades or perhaps a lifetime if antibiotic treatment is not received [13]. About 50% of all people on the planet are infected with *H. pylori*, which is common across the world [14]. Most infected individuals [13] do not exhibition symptoms due to *H. pylori*-induced stomach inflammation; nonetheless, it is related to a higher risk of developing gastric ulcer disease, duodenal ulcer disease, gastric adenocarcinoma, and gastric lymphoma [15]. This study aims to provide fresh light on population genetic variability and the relationship between IL-4 and *H. pylori* infection.

2. Materials and methods

2.1. Study area and groups

The study was conducted between September 2022 and June 2023 at the public hospital and several private clinics in Al-Najaf province, Iraq. A total of 100 samples were taken, 50 men and 50 women spanning 15-85 years of age (50 patients and 50 healthy controls). Samples were gathered. Individuals with blood, stool, and biopsies have a variety of problems, including dyspepsia, vomiting, diarrhea, and appetite loss.

Patients were employed consecutively from outpatient clinics. Those presenting with gastrointestinal complaints such as dyspepsia, vomiting, diarrhea, or loss of appetite were registered after procurement informed consent. Inclusion criteria for patients comprised being between 15 and 85 years of age, having no prior treatment for *H. pylori* infection, and not having conventional antibiotics, proton pump inhibitors, or bismuth mixtures within the preceding 4 weeks. Exclusion criteria included a history of kidney or chronic liver disease, recent use of immunosuppressive therapy, or autoimmune disorders.

The healthy control group contained of healthy volunteers deprived of any known history of *H. pylori* infection or gastrointestinal symptoms. Specimen's size was determined based on the predictable prevalence of *H. pylori* infection (∼70%) with a 10% margin of error and a 95% confidence level.

For each contributor, syringes, 3-5 ml of venous blood were extracted from each human being's radial vein. After that, the blood was put into gel tubes, left to coagulate at room temperature, then spun for fifteen minutes at 1,500 RPM. The sera were then stored at -20°C until additional analyses were carried out [16].

2.2. Rapid urease test (RUT) for *H. pylori* detection

The attendance of *H. pylori* in gastric biopsy sample was firstly screened by the rapid urease test (RUT), achieved due to the manufacturer's instructions [17]. Color change from yellow to pink within 30 minutes was considered positive for *H. pylori*.

2.3. Molecular detection of *H. pylori*

Genomic DNA was extracted from gastric biopsy samples following the protocol described by [18, 19]. A specific primer pair targeting the 16S rRNA gene of *H. pylori* (110 bp amplicon) was used: Forward: 5′-CTGGAGAGACTAAGCCCTCC-3′ and Reverse:5′-ATTACTGACGCTGATTGTGC-3′ as shown in Table 1 and 2.

Table 1: PCR Reaction Mixture

Component	Volume (μL)	Final Concentration
Green Master Mix (2×)	12.5	1×
Forward Primer (10 μM)	2.0	0.4μM
Reverse Primer (10μM)	2.0	0.4μLM
Template DNA	5	–
Nuclease-free Water	3.5	–
Total Volume	25.0μL	–

Table 2: Thermal Cycling Conditions

Step	Temperature (°C)	Time	Number of Cycles
Initial denaturation	95	5 min	1
Denaturation	95	20 sec	35
Annealing	54	45 sec	35
Extension	72	45 sec	35
Final extension	72	7 min	1
Hold	4	∞	–

After amplification, 5μL of each PCR product was electrophoresed on a 1.5% agarose gel in 1× TBE buffer at 70 V for 45 minutes. The gel was stained with RedSafe™ Nucleic Acid Staining Solution (1 mg/L) and visualized under UV illumination to confirm the expected 110 bp product.

2.4. Estimation of serum IL-4 by ELISA

Serum levels of interleukin-4 (IL-4) were determined using a human IL-4 ELISA kit (Elabscience®, Houston, TX, USA; Cat. No. E-EL-H0082) due to the manufacturer's instructions. Specimens were examined in replica, and absorbance was measured at 450 nm using a microplate reader. The assay's detection range was 15.6–1000 pg/mL, and results were expressed in picograms per milliliter (pg/mL). The limit of detection (LOD), as well as intra- and inter-assay coefficients of variation, were within the manufacturer's acceptable specifications (<10%).

2.5. Statically analysis

The common statistical package Graph Pad Prism version 7 was used, and the data was summarized using the (Mean Stander division) format [20].

2.6. Ethical Approval Statement

Ethical approval for this study was granted by The Ethics Committee of Al-hakim General Hospital In Najaf, Iraq (REF. NO. 37922).

3. Results and Discussion

3.1. Demographic study

At the current study, 100 samples were divided into two groups—50 samples from the gastric patient group (50 percent) and 50 samples from the control group (50 percent)—for additional analysis at the hospital and a few private clinics in the Al-Najaf area, Iraq. The study's findings show that, for gastric patients, the proportion of male patients exceeded that of female patients by a margin of 35 (70%) to 15 (30%), while for the control group, the numbers were male 28 (56%) and female 22 (44%) Figure 1.

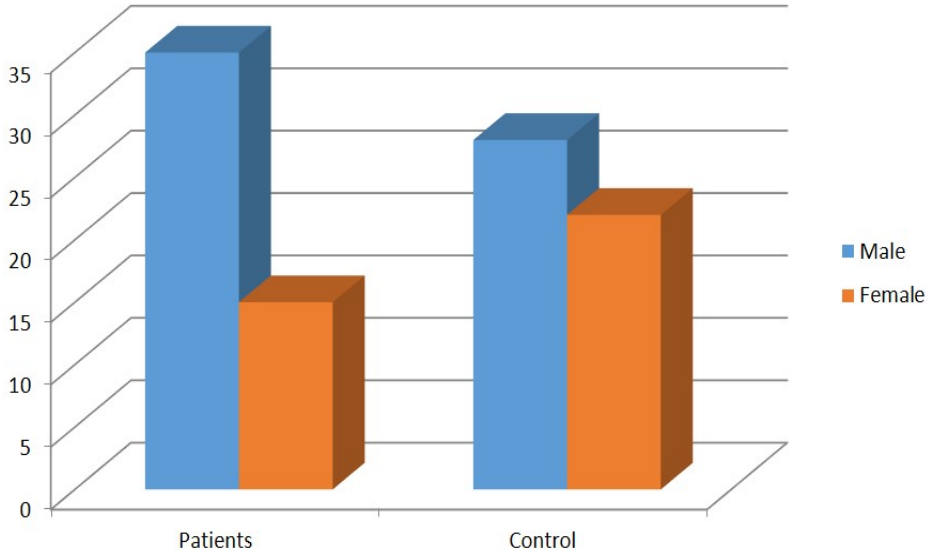

Figure 1: Distribution of patients and control according to sex

Helicobacter pylori may colonise the stomach, thrive in the lumen's acidic pH, and enter the stomach's epithelial cell layer through the mucus layer [21, 22] by this method. *H. pylori* need the urease enzyme to survive in the acidic stomach, since it may neutralise gastric acidity and cause damage to the gastric mucosa via the production of toxins such Cag A and Vac A [23] (emphasis added). *H. pylori* uses a variety of host tissue colonisation cues, including as a high salt consumption, smoking, and low iron levels [22]. The relationship between *H. pylori* isolation from a gastrointestinal patient and many immunological characteristics was investigated, as was the efficacy of molecular vs serological approaches for identification of *H. pylori*.

3.2. Rapid urease test (RUT)

Of the 50 patients, 34 (68%) had a positive result for RUT, whereas 16 (32%) had a negative result. Significant statistical analysis was performed ($p<0.001$). As seen in Figure 2, the positive result is displayed as a pink hue, indicating that the patient has *H. pylori*.

Figure 2 illustrates the occurrence of *H. pylori* in the current study: the positive result was 34 (68%), whereas the negative result was 16 (32%). According to reports, 42% and 58% of people tested positive for RUT [24, 25]. Several studies conducted in Basrah reveal that 74% of patients had varying levels of *H. pylori* detection by RUT, The reason is due to sample processing problems, small sample sizes, and low

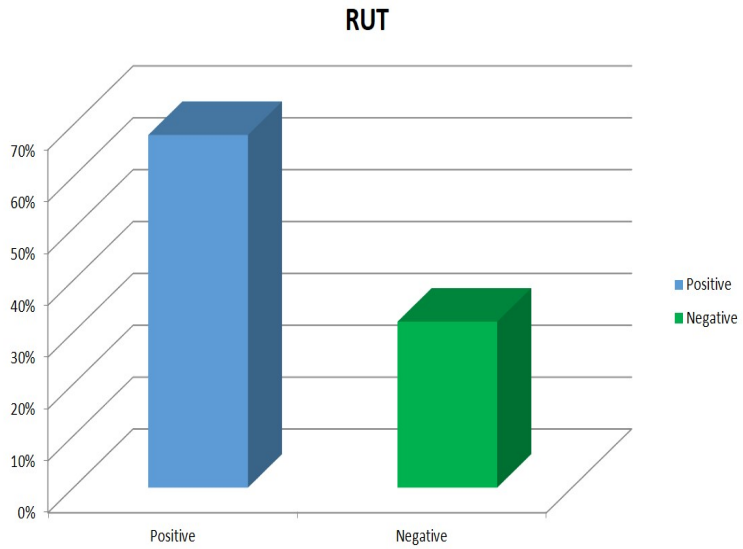

Figure 2: Result of specimens by rapid urease test

H. pylori bacterial densities, all of which might result in misleading negative findings. Furthermore, if another bacterium capable of releasing the urease enzyme is present, it may cause false positive findings by prolonging the incubation duration beyond 24 hours [26].

The requirement for a minimum bacterial load of 10^5 in the same sample as the histopathological tissue to yield a positive outcome impacts the sensitivity and specificity of the test [27]. Studies cited in [28] have demonstrated that the Rapid Urease Test (RUT) achieves high accuracy in detecting *H. pylori*, with both sensitivity and specificity exceeding 90%. Consequently, the urease test proves to be a reliable method for identifying *H. pylori* and serves effectively as an initial screening tool [29].

3.3. *H. pylori* detection by PCR technique

Determination of 16SrRNA gene

In 40 (80%) of the 50 biopsy samples, PCR results for the 16S rRNA gene's 110 bp region were positive. The 2% agarose gel electrophoresis examination of the PCR produces, which revealed bands of the amplified DNA for 16SrRNA, is presented in Figure 3.

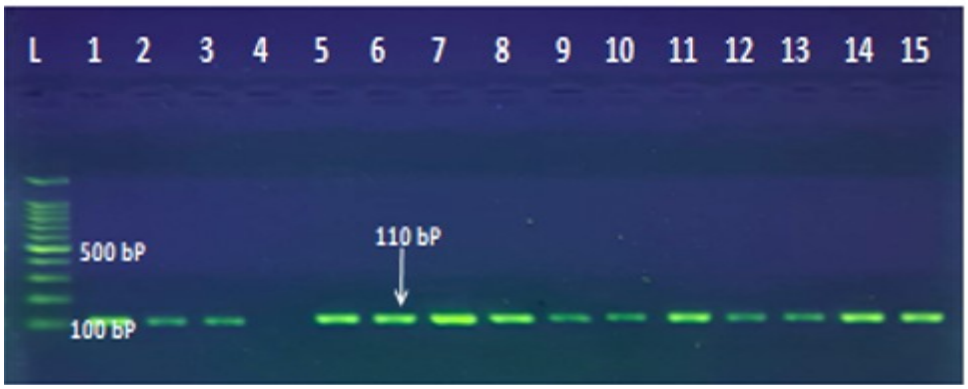

Figure 3: The band size of the PCR result is 110 bp. The end result was electrophoresis at 5 volts per cent agarose. One hour with one TBE buffer. N is the DNA ladder (100)

The findings of this test for identifying *H. pylori* in biopsy samples are shown in Figure 4, out of 50 biopsy specimens, Eighty percent of samples had a positive result in the 16S rRNA gene's 110 bp region. The presence of the pathogen may be directly demonstrated by a positive amplification of *H. pylori* -specific DNA, and one specific target to confirm *H. pylori* infection is the 16S rRNA [30]. The 16S rRNA gene is widely employed for the identification of *H. pylori* because of its high conservation and evolutionary significance across bacterial species, making it a reliable target for microbial classification [31].

Unlike the findings of the present study, research conducted at the Regional Specialist Hospital in Wroclaw, Poland [32] utilized the 16S rRNA gene for bacterial detection, reporting a positive rate of 32.5% among tested samples. Similarly, other investigators in Sudan have applied this molecular approach for bacterial identification [33]. Using this gene, 37.33% of positive specimens were identified; however, the findings of this study differed from the findings of the current investigation. Using the 16 srRNA gene, researchers at the Regional Specialist Hospital in Wroclaw, Poland, detected bacteria in a study conducted in contrast to the present one [32]. 32.5% of the samples were diagnosed. In addition, a study in Sudan was conducted by several researchers to find microorganisms [33]. 37.33% of positive specimens were detected using this gene; however, the results of this study were not consistent with the results of the current inquiry.

3.4. Immunological markers

Evaluation of IL-4 in patients

The present study identified statistically significant differences between the patient and healthy groups, with a P-value ≤ 0.05, as demonstrated in Figure 4. It also noticed that the patient group's IL-4 (pg/ml) concentration increased in comparison to the healthy group (24.460 ± 1.140; 5.632 ± 0.219) pg/ml.

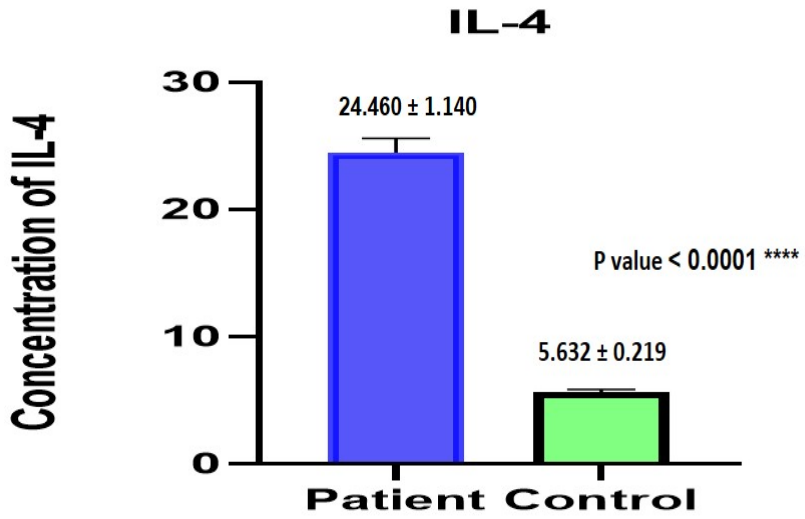

Figure 4: Il-4 level in patients and control group

Interlukin-4, a type 2 cytokine in general, has the ability to suppress the human immune system's cellular response to infection. It is generally accepted that IL-4 inhibits a variety of lymphocyte responses [34]. But compared to IL-I0, another type 2 cytokine that often serves as a deactivating factor, the activity of IL-4 may really be far more complicated [35].

It has been suggested that *H. pylori* infections are persistent, often persisting for decades [36]. One of the causes of the chronic infection appears to be the increased TH2 response, as seen by lymphocyte proliferation and cytokine production in *H. pylori*-infected individuals [37]. On the one hand, by downregulating the inflammatory response, the human host tries to reduce the tissue damage. However, certain hosts' THl response is repressed, making it impossible for them to get rid of the organism [36]. Because IL-4 is known to trigger the formation of the TH2 immune response, which may down-regulate cellular immunological responses to pathogenic pathogens, its capacity to increase lymphocyte proliferation is therefore surprising. This finding does not, however, refute IL-4's down-regulatory function in immune responses.

In summary, IL-4 is cytokine that play important role in the immune system, including the regulation of immune responses against bacterial pathogens.

Article Information

Disclaimer (Artificial Intelligence): The author(s) hereby declare that NO generative AI technologies such as Large Language Models (ChatGPT, COPILOT, etc.), and text-to-image generators have been used during writing or editing of manuscripts.

Competing Interests: Authors have declared that no competing interests exist.

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