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Physiological Characterization for Gastritis with *Helicobacter Pylori*

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Abstract

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H.pylori affects around half of the world's population and colonizes the stomach mucosa. This study aims to investigate the relation between white blood cells (neutrophils, lymphocytes, monocytes, eosinophils, and basophils) in patients with H.*pylori*. A case-control study included 150 patients who had *H. pylori* infection who entered the center of gastrointestinal disease in Nasiriyah city, southern Iraq, and 100 healthy controls. The samples were collected during a period extending from September 2024 to January 2025, and the patients were tested by using UBT to determine the presence of *H.pylori* ,while healthy controls were tested by ELISA; only 25 of them were tested by UBT. The results showed all patients infected with *H.pylori* after being diagnosed by UBT, while healthy controls were non-infected. Also, the results show that patients are infected with *H.pylori* more in urban areas when compared with rural areas (p = 0.0463). The result showed significant differences in WBC count and percentage of neutrophils among patients with gastric disorders compared to control subjects (WBC p = 0.02, neutrophils p = 0.01).

1.Introduction

Helicobacter pylori ,also known as Campylobacter pylori, is a highly motility, spiral-shaped, Gram-negative bacterium with a cilium at one end. It grows in an aerobic environment with low oxygen content and can survive in the stomach, even in acidic conditions [1]. It is estimated that 50% of the world's population is infected, with up to 80% in poor and developing regions and 20–50% in wealthy countries. The prevalence of *H. pylori* is likely to vary greatly due to socioeconomic conditions, level of urbanization, sanitary conditions, and access to clean water sources [2]. The infection often causes chronic gastritis, which varies in severity depending on host genetics, immune responses, and *H. pylori* strain, even though the majority of infected individuals show no symptoms [3]. *H. pylori* has developed a number of defenses against the hostile environment of the stomach. The enzyme urease neutralizes stomach acidity by converting urea into ammonium and carbonic acid [4]. while its spiral structure and flagella-driven motility aid in colonizing the gastric mucosa [5]. The World Health Organization has classified *H. pylori* infection as a risk group 1 carcinogenic, and for this reason, the diagnosis and eradication of *H. pylori* infection are goals in the prevention of gastric cancer [6]. UBT is one of the most important non-invasive diagnostic methods used to detect active *H.pylori* infections, as is widely known. Millions of people are examined each year for the non-invasive test[7]. It is a simple method that is used for diagnosing based on the simple principle that after patients ingest urea that is labeled with 13C or 14C, the bacteria produce urease, an enzyme that breaks down the urea into ammonia and 13C-labeled carbon dioxide. The carbon dioxide is then taken up by the bloodstream, travels to the lungs, and is expelled along with the air that has been exhaled [8].

The body's defense system against the invasion of foreign pathogenic microorganisms is known to be white blood cells (WBC). Different responses to inflammation and infections can manage WBC levels [9].*H. pylori* infection is one such example where the immune system plays a critical role. Among the virulence components of *H. pylori* that contribute to the infection's pathogenesis are kinase inhibitors, which also participate in the reduction in the stomach epithelial cells' polarity and length. Furthermore, the production of pro-inflammatory cytokines causes neutrophils, which are crucial to the active inflammation, to become activated [10]. This neutrophil activation, along with other immune responses, leads to an increase in WBCs and contributes to mucosal inflammation and tissue damage associated with *H. pylori*-induced gastritis and ulceration**.**

2.Materials and Methods

A study of case-control design included 250 cases (150 patients and 100 healthy controls) and the age ranged from 15 to 70 years old. The current investigation was carried out in compliance with the Declaration of Helsinki's ethical guidelines. Before taking a sample, the patient's verbal and analytical consent was obtained. The samples were collected during a period extending from September 2024 to January 2025 from the center of gastrointestinal disease in Nasiriyah city, southern Iraq. Five ml of blood were collected from patients with gastric diseases and healthy control. Whole blood was placed in a tube (EDTA) in order to use it for estimation of complete blood count (CBC) for detection of differential WBC. Exclusion criteria include patients with diabetes mellitus, hypertension, hyperthyroidism, gastric cancer, and patients who were treated with antimicrobial , anti-inflammatory medications and non-steroidal anti-inflammatory drugs (NSAIDs).

3.Determination of *H.pylori* and differential White Blood Cells

The ^{14}C urea breath test is a non-invasive functional test that detects active H. *H.pylori* infection. The patient ingests urea labeled with radioactive (^{14}C). If *H.pylori* is present, its urease enzyme metabolizes the urea into labeled carbon dioxide, which is absorbed into the bloodstream and exhaled. This labeled carbon dioxide is measured in the patient's breath using a Helicobacter pylori detector device, and the units are (DPM). The patient must fast for at least 4-6 hours before the test, stop taking proton pump inhibitors (PPIs) for two weeks, and stop taking antibiotics and bismuth subsalicylate for four weeks. Estimation of differential white blood cells (neutrophils, lymphocytes, monocytes, eosinophils, and basophils) detected in patients and healthy controls by using a hemolyzer instrument (Genrui Biotech) from China .

4.Results

The results shown in Table 1 indicated a statistically significant difference in UBT levels between patients and controls (t = 77.73, *p* = 0.001). The mean UBT level among patients with *H. pylori* (436.9±61.49) was in controls (33.52±6.57).

Table 1. level of urea breath test among patient with *H.pylori* and control

Cases	No.	Level of UBT(DPM) mean± SD
Patients with *H.pylori*	150	436.9±61.49
Control	25	33.52± 6.57

t = 77.73 *p*-value = 0.001 (significant difference P≤ 0.05)

The results in Table 2 show the prevalence of H. pylori in relation to residence area. Among patients with gastritis diseases, the high positivity rate of 70% (n=105) was observed in urban areas, while the low positivity rate of 30% (n=45) was recorded in rural areas. Statistical analysis showed a significant difference between the two groups (*p* = 0.0463).

Table 2. Frequency and percentage residency's of patient with *H.pylori*

Residency	patients with *H.pylori* NO.(%)	Controls NO.(%)
Urban	105 (70%)	82(82%)
Rural	45 (30%)	18(18%)
Total	150	100

X^2 = 3.97 df =1 *p*-value= 0.0463 (Significant differences P>0.05)

The results in Table 3 showed significant differences in WBC count and percentage of neutrophils among patients with gastric disorders compared with control subjects (WBC $P = 0.02$, neutrophils $P = 0.01$) and also showed decreased frequency of lymphocytes in patients compared to controls ($P = 0.08$).

Table 3. Complete blood count among patient with *H.pylori* and control

Cases	Patients	Control	*P*-value	*t*-value
WBC(10^3/ µL)	9.4632±1.2	5.09±0.7	0.02	36.32
Neutrophils(10^3/ µL) Mean ± SD	66.832±3.2	55.83±5.3	0.01	18.62
Lymphocytes(10^3/µL) Mean ± SD	25.9626±1.8	33.35±3.1	0.08	21.53
Monocytes(10^3/ µL) Mean ± SD	4.9444±0.3	5.94±0.2	0.01	31.48
Eosinophil(10^3/ µL) Mean ± SD	2.0809±0.11	4.75±0.4	0.04	65.11
Basophil(10^3/ µL) Mean ± SD	0.21533±0.01	0.11±0.01	0.02	81.59

Significant differences *p*-value less than 0.05

5.Discussion

pylori affects around half of the world's population and colonizes the stomach mucosa [11] .Its prevalence is influenced by socioeconomic variables, urbanization, access to clean water, and sanitation [12].Usually contracted in early childhood by oral-oral or fecal-oral pathways, the illness normally lasts a lifetime and is seldom cleared on its own [13].

The results shown in Table 1 indicated a statistically significant difference in UBT levels between patients and controls (t = 77.73, *p*-value = 0.001). UBTs are the first method of identifying *H. pylori* infection . It is the preferred non-invasive test because of its simplicity, quick results, high accuracy, and specificity. Furthermore, the distribution of *H. pylori* in the stomach has no effect on its effectiveness, enabling a large number of patients to be examined [14]. In a study conducted by Liao, E. C., et al. (2023), it was revealed that the UBT test was definitely the best non-invasive test[15]. Another showed that the UBT test is more important than non-invasive tests [16].also study of Ferwana, M. M., et al. (2015) showed that the UBT test is highly diagnostic in adult patients [17]. Stefano et al. (2018) found a sensitivity of 96% for the UBT test [18] .

The present study shows in Table 2 the prevalence of *H. pylori* in relation to residence area. Which found Among patients with gastritis diseases, the high positivity rate of 70% (n=105) was observed in urban areas, while the low positivity of 30% (n=45) was recorded in rural areas. Statistical analysis showed a significant difference between the two groups (*p*-value = 0.0463).

The current study agrees with the study of Hussain *et al*. (2023), who found a high prevalence rate in urban areas when compared to rural area [19]. Also the present study agree with study of Lindkvist *et al*.,(1998) that found high prevalence of *H.pylori* in an urban population ,This is due to the association of urban areas with risk factors such as environmental contamination and crowded lifestyles [20].A research study disagree with study conducted by Fok *et al*. (2010). According to their study results, the highest prevalence rate (63.4%) was recorded in rural areas of Turkey, compared to 40.5% in urban areas, where the incidence rates were lower [21].

The current study showed in Table 3 significant differences in WBC count and percentage of neutrophils among patients with gastric disorders compare with healthy control subjects(WBC *p*-value =0.02, neutrophils *p*-value=0.01) This indicates an active

inflammatory or immune response. The result corresponds to studies of Thomas, T.C., *et al*. (2022) that demonstrated elevated WBC and neutrophil counts in individuals with gastrointestinal diseases, including peptic ulcers or gastritis [22]. It also correlates with the higher percentage of neutrophils, as reported [23].

This study is consistent with a study by Jiao, R., *et al*. (2024) that found an increase in the number of white blood cells in people infected with *Helicobacter pylori*, with the spreading gradually as the number of white blood cells increases [24]. These findings provide evidence of how *Helicobacter pylori* infection affects the immune environment, leading to the development of many other chronic diseases. also the current study disagrees with the study of Farah, R., et al.'s findings that patients with *Helicobacter pylori*-positive against *H.pylori*-negative conditions had a statistically significant difference in neutrophil counts [25]

On the other hand, the present study indicated the mean value of lymphocyte counts was decreased in gastritis patients compared to the control group (25.9626 ± 1.8 vs. 33.35 ± 3.1). This result is similar to studies such as Kuo, S.-H. et al. (2019), who found a decrease in lymphocytes in patients with H. pylori infection [26]. Although this study indicates a decrease in lymphocyte count, it contradicts the findings of Elkhalifa, A. M. E. et al. (2021), who reported an increase in lymphocytes in patients with chronic *Helicobacter pylori* infection [27]. Continuous exposure to bacteria activates and encourages the immune system to produce more T helper cells ($CD4^+$) and cytotoxic T cells ($CD8^+$) as part of the adaptive immune response. Lymphocytes migrate to the gastric mucosa in an attempt to combat the bacteria. But because the bacteria can avoid total immunological clearance, this immune response is maintained, which results in chronic inflammation. Prolonged lymphocytic infiltration is a histological feature of *H.pylori*-induced chronic gastritis over time [28].

Conclusion: These findings indicated increased prevalence *H.pylori* infection among urban area than the rural area and an elevated WBC count and neutrophils among patients with gastric disorders.

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