

Evaluation of Some Physiological Parameters in Patients with Gastric Ulcer Associated and Not Associated with *H. pylori* Infection in Al-Rifai District, Iraq

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Abstract

This study aimed to evaluate selected physiological parameters in patients with gastric ulcer with and without *H. pylori* infection in Al-Rifai district, Dhi-Qar, Iraq. The study included 320 participants (180 males, 120 females); demographic data were collected from 300 participants, while laboratory tests were performed on 88 participants (68 ulcer patients with or without *H. pylori*, and 20 healthy controls). Liver enzymes (AST, ALT, ALP), gastrin, secretin, and blood parameters (WBC, LYM, GRA, MID) were measured.

Results showed significantly elevated ALP and ALT in *H. pylori*-infected males, and elevated AST in females with ulcer only. No significant differences were found in gastrin or secretin levels. WBC, LYM, and GRA were significantly increased in both patient groups compared to Healthy groups. Infections were most concentrated in the 15–40 years age group, among normal-weight individuals, and in blood group O carriers of both sexes. These findings indicate that gastric ulcer, particularly when associated with *H. pylori*, triggers a systemic inflammatory response with limited effects on liver function and no significant hormonal changes.

Keywords: Gastric ulcer, *H. pylori*, liver function, hormones.

دراسة بعض المعايير الفسيولوجية والمناعية لدى المصابين بالقرحة المعدية

المتراكفة وغير المتراكفة مع عدوى جرثومة المعدة (*H. pylori*) في قضاء الرفاعي

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الخلاصة

هدفت الدراسة الحالية إلى تقييم بعض المعايير الفسيولوجية لدى مرضى القرحة المعدية المتراكفة وغير المتراكفة بعدوى جرثومة المعدة (*H. pylori*) في قضاء الرفاعي، محافظة ذي قار، العراق. شملت الدراسة 320 مشاركاً من كلا الجنسين، خُصص منهم 300 مشارك لجمع وتحليل البيانات الديموغرافية، في حين أُجريت الفحوصات المختبرية على 88 مشاركاً قُسموا إلى مرضى القرحة المعدية بوجود أو عدم وجود العدوى (68) ومجموعة سيطرة تضم (20) من الأصحاء. تم قياس إنزيمات الكبد ALT، AST، ALP، هرموني الغاسترين والسكرتين وبعض معايير الدم المناعية (WBC، LYM، GRA، MID). أظهرت النتائج ارتفاعاً معنوياً في مستويات ALP و ALT لدى الرجال المصابين بعدوى الجرثومة، وارتفاع معنوي في مستوى AST لدى النساء المصابات بالقرحة فقط. بينما لم تُسجل فروق معنوية في مستويات هرمونات الغاسترين والسكرتين، كما ارتفعت مؤشرات الدم المناعية (WBC، LYM، GRA) لدى المرضى مقارنة بالأصحاء ولكلا الجنسين. تركزت الإصابات في الفئة العمرية (15-40 سنة)، وبين ذوي فئة الوزن الطبيعي وبانتشار ملحوظ لدى حملة فصيلة الدم O ولكلا الجنسين. مما يشير إلى أن القرحة المرتبطة بجرثومة المعدة تُحدث استجابة التهابية جهازية مع تأثير محدود في وظائف الكبد والهرمونات.

1. Introduction

Gastric ulcer is one of the most widespread digestive disorders globally, with estimates indicating it affects 5-10% of the world's population, posing a significant health and economic burden on healthcare systems [1]. An ulcer is defined as a localized erosion of the gastric or duodenal mucosa extending beyond the muscularis mucosae. Despite therapeutic

advances, it remains a clinical challenge due to its multiple etiologies, high recurrence rates, and potential for serious complications such as bleeding and perforation [2]. The injury arises from an imbalance between aggressive factors (e.g., gastric acid and *H. pylori*) and protective defensive factors of the gastric mucosa (e.g., mucus, bicarbonate, and prostaglandins) [3]. *H. pylori* infection is the leading cause of ulcers, infecting more than 40% of the world's population [4].

The bacterium produces the enzyme urease to create an alkaline environment around itself and stimulate gastrin secretion, leading to gastric acid hypersecretion. Non-steroidal anti-inflammatory drugs (NSAIDs) emerge as another major causative factor by inhibiting COX-1 and COX-2 enzymes, thereby reducing the synthesis of protective prostaglandins [5].

Inflammation plays a pivotal role in inducing gastric ulcer, as levels of inflammatory cytokines such as interleukin-6 and tumor necrosis factor-alpha are elevated in ulcer patients [6]. *H. pylori* also activates the TLR/NF- κ B signaling pathway, increasing the intensity of the inflammatory response [7]. Furthermore, oxidative stress is considered a mechanism of epithelial cell damage through lipid, protein, and DNA oxidation [8]. Gastric ulcer is associated with changes in hematological parameters; studies have shown that inflammatory indices derived from complete blood count (CBC), such as the neutrophil-to-lymphocyte ratio and platelet-to-lymphocyte ratio, are directly correlated with ulcer severity [9-10]. Studies have also demonstrated a significant association between *H. pylori* infection and liver metabolic disorders, including Metabolic dysfunction-associated steatotic liver disease (MASLD), where mechanisms of chronic inflammation, insulin resistance, and the gut-liver axis contribute to the development of this disease [11]. Signs of hepatocyte damage and cholangitis have been observed in some patients with peptic ulcers [12-13], in addition to hormonal disturbances in gastrin and secretin levels that regulate acid secretion [6].

Gastric ulcer represents a common health problem in Iraq, exacerbated by lifestyle factors, dietary habits, and psychological stress [14]. Moreover, the local treatment of gastric ulcer has been suboptimal [15]. However, data on physiological parameters in patients with gastric ulcer in Dhi-Qar province generally, and Al-Rifai district specifically, remain very limited. Therefore, the current study aimed to evaluate and compare some physiological parameters between gastric ulcer patients and healthy individuals in Al-Rifai district.

2. Materials and Methods

2.1 Study Design

This study was conducted on patients attending Al-Rifai Teaching Hospital in Dhi-Qar province, during the period from September 8, 2025, to February 3, 2026. The total study included 320 participants of both sexes, of whom 300 participants were allocated for collecting and analyzing demographic data, distributed as 180 males and 120 females.

Laboratory tests for physiological parameters were performed on a subsample of 88 participants who met the inclusion criteria (absence of chronic liver, renal, or cardiac diseases, no use of medications affecting liver enzymes or blood counts within the last month, and voluntary consent for blood sampling).

This subsample was divided into three groups, equally distributed between sexes (i.e., equal numbers of males and females in each group), as follows: first group (patients with gastric ulcer only) included 34 participants (17 males + 17 females), second group (patients with gastric ulcer associated with *H. pylori* infection) included 34 participants (17 males + 17

females), and third group (healthy control group) included 20 participants (10 males + 10 females). Cases were diagnosed clinically and confirmed by specialist physicians using laboratory tests, based on diagnostic criteria including esophagogastroduodenoscopy (EGD) and *H. pylori* detection tests. Due to the known physiological and hormonal differences between sexes that influence hematological and hepatic parameters, all statistical comparisons were performed separately for each sex. No direct statistical comparisons were made between male and female groups.

2.2 Laboratory Measurements and Studied Parameters

Cases were diagnosed clinically and laboratory by specialist physicians based on diagnostic criteria including esophagogastroduodenoscopy (EGD) and *H. pylori* detection tests. Liver enzymes AST, ALP, and ALT were measured using a semi-automatic biochemistry analyzer manufactured by BioSystems S.A., Spain, according to the measurement kits provided by the same company. Gastrin and secretin hormones were measured using ELISA technique using ready-made kits from Cusabio, China, following the attached instructions. Some blood parameters including WBC, LYM, and GRA were measured using an auto hematology analyzer manufactured by Boule medical AB (Medonic), Sweden, according to the manufacturer's instructions. Data on demographic characteristics (sex, blood group, and body mass index) were collected using a questionnaire form designed for this purpose; height and weight were measured for each participant, and body mass index was calculated according to the equation dividing body mass (kg) by the square of height (m²), while blood group was documented based on medical records or participant statements.

2.3 Statistical Analysis

Data from all tests were statistically analyzed using the Statistical Package for Social Science (SPSS) version 27, using one-way ANOVA followed by the Least Significant Difference (LSD) test. Results were considered statistically significant at probability level (0.05) to determine the significance of differences between group means.

3. Results and Discussion

3.1 Relationship between Gastric Ulcer and Some Demographic Parameters

3.1.1 Relationship between Gastric Ulcer Disease Prevalence and Age Group

The results of the Chi-square test showed significant differences in the prevalence rates of gastric ulcer according to different age groups for both sexes. The Chi-square value for men was (88.43) and for women (48.56) at probability level ($P \leq 0.001$) for both. It is noted from the results that infection is concentrated in younger age groups (15-40 years), where the percentage recorded was 66.11% in men and 65.83% in women (69 cases), while the age group over 66 years recorded the lowest percentages, with 13.33% for men and 14.17% for women, as shown in Table (1) for men and women respectively.

Table 1- Relationship between gastric ulcer and age group in men and women

For men			
Categories	> 66	66-41	40-15
Number	24	37	119
Percentage %	13.33	20.56	66.11
For women			
Number	17	24	79
Percentage %	14.17	20.00	65.83

The results show a similar pattern of gastric ulcer disease between men and women regarding age, with a concentration of infections in younger age groups (15-40 years) in both sexes. This marked similarity in percentages between sexes for the young group (about 66%) reflects the possibility that this age group is affected by life stresses or common dietary and behavioral patterns that create an environment predisposing to ulcer development, as observed by [16] in his study that dietary patterns and increased consumption of spicy foods in this age group are among the most important causes of ulcer development. The study results also agreed with [17], who observed the prevalence of gastric ulcer in the young age group, and [18] indicated that nearly 47% of patients with ulcer complications fall within the age group (18-40 years). The gradual decrease in infection rates with advancing age, reaching its lowest levels in the age group over 66 years, can be explained by the fact that gastric ulcer disease in the study community is closely linked to the stage of giving and age activity, requiring the focus of awareness and preventive efforts towards young age groups to limit the spread of the disease.

3.1.2 Relationship between Gastric Ulcer Disease Prevalence and Body Mass Index

Table (2) shows the relationship between gastric ulcer disease prevalence and body mass index for men and women respectively. The Chi-square test results showed significant differences in the distribution of the study sample across BMI categories, with values of ($X^2=42.356$) for men and ($X^2=29.467$) for women at probability level ($P \leq 0.001$) for both. It is noted from the results that the largest proportion of individuals is concentrated in the normal weight category, recording (76) cases and a percentage of 42.22% and (51) cases and a percentage of 42.50% for men and women respectively, followed by the underweight category with (55) cases and a percentage of 30.56% and (37) cases and a percentage of 30.83% respectively, while the overweight and obesity categories recorded the lowest percentages in both groups.

Table 1- Relationship between gastric ulcer and body mass index in men and women

For men				
Body mass	Obese	Overweight	Normal weight	Underweight
Number	23	26	76	55
Percentage %	12.78	14.44	42.22	30.56
For women				
Number	15	17	51	37
Percentage %	12.50	14.17	42.50	30.83

These results are an important indicator of the role of physical balance in the stability of the health status of gastric ulcer patients, as they reflect the dominance of the normal weight category in the sample, indicating health awareness or perhaps a physiological nature of patients in the study area. This agrees with [19], who indicated that the majority of patients with gastrointestinal disorders tend to maintain normal weight due to caution in dietary habits to avoid pain episodes, and with [20], who showed a close association between low BMI and increased likelihood of gastric ulcer, unlike other gastrointestinal diseases. [19] also pointed out that pain associated with eating drives patients to reduce the amount of food consumed, leading to a marked loss in body weight, which explains the absence of morbid obesity and the concentration of the sample in low-weight categories among gastric ulcer patients. Additionally, studies have indicated that chronic upper gastrointestinal disorders significantly increase the risk of malnutrition and marked weight loss, due to malabsorption resulting from mucosal damage or dysfunction in the production of acids and digestive enzymes [21], and

decreased appetite due to annoying symptoms such as pain, nausea, early satiety, and food aversion, which drives patients to avoid eating meals to avoid these painful symptoms [22-23]. This interaction between malabsorption and reduced food intake explains why the statistical distribution in the current study results deviates away from overweight categories, i.e., towards underweight and normal weight patterns.

3.1.3 Relationship between Gastric Ulcer Disease Prevalence and Blood Groups

The results in Table (3) indicate significant differences in the study sample between blood groups and the prevalence of gastric ulcer for both sexes, with blood group (O+) showing the highest prevalence of infections at a rate of 48.33% in men and 45.83% in women, followed by group (O-) at rates of 12.78% and 14.16% in men and women respectively, while group (AB) recorded the lowest infection rates ranging between 3.89% and 4.17% in men and women respectively.

Table 3- Relationship between gastric ulcer and blood groups in men and women

For men							
Blood groups	A-	A+	B-	B+	AB	O-	O+
Number	14	17	13	19	7	23	87
Percentage %	7.78	9.44	7.22	10.56	3.89	12.78	48.33
For women							
Number	8	12	9	14	5	17	55
Percentage %	6.67	10.00	7.50	11.67	4.17	14.17	45.83

The results showed a significant relationship between blood groups and the prevalence of gastric ulcer disease in both sexes, with blood group O+ recording the highest infection rates, followed by O-, while the lowest rates were in group AB. This distribution reflects a greater biological and genetic susceptibility among blood group O carriers to gastric ulcer compared to other groups, which aligns with the hypothesis linking ABO antigens to the ability of *H. pylori* to adhere to the gastric mucosa and induce chronic inflammatory damage [24-25]. Studies have indicated that the increased incidence of gastric ulcer in O+ blood group individuals is due to increased binding of *H. pylori* to Lewis b receptors and H antigens on the mucosal surface, enhancing bacterial colonization intensity and chronic inflammation. This group is also associated with increased secretion of inflammatory cytokines such as IL-6 and TNF- α , which weakens the mucosal barrier and raises the risk of ulceration compared to other groups [26-27]. The lower infection rates in groups like AB and B are explained by reduced bacterial adhesion and different molecular characteristics of the mucosal surface, which may provide partial protective effects against inflammation and mucosal damage. Studies have also supported that the ABO system remains an influential genetic factor in susceptibility and severity of gastric ulcer, especially when associated with *H. pylori* infection, where group O remains the highest risk compared to other groups [28]. These results agree with [29], who observed that blood group O is still associated with an increased risk of gastric ulcer, especially in the presence of *H. pylori* infection, as a result of the interaction between genetic, immune, and microbial factors. Large-scale genetic analyses have also shown that ABO system genes are among the fundamental factors influencing susceptibility and severity of ulcer development through their effect on host mucosal and inflammatory responses.

Furthermore, studies have indicated the continued association of blood group O with increased susceptibility to gastric ulcer compared to other groups [30], which is consistent

with the results of the current study for both sexes, although the strength of this association may vary depending on environmental and geographical factors, especially when combined with *H. pylori* infection and accompanying inflammatory factors [31].

3.2 Relationship between Gastric Ulcer and Liver Function

Tables (4) and (5) show the relationship between gastric ulcer and liver function in men and women at probability level $P \leq 0.05$. The results indicate a relationship between gastric ulcer (with or without *H. pylori*) and liver function in both men and women at a significance level of ($P \leq 0.05$), with a different pattern of effect depending on sex and type of infection. No significant differences in AST were recorded between all groups in men, whereas men with both gastric ulcer and *H. pylori* (second group) showed a significant elevation in ALP and ALT levels compared to the healthy group.

Table 4- Relationship between gastric ulcer and liver function in men (mean $\pm$ SD)

Groups	Parameter	AST U/L	ALP U/L	ALT U/L
Groups 1 (Patients with gastric ulcer)		23.58 a ± 2.91	69.55 ab ± 5.05	30.58 ab ± 4.35
Groups 2 (Patients with gastric ulcer and <i>H. pylori</i> infection)		33.49 a ± 4.87	72.74 a ± 3.61	44.26 a ± 5.47
Groups 3 (Healthy controls)		24.79 a ± 4.10	56.15 b ± 6.48	25.94 b ± 2.43
P-Value		0.1650	0.031	0.0287

Different superscript letters (a, b, c) in the same column indicate significant differences at $P \leq 0.05$, while similar letters indicate no significant differences.

Regarding women, a significant elevation in AST level was observed in those with gastric ulcer only (first group) compared to other groups, and a significant elevation in ALT level in both the first and second groups (patients with gastric ulcer and those with gastric ulcer associated with *H. pylori*) compared to the healthy group.

Table 5- Relationship between gastric ulcer and liver function in women (mean $\pm$ SD)

Groups	Parameter	AST U/L	ALP U/L	ALT U/L
Groups 1 (Patients with gastric ulcer)		39.06 a ± 4.39	53.97 a ± 5.23	34.8 a ± 3.81
Groups 2 (Patients with gastric ulcer and <i>H. pylori</i> infection)		27.21 b ± 2.71	63.22 a ± 5.41	35.69 a ± 4.07
Groups 3 (Healthy controls)		26.99 b ± 2.85	57.28 a ± 6.50	23.02 b ± 2.60
P-Value		0.0302	0.4601	0.0386

Different superscript letters (a, b, c) in the same column indicate significant differences at $P \leq 0.05$, while similar letters indicate no significant differences.

These results generally indicate that gastric ulcer, especially when associated with *H. pylori* infection, is linked to disturbances in some liver function indicators; however, the nature of these disturbances differs between men and women. The effect is clearer on ALT and ALP in men, while a greater effect on AST and ALT is observed in women. However, there was generally an elevation in these enzymes in individuals with gastric ulcer associated and not associated with *H. pylori* infection.

The marked elevation in ALP and ALT levels in men with gastric ulcer accompanied by *H. pylori* is an indicator of liver cell involvement [32] and may be attributed to the oxidative stress and inflammatory response resulting from the infection. *H. pylori* stimulates the

production of inflammatory cytokines that reach the liver via the portal circulation, leading to cellular stress and elevated enzyme levels [33]. In contrast, the absence of a significant elevation in AST levels in men may indicate that the liver effect is still in its early stages, or that the damage is not deep histologically, especially since this enzyme is not limited to the liver but is also found in the heart and muscles [34].

In women, the significant elevation in AST in the group with gastric ulcer only, along with elevated ALT in both infected groups, indicates a clear effect of gastric ulcer and *H. pylori* infection on liver function, which agrees with a study [35] that reported an association between *H. pylori* infection and elevated AST levels.

Furthermore, [36] indicated that *H. pylori* infection increases the risk of non-alcoholic fatty liver disease (NAFLD). Similarly, [37] observed in another study that treating this bacterium improves liver function with a marked decrease in ALT levels.

On the other hand, the absence of significant differences in ALP levels in women may suggest that the inflammatory effect has not reached the stage of causing obvious tissue damage in the liver, especially in the bile ducts. The variation in elevation patterns between AST and ALT may be due to AST being present in tissues other than the liver, such as the heart and muscles, which might reflect a stress response in addition to liver involvement [16]

Moreover, [38] reported that *H. pylori* infection is associated with changes in serum, supporting the presence of an inflammatory response and elevated enzyme levels due to general stress. However, this effect may remain limited in its early stages, or the damage may not be deep in liver tissue. Enzyme levels also rise due to the general stress resulting from the systemic inflammation accompanying the infection. This result agrees with a study [39] that found no significant differences in some cases, suggesting that the liver effect may be stage-dependent or variable according to infection severity and individual factors. The difference between men and women in response patterns can be explained by physiological or hormonal differences that affect the severity of the inflammatory response or liver sensitivity to oxidative stress, leading to differences in liver function indicators between sexes.

3.3 Relationship between Gastric Ulcer and Gastrin and Secretin Hormone Concentrations

Tables (6) and (7) show the relationship between gastric ulcer and concentrations of gastrin and secretin in men and women, respectively. The results revealed no significant differences in the levels of these hormones among all groups for both sexes at probability level $P \leq 0.05$.

Table 6- Relationship between gastric ulcer and gastrin and secretin hormones in men (mean $\pm$ SD)

Groups	Parameter	Gastrin U/L	Secretin U/L
Groups 1 (Patients with gastric ulcer)		0.06 a ± 0.04	0.18 a ± 0.06
Groups 2 (Patients with gastric ulcer and <i>H. pylori</i> infection)		0.09 a ± 0.03	0.09 a ± 0.04
Groups 3 (Healthy controls)		0.07 a ± 0.02	0.16 a ± 0.08
	P-Value	0.7293	0.5429

Similar letters in the same column indicate no statistically significant differences at $P \leq 0.05$.

Table 7- Relationship between gastric ulcer and gastrin and secretin hormones in women (mean $\pm$ SD)

Groups	Parameter	Gastrin U/L	Secretin U/L
Groups 1 (Patients with gastric ulcer)		0.20 a 0.06 $\pm$	0.12 a 0.06 $\pm$
Groups 2 (Patients with gastric ulcer and <i>H. pylori</i> infection)		0.33 a 0.13 $\pm$	0.10 a 0.04 $\pm$
Groups 3 (Healthy controls)		0.07 a 0.02 $\pm$	0.04 a 0.02 $\pm$
	P-Value	0.2047	0.5930

Similar letters in the same column indicate no statistically significant differences at $P \leq 0.05$.

Despite the known physiological role of gastrin and secretin in regulating gastric acidity, the current results did not show significant differences, which may indicate that hormonal levels may not be acutely affected in the stages covered by the sample, or due to interference with other factors such as diet, medications used, or the stage of infection (chronic or acute). Gastric acid secretion is regulated through a complex system of neural and hormonal interactions, not through gastrin alone, which may weaken the appearance of clear hormonal differences between patients and healthy individuals [40]. Studies have also shown that changes in gastrin levels are not constant in all cases of gastric ulcer and may remain within normal limits in many patients [41], and that hormonal imbalance is not necessarily a consistent diagnostic criterion for all ulcer cases [42].

3.4 Relationship between Gastric Ulcer and White Blood Cell Parameters

Tables (8) and (9) show the relationship between gastric ulcer (with or without *H. pylori*) and various blood parameters in men and women, respectively, at probability level $P \leq 0.05$. Overall, the results showed a similar pattern between sexes, characterized by a significant increase in white blood cell count (WBC), lymphocytes (LYM), neutrophils (GRA), and middle cells (MID) (which refer to monocytes, eosinophils, and basophils) in both the first group (patients with ulcer only) and the second group (patients with ulcer and *H. pylori*) compared to the third group (healthy).

Table 8- Relationship between gastric ulcer and blood parameters in men (mean $\pm$ SD)

Groups	Parameter	WBC $10^9/l$	LYM $10^9/l$	MID $10^9/l$	GRA $10^9/l$
Groups 1 (Patients with gastric ulcer)		8.57 $\pm$ a 0.17	2.48 $\pm$ a 0.03	0.81 $\pm$ a 0.02	5.28 $\pm$ a 0.15
Groups 2 (Patients with gastric ulcer and <i>H. pylori</i> infection)		8.84 $\pm$ a 0.17	2.51 $\pm$ a 0.03	0.82 $\pm$ a 0.02	5.5 $\pm$ a 0.15
Groups 3 (Healthy controls)		7.91 $\pm$ b 0.12	2.26 $\pm$ b 0.03	0.82 $\pm$ a 0.02	4.83 $\pm$ b 0.11
	P-Value	0.003	0.001	0.781	0.023

Different superscript letters (a, b, c) in the same column indicate significant differences at $P \leq 0.05$, while similar letters indicate no significant differences.

Table 9- Relationship between gastric ulcer and blood parameters in women (mean $\pm$ SD)

Groups	Parameter	WBC $10^9/l$	LYM $10^9/l$	MID $10^9/l$	GRA $10^9/l$
Groups 1 (Patients with gastric ulcer)		8.24 $\pm$ a 0.16	2.4 $\pm$ a 0.04	0.79 $\pm$ a 0.03	5.05 $\pm$ a 0.11
Groups 2 (Patients with gastric ulcer and <i>H. pylori</i> infection)		8.48 $\pm$ a 0.11	2.48 $\pm$ a 0.03	0.83 $\pm$ a 0.03	5.17 $\pm$ a 0.05
Groups 3 (Healthy controls)		7.78 $\pm$ b 0.19	2.25 $\pm$ b 0.06	0.79 $\pm$ a 0.04	4.74 $\pm$ b 0.14
	P-Value	0.014	0.001	0.121	0.023

Different superscript letters (a, b, c) in the same column indicate significant differences at $P \leq 0.05$, while similar letters indicate no significant differences.

The study results clearly show a significant increase in white blood cell counts in both men and women, indicating a systemic inflammatory response associated with gastric ulcer and *H. pylori* infection. These results agree with a study [43] that showed a positive association between *H. pylori* infection and elevated WBC count in a large cross-sectional study, and are supported by a study [44] that confirmed significant differences between infected and non-infected individuals in several hematological parameters. Furthermore, elevated WBC has been identified as an independent risk factor for upper gastrointestinal bleeding in a study [45]. Notably, the elevation was more pronounced in men than in women, which may be attributed to hormonal influences, as a study [46] indicated that estrogen enhances innate immunity but may attenuate the severity of some inflammatory responses.

The results also showed a highly significant increase in lymphocyte counts in men and women in the first and second groups compared to healthy individuals. This increase reflects the activation of adaptive immunity in response to *H. pylori* antigens. A study [47] elucidates the molecular mechanism, where bacterial entry into gastric epithelial cells activates the NF- κ B pathway, stimulating the release of IL-8 and other cytokines that recruit lymphocytes.

A significant increase in absolute neutrophil count was recorded in men and women in the first and second groups compared to healthy individuals; neutrophils are the first line of defense against bacterial infection. These results agree with a study [48], and a study [32] showed that an elevated neutrophil-to-lymphocyte ratio is an important biomarker of systemic inflammation associated with *H. pylori* infection.

Comparing the two tables, it was observed that the magnitude of increase in WBC and GRA was slightly higher in men than in women, although the differences are minor. Studies explain this phenomenon through the sex hormone-gut microbiome axis, which indicates an interaction between sex hormones and microbiome composition, affecting the regulation of immune and inflammatory responses [49]. Furthermore, [50] indicated that androgens play a role in inducing sex differences in inflammation resulting from *H. pylori* infection, contributing to reducing inflammation severity in males. Conversely, a study [46] showed that estrogen enhances innate immunity and microbial diversity, while androgens and progesterone exert immunosuppressive effects, creating a difference in inflammation severity between sexes. Thus, the lower elevation in women may reflect a moderate effect of estrogen on the response, without completely abolishing it.

Although the analysis was performed separately for each sex to avoid bias from physiological differences, presenting data for both sexes provides a comprehensive overview of the disease profile in the Al-Rifai district population.

4. Conclusions

Gastric ulcer, especially when accompanied by *H. pylori* infection, is associated with a clear inflammatory response affecting liver function in both sexes. Infection is more concentrated in younger age groups, while most patients remain within normal weight limits, in addition to a higher infection rate among blood group O carriers.

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