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Research article

The Influence of *Helicobacter pylori* Infection on Hepatic Enzymes, Inflammatory Markers, and Hematological Parameters in Patients with Gastritis

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Abstract: *Helicobacter pylori* (HP) colonizes the gastric mucosa and causes gastric and extra-gastric disorders. Several liver diseases, such as Non-alcoholic fatty liver disease, seem to be caused by the gastrointestinal tract, which is primarily determined by the presence of bacteria like *Helicobacter pylori.* The study aims to evaluate the relationship between *Helicobacter pylori* infection and liver diseases by measuring liver enzymes, inflammatory markers, and hematological parameters among patients with gastritis and without *Helicobacter pylori* infection. A total of 1,325 patients who had gastritis diagnosed by gastroscopy were included in our study. They were divided into the HP– group (340 women, 323 men) and the HP+ group (330 women, 332 men). A control group (360 women, 260 men) without gastritis or HP infection was included in this study. HP was determined using the 13C urea breath test, which requires patients to swallow a capsule with water. Measurements of liver enzymes and complete blood cell counts were done in addition to interleukin-6(IL-6) and C-reactive protein (CRP). The results showed a significant increase in IL-6 and CRP in the HP+ group. Additionally, the mean levels of leukocytes and platelets were significantly increased in the HP+ group, but liver enzymes were within the normal range in both the HP+ & HP– groups. Although *Helicobacter pylori* causes elevation of indices of systemic inflammation, the liver did not show evidence of fatty infiltration, as liver enzymes were within normal limits.

Keywords: *Helicobacter pylori*; inflammatory markers; liver enzymes; hematological parameters

1. Introduction

Helicobacter pylori (HP) is a Gram-negative, helical-shaped pathogenic bacterium that attacks the stomach mucosa. It is believed that 50% of people worldwide are infected with HP, and it is well known that a persistent HP infection can lead to chronic gastritis, peptic ulcers, and stomach cancer [1,2]. In recent years, HP has been reported to be associated with the development of a variety of extra-alimentary disorders, including type 2 diabetes, and cardiovascular and hepatic diseases [3-5]. Since the "Gut-Liver Axis" has attracted a lot of attention, many liver diseases, such as Non-alcoholic fatty liver disease (NAFLD), are believed to be influenced by the environment of the gastrointestinal tract, which is largely determined by the presence of bacteria such as HP [6,7]. NAFLD is a category of disorders of the liver that includes

cirrhosis, nonalcoholic steatohepatitis, and steatosis. The most frequent cause of increased liver enzymes in adults in the US is nonalcoholic fatty liver disease[8]. Research on the relationship between HP infection and NAFLD has provided conflicting findings. According to certain research, NAFLD was considerably more common among HP+ individuals [9-11]. Other research, however, found no link between HP infection and NAFLD [12-14]. Our study aims to determine whether HP is a contributing factor in the natural progression of NAFLD or merely a coincidental finding by measuring liver enzymes, inflammatory markers, and hematological parameters in patients with gastritis with and without HP infection.

2. Materials and Methods

2.1 Study design

The study population was recruited from Iraqi adults who visited a clinic in Baquba, Iraq, between December 2020 and April 2022. A total of 1,325 Iraqi adults who had gastritis diagnosed by endoscopy were included in our study. They were divided into two groups: HP+ (330 women, 332 men) and HP- (340 women, 323 men), aged 30-65 years. A control group (360 women, 260 men) aged 30-60 years was included in this study who had neither gastritis nor HP infection. The study excluded patients taking medication for hypertension, hyperlipidemia, diabetes, hyperuricemia, or cardiovascular disease. Patients with chronic renal or hepatic failure were also ruled out. This work has been approved by the University of Bilad Al-Rafidain's ethical committee, College of Health and Medical Techniques, Iraq. On 20/11/2020, the authorization was acquired under reference number e.125.

2.2 HP Infection Detection

HP was determined using the 13C urea breath test, which requires patients to swallow a capsule with water. After a few minutes, a breath sample is collected. This sample is then processed to determine whether you have an active *H. pylori* infection[7]. Each patient completed a comprehensive questionnaire that included lifestyle factors, medical history, family history, and gastrointestinal symptoms. Regular measurements were taken of each participant's height, weight, and blood pressure.

2.3 Determination of body mass index

The Body mass index (BMI) was calculated as weight (kg) divided by height squared (m^2).

2.4 Determination of liver enzyme levels

Morning venous blood of all participants was obtained to detect the levels of serum alanine aminotransferase (ALT), aspartate aminotransferase (AST), gamma-glutamyl transferase (GGT) & total serum bilirubin (TSB) using an autoanalyzer (Beckman, Palo Alto, CA, United States).

2.5 Determination of inflammatory and hematological parameter levels

By using Cobas e 411, the levels of serum CRP and IL-6 were determined. A Sysmex XE-2100 analyzer was used to perform whole-blood cell counts (Sysmex, Kobe, Japan).

2.6 Statistical analysis

Regarding numerical aspects, statistical analysis was conducted using SPSS (Statistical Package for the Social Sciences) version 22. For determining the samples under investigation. The normality of the data distribution was tested before selecting parametric tests. The mean and standard deviation of the mean were used, and the averages were compared at the 0.05 level. ANOVA is a statistical test used to compare means across three groups. A post hoc test was used because multiple groups were compared.

3. Results and Discussion

3.1 Liver enzyme levels

Results of the current study (Table 1) showed that liver enzymes were within the normal range in both the control and experimental groups, and there were no significant differences ($p>0.05$) in liver function indexes (TSB, ALT, AST, GGT).

Table 1. The relationship between the HP status and liver function indexes (mg/dL).

		Mean	SD	Statistics
TSB (mg/dL)	**control**	0.86	0.36	P>0.05
0.2-1.2	**Gastritis with *H. pylori***	0.88	0.31	
	Gastritis without *H. pylori*	0.73	0.29	
ALT (mg/dL)	**Control**	30.69	12.38	P>0.05
10.0-55.0	**Gastritis with *H. pylori***	35.37	13.45	
	Gastritis without *H. pylori*	33.88	13.65	
AST (mg/dL)	**Control**	28.46	6.80	P>0.05
10.0-40.0	**Gastritis with *H. pylori***	27.98	7.57	
	Gastritis without *H. pylori*	25.50	6.15	
GGT (mg/dL)	**Control**	21.80	7.31	P>0.05
8.0-35.0	**Gastritis with *H. pylori***	23.43	7.20	
	Gastritis without *H. pylori*	23.31	7.02	

Abbrev: TSB: total serum bilirubin, ALT: alanine transaminase, AST: aspartate aminotransferase, GGT: gamma- glutamyl transpeptidase.

By dividing the study sample by sex in Table 2, we found no significant association between liver function indicators and HP infection status.

Table 2. Comparison of liver function indexes (mg/dL) in different genders.

		Gastritis with HP		Gastritis without HP		Statistics
		Mean	SD	Mean	SD	
TSB	**Males**	0.74	0.20	0.72	0.33	P>0.05
	Females	1.02	0.33	0.74	0.26	
ALT	**Males**	39.29	6.79	34.28	14.91	P>0.05
	Females	31.46	17.37	33.48	13.06	
AST	**Males**	29.78	5.83	26.64	6.97	P>0.05
	Females	26.18	8.94	24.36	5.33	
GGT	**Males**	22.60	7.05	22.97	7.60	P>0.05
	Females	24.25	7.64	23.65	6.79	

Abbrev: TSB: total serum bilirubin, ALT: alanine transaminase, AST: aspartate aminotransferase, GGT: gamma- glutamyl transpeptidase.

3.2 Inflammatory and hematological parameter levels

The mean serum levels of CRP and IL-6 were significantly (P<0.001) higher in the HP+ group than in the HP- & control groups, respectively. Also, there were no differences in hemoglobin measurements among the HP+, HP-, and control groups (p>0.05); however, there were significant differences in white blood cells (P<0.001) and platelets (P<0.05). The mean serum white blood cell levels were significantly higher in the HP+ group than in the HP– and control groups. The mean platelet count was within the normal range. However, their mean levels were significantly higher in the HP + group, followed by the HP – and control groups (Table 3).

Table 3. Correlation between HP infection and inflammatory and hematological measures in patients with gastritis.

		Mean	SD	Statistics
IL-6 (pg/ml) 7 ≥ pg/mL	**Control**	7.94	1.63	P<0.001*** LSD=3.34
	Gastritis with *HP*	19.33	2.06	
	Gastritis without *HP*	14.64	2.59	
CRP (mg/L)	**Control**	4.09	1.02	P<0.001*** LSD=2.92
	Gastritis with *HP*	15.62	3.39	
	Gastritis without *HP*	13.37	4.65	
WBCs *109 (4.0-11.0)	**Control**	9.59	2.58	P<0.001*** LSD=0.37
	Gastritis with *HP*	13.76	1.57	
	Gastritis without *HP*	12.20	1.63	
Hb (g/dL) (11.0 - 16.0)	**Control**	12.33	2.92	P>0.05
	Gastritis with *HP*	12.92	0.88	
	Gastritis without *HP*	13.24	1.24	
PLTs (m/mm^3) (150-450)	**Control**	309.70	47.53	P<0.05* LSD=29.45
	Gastritis with *HP*	335.79	48.72	
	Gastritis without *HP*	285.75	69.99	

Abbrev: IL-6: interleukin-6, CRP: C-reactive protein, WBCs: white blood cell count, Hb: hemoglobin, PLTs: platelet count.

Table 4 showed a comparison of hematological and inflammatory parameters by gender. Males had higher Hb, but females had higher CRP, IL-6, WBC, and platelets.

Table 4. Correlation between HP infection and inflammatory and hematological measures in patients with gastritis.

		Gastritis with *H. pylori*		Gastritis without *H. pylori*		Statistics
		Mean	SD	Mean	SD	
IL-6 [15]	**Males**	18.53	1.38	14.09	1.90	P<0.001***
	Females	20.14	2.37	15.19	3.14	LSD=3.88
CRP	**Males**	15.42	4.54	10.02	3.88	P<0.001***
	Females	15.82	1.89	16.72	2.38	LSD=2.68
WBCs	**Males**	12.48	0.85	11.34	1.69	P<0.001***
	Females	15.04	0.93	13.05	1.06	LSD=1.13
Hb	**Males**	12.97	0.73	12.76	1.23	P>0.05
	Females	12.86	1.04	13.72	1.11	
PLTs	**Males**	314.59	26.85	246.90	51.58	P<0. 05*
	Females	356.99	57.38	324.60	65.78	LSD=41.39

Abbrev:IL-6: interleukin-6, CRP: C-reactive protein, WBCs: white blood cell count, Hb: hemoglobin, PLTs: platelet count

The presence of the *H. pylori* bacteria influences the composition, balance, and activity of the microbiota, which, in turn, influences metabolic capability, immune response, and homeostasis in the human stomach. Because the interrelationships between bacteria dictate the illness burden, treating and eradicating H. pylori may result in additional chronic human diseases whose pathogenic mechanisms have yet to be confirmed [16]. Socioeconomic status may serve as a proxy for additional, undisclosed risk factors for *H. Pylori* infection. According to epidemiological studies, people with low socioeconomic positions are more likely to develop peptic ulcer disease [17]. The current study showed that, while liver enzyme levels in the experimental and control groups were within the normal range, HP infection was not associated with an elevated risk of NAFLD. Numerous clinical studies have been conducted to investigate the association between NAFLD and HP infection [18]. According to [18] Wijarnpreecha et al., 2018 comprehensive review and meta-analysis, there is a substantial association between NAFLD and HP infection in patients [19]. Additionally, a cohort study

conducted by Kim *et al. (2017) identified an association between HP and* NAFLD [20]. On the other hand, a large-scale cross-sectional study by Okushin et al. (2015) found that HP infections were not significantly associated with NAFLD11. Additionally, a retrospective cohort study revealed that NAFLD risk was not associated with HP infection [21]. There were two other studies that demonstrated a negative association between HP infection and NAFLD [22-23]. The inconsistency between the above studies may be due to geographical or ethnic variations, with genetic predisposition as the differentiating factor between patients. Regarding HP infection and NAFLD, there was no gender difference between men and women. This result is inconsistent with the finding that HP infection is linked to NAFLD in female patients [24]. Changes in WBC count with HP+ may reflect the severity of gastric mucosal inflammation, which can cause both local and systemic inflammation, a contributing factor to its involvement in extra-gastric disorders [25-26,27]. In turn, this systemic inflammation (IL-6 and CRP) can trigger several extra-gastrointestinal conditions, including metabolic syndrome, diabetes, insulin resistance, and cardiovascular disease [28-29]. In our study, in addition to the significant increases in IL-6 and CRP, the mean levels of leukocytes and platelets were significantly higher in the HP + group, but liver enzymes were within the normal range in both the HP + & HP – groups.

4. Conclusions

Although *Helicobacter pylori* causes elevation of indices of systemic inflammation, the liver does not show evidence of fatty infiltration, as liver enzymes were within normal limits. Further research should investigate additional variables to reveal the effect of *H. pylori* infection on liver disease.

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