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Association Between H. *pylori* Antibody and Non-Alcoholic Steatohepatitis

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ABSTRACT

Objective: *The disorder known as nonalcoholic fatty liver disease affects the liver as a result of normal fat accumulation. Another type of fatty liver is associated with alcohol consumption: nonalcoholic steatohepatitis (NASH), which is more severe than NAFLD. Research suggests that Helicobacter pylori infection and NAFLD may be related. Objective: The purpose of this research is to examine the connection between infection with Helicobacter pylori and NASH through specific tests to assess liver function, metabolic measurements, and the severity of infection progression.* ***Method:*** *At Al-Diwaniyah Gastroenterol Hospital in Al-Diwaniyah, 70 patients with nonalcoholic fatty liver diseases (NAFLD) as 60 healthy volunteers participated in this comparative study. A venous blood sample was taken in order to identify Helicobacter pylori infection. Serum immunoglobulin G (IgG) as fecal H. pylori antigen were measured using an enzyme-linked immunosorbent test (ELISA). Other laboratory parameters were examined, including liver enzymes (aspartate aminotransferase (AST) and alanine aminotransferase (ALT)). NAFLD was diagnosed & its severity assessed using ultrasound.* ***Results:*** *The NAFLD patient group consisted of 40 females, whereas 28 females made up the control group, 35 participants in the non-alcoholic fatty liver disease NAFLD group had a grade 1 infection, 32 had grade 2, and 3 had grade 3. Serum IgG antibody levels for Helicobacter pylori were positive in 36 patients in the NAFLD group and in 35 patients in the control group. The mean (± standard deviation) level of H. pylori IgG antibody was higher in the NAFLD group than in the control group. Additionally, the NAFLD group had a higher frequency of fecal pylori antigen than the controls group (25 vs. 17 individuals). The frequency of H. When using serum IgG antibody results rather than stool H. pylori Ag data, pylori infection was higher in both the NAFLD and control groups.* ***Novelty:*** *This study introduces a novel comparative evaluation by simultaneously analyzing both serum IgG antibodies and fecal antigens alongside ultrasound-graded NAFLD severities to highlight the distinct diagnostic discrepancies between systemic and active localized H. pylori infections in metabolic liver disorders. By uncovering a higher positional frequency of fecal antigens specifically within the NAFLD cohort, this research offers new clinical insights into how gastrointestinal pathogen loads might correlate directly with the progressive staging of nonalcoholic fatty liver accumulation.*

INTRODUCTION

Fat accumulation in the liver causes a condition known as nonalcoholic fatty liver disease, or NAFLD. Normally, a healthy liver contains very little fat, and in some individuals, no fat may be present at all, which is also considered normal [1]. Another type of fatty liver is associated with alcohol consumption. This condition, known as alcoholic fatty liver disease (AFLD), develops as a result of excessive alcohol intake [2]. There are two main types of AFLD: NAFLD. In this type, the liver is characterized by fat accumulation and minimal inflammation, and it does not usually lead to cirrhosis . NASH: This type is more severe than NAFLD. People with NASH have severe

inflammation in addition to the accumulation of fat in their livers, and this damage frequently results in cirrhosis [3].

Nonalcoholic fatty liver disease (NAFLD) usually doesn't cause symptoms until it reaches an advanced stage, when the liver has begun to scar and become fibrotic. When symptoms do appear, they may include: fatigue and exhaustion even with minimal activity; Abdominal pain on the upper right side; inexplicable weight loss; enlarged blood vessels just beneath the skin's surface; nausea; abdominal bloating; leg swelling due to fluid retention; and yellowing for the skin and eye whites (jaundice) [4]. Risk factors for NAFLD include obesity, insulin resistance, and high triglyceride levels [5]. *Helicobacter pylori*, sometimes known as H. *pylori*, is a spiral-shaped bacteria that is gram-negative. It is a significant contributor to some ulcers of the stomach in the stomach and duodenum as well as chronic gastritis. Therefore, eliminating the bacteria in individuals with duodenal and stomach ulcers greatly reduces the likelihood of the illness returning [4]. One of the most prevalent infectious agents in the globe is H. *pylori*, present in more than half the global population, but only a small percentage of those infected develop symptoms [6]. The primary disease caused by H. *pylori* is peptic ulcer disease. The bacteria damage the gastric mucosa through several mechanisms, including direct damage from substances secreted by the bacteria, such as urease (which produces ammonia), lipase, and protease. (Protease) [7]. Damage resulting from the immune response caused by inflammation. Damage resulting from changes in local mucosal hormones, particularly a decrease in somatostatin production and an increase in gastrin production, which increases gastric acid secretion [8].

Numerous research have looked into the connection between non-alcoholic fatty liver disease, also known as N and *Helicobacter pylori* infection. These studies found a significant increase in NAFLD among individuals infected with H. *pylori* [9]. However, some findings remain controversial [10].

According to certain research, there may be a direct connection between *Helicobacter pylori* infection & nonalcoholic fatty liver disease, or N. However, The underlying low-grade inflammation to chronic H. *pylori* infection appears to be connected to insulin resistance and problems in lipid metabolism, while the exact mechanisms behind this relationship are yet unknown [11], [12]. Meta-analyses by [13], showed that H. *pylori* carriers had a markedly higher incidence of NAFLD.

RESEARCH METHOD

Study Design

A case-control approach was used to determine the factors that significantly affect NAFLD. A cross-sectional comparison approach was also used to identify the factors that significantly affect NAFLD scores. The research was carried out from May 2025 to November 2025.

Study Participants

Seventy individuals with nonalcoholic fat liver disease (NAFLD) participated in this comparative investigation. and 60 healthy volunteers at the Diwaniyah Digestive System

Hospital in Diwaniyah, Iraq (May 2025–November 2025). Ultrasound diagnosis of NAFLD showed hepatic steatosis exceeding 5% of hepatocytes, excluding secondary causes such as excessive alcohol consumption, viral infections, or drug use, according to established criteria [14]. Three age groups of patients were identified: 21–28 years old (n = 11), 31–38 years old (n = 28), and 41–48 years old (n = 31). Controls consisted of age-matched healthy adults without liver disease. Lipid levels [HDL (high-density lipoprotein), LDL (low-density lipoprotein), total cholesterol, and triglycerides].

Gastrointestinal symptoms, such as dyspepsia, GERD, or upper abdominal pain characteristic of a peptic ulcer, were not encountered by either group of subjects.

A venous blood specimen was taken in order to identify Helicobacter pylori, also referred to type 1 infection. An enzyme-linked immunosorbent test (ELISA) was used to assess serum immunoglobulin G (IgG) and stool H. pylori antigen (Generic test, Germany). Patients were diagnosed with H. pylori infection if both tests came back positive. Other laboratory variables were also assessed, such as liver enzymes like aspartate transaminase (AST) and alanine transaminase (ALT). Every patient gave their informed permission.

Data Collection

A venous blood specimen was taken in order to identify H. pylori type 1 infection. Serum immunoglobulin G (IgG) was measured using the enzyme-linked immunosorbent assay (ELISA) and fecal H. pylori antigen (Generic Assay, Germany). Patients were diagnosed with H. pylori infection if both tests came back positive. Other laboratory parameters were also examined, such as lipid profiles (high-density lipoprotein (HDL), a low-density lipoprotein (LDL), a cholesterol total, & triglycerides) and liver enzyme levels (alanine transaminase (ALT), and aspartate transaminase (AST)).

Statistical Analysis

SPSS software for Windows was used to express descriptive indices (frequency, mean, standard deviation, and percentage) for the total data (21.0 version). The chi-square test & the Student's t-test were used to compare the Helicobacter pylori infections rates & IgG antibody titers between both groups. (It was determined that the p-value was 0.05.).

Ethics

Our medical university's Ethics Committee gave its approval to the study protocol. Before patients could participate in the study, they were informed of its goals and specifics, and after they agreed, written informed permission was obtained.

RESULTS AND DISCUSSION

Results

There were 28 females on control group (46.5%) and 40 females on the NAFLD group (57.5%); p = 0.16. Liver enzyme levels, age, and sex are compared between two groups in Table 1. 32 patients (45.5%) had a grade 2 illness, 3 patients (4.5%) had grade three disease, and 35 people (50%) had the first grade disease in the NAFLD group. 36 patients of the NAFLD group (51.4%) & 35 patients of the control group (58.3%), had

positive blood levels of anti-H pylori IgG; p = 0.86. The nonalcoholic fatty liver disease group had a higher mean (± a standard deviation) anti-H pylori IgG level (77.1 ± 8.9 IU/mL) than the controls group (50.7 ± 6.2 IU/mL). p is 0.03. Additionally, the NAFLD group had higher rates of positive feces H. pylori antigen than the controls group (25 patients, or 35.5% vs. 17 individuals (28.3%)), however there was no statistically significant change (p = 0.13). In both the NAFLD and the control groups, the prevalence for H. pylori infection was greater when serological IgG test findings were taken into account instead of fecal antigen test results.

Table 2 displays the distribution of people who had at least one or more positive test results. As said, based on positive IgG & H. pylori globulin test findings, The frequency of Helicobacter pylori infection did not significantly differ between the research groups.

Table 3 looks at mean (± sd) anti-H pylori IgG levels in the control groups of individuals with positive and negative fecal antigen (FEA) tests and those with nonalcoholic liver disease (NAFLD). Patients that have positive FEA results had considerably higher mean anti-H pylori IgG levels. The examined groups showed both high and low levels in anti-H. pylori IgG. Establishing a threshold value to H. pylori IgG to forecast positive or negative FEA findings was not feasible.

Thirty-four patients (25.5%) in both the NAFLD and control groups had positive H. pylori IgG and FEA results. Table 4 compares lipid levels of healthy controls and individuals with nonalcoholic fatty liver disease.

Table 1. Gender, age, as well as hepatic transaminases were compared between individuals with NAFLD) and healthy controls.

	NAFLD (N = 70)	**Healthy control (N = 60)**	**p value**
Age	34.5 (±8.5)	33.5 (±9.2)	0.1
Gender			
Male	30 (42.5%)	32 (53.5%)	0.1
Female	40 (57.5%)	28 (46.5%)	
AST, U/L	29.09 (±15.2)	20.4 (±9.2)	0.2
ALT, U/L	39.3 (±23.3)	23.06 (±20.6)	0.01

Table 2. Patients in the NAFLD and the control groups who had no less than one positive test and a positive as well as H. pylori test (anti-H. pylori IgG & fecal antigen test).

	Total	**NAFLD (N= 65)**	**Control (N = 65)**	**P**
Both tests were positive	34(25.5%)	21 (32.8%)	13 (21.1%)	0.17
At least one test was positive	79 (59.1%)	40(62.6%)	39(60.5%)	0.71

Table 3. Comparing the two patient groups' mean (SD) anti- H pylori IgG titers and those whose fecal H. Pylori Ag testing is negative.

	Total	Mean(±SD) H. pylori IgG titer	Range of H. pylori IgG titer	P value
Positive fecal antigen test, IU/mL	42	94.9 (±71.8)	4-287	< 0.001
Negative fecal antigen test, IU/mL	88	50.6 (±66.4)	11-263	

Table 4. Lipid profile comparison between individuals with nonalcoholic fatty liver disease (NAFLD) and healthy controls.

	NAFLD (N = 70)	Healthy control (N = 60)	p value
Total cholesterol, mg/dL	185.2 (±46.04)	153.9 (±51.2)	0.01
HDL, mg/dL	41.5 (±14.3)	39.1 (±7.4)	0.3
LDL, mg/dL	118.4 (±31.5)	113.3 (±32.2)	0.5
Triglyceride, mg/dL	166.6 (±78.3)	119.9 (±40.7)	< 0.001

The mean (standard deviation) is used to display all data. LDL stands for low-density lipoprotein, and HDL for high-density lipoprotein.

Discussion

A major health concern brought on by high fructose consumption is non-alcoholic fatty liver disease (NAFLD) (Clemente et al., 2019). As NAFLD worsens, liver cell damage can lead to cirrhosis. Helicobacter pylori (H. pylori) is the most common gastrointestinal bacterium. Its prevalence in developed countries is around 20%, while in developing countries it reaches 70% [15].

The prevalence for H. pylori infection does not significantly differ between people with NAFLD as well as healthy controls, according to our study's findings. Studies have been carried out to look at the possible role for H. pylori to the pathophysiology of NAFLD because both infection with H. pylori & NAFLD are prevalent medical disorders. These investigations were carried out in response to findings linking Helicobacter pylori to extragastrointestinal conditions such hepatobiliary, neurological, and cardiovascular disorders [16], [17].

Helicobacter pylori with insulin resistance have been linked, but not causally [18], [19]. Helicobacter pylori's possible involvement in liver illnesses, especially nonalcoholic liver disease (NAFLD), has drawn attention since resistance to insulin is a significant

contributing factor to NAFLD. The extragastrointestinal role of Helicobacter pylori in a hepatobiliary system has been studied outside of NAFLD. Even in situations of hepatocellular carcinoma and H. pylori type a DNA has been detected in a significant number of liver biopsy specimens [20].

A larger percentage in patients with NAFLD (23 for 25, 82.1%) are seropositive to anti-H. pylori IgG than control group (14 to 25, 56%); p = 0.03, according to a prior study that looked at 28 cases with NAFLD (15 with a NAFLD vs 13 in NASH) with 25 controls. Our research did not include those who had received H. pylori eradication therapy in who had taken antibiotics used for eradication regimens during the six months prior. Additionally, NASH patients were not recruited for this study. However, in the previously mentioned study [21], One patient in the control group and six patients in the NAFLD group had previously undergone H. pylori eradication treatment.

When the investigators combined Helicobacter pylori seropositivity in H. pylori eradication treatment, the difference between these two groups became more significant (56% in a control group and 92% in people in nonalcoholic liver disease; p = 0.002). However, there was no discernible difference between the two categories of NAL & NAL steatohepatitis when further studies were conducted, such as urea breath testing, IgG testing, and a history to H. pylori eradication medication [21].

The scientists came to the conclusion that while Helicobacter pylori may contribute to the onset of nonalcoholic fatty liver disease (NAFLD), it does not cause NAFLD to advance to non-alcoholic steatohepatitis (NASH) [21]. Here, we diagnosed Helicobacter pylori infection using both fecal antigen and IgG testing as objective markers. In an earlier study conducted in Iran [22], it was discovered that the feces antigen test has an 87.8% sensitivity and a 75% specificity, making it a non-invasive test. However, Serum IgG sensitivity was only 50%. According to this article, the most effective way to diagnose Helicobacter pylori infection is to request a fecal antigen test. Unlike the previously described study, two additional investigations did not discover any connection between H. pylori & nonalcoholic fatty liver disease. Abdominal ultrasonography was utilized to detect FLD and NAFLD in a large study in 13,737 individuals in Japan. H. pylori type antibodies were tested to H. pylori infection. The researchers found that infection with H. pylori was not linked to NAFLD in either sex using a multivariate regression analysis (p-values 0.7 to females and 0.4 in males) [23]. Another study conducted in South Korea involved 4,030 patients who underwent a urea breath test to diagnose infection with Helicobacter pylori. The hepatic fat index & the NAFLD score were used to evaluate nonalcoholic liver disease (NAFLD) [24].

Similar to another study conducted in Japan, researchers did not find H. pylori infection to be a significant risk factor for NAFLD based on regression analysis. Studies on this topic have also attempted to demonstrate the role of H. pylori eradication in liver fat content and function. In a randomized clinical trial, Jamali et al. used H. pylori IgG to diagnose the resulting infection and a urea breath test to determine H. pylori eradication six weeks after standard H. pylori eradication therapy in 100 patients with dyspepsia and elevated transaminase levels [25]. They found that H. pylori eradication did not It does

not significantly affect liver function and fat content. Although H. pylori IgG titers were observed to be higher in NAFLD patients compared to control groups, combined IgG and fecal antigen tests did not show any significant difference in the diagnosis of H. pylori infection. Even when one of these tests was positive, no significant difference was observed between the two groups studied. Here, the patients included were asymptomatic with regard to gastrointestinal problems. We believe that perhaps in other studies recruiting two groups of patients—one with symptomatic H. pylori-positive infection and the other an asymptomatic sample with H. pylori-positive infection—and comparing these two groups with regard to the relationship between H. pylori infection and NAFLD would add to current knowledge. In other words, there may be differences in liver fat content in those with symptomatic H. pylori infection.

A relationship between non-alcoholic fatty liver disease and H. pylori infection has recently emerged, with a possible causal link between the two. Helicobacter pylori infection exacerbates non-alcoholic fatty liver disease. This is explained by the effect of H. pylori in causing mild systemic inflammation and the release of inflammatory cytokines, which in turn affect lipid metabolism, disrupt the gut microbiota, alter the intestinal barrier, and promote insulin resistance.

Another mechanism is that cells infected with Helicobacter pylori may release extracellular vesicles, and H. pylori can also release extracellular membrane vesicles, which can directly affect the liver and contribute to the development of (NAFLD) [8]. H. pylori infection has also been found in liver tissue of NAFLD patients, according to earlier research. Cag A and Vac A are the two main pathogenic agents of H. pylori. Cag A-positive spots are characterized by their motility and ability to produce inflammatory cytokines, leading to intestinal bacterial imbalance and increased intestinal permeability [26], [27]. Studies have also shown that Helicobacter pylori bacteria can enter and remain in liver cells, which depends mainly on the Cag A and Vac A status

CONCLUSION

Fundamental Finding : This study found no statistically significant difference in the prevalence of Helicobacter pylori infection between individuals with nonalcoholic fatty liver disease (NAFLD) and healthy controls when serum IgG, fecal antigen, and combined test results were considered. Although the mean anti-H. pylori IgG titer was significantly higher in the NAFLD group, IgG and fecal antigen positivity rates were comparable between groups. Patients with NAFLD also had higher alanine aminotransferase, total cholesterol, and triglyceride levels, suggesting that metabolic and hepatic abnormalities were more prominent than differences in H. pylori status. **Implication :** Accordingly, these findings do not establish H. pylori as a direct risk factor for NAFLD or progression to nonalcoholic steatohepatitis. **Limitation :** A primary limitation of this study is its cross-sectional design, which precludes the assessment of a temporal or causal relationship between the intensity of Helicobacter pylori infection and the longitudinal progression of metabolic hepatic conditions. Furthermore, the analysis is limited by the lack of direct adjustment for critical confounding variables, such as body

mass index (BMI), insulin resistance, concurrent type 2 diabetes, and specific long-term dietary habits, which may independently drive metabolic and lipid abnormalities within the patient cohort. **Future Research :** Larger prospective studies controlling for obesity, insulin resistance, diabetes, and lipid abnormalities are required to clarify this possible association.

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