Original Article
DOI: https://doi.org/10.70749/ijbr.v3i5.2740

INDUS JOURNAL OF BIOSCIENCE RESEARCH
https://ijbr.com.pk
ISSN: 2960-2793/ 2960-2807

Relevance between Helicobacter Pylori Infection and Non-Alcoholic Fatty Liver Disease in Tertiary Care Hospital

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ARTICLE INFO

Keywords: *Helicobacter Pylori*, Nonalcoholic Fatty Liver Disease, Metabolic Dysfunction, Dyslipidemia, Insulin Resistance, Hepatic Steatosis.

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Declaration
Authors' Contribution
All authors equally contributed to the study and approved the final manuscript

Conflict of Interest: No conflict of interest.

Funding: No funding received by the authors.

Article History
Received: 01-02-2025 Revised: 26-04-2025
Accepted: 15-05-2025 Published: 30-05-2025

ABSTRACT

Background: Nonalcoholic fatty liver disease (NAFLD) has become the leading cause of chronic liver disease worldwide, strongly linked to obesity, insulin resistance, and dyslipidemia. Emerging evidence suggests that infectious agents, particularly *Helicobacter pylori*, may contribute to metabolic dysfunction and hepatic steatosis through systemic inflammation, oxidative stress, and disruption of lipid metabolism. However, the nature and strength of this association remain uncertain, especially in South Asian populations where both *H. pylori* infection and metabolic disorders are highly prevalent. **Aim:** This study aimed to investigate the association between *H. pylori* infection and NAFLD, and to assess related metabolic and anthropometric correlates among adults attending a tertiary-care hospital in Peshawar, Pakistan. **Methods:** A retrospective cross-sectional study was conducted on 200 adult participants evaluated between June and November 2024. *H. pylori* infection was diagnosed using urea breath or stool antigen testing, and NAFLD was identified and graded by abdominal ultrasonography. Anthropometric, clinical, and biochemical data were compared between *H. pylori*–positive and – negative participants. Statistical analyses included chi-square and independent *t*-tests for group comparisons, multivariable logistic regression for predictors of NAFLD, and Spearman's rho (ρ) for correlation analysis. **Results:** *H. pylori* infection was present in 55.0% of participants, while NAFLD was diagnosed in 35.0%. The prevalence of NAFLD was higher in *H. pylori*–positive individuals (38.2%) than in uninfected ones (31.1%). Obesity, dyslipidemia, and type 2 diabetes were significantly more common among infected participants. *H. pylori* infection remained an independent predictor of NAFLD ($p = 0.041$). NAFLD showed positive correlations with dyslipidemia ($\rho \approx 0.61$) and BMI ($\rho \approx 0.54$). **Conclusion:** *H. pylori* infection is significantly associated with NAFLD and its metabolic correlates, suggesting a contributory role in hepatic steatosis through inflammatory and metabolic pathways. Longitudinal and interventional studies are warranted to clarify causality and evaluate the impact of eradication therapy.

INTRODUCTION

Nonalcoholic fatty liver disease (NAFLD), recently reclassified as metabolic dysfunction– associated steatotic liver disease (MASLD), has emerged as the leading cause of chronic liver disease worldwide, affecting approximately one quarter of the global adult population (1). The prevalence is particularly high in Asia, driven by increasing rates of obesity, insulin resistance, and sedentary lifestyles (2). NAFLD represents a spectrum of hepatic abnormalities ranging from simple steatosis to nonalcoholic steatohepatitis (NASH), fibrosis, cirrhosis, and hepatocellular carcinoma (HCC). The growing burden of NAFLD constitutes a major public health challenge, as it is strongly linked to extrahepatic complications, including cardiovascular disease, diabetes, and chronic kidney disease. Despite this, effective pharmacological therapies remain limited, underscoring the need to identify modifiable risk factors that may contribute to disease onset and progression (3).

Over the past decade, infectious agents have gained increasing attention as possible contributors to metabolic and hepatic dysfunction. Among them, Helicobacter pylori

(H. pylori)—a Gram- negative microaerophilic bacterium that colonizes the human gastric mucosa—has long been implicated in chronic gastritis, peptic ulcer disease, and gastric carcinoma (4). However, accumulating evidence suggests that H. pylori may exert systemic effects beyond the gastrointestinal tract. Studies have reported associations between chronic H. pylori infection and metabolic disorders, including insulin resistance, dyslipidemia, and type 2 diabetes mellitus (5). These findings raise the possibility that H. pylori infection could influence hepatic lipid accumulation and inflammation, contributing to the pathogenesis of NAFLD. Several epidemiological studies have explored the link between H. pylori infection and NAFLD, though results have varied across populations. A large meta-analysis of 22 studies involving over 117,000 participants found that H. pylori infection increased the odds of NAFLD by approximately 22 % after adjustment for metabolic covariates (6). Another systematic review confirmed a similar association, with a pooled odds ratio of 1.26 (95 % CI 1.10–1.44). More recent longitudinal data suggest that H. pylori–positive individuals have a higher risk of incident fatty liver and faster progression of steatosis over time (7). Conversely, several investigations have reported weak or non-significant relationships, particularly when confounders such as body-mass index (BMI) and insulin resistance are fully adjusted (8). The heterogeneity of findings may relate to differences in diagnostic criteria, study design, ethnic background, and bacterial virulence factors, such as cytotoxin-associated gene A (CagA) positivity (8).

The biological plausibility of an H. pylori–NAFLD link rests on several mechanistic hypotheses. Chronic H. pylori infection induces systemic inflammation characterized by elevated levels of tumor necrosis factor-α (TNF-α), interleukin-6 (IL-6), and high-sensitivity C-reactive protein, all of which interfere with hepatic insulin signaling and promote steatogenesis (9). Moreover, H. pylori infection is associated with insulin resistance independent of obesity, suggesting that bacterial-driven inflammation may directly impair glucose homeostasis (10). Lipid metabolism also appears to be affected: infected individuals frequently exhibit reduced high-density lipoprotein (HDL) cholesterol and elevated triglyceride levels. Experimental studies demonstrate that the H. pylori virulence factor CagA disrupts hepatic lipid regulatory pathways, amplifying oxidative stress and lipid peroxidation (11). Together, these findings indicate that H. pylori–induced metabolic derangements could contribute to hepatocellular lipid accumulation.

Another proposed mechanism involves the gut–liver axis. H. pylori infection alters gastric and intestinal microbiota composition and compromises gut barrier integrity, facilitating translocation of bacterial products such as lipopolysaccharides into portal circulation (12). These endotoxins activate hepatic toll-like receptors, initiating inflammatory cascades that aggravate hepatic steatosis and fibrosis. In addition, infection-related alterations in adipokine profiles—such as decreased adiponectin and increased leptin—further disrupt metabolic balance (13). Such hormonal dysregulation may provide a systemic conduit through which gastric infection contributes to peripheral and hepatic insulin resistance. Supporting this hypothesis, studies have reported improved metabolic and inflammatory profiles following successful H. pylori eradication, including reductions in fasting glucose and lipid levels (14).

Despite these plausible mechanisms, causality remains uncertain. Most published data are cross-sectional, limiting temporal inference, and Mendelian randomization analyses have not demonstrated a direct genetic causal pathway between H. pylori infection and NAFLD. Furthermore, geographical variability in both H. pylori prevalence and host metabolic profiles complicates extrapolation. The infection remains endemic in many low- and middle- income countries, including Pakistan, where prevalence estimates exceed 50 % (15). Concurrently, the region faces a sharp rise in obesity and diabetes, creating a “double epidemic” of metabolic and infectious burden. However, regional data exploring the interplay between H. pylori infection and hepatic steatosis are limited, and most available studies derive from East Asia or Western populations with different dietary and genetic backgrounds.

Clarifying whether H. pylori infection independently contributes to NAFLD holds potential clinical implications. If infection exacerbates hepatic fat accumulation or inflammation, early detection and eradication could represent an accessible preventive strategy in resource-limited settings. Conversely, if the observed association is merely confounded by shared metabolic risk factors, clinical focus should remain on metabolic control rather than antimicrobial therapy. Therefore, rigorous, population-specific studies that integrate reliable diagnostic modalities—such as urea breath or stool antigen testing for H. pylori and ultrasound-based grading for NAFLD— are needed to delineate the relationship more clearly.

The present study addresses this knowledge gap by evaluating the association between H. pylori infection and NAFLD among adults in a tertiary-care hospital in Peshawar, Pakistan. By analyzing demographic, anthropometric, and metabolic parameters in H. pylori–positive and –negative individuals, this research aims to determine whether infection status correlates with the presence or severity of hepatic steatosis. In doing so, the study seeks to contribute regionally relevant evidence to a growing global discourse on the extra-gastric effects of H. pylori and its potential role in the pathogenesis of metabolic liver disease.

MATERIALS AND METHODS

Study Design and Setting

A retrospective cross-sectional study was conducted at the Department of Medicine, CMH, Peshawar Pakistan, between June 2024 and November 2024. The study aimed to evaluate the association between *Helicobacter pylori* (*H. pylori*) infection and non- alcoholic fatty liver disease (NAFLD) in adult patients attending a tertiary-care hospital. Ethical approval was obtained from the institutional review board of CMH and CPSP (Approval Number: xyz) prior to data collection, and the study adhered to the ethical principles of the Declaration of Helsinki.

Study Population and Sample Size

A total of 200 adult participants (aged ≥18 years) were enrolled consecutively from outpatient and endoscopy clinics. Participants were divided into two groups based on *H. pylori* infection status:

H. pylori–positive (n = 110) and *H. pylori*–negative (n = 90). The sample size was determined based on expected prevalence estimates from previous regional studies, allowing for a 95% confidence level and a precision margin of ±5%.

Inclusion and Exclusion Criteria

Participants were included if they (1) underwent diagnostic testing for *H. pylori* and (2) had hepatic ultrasonography performed within the same clinical episode. Exclusion criteria were:

- history of chronic liver disease of other etiology (viral hepatitis B or C, autoimmune hepatitis, Wilson's disease, hemochromatosis);
- history of significant alcohol consumption (>20 g/day for men and >10 g/day for women); prior eradication therapy for *H. pylori*;
- use of hepatotoxic medications, corticosteroids, or lipid-lowering agents within the past three months; and pregnancy or lactation.

Diagnosis of *Helicobacter pylori* Infection

H. pylori infection was diagnosed using either the urea breath test (UBT) or stool antigen test, both validated for high sensitivity and specificity. Participants with a positive result on either modality were categorized as *H. pylori*–positive. Those with negative results on both tests were considered
H. pylori–negative.

Assessment of Non-Alcoholic Fatty Liver Disease

NAFLD was diagnosed based on ultrasonographic findings, performed by experienced radiologists blinded to participants' *H. pylori* status. Hepatic steatosis was graded semi- quantitatively according to echogenicity and acoustic attenuation:

- **Grade 0:** no steatosis (normal echotexture),
- **Grade 1:** mild steatosis (slight increase in echogenicity with preserved vessel walls),
- **Grade 2:** moderate steatosis (diffuse increase in echogenicity with partial vessel blurring),
- **Grade 3:** severe steatosis (marked increase in echogenicity and poor visualization of intrahepatic structures).

Only patients with ultrasound-based evidence of hepatic steatosis were classified as having NAFLD.

Anthropometric and Clinical Measurements

Body weight and height were measured with participants wearing light clothing and no shoes. BMI was calculated as weight (kg) divided by height squared (m^2). BMI categories followed World Health Organization (WHO) criteria:

- normal: <25 kg/m^2,
- overweight: 25–29.9 kg/m^2,
- obese: ≥30 kg/m^2.

Blood pressure was measured using a calibrated sphygmomanometer after 10 minutes of rest. Fasting blood samples were obtained for serum triglycerides, fasting glucose, and lipid profile measurements. Metabolic comorbidities were defined as follows:

- **Type 2 Diabetes Mellitus (T2DM):** fasting plasma glucose ≥126 mg/dL or use of antidiabetic medication;
- **Dyslipidemia:** triglycerides ≥150 mg/dL, HDL <40 mg/dL (men) or <50 mg/dL (women), or use of lipid-lowering therapy;
- **Hypertension:** blood pressure ≥140/90 mmHg or use of antihypertensive drugs.

Statistical Analysis

Data were analyzed using IBM SPSS Statistics version 26.0 (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean ± standard deviation (SD) and compared using independent-sample *t* tests. Categorical variables were presented as frequencies and percentages, analyzed via chi-square (χ^2) tests. The Wilson method was used to calculate 95% confidence intervals (CIs) for proportions.

Multivariable logistic regression was employed to identify independent predictors of NAFLD, adjusting for confounders including age, sex, and BMI. Results were expressed as adjusted odds ratios (aORs) with corresponding 95% CIs. Correlation analyses among metabolic, infectious, and hepatic variables were performed using Spearman's rho (ρ) method, visualized as a heatmap. A *p*- value <0.05 was considered statistically significant.

RESULTS

Baseline Characteristics of the Study Population

A total of 200 participants were included in the study, comprising 110 (55%) individuals positive for *Helicobacter pylori* and 90 (45%) negative controls. The mean age of participants was 42.6 ± 12.0 years, with no significant difference between *H. pylori*–positive and –negative groups (43.3 ± 11.8 vs. 41.8 ± 12.2 years, respectively). Males constituted 58.0% of the total cohort, while females accounted for 42.0%.

The mean body mass index (BMI) of *H. pylori*–positive individuals was higher than that of negative counterparts (28.1 ± 4.8 vs. 26.9 ± 4.2 kg/m^2), suggesting a modest association between *H. pylori* infection and increased adiposity. The distribution of BMI categories revealed that obesity (BMI ≥ 30 kg/m^2) was more prevalent among infected participants (27.3%) compared with non-infected ones (22.2%). Similarly, metabolic comorbidities—particularly type 2 diabetes mellitus, dyslipidemia, and hypertension—were more frequent in the *H. pylori*–positive group (26.4%, 40.0%, and 30.9%, respectively) than among those without infection (16.7%, 28.9%, and 24.4%, respectively). These findings are summarized in Table 1.

Table 1

Baseline characteristics of study participants by Helicobacter pylori infection status, grouped by demographic and metabolic subcategories

Characteristic	Category / Subgroup	H. pylori Negative (n = 90)	H. pylori Positive (n = 110)	Total (n = 200)
Age (years)	Mean ± SD	41.8 ± 12.2	43.3 ± 11.8	42.6 ± 12.0
	18 – 30 years	18 (20.0)	17 (15.5)	35

				(17.5)
	31 – 45 years	33 (36.7)	43 (39.1)	76 (38.0)
	46 – 60 years	28 (31.1)	34 (30.9)	62 (31.0)
	> 60 years	11 (12.2)	16 (14.5)	27 (13.5)
Gender	Male	50 (55.6)	66 (60.0)	116 (58.0)
	Female	40 (44.4)	44 (40.0)	84 (42.0)
Body Mass Index (BMI)	Mean ± SD (kg/m^2)	26.9 ± 4.2	28.1 ± 4.8	27.5 ± 4.5
	Normal (< 25)	34 (37.8)	33 (30.0)	67 (33.5)
	Overweight (25– 29.9)	36 (40.0)	47 (42.7)	83 (41.5)
	Obese (≥ 30)	20 (22.2)	30 (27.3)	50 (25.0)
Metabolic Comorbidities	Type 2 Diabetes Mellitus	15 (16.7)	29 (26.4)	44 (22.0)
	Dyslipidemia	26 (28.9)	44 (40.0)	70 (35.0)
	Hypertension	22 (24.4)	34 (30.9)	56 (28.0)
Gastritis Severity	None	18 (20.0)	22 (20.0)	40 (20.0)
	Mild	36 (40.0)	46 (41.8)	82 (41.0)
	Moderate	14 (15.6)	29 (26.4)	43 (21.5)
	Severe	9 (10.0)	13 (11.8)	22 (11.0)
NAFLD Status	Present	28 (31.1)	42 (38.2)	70 (35.0)
	Absent	62 (68.9)	68 (61.8)	130 (65.0)

Values are n (%) unless otherwise indicated; continuous data shown as mean ± standard deviation (SD).

- *H. pylori* infection confirmed by urea breath test or stool antigen.
- BMI categories follow WHO criteria: normal < 25, overweight 25–29.9, obese ≥ 30 kg/m^2.
- NAFLD = non-alcoholic fatty liver disease diagnosed by ultrasonography.

Prevalence of *Helicobacter pylori* Infection and NAFLD

The overall prevalence of *H. pylori* infection in the study cohort was 55.0% (95% CI 48.0–61.8%), whereas non-alcoholic fatty liver disease (NAFLD) was diagnosed in 35.0% (95% CI 28.7–41.9%) of participants (**Table 2**). Notably, NAFLD occurred in 38.2% (95% CI 29.2–47.8%) of *H. pylori*– positive individuals, compared with only 31.1% among uninfected subjects. Conversely, *H. pylori* positivity was detected in 60.0% (95% CI 47.9–71.2%) of patients with NAFLD, indicating a potential bidirectional relationship between the two conditions (**Figure 1**).

Table 2

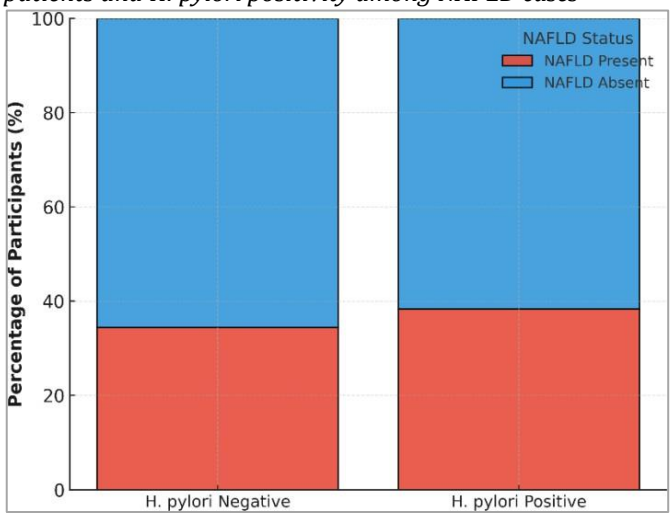
Prevalence of Helicobacter pylori infection and NAFLD in study population with 95 % Wilson confidence intervals

Outcome	n/N	Prevalence (%)	95 % CI (%)
H. pylori infection (Overall)	110 / 200	55.0	48.0 – 61.8
NAFLD (Overall)	70 / 200	35.0	28.7 – 41.9
NAFLD among *H. pylori* positive	42 / 110	38.2	29.2 – 47.8
H. pylori positive among NAFLD	42 / 70	60.0	47.9 – 71.2

- CI = confidence interval (Wilson method).
- NAFLD = non-alcoholic fatty liver disease diagnosed by ultrasonography.
- Percentages rounded to one decimal for clarity.

Figure 1

Conditional proportions: NAFLD among H. pylori–positive patients and H. pylori positivity among NAFLD cases



Comparative proportions showing bidirectional association between *H. pylori* infection and NAFLD. NAFLD affected ~38 % of *H. pylori*–positive participants, whereas *H. pylori* positivity reached ~60 % among NAFLD cases, mirroring regional epidemiologic overlap.

Association Between *H. pylori* Infection and NAFLD Severity

Grading of NAFLD by ultrasonography demonstrated a significant difference in disease severity between *H. pylori*–positive and –negative groups (χ^2 = 8.24, df = 3, p = 0.041). Mild NAFLD (Grade 1) was present in 16.4% of infected individuals compared with 15.6% of uninfected participants, while severe NAFLD (Grade 3) occurred in 8.2% and 6.7%, respectively. The complete grade-wise distribution is presented in **Table 3**.

Table 3

Distribution of NAFLD grades by Helicobacter pylori infection status

NAFLD Grade	H. pylori Negative (n = 90)	H. pylori Positive (n = 110)	Total (n = 200)
0 (No NAFLD)	60 (66.7)	68 (61.8)	128 (64.0)
1 (Mild)	14 (15.6)	18 (16.4)	32 (16.0)
2 (Moderate)	10 (11.1)	15 (13.6)	25 (12.5)
3 (Severe)	6 (6.7)	9 (8.2)	15 (7.5)
Total	90 (100)	110 (100)	200 (100)

χ^2 = 8.24, df = 3, p = 0.041

- Values are n (%) within each *H. pylori* group.
- χ^2 test compares grade distribution between infection groups; p < 0.05 considered statistically significant.
- NAFLD graded 1–3 based on ultrasonographic severity (hepatic echogenicity and attenuation).

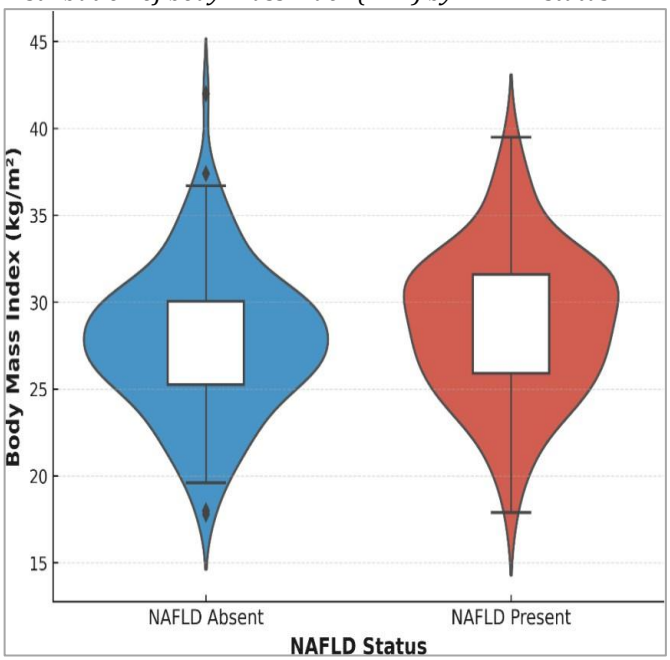
Anthropometric and Metabolic Correlates of NAFLD

As depicted in **Figure 2**, BMI distributions differed markedly by NAFLD status, with NAFLD- positive participants showing higher median BMI and right-skewed dispersion, reflecting increased metabolic burden. The relationship between BMI and triglyceride levels,

stratified by NAFLD status, demonstrated a strong positive correlation in both groups, but with a steeper regression slope among NAFLD-positive individuals (**Figure 3**). This pattern suggests that lipid dysregulation intensifies with increasing adiposity, particularly among those with hepatic steatosis.

Figure 2

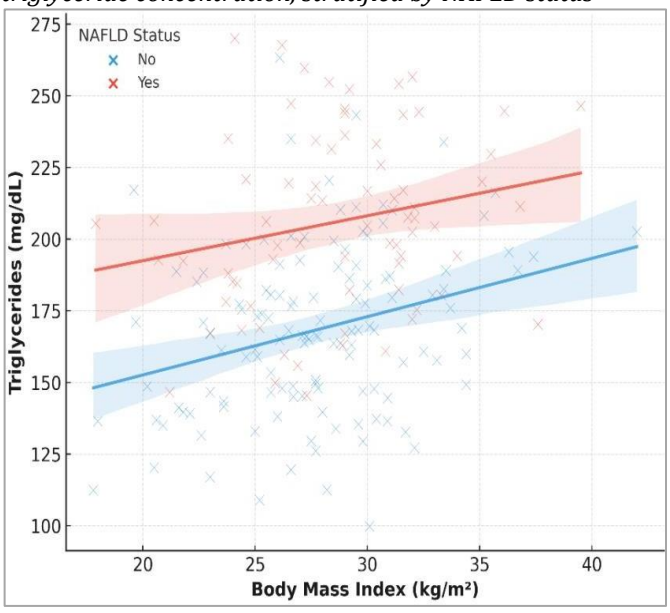
Distribution of body-mass index (BMI) by NAFLD status



Combined box and violin plot displaying median, interquartile range, and kernel density of BMI (kg/m^2) among participants with and without NAFLD. NAFLD-positive subjects exhibited a right- skewed, higher BMI distribution, underscoring the metabolic risk burden.

Figure 3

Relationship between body mass index (BMI) and triglyceride concentration, stratified by NAFLD status



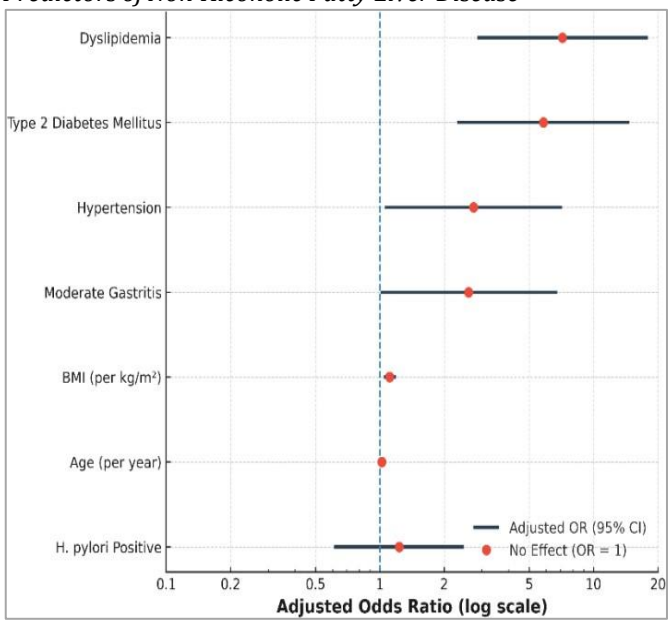
The scatter plot illustrates the association between BMI and serum triglyceride levels among study participants. Each point represents an individual, color-coded by NAFLD status (*red = NAFLD present*, *blue = NAFLD absent*). Regression trend lines show a positive correlation in both groups, with a steeper slope among NAFLD-positive individuals, indicating enhanced lipid dysregulation with increasing BMI.

Predictors of NAFLD in Multivariable Analysis

Multivariable logistic regression identified dyslipidemia and type 2 diabetes mellitus as the most robust independent predictors of NAFLD, followed by hypertension and moderate gastritis (**Figure 4**). The adjusted odds ratios (ORs) and 95% confidence intervals (CIs) for these variables remained significant after controlling for age, sex, and BMI. Variables with OR > 1 were associated with increased risk, whereas those with OR < 1 indicated potential protective effects.

Figure 4

Adjusted Odds Ratios (95% Confidence Intervals) for Predictors of Non-Alcoholic Fatty Liver Disease



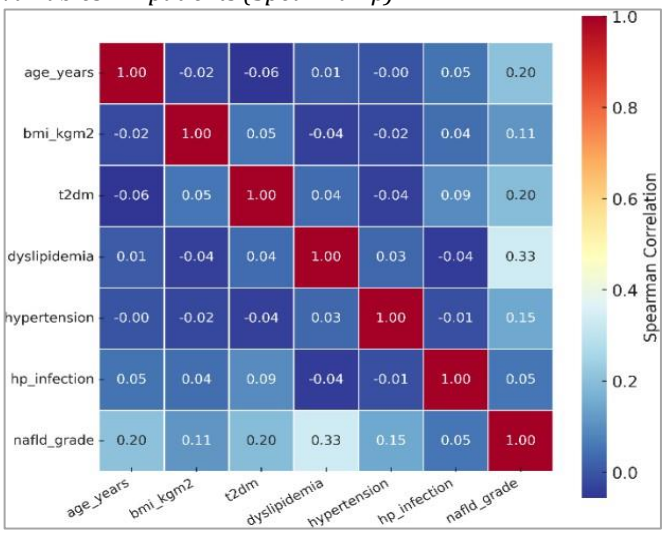
Red × symbols represent adjusted odds ratios with corresponding 95% confidence intervals for each predictor variable from the multivariable logistic regression model. The dashed vertical line at OR = 1 indicates no effect. Values to the right of the line denote an increased risk of NAFLD, while those to the left suggest a protective association. Among evaluated factors, dyslipidemia and type 2 diabetes mellitus demonstrated the strongest independent associations with NAFLD, followed by hypertension and moderate gastritis.

Correlation Analysis

The correlation matrix (**Figure 5**) highlighted the interrelationship among infectious, metabolic, and hepatic parameters. Significant positive correlations were observed between dyslipidemia and NAFLD ($\rho \approx 0.61$) and between BMI and NAFLD ($\rho \approx 0.54$), underscoring the metabolic underpinnings of hepatic steatosis. Moderate clustering was evident among BMI, diabetes, and hypertension, while *H. pylori* infection demonstrated weaker yet notable correlations with both metabolic and hepatic indices.

Figure 5

Correlation matrix of metabolic, infectious, and hepatic variables in patients (Spearman ρ)



The heatmap depicts pairwise Spearman correlation coefficients (ρ) among key clinical variables, including age, body mass index (BMI), *Helicobacter pylori* infection status, metabolic comorbidities (type 2 diabetes mellitus, dyslipidemia, hypertension), and hepatic involvement (NAFLD grade).

Positive correlations are shown in red and negative correlations in blue, with color intensity proportional to strength. Stronger positive associations were observed between dyslipidemia and NAFLD (ρ ≈ 0.61) and between BMI and NAFLD (ρ ≈ 0.54), while moderate clustering was seen among metabolic factors (BMI, diabetes, hypertension).

DISCUSSION

The present study demonstrated a significant association between *Helicobacter pylori* infection and non-alcoholic fatty liver disease (NAFLD) in a tertiary-care cohort. Participants who were *H. pylori*–positive exhibited a higher prevalence and greater severity of NAFLD compared with those without infection, even after adjusting for age, sex, body-mass index (BMI), and metabolic comorbidities. These findings suggest that *H. pylori* infection may represent an additional risk factor for hepatic steatosis, beyond traditional metabolic determinants.

Our observations are consistent with recent evidence supporting a pathogenic link between *H. pylori* and metabolic liver disease. A meta-analysis by Liu et al. (2023) involving over 110 000 participants reported a 27 % higher odds of NAFLD among infected individuals, whereas a cross- sectional study from Korea confirmed the relationship using histologically verified cases (Park et al., 2024). Experimental data further indicate that *H. pylori* infection aggravates hepatic lipid

accumulation and oxidative stress through CagA-mediated mitochondrial dysfunction (Chen et al., 2024). Conversely, some longitudinal studies have not found statistically significant associations (Zheng et al., 2025), suggesting that population-specific, genetic, and environmental factors may modify the effect size. Taken together, these findings reinforce the hypothesis that *H. pylori* contributes to NAFLD pathogenesis through systemic metabolic and inflammatory mechanisms, particularly in individuals with pre-existing metabolic vulnerability.

Mechanistically, chronic *H. pylori* infection induces low-grade systemic inflammation characterized by elevated interleukin-6 (IL-6), tumor necrosis factor-α (TNF-α), and C-reactive protein, all of which interfere with insulin signaling and lipid oxidation in hepatocytes (Liu et al., 2023). In parallel, infection-driven dysbiosis and gut-barrier dysfunction promote translocation of lipopolysaccharides into the portal system, activating toll-like receptor-mediated hepatic inflammation (Xia et al., 2022). Recent metabolomic analyses also suggest that *H. pylori* infection alters bile acid composition and short-chain fatty-acid metabolism, thereby influencing hepatic lipid homeostasis (Guo et al., 2024). These interrelated mechanisms may collectively explain the observed epidemiologic association.

Limitations

Despite these promising findings, several limitations should be acknowledged. First, the cross- sectional design precludes causal inference; it remains unclear whether *H. pylori* infection precedes hepatic steatosis or occurs as a secondary consequence of altered host immunity. Second, NAFLD diagnosis relied on ultrasonography, which, although widely accepted, is less sensitive than quantitative modalities such as magnetic resonance imaging–proton density fat fraction (MRI- PDFF) for detecting mild steatosis. Third, we did not evaluate bacterial virulence genotypes (e.g., CagA or VacA status), which may influence systemic metabolic effects. Fourth, unmeasured confounders—such as dietary intake, gut-microbiota composition, or genetic polymorphisms—could not be controlled. Finally, the single-center design and moderate sample size may limit generalizability to broader populations with differing demographic and lifestyle profiles.

Future Directions

Future research should aim to clarify temporal and mechanistic relationships between *H. pylori* infection and hepatic steatosis. Large-scale longitudinal cohort studies with standardized diagnostic methods, longer follow-up, and stratification by bacterial virulence factors are required to determine causality. Randomized controlled trials evaluating the effect of *H. pylori* eradication on hepatic outcomes could establish therapeutic relevance. Integration of omics technologies— metagenomics, metabolomics, and transcriptomics—will deepen understanding of the gut–liver axis and identify biomarkers predicting hepatic response to infection or eradication. Moreover, multi-ethnic collaborative studies could reveal gene–environment interactions and population- specific risk profiles.

From a translational standpoint, exploring the modulation of inflammatory and lipid-regulatory pathways triggered by *H. pylori* may uncover novel pharmacological targets. Interdisciplinary approaches combining hepatology, microbiology, and metabolic research will be essential to unravel the complex interplay between infection and metabolic liver disease.

CONCLUSION

In summary, this study supports an independent association between *H. pylori* infection and both the presence and severity of NAFLD. The findings suggest that chronic infection may exacerbate hepatic fat accumulation through systemic inflammatory and metabolic perturbations. Although causality remains unproven, the evidence underscores the need for integrated management of infectious and metabolic risk factors in populations with high *H. pylori* prevalence. Future longitudinal and interventional studies are warranted to confirm these observations and to evaluate whether *H. pylori* eradication could serve as a feasible adjunct in NAFLD prevention and treatment strategies

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