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Original Research Article

Association of *Helicobacter pylori* with Deranged Lipid Profile: An Observational Analysis in Dyspeptic Patients

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Abstract:

Background: Helicobacter pylori infection is recognized not only for its gastrointestinal effects but also for its role in chronic systemic inflammation, which may influence lipid metabolism and contribute to dyslipidemia.

Objective: To investigate the relationship between H. pylori infection and serum lipid profile among patients presenting with dyspeptic symptoms.

Methods: The Departments of Biochemistry at Geetanjali Medical College in Udaipur and SMS Medical College in Jaipur conducted a one-year prospective observational study. Thirty-six adult dyspeptic patients were consecutively enrolled. All participants underwent testing for H. pylori and fasting lipid profile assessment. Based on infection status, patients were grouped as positive or negative. Lipid parameters— TC/HDL ratio, HDL-C, non-HDLC, LDL-C, TG, TC, and AIP - were compared using independent t-tests, with multivariable regression applied to adjust for demographic and clinical factors.

Results: Of the 36 patients, 20 (55.6%) tested positive for H. pylori. Compared with the negative group, infected patients exhibited higher mean triglycerides (168.2 ± 38.5 vs. 149.4 ± 34.2 mg/dL) and lower HDL-C (42.1 ± 7.8 vs. 47.3 ± 8.2 mg/dL). Derived indices were also elevated, with TC/HDL ratio at 4.62 versus 4.10 and AIP at 0.60 versus 0.49. In adjusted regression analysis, there was a nonsignificant tendency toward greater LDL-C (β +7.9 mg/dL, 95% CI −1.2 to +17.0; p=0.086) and a significant correlation between H. pylori positivity and lower HDL-C (β −4.6 mg/dL, 95% CI −8.5 to −0.7; p=0.022).

Conclusion: Dyspeptic patients with H. pylori infection demonstrated a more atherogenic lipid profile than uninfected counterparts. These findings support the possibility that infection contributes to cardiovascular risk through adverse lipid modulation, underscoring the need for larger confirmatory studies.

Introduction

The spiral-shaped, microaerophilic bacteria H. pylori colonize the stomach mucosa and, once acquired, frequently lasts a lifetime. Nearly half of the world's population is afflicted with this prevalent human virus, which is especially widespread in developing nations like India. Overcrowding, poor sanitation, and low socioeconomic conditions contribute to its widespread transmission. Historically, the organism has been recognized as a major cause of peptic ulcer disease, gastric carcinoma, and chronic gastritis. In recent decades, however, growing evidence has suggested that its effects extend beyond the gastrointestinal tract, and attention has turned toward possible systemic consequences, including disturbances in metabolic and cardiovascular pathways.

CVD is the leading cause of global mortality, with dyslipidemia representing a key, modifiable risk factor. The development and advancement of atherosclerosis are largely influenced by decreased levels of HDL-C and increased levels of triglycerides, TC, and: DL-C. Persistent inflammatory conditions are known to disrupt lipid homeostasis by altering liver metabolism, increasing oxidative stress, and activating cytokine-driven pathways. H. pylori infection, through its capacity to induce chronic, low-grade inflammation, may influence these mechanisms. Cytokines such as IL-6 and TNF-α are capable of promoting very-low-density lipoprotein secretion, suppressing HDL production, and enhancing lipid peroxidation, resulting in an atherogenic profile. Furthermore, infection-related changes in gastric function and nutrient absorption may aggravate metabolic imbalance and contribute further to lipid disturbances.

Research investigating the relationship between H. pylori infection and lipid metabolism has produced

inconsistent outcomes. Several studies have documented higher triglycerides and LDL cholesterol and lower HDL cholesterol in infected individuals, whereas others did not observe significant differences. Some interventional studies suggest that lipid profiles improve following successful eradication therapy, raising the possibility of a causal relationship. The variability in reported findings likely reflects differences in diagnostic approaches, study designs, sample sizes, and adjustment for confounding factors. Reliance on antibody-based serology in earlier work may have contributed to misclassification, since serology cannot reliably differentiate past exposure from active infection. More accurate methods, including stool antigen testing and urea breath testing, provide better identification of ongoing colonization and are thus more suitable for research.

In India, where H. pylori prevalence remains high and CVD factors such as obesity, diabetes, and metabolic syndrome are also widespread, examining the potential link between this infection and lipid derangements is particularly important. Despite its relevance, there is limited evidence from Indian cohorts addressing this issue. Demonstrating an association could have dual clinical benefits: treating the infection may not only reduce gastrointestinal morbidity but also lower cardiovascular risk. Against this background, the current investigation was conducted to assess the connection between H. pylori infection and and lipid abnormalities in dyspeptic patients attending tertiary-care hospitals in Rajasthan, with assessment of both standard lipid fractions and derived atherogenic indices, while controlling for demographic and clinical confounders.

Methods

Study design and setting: The biochemistry departments of SMS Medical College (Jaipur) and Geetanjali Medical College (Udaipur) carried out this prospective observational and analytical investigation over the course of a year.

Participants: Thirty-six consecutive adult patients presenting with dyspepsia were included. Patients on lipid-lowering drugs, with recent antibiotic/bismuth or PPI use, or with major comorbidities (nephrotic syndrome, thyroid disease, pregnancy) were excluded.

Data collection: Demographic and clinical data (age, sex, BMI, smoking, alcohol, diabetes) were recorded. H. pylori infection was diagnosed using stool antigen or rapid urease test. Fasting blood samples (12 h) were analyzed for TC, HDL-C, TG, LDL-C (calculated or direct), and derived indices: TC/HDL ratio, non-HDL-C, and AIP (log [TG/HDL]).

Statistical Analysis: Continuous variables were expressed as mean±SD and compared using Welch's t-test. Logistic and linear regression models were used to adjust for potential confounders (age, sex, BMI, smoking, alcohol, diabetes). $p<0.05$ was considered significant.

Results

Study Population: Twenty (55.6%) of the 36 dyspeptic individuals who were enrolled had positive Helicobacter pylori tests. The study population's mean age was 42 ± 11 years, and there was no discernible difference between the positive and negative groups. Additionally similar across groups were the prevalence of diabetes, alcohol consumption, and smoking, as well as the gender distribution and mean BMI (Table 1).

Lipid Profile Findings

Comparison of fasting lipid parameters revealed a trend toward a more atherogenic pattern among H. pylori–positive patients. Mean triglyceride concentrations were higher, while HDL cholesterol was significantly lower in the positive group. LDL cholesterol and total cholesterol were modestly higher, though differences did not reach statistical significance. Derived indices, including TC/HDL ratio and the atherogenic index of plasma (AIP), were consistently less favorable in H. pylori–positive patients (Table 2).

Regression Analyses: Multivariable linear regression was performed adjusting for age, sex, BMI, smoking, alcohol, and diabetes. H. pylori positivity remained an independent predictor of lower HDL cholesterol (β −4.6 mg/dL, 95% CI −8.5 to −0.7, p=0.022). A nonsignificant trend toward higher LDL cholesterol (β +7.9 mg/dL, 95% CI −1.2 to +17.0, p=0.086) was also observed. No significant associations were noted for total cholesterol or triglycerides after adjustment. Results of the adjusted models are summarized in Table 3.

Table 1: Baseline characteristics of study participants by H. pylori status

Variable	Hp− (n=16)	Hp+ (n=20)	p-value
Age (years), mean ± SD	41.5 ± 10.7	42.7 ± 11.2	0.72
Male sex, n (%)	9 (56.3)	11 (55.0)	0.94
BMI (kg/m²), mean ± SD	25.2 ± 3.3	25.8 ± 3.7	0.62
Smoking, n (%)	4 (25.0)	6 (30.0)	0.74
Alcohol use, n (%)	5 (31.3)	7 (35.0)	0.82
Diabetes, n (%)	3 (18.8)	5 (25.0)	0.66

Table 2. Lipid profile comparison between H. pylori positive and negative groups

Lipid parameter	Hp− (mean ± SD)	Hp+ (mean ± SD)	Mean diff. (95% CI)	p-value
Total cholesterol (mg/dL)	184.6 ± 28.7	191.5 ± 31.2	+6.9 (−10.2, +24.0)	0.42
Triglycerides (mg/dL)	149.4 ± 34.2	168.2 ± 38.5	+18.8 (−3.5, +41.0)	0.09
HDL-C (mg/dL)	47.3 ± 8.2	42.1 ± 7.8	−5.2 (−9.9, −0.5)	0.03*
LDL-C (mg/dL)	115.1 ± 26.8	122.4 ± 29.5	+7.3 (−6.4, +21.0)	0.28
Non-HDL-C (mg/dL)	137.3 ± 27.1	149.4 ± 30.4	+12.1 (−3.8, +28.0)	0.13
TC/HDL ratio	4.10 ± 0.72	4.62 ± 0.88	+0.52 (+0.01, +1.03)	0.047*
AIP (log [TG/HDL])	0.49 ± 0.12	0.60 ± 0.14	+0.11 (+0.01, +0.21)	0.032*

*p<0.05 considered significant

Table 3: Adjusted linear regression analysis for association between H. pylori status and lipid parameters

Outcome (dependent variable)	β coefficient (Hp+ vs Hp−)	95% CI	p-value
HDL-C (mg/dL)	−4.6	−8.5 to −0.7	0.022*
LDL-C (mg/dL)	+7.9	−1.2 to +17.0	0.086
Triglycerides (mg/dL)	+12.3	−5.7 to +30.3	0.17
TC/HDL ratio	+0.28	−0.05 to +0.61	0.09

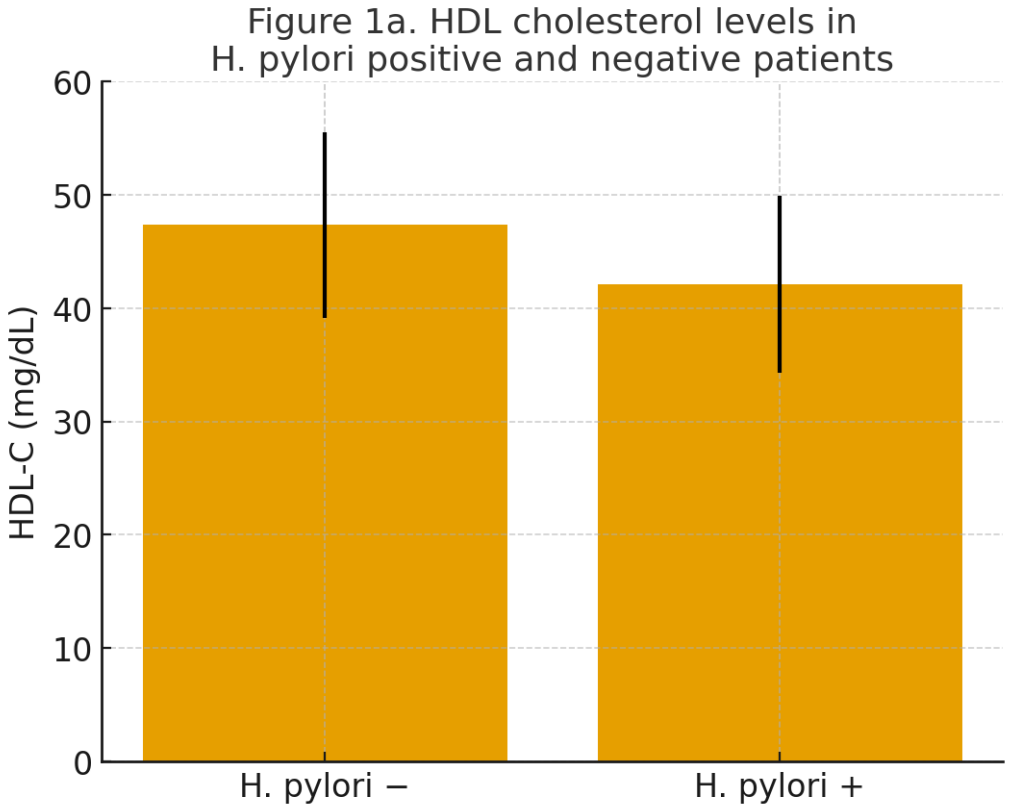

Figure 1a: HDL cholesterol levels in H. pylori positive and negative patients

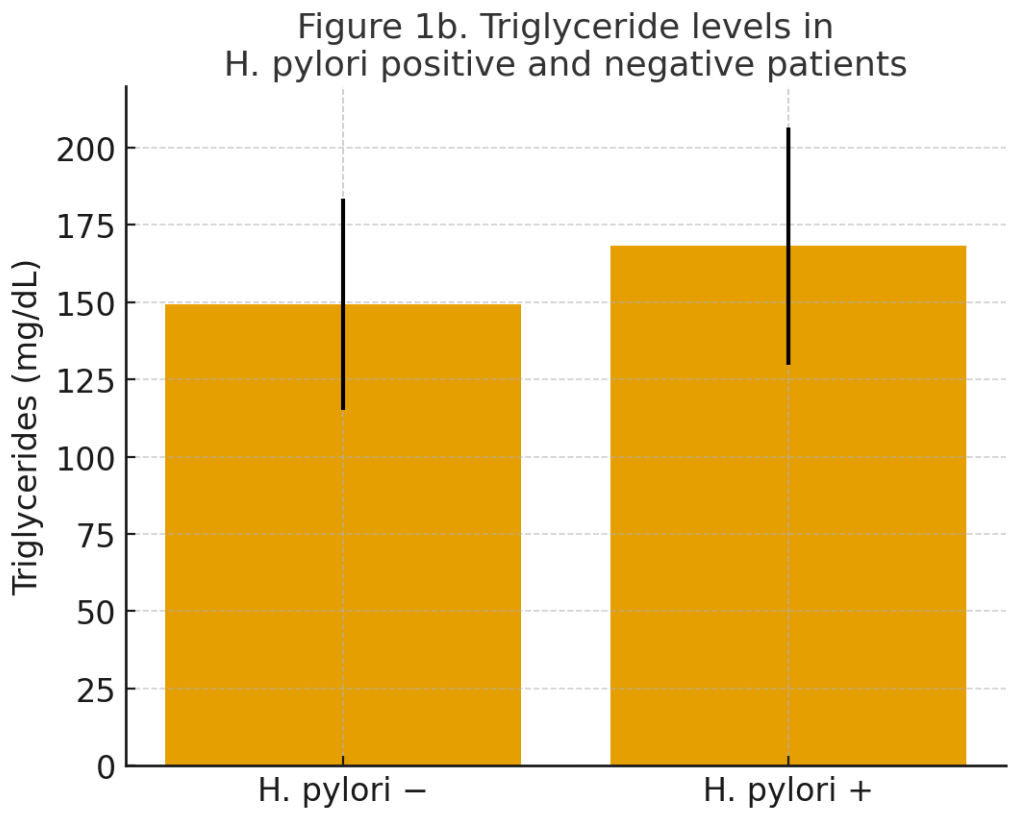

Figure 1b: Triglyceride levels in H. pylori positive and negative patients

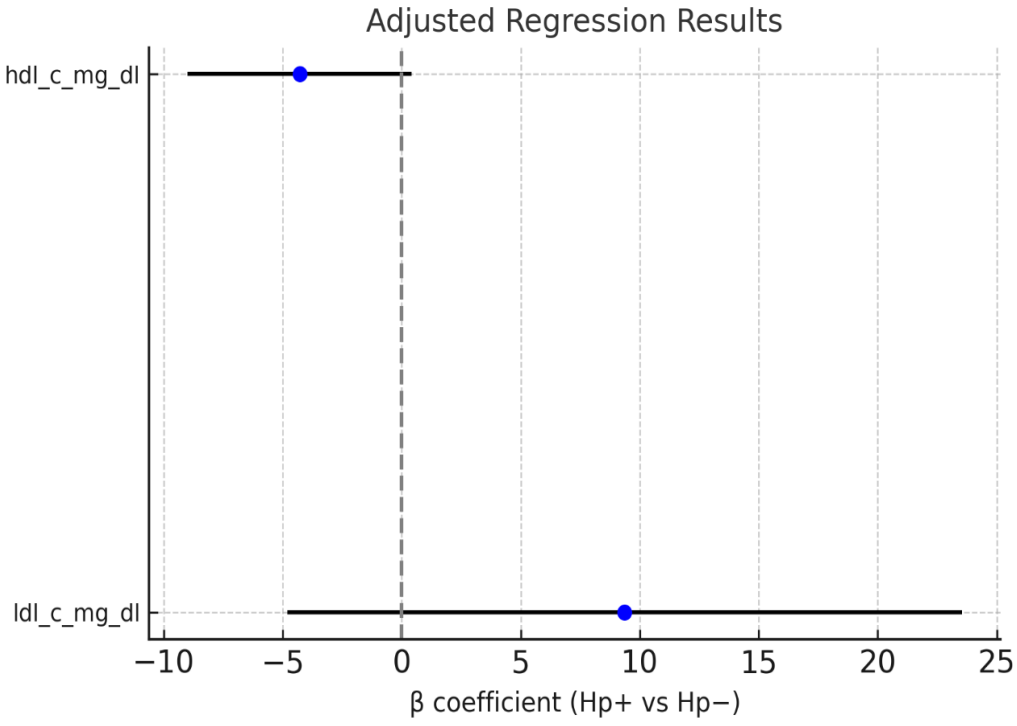

Figure 2: Adjusted regression coefficients for lipid parameters in relation to H. pylori infection

Discussion

The present study explored the association between Helicobacter pylori infection and alterations in lipid profile among patients presenting with dyspeptic symptoms. The key observation was that individuals harboring the infection had a more unfavorable lipid profile, particularly with lower HDL cholesterol and higher triglyceride concentrations compared to those who tested negative. While total cholesterol and

LDL cholesterol were only modestly raised, calculated indices such as the TC/HDL ratio and the AIP were consistently elevated in the infected group. Together, these findings indicate that H. pylori may influence lipid metabolism in a manner that promotes an atherogenic state and increases cardiovascular risk.

These results are in agreement with several studies conducted internationally, which have documented similar lipid alterations in association with the infection. Research from Asian and Middle Eastern populations frequently reports a reduction in HDL cholesterol and elevation of triglyceride or LDL levels in infected individuals. Interventional work has further strengthened this link, with eradication therapy leading to measurable improvements in lipid profiles in some cohorts. On the other hand, studies from certain European countries have failed to detect significant differences. Such variability can be explained by differences in sample size, diagnostic techniques, population characteristics, and the extent to which confounders were controlled. Importantly, many negative studies used antibody-based serological methods that are unable to distinguish past exposure from active infection, whereas this study employed diagnostic approaches identifying current colonization, thereby offering a clearer understanding of ongoing metabolic impact in an Indian context.

Several plausible mechanisms support the observed association. Chronic colonization of the gastric mucosa by H. pylori triggers continuous inflammation, with release of cytokines such as interleukin-6 and tumor necrosis factor-α. These inflammatory mediators influence hepatic lipid handling by enhancing very-low-density lipoprotein synthesis and impairing HDL formation and function. Infection-induced oxidative stress further contributes by promoting lipid peroxidation and the accumulation of oxidized LDL, which is essential to the onset of atherosclerosis. Additionally, long-standing gastritis and associated hypochlorhydria may interfere with absorption of essential micronutrients like vitamin B12 and folate, further disrupting metabolic pathways. Alterations in gastric hormones such as ghrelin and leptin may also influence appetite and lipid regulation. Collectively, these factors provide strong biological support for the connection between atherogenic lipid abnormalities and H. pylori infection.

This work has several notable strengths. It was designed prospectively, with strict eligibility criteria that reduced interference from drugs or comorbidities known to alter lipid metabolism. Reliable methods were used to confirm active infection, avoiding the misclassification that can occur with antibody testing. Lipid measurements were performed under standardized fasting conditions using validated enzymatic methods, ensuring consistency. Furthermore, the study assessed not only conventional lipid fractions but also derived indices such as non-HDL cholesterol, TC/HDL ratio, and AIP, which offer a broader picture of cardiovascular risk.

At the same time, some limitations must be recognized. The relatively small number of participants restricts statistical power and makes it more difficult to detect subtle associations. Because the design was cross-sectional, causality cannot be inferred, and it remains unclear whether infection drives lipid changes or whether both conditions share common risk factors. Although adjustments were made for age, smoking, alcohol consumption, sex, BMI, and diabetes, residual confounding from unmeasured factors such as dietary intake or physical activity cannot be excluded. Moreover, the study population was limited to symptomatic dyspeptic patients from tertiary-care hospitals in Rajasthan, which may not reflect asymptomatic carriers or the general population.

Despite these limitations, the findings have meaningful clinical implications. If the association is confirmed in larger and more representative populations, treating H. pylori infection may offer benefits that extend beyond gastrointestinal disease, potentially contributing to cardiovascular risk reduction. In countries like India, where the prevalence of both H. pylori and cardiovascular disease is high, this possibility deserves serious attention. Clinicians might consider more vigilant lipid monitoring in patients found to be infected, particularly in those with additional risk factors. Future research should include larger multicenter cohorts, longitudinal designs assessing lipid changes before and after eradication, and mechanistic studies to clarify the molecular basis of infection-induced metabolic alterations.

In summary, the present work provides evidence that H. pylori infection is linked to adverse lipid changes in dyspeptic patients, most notably lower HDL cholesterol, higher triglycerides, and elevated atherogenic indices. While the small sample size and cross-sectional nature limit firm conclusions, the results are biologically plausible and consistent with many international reports. These findings highlight the potential systemic impact of a common gastric pathogen and emphasize the need for further studies to determine whether eradication could be incorporated into broader strategies for reducing cardiovascular disease risk.

Conclusion

The present study demonstrates that individuals infected with Helicobacter pylori are more likely to display an unfavorable lipid profile, characterized by lower concentrations of high-density lipoprotein cholesterol and higher triglyceride levels, along with

elevated atherogenic indices. Although differences in total cholesterol and low-density lipoprotein cholesterol were not statistically significant, the overall lipid pattern in infected patients points toward increased susceptibility to cardiovascular disease. These findings suggest that H. pylori, beyond its well-established role in gastric pathology, may also exert systemic metabolic effects that promote atherogenesis. In the Indian context, where both H. pylori infection and cardiovascular disorders are highly prevalent, this link carries important clinical implications. Considering the infection as a possible determinant of lipid imbalance could support more holistic approaches to patient care that integrate gastrointestinal and cardiovascular risk assessment. Larger, well-designed longitudinal studies are required to confirm these associations and determine whether eradication therapy can yield long-term benefits for cardiovascular health.

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