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ORIGINAL ARTICLE

Association of H. pylori gastritis with Non-alcoholic Hepatic Steatosisin Patients - A Cross-Sectional Study in Tertiary Care Hospitals

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ABSTRACT

Background: Helicobacter pylori is a common gastric pathogen that is more and more associated with systemic metabolic disorders. Obesity and insulin resistance are known as conditions that are associated with non-alcoholic hepatic steatosis, or liver fat. T

Aim: The recent studies suggest the presence of possible correlation of H. pylori gastritis with hepatic fat accumulation.

Methodology: Between September to December 2024, this study was conducted in two tertiary care hospitals in Lahore, Pakistan. Upper gastrointestinal endoscopy and abdominal ultrasonography were performed in a total of 100 adult patients with dyspeptic symptoms. A diagnosis of H. pylori infection was confirmed by rapid urease test and histopathology of gastric biopsies and hepatic steatosis was diagnosed based on ultrasounds which were suggestive of a fatty liver. Those who had previously used alcohol, who had viral hepatitis or who had known liver diseases were excluded.

Results: Sixty two percent of enrolled patients (n=62) tested positive for H. pylori infection and 48 percent (n=48) had ultra-sonographic evidence of nonalcoholic hepatic steatosis. Of the 35 patients with H. pylori gastritis 56.4% showed concurrent hepatic steatosis as compared to 13 (30.9%) in H. pylori negative patients. This was a statistically significant association (p = 0.012). Patients with both conditions had elevated BMI and fasting blood glucose.

Conclusion: The association between H. pylori gastritis and nonalcoholic hepatic steatosis in dyspeptic patients was found in this study. These findings indicate a possibility of a chronic H. pylori infection having an extra-gastric metabolic influence. Longitudinal studies to explore the causal relationship and the underlying pathophysiological mechanisms are recommended further.

Keywords: Helicobacter pylori, NAFLD, Dyspepsia, Liver Steatosis, Gastroenterology, ultrasonography

INTRODUCTION

Helicobacter pylori (H. pylori) is a gram negative, spiral shaped bacterium which is a chronic infection of the gastric mucosa of over half of the global population, especially in developing countries[2]. It is primarily associated with gastritis, peptic ulcer disease and gastric carcinoma. But recently evidence is emerging that H. pylori also has a role in extra gastric disease like metabolic disorders[3].

Non-alcoholic hepatic steatosis (NAHS) is commonly referred to as non-alcoholic fatty liver disease (NAFLD) and is the most common cause of chronic liver disease worldwide[4]. Fat accumulation in hepatocytes without

considerable alcohol intake is a characteristic of this condition; it is closely associated with obesity, dyslipidemia, insulin resistance, and type 2 diabetes. In early stages, NAHS is usually asymptomatic; however, it can evolve to steatohepatitis, fibrosis, and cirrhosis[6].

Chronic H. pylori infection has been proposed to be involved with NAHS through systemic inflammation, oxidative stress and insulin resistance in several studies[7]. In chronic infection, production of inflammatory cytokines may cause lipid metabolism disturbance and hepatic fat deposition. The association with this potential is the subject of interest worldwide, however, data from Pakistan is scant. There are paucity of data to evaluate this association in Pakistan[20], particularly in dyspeptic patients who constitute a huge portion of gastroenterology outpatient visits and are routinely screened for H. pylori[7]. This potential link is important, as it may provide the opportunity for early identification of potential liver steatosis cases for early lifestyle or pharmacologic interventions[8].

Different Studies estimate prevalence of H. pylori between 60 and 70% in adult population in Pakistan, which may be identified during upper gastrointestinal endoscopies performed for dyspepsia[9].

The aim of this study is to evaluate the association of H. pylori gastritis and non-alcoholic hepatic steatosis in dyspeptic patients visiting tertiary care hospitals in Lahore. Knowing this relationship could shed light on the global metabolic burden of H. pylori infection in the local population[10].

MATERIALS AND METHODS

Study Design and Duration

Present Cross-sectional observational study was conducted from September to December 2024 in tertiary care hospital.

Setting

Endoscopy and Radiology Units, Mayo Hospital and Services Hospital, Lahore.

Sample Size

100 adult patients with dyspepsia.

Inclusion Criteria:

- Age 18–65 years
- Patients undergoing endoscopy for dyspepsia
- No prior H. pylori eradication therapy

Exclusion Criteria

- Alcohol use
- Chronic liver disease (HBV, HCV, autoimmune hepatitis)
- Use of hepatotoxic medications

Procedure

Patients were undergone through the procedure of endoscopy. Rapid urease test and gastric biopsy tested for H. pylori status. Using abdominal ultrasound, NAFLD was diagnosed. Fasting glucose, lipid profile, AST, ALT and insulin levels were measured from blood samples.

Statistical Analysis

The raw data of present study was processed for presentation with the help of SPSS version 0.26 and Mean slandered deviation was applied in which ($P<0.05$) value was considered for significant and on-significant levels.

RESULTS

This cross sectional study was carried out at two tertiary care hospitals of Lahore on a total of 100 dyspeptic patients. Participants were 42.6±11.8 years of age with a male-to-female ratio of 1.1:1. The H. pylori infection status was confirmed by endoscopic biopsy and rapid urease testing in 62% (n = 62) of patients and negative in 38(38%).

Forty eight percent (n = 48) of the total population had ultra-sonographic evidence of non-alcoholic hepatic steatosis (NAHS). Among these H. pylori positive patients, 35 (56.4 %) had concomitant NAHS as compared to 13 (34.2 %) in the H. pylori negative group. Statistically significantly ($p = 0.012$), there was a strong association between H. pylori gastritis and hepatic fat accumulation.

Both H. pylori infection and NAHS were also associated with significantly higher levels of BMI and fasting blood glucose. Mean BMI in this subgroup was 28.6 ± 2.4 kg/m² versus 25.3 ± 2.1 kg/m² in those without NAHS ($p < 0.001$). Mean fasting blood glucose levels were also increased (112.4 ± 15.2 mg/dL) in co-affected compared with isolated gastritis or steatosis ($p = 0.019$) (Table 1).

Distribution of non- alcoholic hepatic steatosis (NAHS) among patients in relation to their H. pylori infection status is shown in Table 2. Of 62 patients with H. pylori infection, 56.4% (35) had NAHS and 34.2% (13) of 38 H. pylori negative patients had hepatic steatosis. On the contrary, 27 patients (43.5%) in the H. pylori positive group and 25 patients (65.8%) in the negative group did not have NAHS. Statistically significant difference ($p = 0.012$) between the two groups indicates that H. pylori infection is highly associated with the presence of hepatic fat accumulation in dyspeptic patients (Table 2).

These findings were correlated with the hypothesis that Hepatic steatosis may be linked to an increased risk of hepatic steatosis development in patients having upper

gastrointestinal symptoms and infection with H. pylori. The association between BMI, glucose and coexisting infections suggests a metabolic interplay which needs further longitudinal assessment.

Table 1: Demographic and Clinical Characteristics of the Study Population (n = 100)

Parameter	H. pylori Positive (n=62)	H. pylori Negative (n=38)	p-value
Age (years, mean ± SD)	42.1 ± 12.3	43.2 ± 11.1	0.638
Male (%)	54.8%	52.6%	0.821
BMI (kg/m², mean ± SD)	27.9 ± 2.6	25.1 ± 2.4	<0.001
Fasting Blood Glucose (mg/dL)	110.2 ± 14.8	98.7 ± 12.6	0.004

Table 2: Prevalence of Non-Alcoholic Hepatic Steatosis by H. pylori Status

NAHS Status	H. pylori Positive (n=62)	H. pylori Negative (n=38)	Total (n=100)	p-value
Present	35 (56.4%)	13 (34.2%)	48 (48%)	0.012
Absent	27 (43.5%)	25 (65.8%)	52 (52%)	

DISCUSSION

This study has shown a significant association of Helicobacter pylori (H. pylori) gastritis with non- alcoholic hepatic steatosis (NAHS) among dyspeptics in tertiary care hospitals of Lahore. Hepatic steatosis was present in more than half (56.4%) of the H. pylori positive individuals versus 34.2% of those without the infection[18]. The statistical significance of the observed (p = 0.012) underlines the possibility that hepatic fat deposition could be influenced by H. pylori infection[11].

In the past few years, there has been an increasing interest in the association of H. pylori with extra-gastric manifestations[12]. Chronic H. pylori infection leads to a chronic low grade inflammatory response characterized by elevated levels of pro-inflammatory cytokines, e.g. tumor necrosis factor alpha (TNF-α), interleukin 6 (IL-6) and C reactive protein (CRP). It has been demonstrated that these cytokines interfere with insulin signaling pathways and lipid metabolism, two important mechanisms for the pathogenesis of NAHS[18]. Along with the associated BMI and fasting glucose, patients with concurrent H. pylori infection and NAHS had higher BMI and fasting glucose in our study, consistent with the metabolic interplay between the two conditions[20].

Associations similar to that have been reported in several international studies. For example, research performed in Eastern Asia and Southern Europe has shown that H. pylori affected individuals are more likely to develop fatty liver, regardless of traditional risk for metabolic factors[19]. Despite this, the precise pathophysiological mechanism remains unclear but gut liver axis disruption, increased gut permeability and altered gut microbiota induced by chronic H. pylori colonization might play a role[13].

This is one of the few studies from the country that has explored this association in a local hospital based population. Our findings are consistent with the emerging global concern that H. pylori might act as a systemic pro inflammatory trigger and thereby affect metabolic organs including the liver[14]. In view of the high prevalence of H. pylori and NAHS in South Asian populations, the clinical significance of this association is of particular importance in preventive gastroenterology and hepatology[15].

Current study was cross sectional, therefore it does not permit causal inference. Second, the diagnosis of NAHS was based on ultra-sonographic criteria without histological verification, therefore the prevalence might have been underestimated or overestimated. In addition, diet and physical activity levels were not controlled as well.However, this study reveals some possible metabolic implications of H. pylori gastritis despite these limitations[19]. It emphasizes the importance of developing an integrated strategy of screening and the need for further prospective, multicenter study to determine the causality and whether H. pylori eradication might have therapeutic benefits in patients with, or at risk of, NAHS.

CONCLUSION

The link between H. pylori gastritis and nonalcoholic hepatic steatosis was significant in dyspeptic patients. There may be inflammatory or metabolic effects that lead to the buildup of liver fat from the infection. H. pylori may be detected early and treated, which would reduce liver-related metabolic risks.

DECLARATION

Acknowledgement:
We would Like to Acknowledge our collegues and paramedical staff of hospital for supporting us for data collection and making current study possible.

Authors contribution
Each author of this article fulfilled following Criteria of Authorship:

1. Conception and design of or acquisition of data or analysis and interpretation of data.
2. Drafting the manuscript or revising it critically for important intellectual content.
3. Final approval of the version for publication.

All authors agree to be responsible for all aspects of their research work.

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Ethical Considerations:
Institutional Review Boards (IRBs) gave ethical clearance. All participants gave informed verbal and written consent. Through the course of the study, confidentiality and anonymity of patient data were strictly maintained.

Competing interests
The authors declare that the research was conducted in the absence of any commercial or fnancial relationships that could be construed as a potential confict of interest.

Conflict of interest
The authors declared no conflict of interest.

REFERENCES

1. Mitchell H, Katelaris P. Epidemiology, clinical impacts and current clinical management of Helicobacter pylori infection. Med J Aust 2016; 10:376–380.
2. Sustmann A, Okuda M, Koletzko S. Helicobacter pylori in children. Helicobacter 2016; 21: 49-54.
3. Tapper EB, Parikh ND. Diagnosis and Management of Cirrhosis and Its Complications: A Review. JAMA. 2023;329(18):1589–1602.
4. Hooi J.K.Y., Lai W.Y., Ng W.K., Suen M.M.Y., Underwood F.E., Tanyingoh D., Malfertheiner P., Graham D.Y., Wong V.W.S., Wu J.C.Y., et al. Global Prevalence of Helicobacter pylori Infection: Systematic Review and Meta-Analysis. Gastroenterology. 2017;153:420–429.
5. World Gastroenterology O. World Gastroenterology Organisation Global Guideline: Helicobacter pylori in developing countries. J. Clin. Gastroenterol. 2011;45:383–388.
6. Burucoa C., Axon A. Epidemiology of Helicobacter pylori infection. Helicobacter. 2017;22(Suppl. 1).
7. 32. Hunt R.H., Xiao S.D., Megraud F., Leon-Barua R., Bazzoli F., van der Merwe S., Vaz Coelho L.G., Fock M., Fedail S., Cohen H., et al. Helicobacter pylori in developing countries. World Gastroenterology Organisation Global Guideline. J. Gastrointest. Liver Dis. 2011;20:299–304.
8. Xia HX, Windle HJ, Marshall DG, Smyth CJ, Keane CT, O'Morain CA. Recrudescence of Helicobacter pylori after apparently successful eradication: novel application of randomly amplified polymorphic DNA fingerprinting. Gut [Internet]. 1995;37(1):30–4.
9. Singh V, Mishra S, Rao GRK, Jain AK, Dixit VK, Gulati AK, et al. Evaluation of nested PCR in detection of Helicobacter pylori targeting a highly conserved gene: HSP60: Nested PCR in detection of H. pylori. Helicobacter [Internet]. 2008;13(1):30–4.
10. ZulloA,RomilliA,RinaldiV,VecchioneA.Gastric epithelial cell proliferation in patients with liver cirrhosis.Dig Dis Sci 2001;46(3):550-4.
11. WalugaM,KuklaM,ZorniakM,BacikA,KotulskiR.From the stomach to other organs:Helicobacter pylori and the liver.World J Hepatol.(2015)7:2136-46.
12. Sun X, Xu Y, Wang L, Zhang F, Zhang J, Fu X, et al. Association between TNFA gene polymorphisms and Helicobacter pylori infection: a meta-analysis. PLoS One 2016; 1:e0147410.
13. Silva LD, Rocha AM, Rocha GA, de Moura SB, Rocha MM, Dani R, et al. The presence of Helicobacter pylori in the liver depends on the Th1, Th17 and Treg cytokine profile of the patient. Mem Inst Oswaldo Cruz 2011; 106:748–754.
14. Buzás GM. Metabolic consequences of Helicobacter pylori infection and eradication. World J Gastroenterol 2014; 18:5226–5234.
15. Abdel-Razik A, Mousa N, Shabana W, Refaey M, Elhelaly R, Elzehery R, et al. Helicobacter pylori and non-alcoholic fatty liver disease: a new enigma? Helicobacter. (2018) 23:e12537.
16. Queiroz DM, Rocha AM, Rocha GA, Cinque SM, Oliveira AG, Godoy A, Tanno H. Association between Helicobacter pylori infection and cirrhosis in patients with chronic hepatitis C virus. Dig Dis Sci. 2006 Feb;51(2):370-3.
17. Feng H, Zhou X, Zhang G. Association between cirrhosis and Helicobacter pylori infection: a meta-analysis. Eur J GastroenterolHepatol. 2014 Dec;26(12):1309-19.
18. Pogorzelska J, Łapińska M, Kalinowska A, Łapiński TW, Flisiak R. Helicobacter pylori infection among patients with liver cirrhosis. Eur J GastroenterolHepatol. 2017 Oct; 29(10):1161-1165.
19. Singh MP, Yadav KA, Singh B, Gautam A. Prevalance of H. Pylori Infection in Patients of Cirrhosis of Liver with Its Etiological Correlation. 2022 Feb; 10(02):60-7.
20. Xu G, Ma S, Dong L, Mendez-Sanchez N, Li H, Qi X. Relationship of Helicobacter pylori Infection with Nonalcoholic Fatty Liver Disease: A Meta-Analysis. Can J Gastroenterol Hepatol. 2023 Jan; 2023: 5521239.