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RESEARCH ARTICLE

Prevalence of *Helicobacter pylori* Infection in Diabetic and Non-Diabetic Patients with Dyspepsia

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Abstract

Helicobacter pylori is one of the most widespread chronic bacterial infections worldwide and plays a significant role in the development of chronic gastritis, peptic ulcer disease, mucosa- associated lymphoid tissue lymphoma and gastric malignancy. Patients with Type 2 Diabetes Mellitus frequently exhibit gastrointestinal disturbances due to autonomic neuropathy, delayed gastric emptying, altered gastric acidity and impaired innate immunity, all of which may increase their susceptibility to *H. pylori* infection. This cross-sectional study aimed to compare the prevalence of *H. pylori* infection between diabetic and non-diabetic patients presenting with dyspepsia. A total of 204 dyspeptic patients were evaluated, consisting of 102 diabetics and 102 non-diabetics. All individuals underwent upper gastrointestinal endoscopy followed by Rapid Urease Testing and histopathological examination of antral and body biopsies. *H. pylori* positivity was defined as the presence of infection by either diagnostic method. The mean age of the diabetic group was 49 years, while that of the non-diabetic group was 42 years, with males outnumbering females in both groups. The prevalence of *H. pylori* infection was significantly higher in diabetics (69.6%) compared to non- diabetics (32.3%). Diabetic patients also exhibited a higher mean HbA1c level of 8.9%, and those with higher HbA1c values demonstrated a greater likelihood of *H. pylori* positivity. Logistic regression confirmed diabetes and higher HbA1c as independent predictors of infection. The study concludes that *H. pylori* infection is considerably more prevalent among diabetic individuals, particularly those with poor glycemic control, and routine screening may be beneficial in diabetic patients presenting with persistent upper gastrointestinal symptoms.

Keywords: Dyspepsia, Gastric Mucosal Infection, Glycemic Control, *Helicobacter pylori,* Histopathology, HbA1c, Rapid Urease Test, Type 2 Diabetes Mellitus

1. Introduction

Type 2 Diabetes Mellitus is a chronic metabolic disorder characterised by insulin resistance, insulin secretory dysfunction and a systemic inflammatory milieu. In addition to its well-known microvascular and macrovascular complications, diabetes significantly affects gastrointestinal function. Autonomic neuropathy alters gastric emptying, decreases gastric acid secretion, impairs mucosal defence and modifies the protective barrier of the stomach. These alterations create an environment conducive to colonisation by organisms such as *Helicobacter pylori*, a gram-negative spiral-shaped bacterium that persistently infects the gastric mucosa[1]. *H. pylori* induces chronic gastric inflammation through cytokine-mediated mechanisms involving interleukins and tumour necrosis factor-alpha, and contributes to several upper gastrointestinal diseases[2,3]. Since dyspeptic symptoms such as epigastric discomfort, bloating, early satiety and nausea occur commonly in both diabetic individuals and those infected with *H. pylori*, exploring the interplay

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between these conditions is clinically relevant. It is biologically plausible that diabetic patients may exhibit increased rates of *H. pylori* infection due to impaired immune responses, defective neutrophil function, delayed gastric transit and disruption of the gastric microenvironment[4-6]. Evaluating the prevalence of *H. pylori* among diabetics, especially in an Indian population with high burdens of both diabetes and infection, will help guide screening practices and therapeutic decisions.

2. Aim and Objectives

The primary aim of this study was to determine the prevalence of *Helicobacter pylori* infection among diabetic and non-diabetic patients presenting with dyspepsia. In addition, this study sought to evaluate differences in age and gender distribution between the groups, examine the relationship between glycemic control (as measured by HbA1c) and *H. pylori* status in diabetics, and determine whether diabetes and higher HbA1c independently increase the likelihood of *H. pylori* positivity.

3. Review of Literature

Helicobacter pylori has remained a central focus of gastroenterological research since its discovery in the early 1980s, owing to its profound clinical significance[1]. It has an established relationship with chronic gastritis, peptic ulcer disease, mucosa-associated lymphoid tissue lymphoma, and gastric carcinoma[2,3]. It is a highly adapted gram-negative, microaerophilic bacterium capable of surviving the harsh acidic environment of the stomach through multiple virulence mechanisms[4]. One of the most important survival strategies of *H. pylori* is its ability to produce urease, an enzyme that converts urea into ammonia, thereby buffering gastric acidity and creating a more favourable microenvironment around the organism. In addition to urease, several virulence factors such as cytotoxin-associated gene A (CagA), vacuolating cytotoxin A (VacA), adhesins like BabA and SabA, and flagellar motility allow the bacterium to firmly adhere to epithelial surfaces, evade immune responses, induce cellular injury, and perpetuate chronic inflammation.

Once colonisation occurs, *H. pylori* triggers a persistent inflammatory response involving neutrophils, lymphocytes, macrophages and dendritic cells, leading to the secretion of pro- inflammatory cytokines including IL-1β, IL-6, IL-8 and TNF-α[4]. This chronic inflammatory state contributes to progressive gastric mucosal damage, epithelial cell apoptosis, and alterations in acid secretion, ultimately resulting in a spectrum of pathological outcomes ranging from superficial gastritis to atrophic gastritis, intestinal metaplasia and malignant transformation. The long-standing nature of *H. pylori* infection makes it one of the most significant modifiable risk factors in gastrointestinal disease worldwide[1].

In parallel, Type 2 Diabetes Mellitus is recognised as a chronic metabolic disorder associated with considerable systemic implications, including those affecting the gastrointestinal tract. Diabetics frequently exhibit delayed gastric emptying (gastroparesis), altered gastric acid output, impaired mucosal healing, and disturbances in gastrointestinal motility due to autonomic neuropathy. Hyperglycemia—both chronic and intermittent—induces oxidative stress, disrupts mucosal microcirculation, reduces nitric oxide production, and impairs epithelial barrier integrity[6]. These mechanisms collectively weaken the host's innate and adaptive immune responses, making diabetic individuals more susceptible to infections, including those by *H. pylori*[5].

The impaired neutrophil chemotaxis, reduced macrophage activation, diminished phagocytic function, and altered cytokine regulation seen in diabetes serve as additional predisposing factors that increase the likelihood of persistent *H. pylori* colonization[5]. Hyperglycemia also influences gastric motility by affecting vagal nerve function, leading to prolonged gastric transit time. This creates a physiological environment that may favour the persistence of *H. pylori* in the stomach[6].

Over the past two decades, multiple observational and cross-sectional studies have been conducted across

different populations have explored the association between diabetes and *H. pylori*. Several of these investigations have reported higher prevalence rates of the bacterium among diabetic patients compared to non-diabetics[7-10]. Possible explanations include impaired immune function, increased susceptibility to colonisation, slower gastric emptying, and poorer gastric mucosal integrity in individuals with diabetes. Some studies have further demonstrated that poor glycemic control, reflected by higher HbA1c levels, correlates with increased *H. pylori* positivity[7,8]. This suggests that the metabolic state of the patient may directly influence the organism's ability to colonise and persist within the gastric mucosa.

However, literature also reflects some heterogeneity in findings, likely due to variations in diagnostic methods used to detect *H. pylori*, such as serological assays, stool antigen tests, urea breath tests and biopsy-based methods, including Rapid urease test and histology[4]. Differences in study populations, geographic variations in prevalence, dietary patterns, socioeconomic factors, nutritional status and antibiotic exposure also contribute to variability in reported associations. Despite these variations, the majority of data suggests that diabetic individuals demonstrate a higher vulnerability to *H. pylori* infection compared to the general population[7-10].

Additionally, research has explored whether eradication of *H. pylori* can improve glycemic control[4]. Some investigators have suggested that chronic infection contributes to systemic inflammation, insulin resistance and dysregulation of appetite hormones such as ghrelin and leptin. Although evidence remains inconclusive, eradication therapy has been associated in some studies with modest improvements in glycemic parameters, indicating a possible bidirectional relationship between the organism and metabolic control[5].

Overall, the accumulated scientific evidence indicates a biologically plausible and clinically relevant association between diabetes mellitus, glycemic control, and *H. pylori* infection[4-10]. Given the high prevalence of both conditions in many parts of the world, especially in regions like India, understanding this relationship is essential for guiding clinical evaluation and management of dyspeptic symptoms in diabetic patients.

4. Materials and Methods

This cross-sectional study was conducted in the Department of Medical Gastroenterology at Kilpauk Medical College, Chennai, between August 2024 and January 2025. A total of 204 consecutive patients aged between 35 and 75 years who presented with dyspeptic symptoms were included after obtaining informed consent. Dyspepsia was defined according to the Rome IV criteria, which describe it as the presence of one or more upper abdominal symptoms—including epigastric pain, epigastric burning, postprandial fullness, or early satiety—occurring for at least three months, with symptom onset at least six months before diagnosis and not explained by structural disease on routine testing.

The study population was divided equally into two groups: 102 diabetic patients and 102 non- diabetic controls. Type 2 Diabetes Mellitus was defined as a metabolic disorder characterised by insulin resistance and relative insulin deficiency, diagnosed based on standard ADA criteria, which include a fasting plasma glucose ≥126 mg/dL, a 2-hour postprandial glucose ≥200 mg/dL during an oral glucose tolerance test, an HbA1c ≥6.5%, or a random plasma glucose ≥200 mg/dL in patients with classic symptoms of hyperglycemia.

Exclusion criteria included patients with Type 1 diabetes, those with a history of prior *H. pylori* eradication therapy, previous gastric surgery, chronic liver disease, chronic kidney disease, malignancy, pregnancy, or refusal to consent. Detailed demographic information, clinical features, medication history, and laboratory parameters were recorded for all participants. HbA1c measurement was performed for all diabetic patients to assess glycemic control.

All patients underwent upper gastrointestinal endoscopy. During the procedure, antral and body biopsies were obtained and sent for histopathological examination. A Rapid Urease Test (RUT) was also performed using the biopsy sample. RUT is a biochemical test that detects *H. pylori* by identifying

its urease activity; the organism hydrolyses urea to produce ammonia, leading to a detectable colour change in the test medium. A patient was considered positive for *H. pylori* infection if either histopathology or the Rapid Urease Test demonstrated the presence of the organism.

Statistical analysis was conducted using SPSS version 22. Continuous variables were analysed using independent t-tests, while categorical variables were evaluated using the chi-square test. Multivariate logistic regression was applied to identify independent predictors of *H. pylori* infection after adjusting for confounding factors. A p-value of less than 0.05 was considered statistically significant.

5. Results (Including Observations)

A total of 204 patients with dyspeptic symptoms were included in the study, divided equally into two groups: 102 diabetic patients and 102 non-diabetic patients. The demographic characteristics of the study population revealed that the mean age of individuals in the diabetic group was 49 years, whereas the non-diabetic group had a lower mean age of 42 years (Table 1). This difference reflects the natural tendency for Type 2 Diabetes Mellitus to manifest progress in older age groups. In both groups, males outnumbered females, although the gender distribution remained broadly comparable (Table 2). When categorised into specific age brackets—35–45 years, 46–55 years, 56–65 years, and 66–75 years—it became evident that a larger proportion of diabetic patients fell into the higher age categories, particularly the 46–55 and 56–65-year groups. This pattern aligns with the expected epidemiological distribution of diabetes and supports the observation of an older average age among diabetics.

The central aim of the study was to evaluate and compare the prevalence of *Helicobacter pylori* infection between diabetics and non-diabetics. The findings demonstrated a marked and statistically significant difference. Of the 102 diabetic patients, 71 individuals (69.6%) tested positive for *H. pylori*, whereas only 33 individuals (32.3%) in the non-diabetic group were positive (Table 3, Figure 1). This indicates more than a twofold increase in *H. pylori* infection among diabetic patients compared to their non- diabetic counterparts. Statistical analysis using the chi-square test confirmed this relationship to be highly significant, with a p-value less than 0.00001, reinforcing the robustness of the association.

Table 1. Age distribution among diabetic and non-diabetic patients

Age group (years)	Diabetics (%)	Non-Diabetics (%)	Total (%)	F test	P value
35-45	27.4	45.1	36.3	1.12	0.292
46-55	37.2	31.4	34.3		
56-65	25.4	15.7	20.6		
66-75	9.8	7.8	8.8		
Total	100	100	100		

Table 2. Gender distribution among diabetic and non-diabetic patients.

Gender	Diabetics (%)	Non-Diabetics (%)	Total (%)	X^2	P value
Male	54.9	51.0	53.0	0.18	0.67
Female	45.1	49.0	47.0		
Total	100	100	100		

Table 3. Prevalence of *H. pylori* in diabetic and non-diabetic patients with dyspepsia

H. pylori	Diabetic patients No (%)	Non-Diabetic patients No (%)	Total no (%)	X^2	OR (95%CI)	P value
Positive	71(69.6 %)	33(32.3%)	104 (51%)	26.85	4.46(2.5-7.8)	<0.0001)
Negative	31 (30.4%)	69 (67.6%)	100 (49%)			
Total	102(50%)	102 (50%)	204 (100%)			

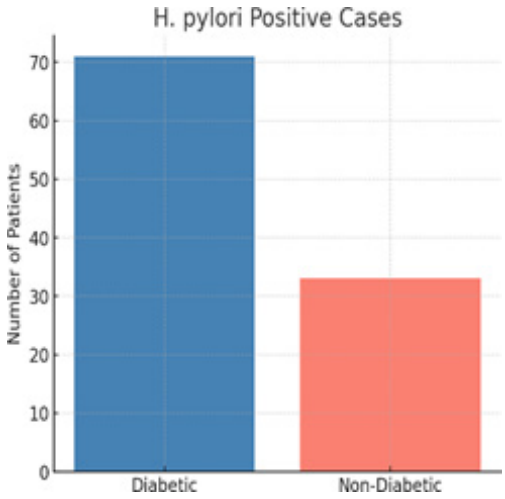

Figure 1. *H. pylori* positivity among diabetic and non-diabetic patients with dyspepsia

A detailed analysis of glycemic control revealed further insights. The average HbA1c level among diabetic patients was 8.9%, indicating poor overall glycemic control in the study cohort (Table 4). When stratified according to glycemic status, diabetic patients with HbA1c values ≥ 7.5% showed substantially higher rates of *H. pylori* positivity compared to those with HbA1c < 7.5%. This trend suggests that the severity of hyperglycemia plays an influential role in susceptibility to colonisation by *H. pylori*. The relationship between higher HbA1c levels and increased infection rates implies that poor metabolic control may impair immune function or alter gastric physiology in a way that favours bacterial survival and persistence.

Multivariate logistic regression analysis was performed to determine whether diabetes independently contributed to *H. pylori* positivity after adjusting for confounding variables such as age, gender, and HbA1c level. The analysis demonstrated that diabetes remained a strong and significant independent predictor of *H. pylori* infection even after statistical adjustment. This reinforces the observation that the association between diabetes and *H. pylori* is not merely due to age or demographic differences but is likely driven by pathophysiological mechanisms intrinsic to diabetes itself. Additionally, elevated HbA1c was also found to be independently associated with *H. pylori* infection, highlighting the direct influence of glycemic control on infection risk. In contrast, age and gender did not show statistically significant associations, suggesting that these demographic factors do not materially affect the likelihood of infection within the context of this study.

Overall, the results indicate a clear and compelling relationship between diabetes, poor glycemic control, and increased prevalence of *H. pylori* infection. The findings support the hypothesis that diabetic patients

Table 4. Mean HbA1c in diabetic patients with and without *H. pylori*

HbA1c test	Diabetes with *H. pylori* (Positive = 71)	Diabetes without *H. pylori* (Negative = 31)	t test	P value
Mean ± SD	9.4 ± 1.1	7.8 ±0.9	3.8	<0.001

are at significantly greater risk of harbouring *H. pylori*, with the risk further amplified in those with uncontrolled blood glucose levels (Figures 2 and 3).

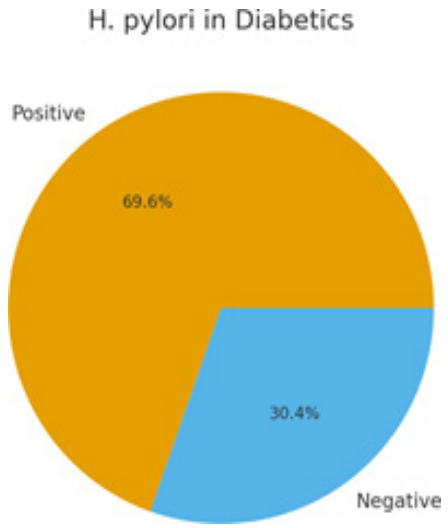

Figure 2. *H. pylori* positivity in diabetic patients with dyspepsia.

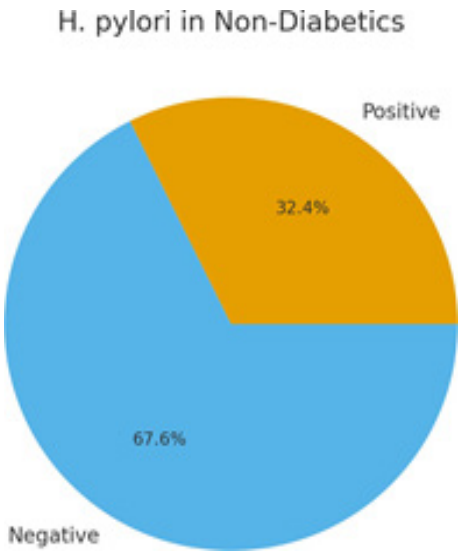

Figure 3. *H. pylori* positivity in non-diabetic patients with dyspepsia.

6. Discussion

The present study clearly demonstrates that *Helicobacter pylori* infection is significantly more prevalent among diabetic patients compared to non-diabetic individuals presenting with dyspepsia[7-10]. Nearly 70% of diabetics were positive for *H. pylori*, which is more than double the prevalence observed in non-diabetics. This substantial difference suggests a meaningful biological interplay between diabetes and increased susceptibility to gastric colonisation. Diabetes mellitus produces several pathophysiological changes that compromise host defences against infection. Chronic hyperglycemia impairs neutrophil chemotaxis and phagocytic function, reduces macrophage activity, alters cytokine responses and interferes with the complement cascade[5]. These immune alterations diminish the host's capability to eliminate pathogens effectively. Furthermore, autonomic neuropathy associated with diabetes leads to delayed gastric emptying, which prolongs gastric stasis and enhances bacterial persistence in the gastric mucosa. Hyperglycemia also disrupts mucosal microcirculation and epithelial integrity, reducing the efficiency of the mucosal barrier and creating a favourable environment for bacterial colonization[6].

The association between higher HbA1c levels and increased *H. pylori* positivity in this study further highlights the profound impact of poor glycemic control on infection susceptibility[7,8]. Patients with elevated HbA1c values exhibited significantly higher rates of *H. pylori* infection, indicating that metabolic dysregulation directly contributes to impaired host defences. Logistic regression analysis confirmed that diabetes remained an independent predictor of infection even after adjusting for confounding variables such as age and gender, emphasising that the relationship is rooted in metabolic and immunological mechanisms rather than demographic factors.

The overlap in symptoms between diabetic gastropathy and *H. pylori*-related dyspepsia also creates diagnostic challenges. Many diabetic patients may attribute their gastrointestinal discomfort to their underlying metabolic condition rather than a superimposed infection, resulting in delayed diagnosis. The findings of this study highlight the importance of maintaining a high degree of suspicion for *H. pylori* infection in diabetic individuals presenting

with dyspeptic symptoms. Early detection and treatment may help reduce complications such as peptic ulcer disease and gastric malignancy, and may potentially improve metabolic outcomes in selected patients[2,3].

This study has a few limitations that should be considered when interpreting the findings. It was conducted in a single tertiary care centre, which may limit the generalizability of the results to the wider population. Cross-sectional design does not allow assessment of a causal relationship between diabetes, glycemic control and *H. pylori* infection. Some potential confounding factors, such as dietary habits, socioeconomic status, and use of medications like proton pump inhibitors or antibiotics, were not evaluated, and these may influence *H. pylori* prevalence. Additionally, diabetic gastroparesis, which can mimic dyspeptic symptoms, was not objectively assessed. Despite these limitations, the study provides valuable insight into the significantly higher burden of *H. pylori* infection among diabetic individuals.

7. Summary and Conclusion

This study of 204 patients presenting with dyspepsia demonstrates that *Helicobacter pylori* infection is significantly more prevalent among diabetic individuals than non-diabetics. The diabetic group exhibited a positivity rate of 69.6%, compared to 32.3% among non-diabetics. Poor glycemic control, as reflected by higher HbA1c levels, was associated with increased infection rates. Logistic regression analysis confirmed diabetes and elevated HbA1c as independent predictors of *H. pylori* infection. The findings suggest that routine *H. pylori* screening should be considered in diabetic patients presenting with dyspeptic symptoms, particularly those with poor glycemic control, to facilitate timely diagnosis and management.

8. References

1. Malaty HM. Epidemiology of *Helicobacter pylori* infection. Best Pract Res Clin Gastroenterol. 2007; 21(2):205-214. https://doi.org/10.1016/j.bpg.2006.10.005
2. Parsonnet J. *Helicobacter pylori* and gastric cancer. Gastroenterol Clin North Am. 1993; 22(1):89-104. https://doi.org/10.1016/S0889-8553(21)00265-X
3. Wotherspoon A, Ortiz-Hidalgo C, Falzon M, *et al.* *Helicobacter pylori*-associated gastritis and primary B-cell gastric lymphoma. Lancet. 1991; 338(8776):1175-1176. https://doi.org/10.1016/0140-6736(91)92035-Z
4. He C, Yang Z, Lu NH. *Helicobacter pylori* infection and diabetes: is it a myth or fact? World J Gastroenterol. 2014; 20(16):4607-4617. https://doi.org/10.3748/wjg.v20.i16.4607
5. Manco M, Putignani L, Bottazzo GF. Gut microbiota, lipopolysaccharides, and innate immunity in the pathogenesis of obesity and cardiovascular risk. Endocr Rev. 2010; 31(6):817-844. https://doi.org/10.1210/er.2009-0030
6. Gentile S, Turco S, Oliviero B, *et al.* The role of autonomic neuropathy as a risk factor of *Helicobacter pylori* infection in dyspeptic patients with type 2 diabetes mellitus. Diabetes Res Clin Pract. 1998; 42(1):41-48. https://doi.org/10.1016/S0168-8227(98)00088-6
7. Al-Awadhi EF, Bahaj S, Al-Oferi B, *et al.* *Helicobacter pylori* infection among patients with type II diabetes mellitus. J Diab Metab Disorder Control. 2020; 7(2):53-56. https://doi.org/10.15406/jdmdc.2020.07.00200
8. Olimy MG. Course of *Helicobacter pylori* infection in patients with type 2 diabetes mellitus in comparison with healthy persons. 2013.
9. Bener A, Micallef R, Afifi M, *et al.* Association between type 2 diabetes mellitus and *Helicobacter pylori* infection. Turk J Gastroenterol. 2007; 18(4):225-229.
10. De Luis D, De La Calle H, Roy G, *et al.* *Helicobacter pylori* infection and insulin-dependent diabetes mellitus. Diabetes Res Clin Pract. 1998; 39(2):143-146. https://doi.org/10.1016/S0168-8227(97)00127-7