



Impact of *Helicobacter pylori* infection on diabetes: A cross-sectional investigation in Northern Iran

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ABSTRACT

Article info:

Received: 18 May 2025
Accepted: 17 Jun 2025

Keywords:

Helicobacter pylori
Gastritis severity
Prevalence
FBS
Diabetes

Globally, *Helicobacter pylori* represents one of the leading chronic infections, and is suspected to contribute to extra-digestive diseases, notably diabetes mellitus (DM). Despite growing interest, data from Middle Eastern populations are limited. This study was conducted to determine the occurrence of diabetes and explore its relationship with *H. pylori* infection. This analytical cross-sectional study included 383 patients with histologically confirmed *H. pylori* infection admitted to a tertiary care center in the North of Iran, between March 2021 and March 2023. Demographic records, diabetes status, fasting blood glucose (FBS), and gastritis severity were extracted from medical archives. Diabetes was defined using international diagnostic criteria. Among the 383 patients, 90 (23.5%) had diabetes. Diabetic patients were significantly older than non-diabetics (63.5 ± 10.8 vs. 56.3 ± 17.8 years, $p < 0.001$), and diabetes prevalence increased with age, reaching 36.7% in patients ≥ 70 years. FBS was markedly higher in diabetic patients compared with non-diabetics (173.8 ± 48.5 vs. 100.6 ± 14.4 mg/dL, $p < 0.001$). No significant association was observed between diabetes and gender ($p = 0.059$) or gastritis severity ($p = 0.692$). Duration of diabetes did not differ significantly across gastritis grades ($p = 0.352$). Nearly one-quarter of patients with *H. pylori* infection in Northern Iran had diabetes, with older age and higher FBS significantly associated with its presence. Our analysis did not reveal a significant link between diabetes and the histological severity of gastritis. These results underscore the necessity for prospective research to elucidate the causal mechanisms linking *H. pylori* infection and diabetes.

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1. Introduction

Globally, *Helicobacter pylori* represents one of the leading chronic infections, particularly common in developing countries [1,2]. Beyond its established gastrointestinal consequences, emerging research suggests potential systemic effects, notably an influence on metabolic disorders such as diabetes mellitus (DM) [3,4].

Several biological mechanisms may explain these associations. Chronic infection by *H. pylori* stimulates systemic inflammation, with elevated cytokines, including C-reactive protein, tumor necrosis factor- α , and interleukin-1 β , which may disrupt insulin signaling and promote insulin resistance [5,6]. Additionally, gastric infection can alter secretion of key hormones such as ghrelin, leptin, gastrin, and somatostatin, thereby influencing insulin secretion and glucose metabolism [7]. Studies in human subjects have also documented impaired insulin secretion and elevated insulin resistance indices among individuals with *H. pylori* infection [8]. DM represents a serious and growing public health challenge, particularly in Iran. Giving to published data, the pooled prevalence of type 2 diabetes (T2D) among Iranian adults over 25 years has reached approximately 10.8 % (95 % CI: 9.05–12.20 %) between 1996 and 2023, with a notable rise from 7.08 % in the late 1990s to over 13.40 % in recent years [9]. At a population level, in Iranian adults aged 35–70 years, diabetes affected 15.0 % (95 % CI: 12.6–17.3 %) and prediabetes 25.4 % (95 % CI: 18.6–32.1 %) of the population; alarmingly, only 41.2 % of those receiving treatment achieved glycemic control [10]. Taken together, these trends underscore the significant and growing strategies to safeguard population health.

Despite the growing data on the connection between *H. pylori* infection and DM [11-13], still, some studies have reported conflicting findings [14,15]. Moreover, data specific to Middle Eastern populations, particularly Northern Iran, are scarce. The city of Rasht, located in Guilan province, exhibits relatively high endemicity of *H. pylori* [16,17]. Therefore, this study was conducted to determine the occurrence of diabetes and explore its relationship with *H. pylori* infection.

2. Materials and Methods

2.1 Study design and setting

This analytical cross-sectional study was directed at Razi Hospital, Rasht, Northern Iran, over a two-year period from March 2021 to March 2023. The study population included hospitalized patients with histologically confirmed *H. pylori* infection, diagnosed according to the Sydney classification system.

2.2 Study population and sample size

Medical records archived at Razi Hospital were

reviewed using convenience sampling. Based on a previous report by Moosazadeh et al. estimating the prevalence of *H. pylori* infection in Iran at 54% [18], and using a standard sample size calculation formula with a 95% confidence level, the minimum sample size required was 382 patients. All eligible cases within the defined period were included in the study.

2.3 Eligibility criteria

Inclusion criteria were: (1) age ≥ 18 years; (2) hospitalization between study periods; (3) histopathological confirmation of *H. pylori* infection; and (4) availability of complete medical records containing demographic, laboratory and clinical data. Exclusion criteria were: (1) absence of histological confirmation of *H. pylori*; (2) incomplete key clinical or laboratory data (e.g., missing fasting blood glucose (FBS), or severity of infection; (3) end-stage renal disease (GFR < 30 mL/min/1.73 m²); (4) comorbid chronic or malignant diseases significantly affecting glucose metabolism; and (5) prior antibiotic therapy before hospitalization that could alter *H. pylori* detection or severity.

2.4 Data collection

After approval from the Ethics Committee of Guilan University of Medical Sciences (IR.GUMS.REC.1401.553), patient files were accessed. A structured checklist was used to extract data including age, sex, duration of diabetes, FBS, and histological severity of *H. pylori* infection (mild, moderate, severe). Diabetes was defined according to international diagnostic criteria: HbA1c $\geq 6.5\%$, FBS ≥ 126 mg/dL, 2-hour plasma glucose ≥ 200 mg/dL during an oral glucose tolerance test, or random plasma glucose ≥ 200 mg/dL with classic hyperglycemia symptoms or hyperglycemic crisis.

2.5 Statistical assessment

Data were analyzed using SPSS version 22 (IBM Corp., Armonk, NY, USA). Continuous variables are reported as mean \pm standard deviation (SD), while categorical variables are presented as frequencies and percentages. Associations between categorical variables were assessed using the chi-square test, and differences in continuous variables were evaluated with the independent t-test. A p-value of less than 0.05 was considered statistically significant.

3. Results

Overall, 383 patients with *H. pylori* infection confirmed by histology were included in the analysis. Of these, 90 patients (23.5%) were diagnosed with diabetes mellitus, while 293 (76.5%) had no history of diabetes. The mean age of the study population was 58.0 \pm 16.7 years, ranging from 19 to 89 years. The majority

of patients were aged ≥ 60 years (51.3%). Female patients represented 56.4% (n=216), while males accounted for 43.6% (n=167). The mean FBS level was 117.8 ± 40.9 mg/dL. Regarding gastritis severity, 59.3% (n=227) had mild inflammation, 33.7% (n=129) moderate, and 7.0% (n=27) severe inflammation (Table 1).

Patients with diabetes were significantly older compared with non-diabetic patients (63.5 ± 10.8 vs. 56.3 ± 17.8 years; $p < 0.001$). Diabetes prevalence increased with advancing age, from 2.2% in patients < 40 years to 36.7% among those ≥ 70 years. Gender distribution showed no statistically significant difference between diabetic and non-diabetic groups ($p = 0.059$). The mean FBS was markedly higher in diabetic patients compared with non-diabetics (173.8 ± 48.5 vs. 100.6 ± 14.4 mg/dL; $p < 0.001$) (Table 2).

No statistically significant association was observed between diabetes and histological severity of gastritis ($p = 0.692$). Similarly, mean FBS values did not differ significantly across the three grades of gastritis ($p = 0.407$). The mean duration of diabetes varied numerically by histological grad, but the difference was not statistically significant ($p = 0.352$) (Table 3).

4. Discussion

In this two-year cross-sectional study conducted in Northern Iran, we investigated the prevalence of diabetes mellitus among patients with histologically confirmed *H. pylori* infection and evaluated its association with demographic and clinical variables. These observations could contribute to clarifying how *H. pylori* infection might influence metabolic dysregulation in this geographic and ethnic context. We found that 23.5% of patients with *H. pylori* infection had diabetes, with a significantly higher prevalence in older individuals. Diabetes was strongly associated with elevated FBS levels but not with gender or histological severity of gastritis. Although the mean duration of diabetes differed numerically across gastritis grades, this association was not statistically significant.

Our findings are in line with several previous studies suggesting a positive association between *H. pylori* infection and diabetes mellitus. Zhou et al. reported in a meta-analysis of 41 studies that the odds of *H. pylori* infection were significantly higher among individuals with type 2 diabetes (OR 1.76; 95% CI 1.40–2.21) compared to non-diabetic controls [11].

Table 1. Demographic, laboratory, and clinical characteristics of patients with *Helicobacter pylori* infection (n=383)

Variable	Group	Frequency	Percent
Age (year)	<40	59	15.4
	40-49	51	13.33
	50-59	76	19.84
	60-69	97	25.23
	≥ 70	100	26.11
	Mean \pm SD		58.01 \pm 16.68
Gender	Male	167	43.6
	Female	216	56.4
Gastritis grade	Mild inflammation	227	59.27
	Moderate inflammation	129	33.68
	Severe inflammation	27	7.05
FBS	Mean \pm SD	117.83 \pm 40.89	
Duration of diabetes (year)	Mean \pm SD	6.04 \pm 9.52	

Table 2. Association of demographic, laboratory, and clinical variables with the occurrence of diabetes in patients with *Helicobacter pylori* infection

Variable	Group	Non-diabetes		Diabetes		P value
		Frequency	Percent	Frequency	Percent	
Age (year)	<40	57	14.3	2	2.2	<0.001
	40-49	42	19.5	9	10.0	
	50-59	59	20.1	17	18.9	
	60-69	64	21.8	33	32.2	
	≥ 70	71	24.2	29	36.7	
	Mean \pm SD		56.32 \pm 17.79		63.51 \pm 10.78	
Gender	Male	173	59	43	47.8	0.059
	Female	120	41	47	58.2	
Gastritis grade	Mild inflammation	171	6.8	56	7.8	0.692
	Moderate inflammation	102	34.8	27	30.0	
	Severe inflammation	20	58.4	7	62.2	
FBS	Mean \pm SD	100.62 \pm 14.42		173.83 \pm 48.47		<0.001

Table 3. Association of fasting blood glucose and duration of diabetes with the severity of gastritis in patients with *Helicobacter pylori* infection

Variable	Group	Gastritis grade			P value
		Mild inflammation	Moderate inflammation	Severe inflammation	
FBS	Mean \pm SD	118.03 \pm 41.83	115.53 \pm 37.33	127.11 \pm 49.17	0.407
Duration of diabetes (year)	Mean \pm SD	9.51 \pm 6.76	10.3 \pm 4.75	6.57 \pm 3.55	0.352

Similarly, Wang et al. demonstrated that *H. pylori* infection was associated not only with type 2 diabetes but also with diabetic nephropathy [12]. More recent evidence from cohort studies has also supported this association. Chen et al., using Taiwanese cohort data, observed that *H. pylori* infection significantly increased the risk of incident type 2 diabetes and metabolic syndrome [19].

Conversely, not all studies have reported consistent results. Cai et al. found no significant overall association between *H. pylori* infection and diabetes in a large Chinese cohort, although a slight increase in risk was noted among women [14]. Similarly, studies from Korea, and Lebanon indicated that while *H. pylori* infection was related with insulin resistance [15,20]. These discrepancies may be attributable to differences in diagnostic methods, study designs, population genetics, or environmental exposures.

The underlying mechanisms linking *H. pylori* infection with diabetes remain under investigation. Chronic infection, particularly *H. pylori*, can promote insulin resistance by inducing systemic inflammation and disrupting gastric hormone secretion involved in glucose regulation [7]. While these mechanisms provide biological plausibility, the lack of significant association between diabetes and histological gastritis severity in our study suggests that systemic effects may be independent of local mucosal inflammation.

From a public health perspective, the coexistence of *H. pylori* infection and diabetes is particularly concerning in Iran, where both conditions are highly prevalent. Recent national data indicate that the prevalence of type 2 diabetes among Iranian adults exceeds 10% and continues to rise [9]. Our finding of a 23.5% diabetes prevalence among *H. pylori*-infected patients underscores the importance of considering potential metabolic consequences in the management of this infection.

The strengths of our study include the use of histological confirmation for *H. pylori* infection and application of standardized diagnostic criteria for diabetes. Furthermore, we analyzed the relationship between diabetes and the histological severity of gastritis, which has been less frequently explored. However, the study has limitations. Its cross-sectional design precludes causal inference, and the use of hospital-based sampling may limit generalizability. Potential residual confounding, such as dietary habits, socioeconomic status, and genetic predisposition, could not be fully controlled.

Our findings suggest that diabetes mellitus is common among patients with *H. pylori* infection in Northern Iran and is strongly associated with older age and elevated FBS. However, no significant association was observed with histological gastritis severity. Future prospective studies with larger and more diverse populations are warranted to clarify the causal relationship and underlying mechanisms between *H. pylori* infection and diabetes.

Authors' contributions

Study supervision, Design, and Resources: PK, BM, HS; Data collection, and Investigation: FB, PK. Analysis and Interpretation: FB, PK, BM; Writing draft, and Critical revision: FB, HS. All authors have read and approved the final version of manuscript.

Conflict of interest

No potential conflict of interest was reported by the authors.

Ethical declarations

Approval for this study was obtained from the Ethics Committee of Guilan University of Medical Sciences (IR.GUMS.REC.1401.553), which also waived the need for informed consent due to the retrospective nature of the research.

Financial support

Self-funded.

References

1. Elbehiry A, Marzouk E, Aldubaib M, Abalkhail A, Anagreyyah S, Anajirih N, et al. Helicobacter pylori Infection: Current Status and Future Prospects on Diagnostic, Therapeutic and Control Challenges. *Antibiotics (Basel)*. 2023;12(2):191. DOI: [10.3390/antibiotics12020191](https://doi.org/10.3390/antibiotics12020191) PMID: [36830102](https://pubmed.ncbi.nlm.nih.gov/36830102/)
2. Salih BA. Helicobacter pylori infection in developing countries: the burden for how long? *Saudi J Gastroenterol*. 2009;15(3):201-7. DOI: [10.4103/1319-3767.54743](https://doi.org/10.4103/1319-3767.54743) PMID: [19636185](https://pubmed.ncbi.nlm.nih.gov/19636185/)
3. Wroblewski LE, Peek RM Jr, Wilson KT. Helicobacter pylori and gastric cancer: factors that modulate disease risk. *Clin Microbiol Rev*. 2010;23(4):713-39. DOI: [10.1128/CMR.00011-10](https://doi.org/10.1128/CMR.00011-10) PMID: [20930071](https://pubmed.ncbi.nlm.nih.gov/20930071/)
4. Valenzano M, Bisio A, Grassi G. Helicobacter pylori and diabetes mellitus: a controversial relationship. *Minerva Endocrinol*. 2019;44(3):301-309. DOI: [10.23736/S0391-1977.19.03021-9](https://doi.org/10.23736/S0391-1977.19.03021-9) PMID: [31304727](https://pubmed.ncbi.nlm.nih.gov/31304727/)
5. Buzás GM. Metabolic consequences of Helicobacter pylori infection and eradication. *World J Gastroenterol*. 2014;20(18):5226-34. DOI: [10.3748/wjg.v20.i18.5226](https://doi.org/10.3748/wjg.v20.i18.5226) PMID: [24833852](https://pubmed.ncbi.nlm.nih.gov/24833852/)
6. Dincă AL, Meliț LE, Mărginean CO. Old and New Aspects of *H. pylori*-Associated Inflammation and Gastric Cancer. *Children (Basel)*. 2022;9(7):1083. DOI: [10.3390/children9071083](https://doi.org/10.3390/children9071083) PMID: [35884067](https://pubmed.ncbi.nlm.nih.gov/35884067/)
7. Hosseinasab Nodoushan SA, Nabavi A. The Interaction of Helicobacter pylori Infection and Type 2 Diabetes Mellitus. *Adv Biomed Res*. 2019;8:15. DOI: [10.4103/abr.abr_37_18](https://doi.org/10.4103/abr.abr_37_18) PMID: [30993085](https://pubmed.ncbi.nlm.nih.gov/30993085/)
8. Vafaemanesh J, Parham M, Seyyedmajidi M, Bagherzadeh M. Helicobacter pylori infection and insulin resistance in diabetic and nondiabetic population. *ScientificWorldJournal*. 2014;2014:391250. DOI: [10.1155/2014/391250](https://doi.org/10.1155/2014/391250) PMID: [25405220](https://pubmed.ncbi.nlm.nih.gov/25405220/)
9. Hazar N, Jekar M, Namavari N, Hosseini S, Rahmanian V. An updated systematic review and Meta-analysis of the prevalence of type 2 diabetes in Iran, 1996-2023. *Front Public Health*. 2024;12:1322072. DOI: [10.3389/fpubh.2024.1322072](https://doi.org/10.3389/fpubh.2024.1322072) PMID: [38638475](https://pubmed.ncbi.nlm.nih.gov/38638475/)

10. Khamseh ME, Sepanlou SG, Hashemi-Madani N, Joukar F, Mehrparvar AH, Faramarzi E, et al. Nationwide Prevalence of Diabetes and Prediabetes and Associated Risk Factors Among Iranian Adults: Analysis of Data from PERSIAN Cohort Study. *Diabetes Ther.* 2021;12(11):2921-2938. DOI: [10.1007/s13300-021-01152-5](https://doi.org/10.1007/s13300-021-01152-5) PMID: [34595726](https://pubmed.ncbi.nlm.nih.gov/34595726/)
11. Zhou X, Zhang C, Wu J, Zhang G. Association between Helicobacter pylori infection and diabetes mellitus: a meta-analysis of observational studies. *Diabetes Res Clin Pract.* 2013;99(2):200-8. DOI: [10.1016/j.diabres.2012.11.012](https://doi.org/10.1016/j.diabres.2012.11.012) PMID: [23395214](https://pubmed.ncbi.nlm.nih.gov/23395214/)
12. Wang F, Liu J, Lv Z. Association of Helicobacter pylori infection with diabetes mellitus and diabetic nephropathy: a meta-analysis of 39 studies involving more than 20,000 participants. *Scand J Infect Dis.* 2013;45(12):930-8. DOI: [10.3109/00365548.2013.844351](https://doi.org/10.3109/00365548.2013.844351) PMID: [24143873](https://pubmed.ncbi.nlm.nih.gov/24143873/)
13. Mansori K, Moradi Y, Naderpour S, Rashti R, Moghaddam AB, Saed L, et al. Helicobacter pylori infection as a risk factor for diabetes: a meta-analysis of case-control studies. *BMC Gastroenterol.* 2020;20(1):77. DOI: [10.1186/s12876-020-01223-0](https://doi.org/10.1186/s12876-020-01223-0) PMID: [32209055](https://pubmed.ncbi.nlm.nih.gov/32209055/)
14. Man S, Ma Y, Jin C, Lv J, Tong M, Wang B, et al. Association between Helicobacter pylori Infection and Diabetes: A Cross-Sectional Study in China. *J Diabetes Res.* 2020;2020:7201379. DOI: [10.1155/2020/7201379](https://doi.org/10.1155/2020/7201379) PMID: [33062714](https://pubmed.ncbi.nlm.nih.gov/33062714/)
15. Pyo JH, Lee H, Choi SC, Cho SJ, Choi YH, Min YW, et al. Lack of Association between Past Helicobacter pylori Infection and Diabetes: A Two-Cohort Study. *Nutrients.* 2019;11(8):1874. DOI: [10.3390/nu11081874](https://doi.org/10.3390/nu11081874) PMID: [31409000](https://pubmed.ncbi.nlm.nih.gov/31409000/)
16. Hojati SA, Kokabpeyk S, Yaghoubi S, Joukar F, Asgharnejhad M, Mansour-Ghanaei F. Helicobacter pylori infection in Iran: demographic, endoscopic and pathological factors. *BMC Gastroenterol.* 2021;21(1):355. DOI: [10.1186/s12876-021-01931-1](https://doi.org/10.1186/s12876-021-01931-1) PMID: [34579657](https://pubmed.ncbi.nlm.nih.gov/34579657/)
17. Mansour-Ghanaei F, Yousefi Mashhour M, Joukar F, Sedigh M, Bagher-Zadeh A, Jafarshad R. Prevalence of Helicobacter Pylori Infection among Children in Rasht, Northern Iran. *Middle East J Dig Dis.* 2011;1(2):5. URL: <https://mejdd.org/index.php/mejdd/article/view/432>
18. Moosazadeh M, Lankarani KB, Afshari M. Meta-analysis of the Prevalence of Helicobacter Pylori Infection among Children and Adults of Iran. *Int J Prev Med.* 2016;7:48. DOI: [10.4103/2008-7802.177893](https://doi.org/10.4103/2008-7802.177893) PMID: [27076886](https://pubmed.ncbi.nlm.nih.gov/27076886/)
19. Chen YY, Fang WH, Wang CC, Kao TW, Chang YW, Wu CJ, et al. Helicobacter pylori infection increases risk of incident metabolic syndrome and diabetes: A cohort study. *PLoS One.* 2019;14(2):e0208913. DOI: [10.1371/journal.pone.0208913](https://doi.org/10.1371/journal.pone.0208913) PMID: [30779804](https://pubmed.ncbi.nlm.nih.gov/30779804/)
20. Naja F, Nasreddine L, Hwalla N, Moghames P, Shoaib H, Fatfat M, et al. Association of H. pylori infection with insulin resistance and metabolic syndrome among Lebanese adults. *Helicobacter.* 2012;17(6):444-51. DOI: [10.1111/j.1523-5378.2012.00970.x](https://doi.org/10.1111/j.1523-5378.2012.00970.x) PMID: [23066847](https://pubmed.ncbi.nlm.nih.gov/23066847/)