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Research Paper

Link Between *Helicobacter pylori* Infection and Colorectal Polyps: Implications for Screening in Northern Iran

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ABSTRACT

Background: *Helicobacter pylori (H. pylori)* is a common gastrointestinal pathogen implicated in gastric malignancies, and emerging evidence suggests a potential link to colorectal neoplasia. Objectives:

Objectives: This study was aimed to investigate the relationship between *H. pylori* infection and colorectal polyps in a Middle Eastern population with high background prevalence of both conditions.

Materials & Methods: In this hospital-based cross-sectional analytical study, 382 adults undergoing both upper endoscopy and colonoscopy at Razi Hospital in Rasht, Iran, were enrolled over a one-year period. *H. pylori* infection was detected via histopathological examination of gastric biopsies. Colorectal polyps were identified during colonoscopy and confirmed by histological evaluation. Demographic, clinical, and lifestyle data were collected. Logistic regression analyses were performed to assess the association between *H. pylori* infection and colorectal polyps, adjusting for age, sex, and other confounders.

Results: The mean age of participants was 56.93±13.51years with aged 18 years and older. The overall prevalence of *H. pylori* infection and colorectal polyps was 48.4% and 33.0%, respectively. No significant association was observed between *H. pylori* infection and colorectal polyps in unadjusted (OR=1.10; 95% CI, 0.72%, 1.68%; P=0.667) or adjusted models (adjusted OR=1.19; 95% CI, 0.76%, 1.87%; P=0.435). Polyp prevalence increased significantly with age (P for trend=0.049) but did not differ by sex or *H. pylori* status.

Conclusion: This study found no significant association between *H. pylori* infection and colorectal polyps. These results underscore the need for further prospective and mechanistic research across diverse populations to clarify this relationship.

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Introduction

Colorectal cancer (CRC) remains a major public health concern worldwide, ranking as the third most commonly diagnosed malignancy and the second leading cause of cancer-related mortality [1]. Colorectal polyps, particularly adenomatous and serrated types, are well-established precursor lesions that play a critical role in the adenoma carcinoma sequence [2, 3]. Identifying and understanding the modifiable risk factors contributing to colorectal polyp formation is vital for the development of effective preventive strategies against CRC [4]. Recent research has increasingly focused on the role of gastrointestinal microbiota and chronic infections in the pathogenesis of colorectal neoplasia. Among these, *Helicobacter pylori (H. pylori)*, a Gram-negative, microaerophilic bacterium that colonizes the human stomach and is implicated in chronic gastritis, peptic ulcer disease, and gastric cancer has drawn attention for its potential association with extragastric malignancies, including CRC [5–7].

Mechanistically, *H. pylori* may influence colorectal carcinogenesis via systemic inflammatory responses, alterations in gastric acid secretion, changes in gut microbiota, or through the release of carcinogenic metabolites such as ammonia and N-nitroso compounds [8–10]. Epidemiological studies examining the link between *H. pylori* infection and colorectal polyps have yielded mixed results. Several meta-analyses and case-control studies have reported a significant association between *H. pylori* seropositivity and increased risk of colorectal polyps and adenomas [11, 12], whereas others have found no clear relationship [13, 14]. These inconsistencies may be attributed to variations in geographic regions, diagnostic methods, *H. pylori* strains, host genetic factors, and environmental exposures. Notably, studies suggest that the effect of *H. pylori* may be more pronounced in populations with high background infection rates and differing gut microbial profiles [6, 15, 16].

Iran, particularly its northern regions along the Caspian Sea, exhibits a relatively high prevalence of both *H. pylori* infection and CRC incidence compared to global averages [16, 17]. Despite this epidemiological overlap, limited population-based studies have explored the potential association between *H. pylori* infection and colorectal polyps in this region. Given the unique demographic, dietary, and microbial characteristics of northern Iran, investigating this relationship is both timely and regionally relevant. Therefore, this study aims to evaluate the association between *H. pylori* infection and the presence of colorectal polyps in individuals undergoing colonoscopy in northern Iran. By identifying potential infectious contributors to colorectal polyp formation, this research may enhance our understanding of CRC risk stratification and inform tailored screening and prevention strategies in high-risk populations.

Materials and Methods

Study design and setting

This cross-sectional study was conducted at Razi Hospital, the main gastroenterology referral center in Rasht, northern Iran between May 2024 and July 2024. All eligible individuals referred for both upper gastrointestinal endoscopy and colonoscopy were recruited using a census and convenience sampling strategy. All procedures complied with the ethical standards of the Declaration of Helsinki. Written informed consent was obtained from all participants prior to enrollment.

Study population and eligibility criteria

Adults aged 18 years and older who were referred for diagnostic upper endoscopy and colonoscopy were considered eligible for inclusion in the study. Based on an expected prevalence of colorectal polyps of 34.6% [18], a 95% confidence level, and 80% power to detect an odds ratio of 1.8 for the association with *H. pylori*, the minimum required sample size was calculated as 370 participants. Individuals were excluded if they were unable to complete adequate bowel preparation, had a history of gastrointestinal malignancy or were under ongoing follow-up for known gastrointestinal cancers, had prior gastrointestinal surgery other than polypectomy, presented with active gastrointestinal bleeding at the time of evaluation, had used proton pump inhibitors (PPIs) without discontinuation for at least two weeks prior to endoscopy to avoid false-negative *H. pylori* results [19], had received antibiotics within the preceding month, or had previously undergone eradication therapy for *H. pylori* infection.

Data collection and instruments

Data were collected using a standardized case-report form and structured questionnaire, administered by a trained research team. The questionnaire gathered demographic (age, sex, education level, marital status, occupation), lifestyle (smoking status, alcohol consumption), clinical (underlying diseases, family history of CRC). and body mass index (BMI). Anthropometric measurements were performed by trained staff using a calibrated digital scale (Seca 803, Germany) for weight and a stadiometer for height.

Assessment of *H. pylori* infection

All participants underwent upper endoscopy using a standard video gastroscope (FUJIFILM, Japan). During the procedure, multiple biopsies were obtained, usually two from the gastric body and two from the antrum, for histopathological examination. The presence of *H. pylori* was confirmed by pathologist reports using Giemsa staining for improved detection sensitivity.

Colonoscopy procedure and polyp evaluation

Colonoscopy was performed on all subjects by gastroenterologists blinded to participants' *H. pylori* status using standard colonoscopy systems (FUJIFILM, Japan). Bowel preparation involved a clear liquid diet for 24 hours, with a regimen including Bisacodyl tablets, Sena-Graph syrup, and Pidrolax powder, in accordance with local institutional protocol. Solid foods and colored liquids were avoided; antidiabetic and cardiovascular medications were managed under specialist guidance. When polyps were detected, polypectomy was performed using cold snare forceps for polyps ≤1 cm and hot snare forceps for polyps >1 cm, in accordance with established endoscopic guidelines [20, 21]. Excised specimens were placed in 10% formalin and transferred to the pathology department, which was blinded to *H. pylori* status.

Polyp size was determined by visual comparison with biopsy forceps and confirmed by direct measurement of the specimen with a surgical ruler. Histopathological classification was based on World Health Organization (WHO) criteria and included [22]: Tubular adenoma, tubulovillous adenoma, serrated adenoma, hyperplastic polyp, inflammatory polyp, and sessile serrated lesions . Polyps were further categorized by size: <0.5 cm, 0.5–1 cm, and >1 cm. Participants were categorized into four groups based on their *H. pylori* infection status and the presence or absence of colorectal polyps: Those with both *H. pylori* infection and polyps, those with *H. pylori* infection but no polyps, those without *H. pylori* infection who had polyps, and those without *H. pylori* infection and no polyps.

Statistical analysis

Categorical variables were summarized as frequencies and percentages, while continuous variables were reported as Mean±SD. Differences in the prevalence of colorectal polyps across sex and age groups were assessed using the chi-square test and the cochran-armitage test for trend. The prevalence of colorectal polyps among participants with and without *H. pylori* infection was also compared using the chi-square test. To evaluate the association between *H. pylori* infection and the presence of colorectal polyps, logistic regression analysis was performed in both crude and adjusted models. Results were reported as odds ratios (ORs) with corresponding 95% confidence intervals (CIs). All statistical analyses were conducted using SPSS software, version 16.0 (SPSS Inc., Chicago, IL), and a two-tailed P<0.05 was considered statistically significant. Graphs were generated using GraphPad Prism software, version 8.0.1 (GraphPad Software, San Diego, CA).

Results

Participant demographics and clinical characteristics

A total of 382 individuals referred to the endoscopy unit at Razi Hospital in Rasht were included in the study. The mean age of participants was 56.93±13.51 years, with 39.3% (n=150) aged above 60 years. Women accounted for 54.5% (n=208) of the study population. The majority were married (82.7%, n=316), and 66.2% (n=253) resided in urban areas. Only 9.4% (n=36) had a university education, while 57.1% (n=218) had not completed high school. The mean BMI was 25.59±4.46 kg/m², and 15.2% (n=58) of participants were categorized as obese. Smoking and alcohol use were reported in 7.3% (n=28) and 1.3% (n=5), respectively. A total of 53.4% (n=204) had at least one underlying medical condition, and 6.8% (n=26) reported a family history of cancer (Table 1).

Prevalence of *H. pylori* infection and colorectal polyps

The overall prevalence of *H. pylori* infection was 48.4% (n=185). The prevalence did not differ significantly between men and women (49.4% vs 47.6%, P=0.722) or among age groups (48.9% in <40 years, 50.8% in 40–60 years, 45.3% in >60 years; P for trend = 0.453) (Table 2). The overall prevalence of colorectal polyps was 33.0% (n=126) (Table 2, Figure 1). No significant sex difference was observed (33.3% in men vs. 32.7% in women; P=0.894). However, polyp prevalence increased significantly with age: 19.1% in participants <40 years, 33.5% in those 40–60 years, and 36.7% in those >60 years (P for trend=0.049) (Table 2).

Table 1. Demographic and clinical characteristics of participants (n=382)

Variables		No. (%)
Age (y)	Mean±SD	56.93±13.51
	<40	47(12.3)
	40–60	185(48.4)
	60	150(39.3)
Sex	Male	174(45.5)
	Female	208(54.5)
Marital status	Single	24(6.3)
	Married	316(82.7)
	Divorced/widowed	42(11.0)
Education level	< High school	218(57.1)
	High school	128(33.5)
	University	36(9.4)
Residence	Urban	253(66.2)
	Rural	129(33.8)
BMI (kg/m²)	Mean±SD	25.59±4.46
	Underweight	8(2.1)
	Normal	199(52.1)
	Overweight	117(30.6)
	Obese	58(15.2)
Smoking		28(7.3)
Alcohol use		5(1.3)
Chronic diseases		204(53.4)
Family history of cancer		26(6.8)

BMI: Body mass index.

Colorectal polyp characteristics

Among participants, 67.0% (n=256) had no polyps, while 28.0% (n=107) had one, 2.9% (n=11) had two, and 1.3% (n=5) had three polyps. A minority (0.8%, n=3) presented with four or more polyps (Table 3, Figure 1). In total, 158 polyps were detected among all participants. Regarding size, 52.5% (n=83) of polyps were classified as diminutive (<0.5 cm), 38.6% (n=61) were small (0.5–1 cm), and 8.9% (n=14) were larger than 1 cm. Anatomical distribution among participants showed the most frequent sites as descending colon (8.1%), transverse colon (7.9%), rectum (7.9%), and sigmoid colon (7.1%). The ascending colon, cecum, and ileum had

Table 2. Prevalence of *H. pylori* Infection and colorectal polyps by sex and age

Variables		H. pylori n/N	H. pylori (%)	P	Polyp n/N	Polyp (%)	P
Total		185/382	48.4		126/382	33.0	
Sex	Male	86/174	49.4	0.722	58/174	33.3	0.894
	Female	99/208	47.6		68/208	32.7	
Age (y)				0.453‡			0.049‡
<40		23/47	48.9		9/47	19.1	
40–60		94/185	50.8	0.606†	62/185	33.5	0.082†
>60		68/150	45.3		55/150	36.7	

n: Number of individuals with *H. pylori* infection in each category; N: Total number of individuals in each category.

†Chi-square test, ‡Cochran-Armitage test for trend, P<0.05 is considered as a significant level.

lower frequencies, 6.3%, 3.7%, and 0.5%, respectively. Histopathologically, 22.8% (n=87) were adenomatous (tubular adenomas), and 10.2% (n=39) were hyperplastic polyps (Table 3).

Association between *H. pylori* infection and colorectal polyps

The prevalence of colorectal polyps among those with and without *H. pylori* infection was 34.1% (63/185) and 32.0% (63/197), respectively, with no statistically significant difference (P=0.667) (Table 4, Figure 2). In unadjusted logistic regression (Model 1), *H. pylori* infection was not significantly associated with colorectal polyps (OR=1.10; 95% CI, 0.72%, 1.68%; P=0.667). After adjusting for age and sex (Model 2), the association remained non-significant (OR=1.14; 95% CI, 0.74%, 1.75%; P=0.555). In the fully adjusted model (Model 3), which included demographic and clinical variables (marital status, education, residence, BMI, smoking, alcohol use, comorbidities, and family history of cancer), *H. pylori* infection still did not demonstrate a statistically significant association with colorectal polyps (adjusted OR=1.19; 95% CI, 0.76%, 1.87%; P=0.435) (Table 4).

Discussion

In this cross-sectional study conducted on individuals undergoing endoscopy and colonoscopy at a hospital in northern Iran, we found no statistically significant association between *H. pylori* infection and the presence of colorectal polyps, even after adjustment for potential confounders such as age, sex, lifestyle, and clinical factors. Our study contributed to this ongoing debate by providing evidence from a Middle Eastern population with high endemic rates of *H.pylori* infection. It is noteworthy that the overall prevalence of colorectal polyps in our study (33%) was consistent with other regional studies [23, 24], and that polyp prevalence increased significantly with age, reinforcing age as a principal determinant in colorectal neoplasia development. These findings validated current guidelines supporting age-based colonoscopy screening [25–27].

Although the prevalence of colorectal polyps was marginally higher among those infected with *H. pylori* (34.1% vs 32.0%), logistic regression analysis did not support a significant association. Our findings aligned with those of Liou et al. in Taiwan, who also reported no significant correlation between *H. pylori* infection and the presence of colorectal polyps in a large population-based study [13]. Similarly, Siddheshwar et al. also reported no significant association between *H. pylori* infection and the presence of colorectal polyps or cancer, suggesting that geographic, genetic, or microbial diversity might moderate the proposed relationship [28]. These consistent results across diverse populations suggest that *H. pylori* may not universally act as a risk factor for colorectal neoplasia, especially when polyp characteristics were not stratified.

In contrast, a growing number of studies report a significant association between *H. pylori* infection and colorectal polyps, particularly adenomatous and advanced lesions. A meta-analysis by Lu et al. including over 320,000 participants, and a cohort study by Basmacı et al. analyzing 4,561 patients, both demonstrated higher rates of *H. pylori* infection in individuals with total, ad-

Table 3. Distribution of colorectal polyp characteristics among participants (n=382)

Polyp Characteristics		No. (%)
Polyp count	0	256(67.0)
	1	107(28.0)
	2	11(2.9)
	3	5(1.3)
	≥4	3(0.8)
Polyp size (cm)	Diminutive (<0.5)	83(52.5)
	Small (0.5–1)	61(38.6)
	Large (>1)	14(8.9)
Polyp location	Descending colon	31(8.1)
	Transverse colon	30(7.9)
	Rectum	30(7.9)
	Sigmoid colon	27(7.1)
	Ascending colon	24(6.3)
	Cecum	14(3.7)
	Ileum	2(0.5)
	Splenic/Hepatic flexures	0
Polyp pathology†	Adenomatous (tubular)	87(22.8)
	Hyperplastic	39(10.2)

†Percentages are calculated based on individuals.

enomatous, or high-risk polyps (ORs ranging from 1.67 to 2.06), particularly for multiple, large, or high-risk histological types [12, 29]. These findings supported the hypothesis that *H. pylori* may contribute to colorectal carcinogenesis, especially in relation to advanced or high-risk polyps. The lack of association in our study might have been partly attributed to the relatively small number of high-risk polyps and absence of detailed subgroup analysis by polyp histology or multiplicity.

The potential biological mechanisms supporting a link between *H. pylori* infection and colorectal neoplasia are multifaceted. Chronic *H. pylori* infection can lead to sustained systemic inflammation and increased levels of proinflammatory cytokines such as IL-6, IL-8, and TNF-α, which can influence epithelial cell proliferation and DNA damage beyond the gastric environment [30, 31]. Additionally, infection-associated hypochlorhydria may alter gut microbial diversity and composition, promoting dysbiosis and the production of carcinogenic N-nitroso compounds in the colon [30]. The CagA virulence factor of *H. pylori* has also been implicated in oncogenic pathways involving β-catenin activation and E-cadherin disruption, mechanisms relevant to colorectal epithelial transformation [32]. These mechanisms may explain why some studies report stronger associations in individuals with advanced or multiple polyps, as seen in the study by Basmaci et al. or in populations with higher inflammatory burdens [29].

A local study by Teimoorian et al. also supported the positive association between *H. pylori* and colorectal neoplasia, reporting significantly elevated IgG and IgA antibody titers against *H. pylori* in patients with adenomatous polyps and CRC compared to healthy controls [33]. Their findings, although based on serologic rather than histologic or stool antigen methods, suggested a possible systemic immune response that might be as-

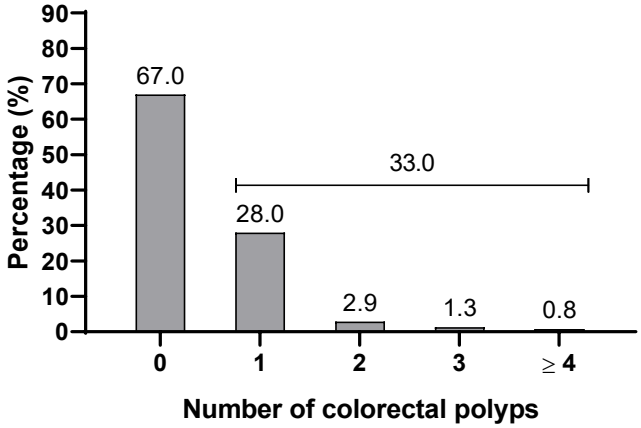

Figure 1. Frequency distribution of the number of colorectal polyps in all participants

sociated with colonic mucosal changes. This contrasted with our findings, where histopathological confirmation of infection did not show a statistically significant relationship. Such discrepancies highlight the influence of diagnostic modality, immune response heterogeneity, and possibly strain-specific virulence in shaping the observed outcomes.

Conclusion

In conclusion, our study found no significant association between *H. pylori* infection and colorectal polyps in a northern Iranian population. However, this does not exclude a potential effect in subgroups, advanced lesions, or in association with specific *H. pylori* strains. Given the complexity of this relationship, further well-designed, multi-parameter studies are warranted to elucidate the role of *H. pylori* in colorectal tumorigenesis. A strength of this study was the use of standardized colonoscopy and endoscopy protocols, *H. pylori* detection using histology, and multivariable adjustment for potential confounders. However, a limitation of this study was the lack of detailed subgroup analysis by polyp histology, size, and multiplicity. Adenomatous and hyperplastic polyps have different risk profiles, and the omission of such stratification may mask potential subtype-specific associations with *H. pylori* infection. Moreover, as a single-center study with convenience sampling, generalizability may have been limited. The absence of virulence profiling (e.g. CagA, VacA) and microbiome assessment was also a notable limitation. Future studies should aim for multicenter designs with larger sample sizes and standardized criteria for polyp classification.

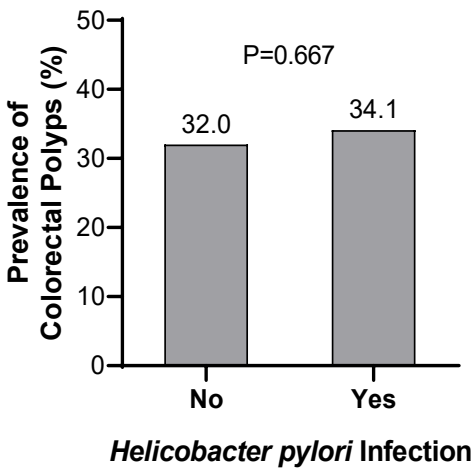

Figure 2. Prevalence of colorectal polyps according to *H. pylori* infection in all participants

P is based on the chi-square test.

Table 4. Association between *H. pylori* Infection and colorectal polyps (logistic regression)

H. pylori Status	n/N (%)	Model 1 (Unadjusted)		Model 2		Model 3	
	Prevalence of Colorectal Polyps	OR (95% CI)	P	OR (95% CI)	P	OR (95% CI)	P
No	63/197 (32.0)	Ref		Ref		Ref	
Yes	63/185 (34.1)	1.10 (0.72–1.68)	0.667	1.14 (0.74–1.75)	0.555	1.19 (0.76–1.87)	0.435

n: Number of individuals with colorectal polyps in each category; N: Total number of individuals in each category; OR: Odds ratio; CI: Confidence interval; Model 1: Crude; Model 2: Adjusted for age and sex; Model 3: Fully adjusted for age, sex, marital status, education, residence, BMI, smoking, alcohol use, chronic illness, and family history of cancer.

P<0.05 is considered as a significant level.

Incorporating *H. pylori* strain typing and microbiome profiling could enhance understanding of host pathogen interactions. Longitudinal cohorts are needed to clarify temporal relationships between infection and neoplastic progression.

Ethical Considerations

Compliance with ethical guidelines

This study was approved by the Ethics Committee of Guilan University of Medical Sciences, Rasht, Iran (Code: IR.GUMS.REC.1404.086), and all participants were fully informed about the aim of the research study and the voluntary nature of participation. This study was conducted in accordance with the ethical principles outlined in the Declaration of Helsinki.

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Authors' contributions

Conceptualization: Kourosh Mojtahedi, Fariborz Mansour-Ghanaei, Alieh Jedinia, and Farahnaz Joukar; Data curation: Kourosh Mojtahedi, Fariborz Mansour-Ghanaei, Alieh Jedinia, Farahnaz Joukar, Mehrnaz Asgharnezhad, and Abed Pourseyedian; Formal analysis: Alieh Jedinia, Farahnaz Joukar, Mehrnaz Asgharnezhad, and Saman Maroufizadeh; Investigation: Kourosh Mojtahedi, Fariborz Mansour-Ghanaei, Alieh Jedinia, Farahnaz Joukar, Saman Maroufizadeh, Mehrnaz Asgharnezhad, and Abed Pourseyedian; Methodology: Kourosh Mojtahedi, Fariborz Mansour-Ghanaei, Alieh Jedinia, Farahnaz Joukar, Mehrnaz Asgharnezhad, and Abed Pourseyedian; Project administration: Kourosh Mojtahedi, Fariborz Mansour-Ghanaei, and Alieh Jedinia; Resources: Kourosh Mojtahedi, Fariborz Mansour-Ghanaei, and Alieh Jedinia; Software: Saman Maroufizadeh, Alieh Jedinia, Farahnaz Joukar, and Mehrnaz Asgharnezhad; Supervision: Kourosh Mojtahedi, Fariborz Mansour-Ghanaei, Alieh Jedinia, and Farahnaz Joukar; Validation: Kourosh Mojtahedi, Fariborz Mansour-Ghanaei, Alieh Jedinia, Farahnaz Joukar, and Saman Maroufizadeh; Visualization: Kourosh Mojtahedi, Fariborz Mansour-Ghanaei, Alieh Jedinia, Farahnaz Joukar, Saman Maroufizadeh, Mehrnaz Asgharnezhad, and Abed Pourseyedian; Writing the original draft: Kourosh Mojtahedi, Fariborz Mansour-Ghanaei, Alieh Jedinia, Farahnaz Joukar, Saman Maroufizadeh, Mehrnaz Asgharnezhad, and Abed Pourseyedian; Review and editing: Kourosh Mojtahedi, Fariborz Mansour-Ghanaei, Alieh Jedinia, Farahnaz Joukar, Saman Maroufizadeh, Mehrnaz Asgharnezhad, and Abed Pourseyedian.

Conflict of interest

The authors declared no conflict of interest.

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