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Retrospective Study

Association of *Helicobacter pylori* infection with colorectal polyps and malignancy in China

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Abstract

BACKGROUND

Gastric *Helicobacter pylori* (*H. pylori*) infection is related to chronic gastritis, gastroduodenal ulcer, and gastric malignancies; whether this infection is related to colorectal polyps and colorectal cancer (CRC), remains debatable.

AIM

To investigate the relationship between gastric *H. pylori* infection and the risk of colorectal polyps and CRC.

METHODS

We retrospectively analyzed 3872 patients with colorectal polyps who underwent colonoscopy and pathological diagnosis. We also analyzed 304 patients with primary CRC. The characteristics of these patients were compared with those of the control group, which included 2362 patients with the normal intestinal mucosa. All subjects completed a ¹⁴C-urea breath test, bidirectional gastrointestinal endoscopy, and a biopsy on the same day. Data on the number, size, location, and pathology of the polyps, the location, and pathology of the CRC, the detection of *H. pylori*, and the incidence of *H. pylori*-associated atrophic gastritis or intestinal metaplasia were obtained. A logistic regression model was used to analyze the relationship between gastric infection due to *H. pylori*, and the incidence of colorectal polyps and CRC.

RESULTS

The prevalence of *H. pylori* infection was higher in the multiple polyps group than in the solitary polyp group and the control group [95% confidence interval

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(CI) = 1.02-1.31, $P = 0.03$; 95%CI: 2.12-2.74, $P < 0.001$]. The patients with adenomatous polyps had a higher incidence of *H. pylori* infection than patients with non-adenomatous polyps [59.95% vs 51.75%, adjusted odds ratio (OR) = 1.41, 95%CI: 1.24-1.60, $P < 0.01$]. Patients with *H. pylori*-associated atrophic gastritis or intestinal metaplasia were at high risk of CRC (adjusted OR = 3.46, 95%CI: 2.63-4.55, $P < 0.01$; adjusted OR = 4.86, 95%CI: 3.22-7.34, $P < 0.01$, respectively). The size and location of the polyps, the histopathological characteristics and the location of CRC were not related to *H. pylori* infection.

CONCLUSION

Our study demonstrates that the incidence of gastric *H. pylori* infection and *H. pylori*-associated atrophic gastritis or intestinal metaplasia elevates the risk of colorectal polyps and CRC.

Key words: *Helicobacter pylori*; Atrophic gastritis; Intestinal metaplasia; Polyps; Colorectal cancer; Gastrointestinal neoplasms

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Core tip: This study investigated the association of gastric *Helicobacter pylori* (*H. pylori*) infection with the risk of colorectal polyps and colorectal cancer (CRC). The results indicated that patients with *H. pylori* infection were 2.19 and 3.05 times more likely to develop colorectal polyps and CRC, respectively, than those without *H. pylori* infection. The prevalence of *H. pylori* infection was higher in the patient group with multiple polyps and colorectal adenomas than in those with a solitary polyp and non-adenomatous polyps, respectively. Gastric *H. pylori* infection and *H. pylori*-associated atrophic gastritis or intestinal metaplasia elevated the risk of colorectal polyps and CRC. Therefore, earlier and frequent colonoscopy is necessary.

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INTRODUCTION

Colorectal cancer (CRC) accounted for 881000 deaths in 2018, representing 9.8% of deaths worldwide. The incidence rate of CRC has increased to the extent that 1.8 million new CRC cases were diagnosed in 2018, according to recent data by the World Health Organization^[1]. However, it is difficult to detect CRC early due to the lack of typical clinical symptoms, and 20% of patients who are asymptomatic at diagnosis may manifest with aggressive metastases^[2]. The appearance of colorectal polyps and malignancy is a complex process that involves a combination of dietary habits, smoking, drinking and other environmental factors, and genetic susceptibility^[3,4]. Colorectal polyps are abnormal protrusions on the surface of the large intestine^[5] that are either non-adenomatous or adenomatous polyps. Colorectal adenomatous polyps are considered to be the most important precancerous lesions, developing into CRC through the adenomatous cancer sequence^[6]. Therefore, early screening and detection of precancerous lesions can prevent the occurrence and development of CRC^[7].

Gastric *Helicobacter pylori* (*H. pylori*) infection is a global public health concern, which has infected approximately^[8] 50% of the world population, according to recent epidemiological studies^[9]. The chronic inflammatory state caused by *H. pylori* infection is closely related to the occurrence of gastric cancer. *H. pylori* is listed as a class I carcinogen by the World Health Organization International Agency for Cancer Research^[10] and is also involved in the tumorigenesis of extragastric target organs, such as lung cancer, and hepatocellular carcinoma^[11]. Previous studies have indicated that gastric *H. pylori* infection increased the risk of colorectal tumors^[12-16]. In contrast, however, several other reports have concluded that gastric *H. pylori* infection was not correlated with colorectal polyps or CRC^[17-21]. Thus, the results of different research studies in different regions vary significantly. Therefore, the present study

investigated the relationship between gastric *H. pylori* infection, colorectal polyps, and CRC in northwestern China. In addition, the study provides answers to questions regarding the necessity for colonoscopy screening in patients with gastric *H. pylori* infection.

MATERIALS AND METHODS

Study population

We reviewed the consecutive electronic medical records of patients who underwent gastroscopy and colonoscopy at a regional institution from January 2014 to January 2019. The inclusion criteria included: (1) Complete general information (including gender, age, ethnicity, past history, family history, *etc.*); (2) Age ≥ 18 years; (3) Patients who underwent bidirectional endoscopy (colonoscopy performed immediately after gastroscopy); (4) A clear pathological diagnosis of colorectal polyps or CRC; and (5) Detection of *H. pylori* infection. Exclusion criteria were as follows: (1) History of gastric cancer, peptic ulcer, and other malignant tumors; (2) Received antibiotics, NSAIDs, proton pump inhibitors or glucocorticoids in the past month; (3) Patients who underwent *H. pylori* eradication therapy previously, or radiation therapy, chemotherapy, and other specific treatment for tumors; (4) No total colonoscopy or biopsy; (5) Previous history of gastrointestinal surgery; (6) Presence of inflammatory bowel disease, familial adenoma, Gardner's syndrome (a disease that affects the incidence of CRC); (7) A history of severe systemic disease; (8) A family history of polyposis, and (9) Patients who underwent repeated hospitalizations and a history of endoscopic polyp therapy. All patients provided consent for the study.

The diagnosis of normal intestinal mucosa, colorectal polyps, CRC, atrophic gastritis, and intestinal metaplasia was mainly based on endoscopic manifestations and histopathological examinations. Pathological diagnoses required confirmation by two pathologists. Referring to the fourth national consensus report on *H. pylori* infection treatment in 2012^[22], *H. pylori* infection was defined as follows: Positive ¹⁴C-urea breath test and/or positive hematoxylin and eosin staining on gastric biopsies.

Study design

All subjects underwent complete colonoscopy and data regarding the location, size, shape, and number of polypoid lesions and the location of tumors were recorded. According to the size of the largest polyp, the patients with colorectal polyps were divided into those with a maximum diameter ≥ 1 cm and those with a maximum diameter ≤ 1 cm. According to the number of polyps, patients with a single polyp were included in the solitary polyp group, and those with ≥ 2 polyps were included in the multiple polyps group. Colorectal polyps and CRC were classified according to the location. The distal colorectum was defined as the anus to the splenic flexure, while the proximal colon was defined as the cecum to the splenic flexure, and patients with multiple lesions on both sides were defined as the whole colon. We divided *H. pylori*-related gastric disease into atrophy and intestinal metaplasia according to a method described previously^[23]. Colorectal polyps including non-adenomatous and adenomatous polyps were considered. The prevalence of gastric *H. pylori* infection in CRC patients, patients with colorectal polyps, and the control group were compared. In addition, the prevalence of atrophic gastritis or intestinal metaplasia with gastric *H. pylori* infection among CRC patients, colorectal polyp patients, and the control group was also compared.

Statistical analysis

SPSS 17.0 was used for statistical analysis. Data for continuous variables were expressed as mean \pm SD, and categorical data as a ratio or percentage. The *t*-test was used for data with a normal distribution. For comparisons among multiple groups of means (*e.g.*, sex, BMI), one-way ANOVA was used. If the hypothesis of homogeneity of variance was not satisfied, Welch's ANOVA was used. In addition, we used the χ^2 test to compare categorical variables. A logistic regression model was applied to estimate the correlation between *H. pylori* infection, colorectal polyps, and CRC. After adjusting for gender and age, we calculated the odds ratio (OR) value and 95% confidence interval (95% CI). All variables with *P* < 0.05 were considered statistically significant and remained in the final models.

RESULTS

Patient characteristics

In total, 6538 patients were enrolled in our study (Figure 1). The characteristics of the participants are outlined in Table 1. Of these patients, 3872 were confirmed to have colorectal polyps and 304 had CRC by colorectal biopsy, and the remaining 2362 patients who had no abnormalities on colonoscopy, no history of polypectomy, and no clinical symptoms were classified as controls. The patient group with colorectal polyps included 2189 men and 1683 women. Their mean age was 53.01 ± 12.60 years. The patient group with CRC comprised 167 men and 137 women with an average age of 61.26 ± 12.94 years. The control group comprised 1355 men and 1007 women with a mean age of 45.81 ± 13.44 years. There were significant differences in age among the three groups (Welch $F = 320.48$, $P < 0.001$). However, there were no significant differences in gender, BMI, smoking, drinking, or family history of CRC among the three groups ($P = 0.66$; $P = 0.06$; $P = 0.39$; $P = 0.28$; $P = 0.16$, respectively). All patients underwent gastroscopy on the day of colonoscopy. The main indications for performing gastroscopy and colonoscopy are shown in Table 2.

Correlation between *H. pylori* infection and colorectal polyps

The colonoscopy findings and pathological characteristics of colorectal polyps are shown in Table 3. The prevalence of *H. pylori* infection in the colorectal polyp group (2134/3872, 55.11%) was higher than that in the control group (890/2362, 37.68%; $P < 0.001$). Multivariate logistic regression demonstrated that the patients with positive *H. pylori* infection in the colorectal polyp group had a higher OR after age and gender adjustment (adjusted OR = 2.19, 95%CI: 1.96-2.44, $P < 0.001$). The prevalence of *H. pylori* infection was higher in the multiple polyps group than that in the solitary polyp group and the control group (adjusted OR = 1.15, 95%CI: 1.02-1.31, $P = 0.03$; adjusted OR = 2.41, 95%CI: 2.12-2.74, $P < 0.001$). The positive rate of *H. pylori* infection in the adenomatous polyp group (952/1588, 59.95%) was higher than that in the non-adenomatous polyp group (1182/2284, 51.75%, adjusted OR = 1.41, 95%CI = 1.24-1.60, $P < 0.001$). The same association was found between the adenomatous polyps and control groups (adjusted OR = 2.53, 95%CI: 2.20-2.89, $P < 0.001$). However, the polyp size and locations were not associated with *H. pylori* ($P = 0.26$; $P = 0.08$). According to the polyp location, the P value of the proximal colon was 0.18, the distal colorectal was 0.23, and the whole colon was 0.51. Furthermore, based on the status of *H. pylori* infection and histopathological findings of the gastroscopy specimens, we found that the incidence of *H. pylori*-related atrophic gastritis in the colorectal polyp group was 34.99% (1355/3872), which was higher than that in the control group (541/2362, 22.90%, $P < 0.001$) (Table 4). *H. pylori*-associated atrophic gastritis was significantly associated with colorectal polyps compared to that in the control group (adjusted OR = 5.42, 95%CI: 4.67-6.30, $P < 0.001$). The prevalence of *H. pylori*-related intestinal metaplasia in the patient group with colorectal polyps was 8.96% (347/3872), which was higher than that in the control group (105/2362, 4.45%) ($P < 0.001$). Overall, *H. pylori*-associated intestinal metaplasia status was positively associated with colorectal polyps (adjusted OR = 5.88, 95%CI: 4.60-7.52, $P < 0.001$).

Correlation between *H. pylori* infection and CRC

The prevalence of *H. pylori* infection in the CRC group (189/304, 62.17%) was higher than that in the control group (890/2362, 37.68%) ($P < 0.001$). After adjustment for age and gender, multivariate logistic regression demonstrated that the patients with positive *H. pylori* infection in the CRC group had a higher OR (adjusted OR = 3.05, 95%CI: 2.33-3.99, $P < 0.001$) and among 304 patients with CRC, 270 cases had adenocarcinoma, including 167 cases with *H. pylori* infection (61.85%); 11 cases had neuroendocrine tumors, including 7 cases with *H. pylori* infection (63.64%); 14 cases had intramucosal cancer, including 9 cases with *H. pylori* infection (64.29%); 9 cases had signet ring cell cancer, including 6 cases with *H. pylori* infection (66.67%). The histopathological findings of CRC were not related to *H. pylori* ($P > 0.05$). Moreover, the χ^2 test showed that CRC location was not correlated with *H. pylori* infection ($P = 0.62$). Compared to the control group, both *H. pylori*-related atrophic gastritis and intestinal metaplasia significantly increased the risk of CRC (adjusted OR = 3.46, 95%CI: 2.63-4.55, $P < 0.001$; adjusted OR = 4.86, 95%CI: 3.22-7.34, $P < 0.001$) (Table 2).

DISCUSSION

H. pylori infection plays an important role in the pathogenesis of gastrointestinal diseases^[24]. However, whether gastric *H. pylori* infection increases the risk of colorectal

Table 1 Baseline characteristics of the participants, *n* (%)

Parameter	Control group (<i>n</i> = 2362)	Colorectal polyps group (<i>n</i> = 3872)	CRC group (<i>n</i> = 304)	<i>F</i> / χ^2	<i>P</i> value
Age, mean \pm SD (yr)	45.81 \pm 13.44	53.01 \pm 12.60	61.26 \pm 12.94	320.48	< 0.001
Male/female	1355/1007	2189/1683	167/137	0.85	0.66
BMI, mean \pm SD (kg/m ²)	24.78 \pm 3.83	24.85 \pm 3.82	25.34 \pm 3.66	2.87	0.06
Smoking	677 (28.66)	1048 (27.07)	94 (30.92)	1.89	0.39
Drinking	271 (11.47)	501 (12.94)	41 (13.49)	2.52	0.28
Family history of CRC	124 (5.25)	243 (6.28)	23 (7.57)	3.73	0.16

BMI: Body mass index; CRC: Colorectal cancer.

polyps and CRC has been debated in various studies. This could be related to differences in the dietary habits of the study population, the patient population susceptible *H. pylori*, study sample size, and other factors. The *H. pylori* infection rate in China is more than 50%^[25]. The relationship between gastric *H. pylori* infection and colorectal polyps and CRC in Northwest China is unclear; thus, we conducted this study. Some studies have shown that gender and age are related to *H. pylori* infection, colorectal polyps, and CRC^[12,26]. Therefore, we conducted a multifactorial logistic regression analysis after adjusting for those factors. No significant differences in BMI, smoking, drinking, or family history of CRC among the subjects were found.

The results showed that the increase in *H. pylori* infection rate was positively correlated with the increase in colonic polyp incidence, polyp number, and malignancy, suggesting that *H. pylori* infection might be a risk factor for colorectal polyps and tumors. Patients with *H. pylori* infection are 2.19 times more likely to develop colorectal polyps and 3.05 times more likely to develop CRC than those who do not have *H. pylori* infection. Additionally, we found that the incidence of *H. pylori* infection coexisting with atrophic gastritis or intestinal metaplasia was higher in patients with colorectal polyps and CRC than in the control group. There was a significant correlation between *H. pylori*-associated gastropathy and colorectal adenomatous polyps or CRC. These results are consistent with those of previous studies^[26,27]. A large-scale population-based study by Sonnenberg *et al*^[28] also supports this argument. However, another study found no significant relationship between *H. pylori* infection and CRC. This may have been due to the broader age range and the limited number of patients^[29].

There are a few plausible theories to explain the distribution of colorectal polyps and CRC and its association with *H. pylori* infection. Hong *et al*^[30] found that gastric *H. pylori* infection was positively associated with an increased risk of proximal colorectal adenomatous polyps. Zhang *et al*^[31] found that *H. pylori* infection mainly increased the risk of distal CRC. However, our study illustrates that gastric *H. pylori* infection could increase the risk in both proximal and distal colorectal neoplasms, which was consistent with the study by Inoue *et al*^[32,33]. *H. pylori* infection causes microbiological changes in the digestive tract, increases the production of bile acids, causes DNA damage and activation, plays an important role in the proximal colonic mucosa, and increases the risk of proximal colonic polyps and malignancy^[34]. Preclinical models have demonstrated that increased gastrin secretion caused by *H. pylori* infection has a mitogenic effect and selectively acts on the distal colon, thereby increasing the risk of distal colon polyps and malignancy^[35]. These mechanisms may work synergistically. In addition, in our study, no significant differences were found among CRC patients with different pathological types and the prevalence of *H. pylori* infection. This may be because the most prevalent type of CRC is adenocarcinoma; however, studies with a larger sample size are needed to confirm this hypothesis.

The mechanism by which gastric *H. pylori* infection increases the incidence of colorectal polyps and CRC is not clear. Some studies have shown that gastrin gene expression is up-regulated in both colorectal polyps and CRC^[36,37], and *H. pylori* infection can cause hypergastrinemia. Gastrin acts on gastrointestinal epithelial cells and can promote the formation of COX-2, which affects the occurrence, development, invasion, and metastasis of colorectal neoplasia^[38]. Gastrin can also induce colonic mucosal cell proliferation to promote the development of CRC^[39]. Moreover, from the perspective of gastrointestinal microecology, chronic gastritis caused by long-term *H. pylori* infection can lead to massive glandular atrophy and decreased gastric acid secretion. Low gastric acid may adversely affect the intestinal flora, cause bacterial overgrowth, colonic disorders, and colorectal carcinogenesis^[40,41]. In addition, *H. pylori* infection may cause damage to colorectal epithelium through the chronic

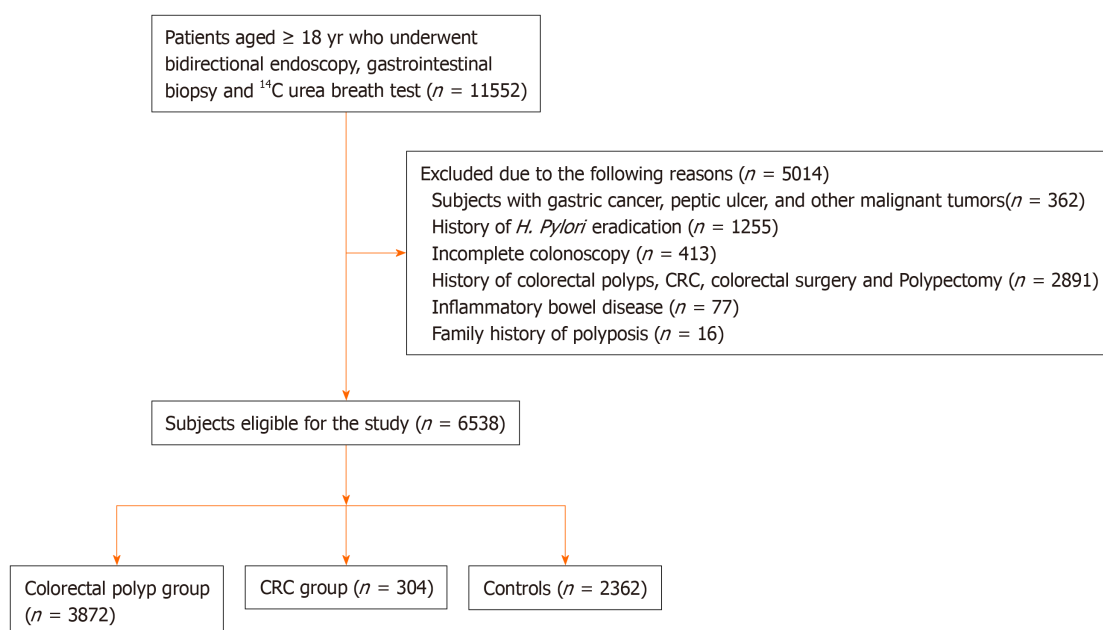


Figure 1 Flowchart of the study participants. Controls were patients with neither colorectal polyps nor colorectal cancer. CRC: Colorectal cancer; *H. pylori*: *Helicobacter pylori*.

inflammatory response mediated by inflammatory factors such as interleukin-8^[42].

The early diagnosis of CRC is relatively difficult. During the development of CRC, the normal mucosa develops into an adenoma and then to adenocarcinoma. This process provides opportunities for early detection and intervention of CRC. Early diagnosis and resection of colonic polyps can reduce the morbidity and mortality of CRC^[43]. Further studies with regard to pathogenic mechanisms should be continued, which can help to develop relevant prevention and early detection strategies.

Some studies found that only the current situation of *H. pylori* infection could stimulate the immune response, thus inducing or perpetuating chronic inflammation of the gastrointestinal tract^[44,45]. The strength of our study was that histopathological results and the ¹⁴C-urea breath test were used to determine *H. pylori*. A histopathological examination has high specificity and sensitivity in the diagnosis of gastric pathological changes and *H. pylori* infection. However, histopathological examination and the ¹⁴C-urea breath test can only diagnose the current infection of *H. pylori*, compared with the serological tests, which do not distinguish current or past infections. Therefore, our study explains the current relationship between *H. pylori* infection, colorectal polyps, and CRC more accurately. In addition, our sample size was relatively large, which is an advantage of this study.

Our study also has several limitations. First, we did not consider the possible effect of the duration of *H. pylori* infection on colorectal polyps and CRC. Secondly, we did not consider other confounding factors, such as constipation, eating habits, and metabolic syndrome. Thirdly, this was a single-center study. More investigation through prospective multicenter studies with large sample sizes should be conducted.

In conclusion, this study showed that gastric *H. pylori* infection and *H. pylori*-related gastric atrophic or intestinal metaplasia increased the risk of colorectal polyps and CRC. Early colonoscopy screening and surveillance is necessary to reduce the risk of colonic polyps and CRC in patients with *H. pylori* infection. Further investigation is required to understand whether the eradication of gastric *H. pylori* can reduce the occurrence of colorectal polyps and CRC.

Table 2 The main indications for gastroscopy and colonoscopy

Indication for gastroscopy and colonoscopy ¹	Gastroscopy (%)	Colonoscopy (%)
Abdominal pain	35.23	27.95
Diarrhea	10.12	20.03
Constipation		8.05
Gastrointestinal bleeding	12.07	14.43
Gastroesophageal reflux disease	16.51	
Dyspepsia	19.22	
Nausea/vomiting	10.16	
Dysphagia	6.2	
Weight loss	7.38	5.24
Anemia	6.47	6.32
Other	6.35	3.26
Gastric cancer/CRC screening	28.19	26.92
Polyp surveillance	7.94	9.21

¹Individual patients could present with more than one indication. CRC: Colorectal cancer.

Table 3 The association between *Helicobacter pylori* infection, colorectal polyps, and colorectal cancer, compared with the control group

Parameter	<i>H. pylori</i> positive [n (%)]	<i>H. pylori</i> negative [n (%)]	Adjusted OR (95%CI)	P value
Control group	890 (37.68)	1472 (62.32)	1	
Colorectal polyp group	2134 (55.11)	1738 (44.89)	2.19 (1.96-2.44)	< 0.01
Polyp size				
≥ 1 cm	529 (56.70)	404 (43.30)	2.33 (1.98-2.74)	< 0.01
< 1 cm	1605 (54.61)	1334 (45.39)	2.15 (1.92-2.41)	< 0.01
Polyp number				
Solitary	942 (53.34)	824 (46.66)	1.98 (1.74-2.25)	< 0.01
Multiple	1192 (56.60)	914 (43.40)	2.41 (2.12-2.74)	< 0.01
Polyp histology				
Adenomatous polyps	952 (59.95)	636 (40.05)	2.53 (2.20-2.89)	< 0.01
Non-adenomatous polyps	1182 (51.75)	1102 (48.25)	2.00 (1.77-2.26)	< 0.01
Polyp location				
Proximal colon	732 (57.28)	546 (42.72)	2.49 (2.15-2.88)	< 0.01
Distal colorectal	1160 (53.51)	1008 (46.49)	1.98 (1.75-2.24)	< 0.01
Whole colon	242 (56.81)	184 (43.19)	2.37 (1.92-2.94)	< 0.01
CRC group	189 (62.17)	115 (37.83)	3.05 (2.33-3.99)	< 0.01
CRC location				
Proximal colon	65 (67.01)	32 (32.99)	3.73 (2.39-5.82)	< 0.01
Distal colorectal	124 (59.90)	83 (40.10)	2.79 (2.04-3.81)	< 0.01

CRC: Colorectal cancer; *H. pylori*: *Helicobacter pylori*; CI: Confidence interval; OR: Odds ratio.

Table 4 The association of *Helicobacter pylori*-associated atrophic gastritis or intestinal metaplasia with colorectal polyps and colorectal cancer, compared with the control group

	<i>H. pylori</i> - associated atrophic gastritis [<i>n</i> (%)]	Adjusted OR (95%CI)	<i>P</i> value	<i>H. pylori</i> - associated intestinal metaplasia [<i>n</i> (%)]	Adjusted OR (95%CI)	<i>P</i> value
Control group	541 (22.90)	1		105 (4.45)	1	
Colorectal polyp group	1355 (34.99)	5.42 (4.67-6.30)	< 0.01	347 (8.96)	5.88 (4.60-7.52)	< 0.01
CRC group	144 (47.37)	3.46 (2.63-4.55)	< 0.01	55 (18.09)	4.86 (3.22-7.34)	< 0.01

CRC: Colorectal cancer; *H. pylori*: *Helicobacter pylori*; CI: Confidence interval; OR: Odds ratio.

ARTICLE HIGHLIGHTS

Research background

Gastric *Helicobacter pylori* (*H. pylori*) infection is a global public health problem. It is associated with chronic gastritis, gastroduodenal ulcer and gastric malignancies. The relationship between *H. pylori* infection and the risk of colorectal polyps and colorectal cancer (CRC) has also received extensive attention in recent years.

Research motivation

There is still no clear conclusion regarding the relationship between gastric *H. pylori* infection and the risk of colorectal polyps and CRC.

Research objectives

Our main purpose was to investigate the correlation between gastric *H. pylori* infection and the risk of colorectal polyps and CRC, which is essential for the early screening and detection of colorectal precancerous lesions.

Research methods

A retrospective analysis of 6538 patients who underwent colonoscopy was conducted. The patients were divided into three groups: The CRC group, colorectal polyps group, and the control group. All subjects completed a ¹⁴C-urea breath test, bidirectional gastrointestinal endoscopy, and a biopsy on the same day. The characteristics of gastrointestinal endoscopy, pathology of gastritis, polyps and CRC, and the detection of *H. pylori* in the three groups were analyzed.

Research results

Patients with *H. pylori* infection were 2.19 times more likely to develop colorectal polyps and 3.05 times more likely to develop CRC than those who did not have *H. pylori* infection. The prevalence of *H. pylori* infection was higher in the multiple polyps group than in the solitary polyp group, and was also higher in the adenomatous polyps group than in the non-adenomatous polyps group. Additionally, we found that the incidence of *H. pylori* infection coexisting with atrophic gastritis or intestinal metaplasia was higher in patients with colorectal polyps and CRC than in the control group. The size and location of polyps, the histopathological characteristics and the location of CRC were not related to *H. pylori* infection.

Research conclusions

The incidence of colonic polyps and CRC in patients with gastric *H. pylori* infection and *H. pylori*-associated atrophic gastritis or intestinal metaplasia was significantly higher than that in the normal population. Early and frequent colonoscopy is necessary to reduce the risk of colonic polyps and CRC in patients with *H. pylori* infection. The mechanism by which gastric *H. pylori* infection increases the incidence of colorectal polyps and CRC should be further studied.

Research perspectives

This study demonstrates that early colonoscopy screening and surveillance are necessary to reduce the risk of colonic polyps and CRC in patients with *H. pylori* infection. The future direction of research is to evaluate whether the eradication of gastric *H. pylori* can reduce the occurrence of colorectal polyps and CRC. Large-scale and long-term follow-up investigations are needed.

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