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ABOUT COVER

Editorial Board of *World Journal of Gastroenterology*, Dr. Angelo Zambam de Mattos is a Professor of Medicine – Gastroenterology at the Federal University of Health Sciences of Porto Alegre (UFCSPA), where he is also a permanent faculty member of the Graduate Program in Medicine: Hepatology (the only Brazilian graduate program specialized specifically in Hepatology). His research focuses on cirrhosis and its complications, culminating in > 50 academic papers. He also carries out clinical work at Irmandade Santa Casa de Misericórdia of Porto Alegre, one of the largest hospital complexes in southern Brazil. Prof. Mattos received his Medical degree in 2005, Master's degree in 2012 and PhD in 2015, all from UFCSPA. He is a member of the Brazilian Federation of Gastroenterology, Brazilian Association of Hepatology, and Brazilian Association of Digestive Endoscopy, and he is past president of the Gastroenterology Association of Rio Grande do Sul, Brazil (2017-2018). (L-Editor: Filipodia)

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ORIGINAL ARTICLE

Retrospective Study

Helicobacter pylori infection with atrophic gastritis: An independent risk factor for colorectal adenomas

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Author contributions: Chen QF and Zhou XD contributed equally to this work; Chen QF, Zhou XD, Lin WH, Wang D and Wu JS designed the study; Chen QF, Zhou XD, Feng XZ and Wang N collected data; Chen QF, Zhou XD and Fang DH did the statistical analyses; Chen QF, Zhou XD, Zhang EG and Lin CJ reviewed the results, interpreted data and wrote the manuscript; Lin WH and Wang D are both corresponding authors; All authors have made an intellectual contribution to the manuscript and approved the submission.

Institutional review board statement: The investigation conforms to the principles outlined in the Declaration of Helsinki. The study was approved by the ethical committee of The First Affiliated Hospital of Wenzhou Medical

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Abstract

BACKGROUND

The significance of *Helicobacter pylori* (*H. pylori*) infection and atrophic gastritis (AG) in the prevalence of colorectal adenomas has been examined in a limited number of studies. However, these studies reported disputed conclusions.

AIM

To investigate whether *H. pylori* infection, AG, and *H. pylori*-related AG increase the risk of colorectal adenomas.

METHODS

This retrospective cross-sectional study included 6018 health-check individuals. The relevant data for physical examination, laboratory testing, ^{13}C-urea breath testing, gastroscopy, colonoscopy and histopathological examination of gastric and colorectal biopsies were recorded. Univariate and multivariate logistic regression analyses were performed to determine the association between *H. pylori*-related AG and colorectal adenomas.

RESULTS

Overall, 1012 subjects (16.8%) were diagnosed with colorectal adenomas, of whom 143 (2.4%) had advanced adenomas. Among the enrolled patients, the prevalence of *H. pylori* infection and AG was observed as 49.5% (2981/6018) and 10.0% (602/6018), respectively. Subjects with *H. pylori* infection had an elevated risk of colorectal adenomas (adjusted odds ratio [OR] of 1.220, 95% confidence interval

University Ethical Committee.

Informed consent statement: Patients were not required to give informed consent to the study because the analysis used anonymous clinical data that were obtained after each patient agreed to examination by verbal consent. Individuals can't be identified according to the data presented.

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(CI): 1.053-1.413, $P = 0.008$) but no increased risk of advance adenomas (adjusted OR = 1.303, 95%CI: 0.922-1.842, $P = 0.134$). AG was significantly correlated to an increased risk of colorectal adenomas (unadjusted OR = 1.668, 95%CI: 1.352-2.059, $P < 0.001$; adjusted OR = 1.237, 95%CI: 0.988-1.549, $P = 0.064$). *H. pylori* infection accompanied by AG was significantly associated with an increased risk of adenomas (adjusted OR = 1.491, 95%CI: 1.103-2.015, $P = 0.009$) and advanced adenomas (adjusted OR = 1.910, 95%CI: 1.022-3.572, $P = 0.043$).

CONCLUSION

H. pylori-related AG was associated with a high risk of colorectal adenomas and advanced adenomas in Chinese individuals.

Key Words: *Helicobacter pylori*; Gastritis; Atrophy; Adenomas; Colorectal; Health-check

Core Tip: The relationship among *Helicobacter pylori* (*H. pylori*), atrophic gastritis (AG), and colorectal adenomas has been inconclusive. We conducted this retrospective study on 6018 health-check individuals and observed that *H. pylori*-related AG is an independent risk factor for colorectal adenomas in Chinese individuals. Clinically, rigorous colonoscopy screening and monitoring may be necessary for individuals with *H. pylori*-positive AG.

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INTRODUCTION

Colorectal cancer is one of the most common human malignancies worldwide, and the fifth common cause of cancer death in China[1]. Due to genetic mutations, colorectal adenomas may develop into carcinoma[2,3]. Common risk factors, such as age, male gender, nonalcoholic fatty liver disease, metabolic syndrome, family history, smoking, alcohol consumption, diet and lifestyle, contribute to the development of colorectal neoplasms[4,5].

Helicobacter pylori (*H. pylori*) is a gram-negative, microaerophilic bacterium generally found in the stomach[6]. *H. pylori* infection is associated with the development of gastric cancer[7]. In addition to its well-known association with gastric adenocarcinoma, *H. pylori* is associated with numerous extragastric malignancies[8,9]. Inconsistent conclusions of the relationship between *H. pylori* infection and colorectal neoplasia were presented in previous studies. In the early years, *H. pylori* infection had been confirmed as a risk factor for colorectal neoplasm[10-14]. However, the association between *H. pylori* infection and development of colorectal neoplasia remains unclear in recent studies[15].

Gastric mucosal atrophy is a typical symptom of atrophic gastritis (AG). AG in 8.1% of patients per year results from a chronic *H. pylori* infection with a ten-fold increased risk[16,17]. It is well established that gastric cancer and/or adenomas are associated with higher rates of colorectal cancer. In addition, precancerous lesions such as dysplasia or AG are important risk factors for gastric adenomas and gastric cancer[18,19]. However, only limited studies have investigated the association between AG and colorectal neoplasia. One study reported that intestinal metaplasia, often accompanied by AG, was closely related to any type of colorectal neoplasia[13].

In contrast, another study showed that the presence of AG has insignificantly increased the risk of colon cancer[20]. In addition, a recent study showed a significant association between colorectal neoplasm and AG, which was diagnosed by Kimura and Takemoto criteria. However, this study did not have the criteria for a histologic diagnosis[21]. The relationship between AG and colorectal neoplasia, especially that between *H. pylori*-related AG and colorectal neoplasia, is still controversial.

Thus, the aim was to assess the relationship between colorectal adenomas and *H. pylori*-related AG based on the histologic diagnosis.

MATERIALS AND METHODS

Eligible subjects

This retrospective study analyzed records between August 2014 and August 2017 that were extracted from the Medical and Health Care Center at The First Affiliated Hospital of Wenzhou Medical University. Relevant information was obtained *via* a survey, utilizing a standard relevant questionnaire. Out of these 13400 individuals, 6086 individuals aged 30 years and older underwent a gastroscopy, colonoscopy, ^{13}C-urea breath test and related pathological examination. Exclusion criteria were: A previous history of *H. pylori* eradication therapy; incomplete colonoscopy; polyp resection; inflammatory bowel disease; and gastrointestinal cancers. Finally, the data of 6018 individuals were included in our analysis. The investigation conforms to the principles outlined in the Declaration of Helsinki. The study was approved by the ethical committee of The First Affiliated Hospital of Wenzhou Medical University Ethical Committee

Data collection

Baseline characteristics, including age, gender, smoking, alcohol consumption, previous medical history and family history, were obtained from the standard questionnaires. Physical parameters and laboratory assays, including body mass index (BMI), systolic blood pressure (SBP), diastolic blood pressure (DBP), fasting blood glucose (FBG), total cholesterol (TC), triglyceride (TG), low-density lipoprotein (LDL), and high-density lipoprotein (HDL) and were collected and recorded from reports of physical examination. All blood samples were drawn from antecubital vein sampling following an overnight fast. The tests for physical parameter measurements were operated by trained nurses.

Diagnostic criteria

H. pylori (HP) infection was diagnosed by the ^{13}C-urea breath test or a histological diagnosis of biopsied stomach specimens. All enrolled subjects were divided into HP (+) group and HP (-) group depending on the above check mentions. Also, subjects were divided into AG (+) group and AG (-) group depending on the histopathological results of the gastric mucosa. For further subgroup analysis, subjects were divided into the nonpolyp group, the nonadenomatous polyp group (including inflammatory polyps and hyperplastic polyps) and the adenoma group based on the results from colorectal biopsies. Advanced colorectal adenoma was diagnosed by an adenoma with a diameter of ≥ 10 mm, a significant villous component, high-grade dysplasia or any combination thereof[21]. Additionally, the size of the polyps was divided into two groups: 0-9 mm and 10 mm +. While the number of polyps was divided into two groups: One and two or more. Following full bowel preparation, GIF-H260 gastroscopy and CF-H260AI colonoscopy (OLYMPUS, Tokyo, Japan) were performed in all eligible subjects. The surgeries were performed by experienced gastroenterologists with standard protocol followed. All examinations were performed in 2 d.

Statistical analysis

SPSS software (SPSS version 23.0 for Windows) was used for analysis. Continuous variables for nonadenomatous polyps, adenoma and advanced adenoma were presented as mean ± standard deviation. Pearson χ^2 tests for categorical variables and one-way analysis of variance or Kruskal–Wallis test for continuous variables were used to compare the baseline of the study population among the previously described groups. Associations of the risk factors with nonadenomatous polyps, adenoma and advanced adenoma were tested using univariate logistic regression and multivariate analysis. A two-sided P value of < 0.05 was considered statistically significant.

RESULTS

Baseline characteristics of eligible subject

As shown in Table 1, a summary of the characteristics stratified by nonpolyp, adenoma, nonadenomatous polyp and advanced adenoma groups are presented. Of 6018 subjects studied, 2035 (33.8%) presented with colorectal polyps, 1012 (16.8%) with adenomas and 1023 (17.0%) with nonadenomatous polyps. Out of 1012 subjects in the adenoma group, there were 143 cases of advanced adenomas. The prevalence of *H. pylori* infection in the nonpolyp group, adenoma group, nonadenomatous polyp group and advanced adenoma group were 48.6% (1936/3983), 53.0% (536/1012), 49.8% (509/1023) and 54.5% (78/143), respectively. The prevalence of AG in the nonpolyp group, adenoma group, nonadenomatous polyp group and advanced adenoma group were 8.7% (347/3983), 13.7% (139/1012), 11.3% (116/1023) and 14.7% (21/143), respectively. Overall, subjects with adenoma were older, had higher values of BMI, SBP, DBP, FBG, TC, TG, LDL, and lower values of HDL-cholesterol.

Association between H. pylori infection and adenoma

Based on the status of the *H. pylori* infection, all 6018 subjects were divided into HP (+) (2981, 49.5%) and HP (-) (3037, 50.5%). As reported in Table 2, the prevalence of adenoma in the HP (+) group was significantly higher than that of HP (-) group [unadjusted odds ratio (OR) = 1.1919, 95% confidence interval (CI): 1.037-1.367, P = 0.013; adjusted OR = 1.220, 95%CI: 1.053-1.413, P = 0.008, Table 3]. The mean age was not significantly different between the HP (+) and HP (-) groups. Compared to the HP (-) group, individuals in the HP (+) group had a higher proportion of men (P = 0.027, Table 2) and a higher prevalence of multiple colorectal polyps (P = 0.045). But the prevalence of nonadenomatous polyp, advanced adenoma, villous adenoma, adenoma size of ≥ 10 mm, single polyps, polyp size and *H. pylori* infection were similar (P > 0.05).

Association between AG and adenoma

Based on the AG status of all the 6018 subjects, we divided our cohort into two groups, the AG (+) group (602, 10.0%) and the AG (-) group (5416, 90.0%). Compared with the AG (-) group, subjects in the AG (+) group were older (P < 0.001, Table 4). The prevalence of adenoma in the AG (+) group was higher than that in the AG (-) group (unadjusted OR = 1.668, 95%CI: 1.352-2.059, P < 0.001, Table 4; adjusted OR = 1.237, 95%CI: 0.988-1.549, P = 0.064; Table 3). The prevalence of nonadenomatous polyps in the AG (+) group and AG (-) group was 19.3% and 16.7%, respectively (unadjusted OR = 1.340, 95%CI: 1.073-1.674, P = 0.010; adjusted OR = 1.103, 95%CI: 0.872-1.394, P = 0.413, Table 3). In addition, the prevalence of advanced adenoma in the AG (+) group and AG (-) group was 3.49% and 2.25%, respectively (unadjusted OR = 1.804 (95%CI: 1.121-2.903, P = 0.015; adjusted OR = 1.320, 95%CI: 0.805-2.165, P = 0.271, Table 3). The association of polyps with AG (+) was highest for individuals with more than one polyp (OR = 1.608, 95%CI: 1.302-1.985, P = 0.003). In patients with a polyp size of 0-9 mm, there existed a significant association between the prevalence of polyps and AG status (OR = 1.519, 95%CI: 1.275-1.809, P < 0.001).

Presence of both H. pylori infection and AG may increase the risk for adenoma significantly

According to the different statuses of *H. pylori* infection and AG, the individuals in our study were divided into HP (-) AG (-) group, HP (-) AG (+) group, HP (+) AG (-) group and HP (+) AG (+) group to understand whether *H. pylori* infection with AG increased the risk of adenoma. As reported in Table 5 and Table 6, the HP (+) AG (+) group had an approximately 1.5-fold risk for colorectal adenomas in comparison with that in the HP (-) AG (-) group (unadjusted OR = 1.964, 95%CI: 1.477-2.610, P < 0.001; adjusted OR = 1.491, 95%CI: 1.103-2.015, P = 0.009).

Presence of both H. pylori infection and AG also increase the risk for advanced adenoma

In subgroup analysis, the risk of colorectal adenomas was similar in either the HP (-) AG (-) group or HP (-) AG (+) group (unadjusted OR = 1.377, 95%CI: 0.618-3.064, P = 0.434), or between the HP (-) AG (-) group and HP (+) AG (-) group (unadjusted OR = 1.184, 95%CI: 0.825-1.699, P = 0.360). However, the presence of *H. pylori*-related AG was related to a significant increased risk for advanced adenomas (unadjusted OR = 2.496, 95%CI: 1.366-4.562, P = 0.003; adjusted OR = 1.910, 95%CI: 1.022-3.572, P =

Table 1 Baseline characteristics of 6018 subjects

Parameter	Nonpolyp, *n* = 3983	Adenoma, *n* = 1012	Nonadenomatous polyp, *n* = 1023	Advanced adenoma, *n* = 143	[a]*P* value	[b]*P* value	[c]*P* value
Male/female	2336/1647	780/232	788/235	110/33	< 0.001	< 0.001	< 0.001
HP (+/-)	1936/2047	536/476	509/514	78/65	0.013	0.512	0.165
AG (+/-)	347/3636	139/873	116/907	21/122	< 0.001	0.016	0.049
Smoker (+/-)	1029/2954	402/610	430/593	61/82	< 0.001	< 0.001	< 0.001
Alcohol (+/-)	1423/2560	494/518	476/547	68/75	< 0.001	< 0.001	0.006
Age in yr	46.430 (10.150)	52.680 (9.981)	50.010 (10.269)	53.310 (9.738)	< 0.001	< 0.001	< 0.001
BMI	23.611 (3.160)	24.355 (2.938)	24.667 (3.099)	24.809 (2.929)	< 0.001	< 0.001	< 0.001
SBP	123.950 (17.747)	129.700 (18.488)	127.810 (18.388)	131.650 (17.271)	< 0.001	< 0.001	< 0.001
DBP	73.410 (12.255)	76.990 (12.036)	75.910 (12.541)	77.520 (11.798)	< 0.001	< 0.001	< 0.001
TC	5.296 (1.076)	5.434 (1.155)	5.387 (1.077)	5.528 (1.083)	< 0.001	0.016	0.012
TG	1.771 (1.586)	1.982 (1.784)	2.001 (1.525)	2.194 (1.693)	< 0.001	< 0.001	0.002
HDL	1.296 (0.330)	1.252 (0.330)	1.216 (0.304)	1.237 (0.335)	< 0.001	< 0.001	0.034
LDL	3.169 (0.841)	3.253 (0.866)	3.258 (0.855)	3.259 (0.905)	0.005	0.003	0.208
FBG	4.818 (1.136)	5.065 (1.422)	5.055 (1.395)	5.091 (1.608)	< 0.001	< 0.001	0.047

[a]Two-sided *P* values for the difference between adenoma and nonpolyp were based on the χ^2 test and *t* test.
[b]Two-sided *P* values for the difference between nonadenomatous polyp and nonpolyp were based on the χ^2 test and *t* test.
[c]Two-sided *P* values for the difference between advanced adenoma and nonpolyp were based on the χ^2 test and *t* test. HP: *Helicobacter pylori*; AG: Atrophic gastritis; BMI: Body mass index; SBP: Systolic blood pressure; DBP: Diastolic blood pressure; TC: Total cholesterol; TG: Triglyceride; HDL: High-density lipoprotein; LDL: Low-density lipoprotein; FBG: Fasting blood glucose.

0.043).

DISCUSSION

In this study, the potential roles of *H. pylori* infection, AG and *H. pylori*-related AG in the progress of colorectal adenomas and advanced adenoma were investigated. According to previous research, the association between *H. pylori* and colorectal adenomas remains unclear[20,22-26]. In our study, *H. pylori* infection was an independent risk factor for colorectal adenomas. The finding is consistent with current studies that indicate a positive correlation was revealed between colorectal adenomas and *H. pylori*. Additionally, HP (+) AG (-) may indicate a higher risk of colorectal adenomas. However, it was not associated with an increased risk of advanced adenomas. In our study, *H. pylori* infection was diagnosed by the results from the ^{13}C-urea breath test or a histological diagnosis of biopsied gastric specimen serology test that can accurately reflect a current *H. pylori* infection[21]. With the development of detection technologies of *H. pylori* infection, the role of *H. pylori* in the colorectal carcinogenesis may be revealed.

No significant association between AG and colorectal adenomas was observed in our cohort. Moreover, HP (-) AG (+) was not an independent risk factor for colorectal adenomas. Some subjects with HP (-) AG (+) may be affected with severe AG following a long-term infection with *H. pylori*. Theoretically, these patients may present with hypergastrinemia and have a higher risk of colorectal adenomas. However, our study did not indicate any correlation based on this hypothetical reasoning. In the multivariate analysis, the relatively small number (*n* = 279) of the HP (-) AG (+) group may have concealed the possible effects on colorectal adenomas.

After controlling all confounding factors, the ORs for colorectal adenomas in eligible individuals with *H. pylori*-related AG were higher than those in individuals of the HP (-) AG (-) group (adjust OR = 1.491, 95%CI: 1.103-2.015, *P* = 0.009). HP (+) AG (+) is independently associated with colorectal adenomas. Additionally, HP (+) AG (+) is significantly associated with an increased risk of advanced adenomas. However, no such association was observed in the HP (-) AG (+) or the HP (+) AG (-) group. This

Table 2 Correlation between *Helicobacter pylori* infection and colorectal neoplasm

Parameter	HP (-), *n* = 3037	HP (+), *n* = 2981	OR (95%CI)	*P* value
Age in yr	48.130 (10.678)	48.040 (10.177)	0.999 (0.994-1.004)	0.745
Female	1026	1088	1	
Male	2011	1893	0.888 (0.798-0.987)	0.027
Nonpolyp	2047	1936	1	
Nonadenomatous polyp	514	509	1.047 (0.913-1.201)	0.512
Adenoma	476	536	1.191 (1.037-1.367)	0.013
Advanced adenoma	65	78	1.269 (0.908-1.774)	0.164
Villous adenoma	24	23	1.013 (0.570-1.801)	0.964
Size of adenoma ≥ 10 mm	49	64	1.381 (0.947-2.014)	0.093
High-grade dysplasia	5	8	1.692 (0.552-5.180)	0.357
Polyps number				
One	509	521	1.082 (0.944-1.241)	0.258
Two or more	481	524	1.152 (1.003-1.323)	0.045
Polyps size				
0-9 mm	925	963	1.101 (0.987-1.228)	0.086
≥ 10 mm	65	82	1.334 (0.958-1.858)	0.088

Correlation between *Helicobacter pylori* (+) and *Helicobacter pylori* (-) by logistic regression analysis. OR: Odds ratio; CI: Confidence interval; HP: *Helicobacter pylori*.

Table 3 Logistic regression model of the association between *Helicobacter pylori* infection, atrophic gastritis and colorectal neoplasm after adjustments for confounding factors

	Nonadenomatous polyp		Adenoma		Advanced adenoma	
	Adjusted OR (95%CI)	*P* value	Adjusted OR (95%CI)	*P* value	Adjusted OR (95%CI)	*P* value
HP (+)	1.033 (0.895-1.193)	0.658	1.220 (1.053-1.413)	0.008	1.303 (0.922-1.842)	0.134
AG (+)	1.103 (0.872-1.394)	0.413	1.237 (0.988-1.549)	0.064	1.320 (0.805-2.165)	0.271

Adjusted for age, gender, systolic blood pressure, diastolic blood pressure, body mass index, smoking habit, alcohol consumption, total cholesterol level, triglyceride level, high-density lipoprotein-C level, low-density lipoprotein-C level and fasting blood glucose level by logistic regression analysis. HP: *Helicobacter pylori*; AG: Atrophic gastritis; OR: Odds ratio; CI: Confidence interval.

finding is consistent with that of a recent study that indicated that *H. pylori* infection along with AG increased the risk of both overall and advanced colorectal neoplasm[21]. Chronic *H. pylori* infection can lead to the occurrence of gastric mucosal atrophy[27]. In our study, the mean age in HP (+) AG (+) group was higher than in the HP (+) AG (-) group (52.3 years *vs* 47.5 years). This can be explained as the individuals in the HP (+) AG (+) group may have *H. pylori* infection for a longer period.

The presence of the *H. pylori* infection and AG increases the risk of colorectal adenoma. This may occur *via* various mechanisms. The cholecystokinin type B/gastrin receptor and gastrin are present in human colorectal polyps, and they are activated in the early stages of the adenoma-carcinoma sequence[28,29]. Persistent exposure to *H. pylori* infection directly induces the atrophic changes of the gastric body mucosa and increases the gastrin secretion. This has a nutritional effect on the growth and proliferation of epithelial cells and ultimately contributes to colorectal carcinogenesis[30,31]. In addition, hypochlorhydria caused by *H. pylori*-related AG may hamper protein assimilation, leading to an increase of some unabsorbed nutrients and metabolites[32]. Hypochlorhydria generates bacterial overgrowth and colorectal disorders, resulting in colorectal carcinogenesis[33].

Table 4 Correlation between atrophic gastritis and colorectal neoplasm

Parameter	AG (-), *n* = 5416	AG (+), *n* = 602	OR (95%CI)	*P* value
Age in yr	47.590 (10.350)	52.530 (10.117)	1.045 (1.037-1.053)	< 0.001
Female	1921	193	1	
Male	3495	409	1.165 (0.973-1.394)	0.097
Nonpolyp	3636	347	1	
Nonadenomatous polyp	907	116	1.340 (1.073-1.674)	0.010
Adenoma	873	139	1.668 (1.352-2.059)	< 0.001
Advanced adenoma	122	21	1.804 (1.121-2.903)	0.015
Villous adenoma	40	7	1.834 (0.815-4.124)	0.143
Size of adenoma ≥ 10 mm	98	15	1.604 (0.921-2.792)	0.095
High-grade dysplasia	12	1	0.873 (0.113-6.735)	0.897
Polyps number				
One	893	137	1.608 (1.302-1.985)	< 0.001
Two or more	887	118	1.394 (1.117-1.739)	0.003
Polyps size				
0-9 mm	1649	239	1.519 (1.275-1.809)	< 0.001
≥ 10 mm	131	16	1.280 (0.753-2.176)	0.362

Correlation between atrophic gastritis (+) and atrophic gastritis (-) by logistic regression analysis. OR: Odds ratio; CI: Confidence interval; AG: Atrophic gastritis.

Table 5 Association between *Helicobacter pylori* infection, atrophic gastritis and colorectal neoplasm

	HP (–) AG (–), *n* = 2758		HP (–) AG (+), *n* = 279			HP (+) AG (–), *n* = 2658			HP (+) AG (+), *n* = 323		
	n (%)	OR (95%CI)	*n* (%)	OR (95%CI)	*P* value	*n* (%)	OR (95%CI)	*P*	*n* (%)	OR (95%CI)	*P*
Age in yr, mean ± SD	47.7 ± 10.6		52.8 ± 10.5			47.5 ± 10.1			52.3 ± 9.8		
Male sex	1818 (65.9)		193 (69.2)			1677 (63.1)			216 (66.9)		
Nonadenomatous polyp	460 (16.7)	1	54 (19.4)	1.339 (0.969-1.851)	0.077	447 (16.8)	1.043 (0.901-1.206)	0.574	62 (19.2)	1.394 (1.027-1.892)	0.033
Adenoma	416 (15.1)	1	60 (21.5)	1.645 (1.202-2.252)	0.002	457 (17.2)	1.179 (1.017-1.367)	0.029	79 (24.5)	1.964 (1.477-2.610)	< 0.001
Advanced adenoma	58 (2.1)	1	7 (2.5)	1.377 (0.618-3.064)	0.434	64 (2.4)	1.184 (0.825-1.699)	0.360	14 (4.3)	2.496 (1.366-4.562)	0.003

Univariate logistic regression was used to analyze the association between *Helicobacter pylori* infection, atrophic gastritis and colorectal neoplasm. HP: *Helicobacter pylori*; AG: Atrophic gastritis; OR: Odds ratio; CI: Confidence interval; SD: Standard deviation.

Table 6 Logistic regression model of the association between *Helicobacter pylori* infection, atrophic gastritis and colorectal neoplasm after adjustments for confounding factors

	HP (–) AG (–), *n* = 2758	HP (–) AG (+), *n* = 279		HP (+) AG (–), *n* = 2658		HP (+) AG (+), *n* = 323	
	OR (95%CI)	OR (95%CI)	*P* value	OR (95%CI)	*P* value	OR (95%CI)	*P* value
Non-adenomatous polyp	1	1.093 (0.776-1.540)	0.612	1.03 (0.884-1.199)	0.707	1.141 (0.830-1.568)	0.417
Adenoma	1	1.216 (0.868-1.705)	0.255	1.213 (1.037-1.419)	0.016	1.491 (1.103-2.015)	0.009
Advanced adenoma	1	0.979 (0.431-2.226)	0.960	1.214 (0.836-1.763)	0.308	1.910 (1.022-3.572)	0.043

Adjusted for age, gender, systolic blood pressure, diastolic blood pressure, body mass index, smoking habit, alcohol consumption, total cholesterol level, triglyceride level, high-density lipoprotein-C level, low-density lipoprotein-C level and fasting blood glucose level by logistic regression analysis. HP: *Helicobacter pylori*; AG: Atrophic gastritis; OR: Odds ratio; CI: Confidence interval.

Generalizability of findings in this study is limited by several factors. First, based on general health check-ups, a potential selection bias may have existed. In addition, the data affecting the changes of gastric mucosa, viz. dietary habit, was insufficient. Second, serum gastrin level, as a key mechanism in the progress of colorectal carcinogenesis, was not included in our analysis. Third, biopsy samples accounted for only 74% of the data. This may have potentially lowered the rate of gastric disease detection. Finally, our analyzable data were derived from a single center and local region in Chinese people, thereby limiting the ability to generalize our finding. Therefore, further multicenter research should be established to determine the potential association of individuals with other nations and ethnic groups. Despite these limitations, it is a novel study as we not only analyzed the relationship between *H. pylori* infection and colorectal adenomas but also further investigated the role of AG in colorectal carcinogenesis.

CONCLUSION

In summary, our study clearly demonstrated that subjects with *H. pylori*-related AG did have an increased risk for colorectal adenoma. Due to the high prevalence of *H. pylori* infection and colorectal cancer in the Chinese population, strict colonoscopy screening and surveillance are necessary for patients with *H. pylori* infection, especially for those with *H. pylori*-related AG.

ARTICLE HIGHLIGHTS

Research background

Several previous studies demonstrated the significance of *Helicobacter pylori* (*H. pylori*) infection and atrophic gastritis (AG) in the prevalence of colorectal adenomas. A recent study showed a significant association between colorectal neoplasm and AG, which was diagnosed by Kimura and Takemoto criteria without the histologic diagnosis. However, the relationship between AG and colorectal neoplasia, especially that between *H. pylori*-related AG and colorectal neoplasia, is still controversial.

Research motivation

Colorectal adenomas may develop colorectal cancer, which is considered to be one of the most common human malignancies worldwide. Early diagnosis of colorectal adenomas is important to reduce mortality. The association of *H. pylori* infection and AG in the prevalence of colorectal adenomas has been examined in a limited number of studies. However, there exists disputed conclusions in the studies reported.

Research objectives

The aim was to investigate the relationship between colorectal adenomas and *H. pylori*-related AG based on the histologic diagnosis.

Research methods

This retrospective cross-sectional study analyzed records between August 2014 and August 2017 and were extracted from the Medical and Health Care Center at The First Affiliated Hospital of Wenzhou Medical University. Based on the relevant inclusion and exclusion criteria, 6018 health-check individuals were eventually enrolled. The relevant data were recorded. Univariate and multivariate logistic regression analyses were performed to determine the association between *H. pylori*-related AG and colorectal adenomas.

Research results

H. pylori infection accompanied by AG was significantly associated with an increased risk of adenomas (adjusted odds ratio = 1.491, 95% confidence interval: 1.103-2.015, P = 0.009) and advanced adenomas (adjusted odds ratio = 1.910, 95% confidence interval: 1.022-3.572, P = 0.043).

Research conclusions

Our research demonstrated that *H. pylori*-related AG is an independent risk factor for colorectal adenomas in the Chinese population.

Research perspectives

The Chinese have a high prevalence of *H. pylori* infection and colorectal cancer. Therefore, strict colonoscopy screening and surveillance are necessary for patients with *H. pylori* infection, especially for those with *H. pylori*-related AG.

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