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Original Article

Relationship between *Helicobacter pylori* infection and different pathological types of chronic atrophic gastritis and analysis of serum gastric function

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Abstract

Objective: This study aimed to compare the *Helicobacter pylori* (Hp) infection rate and serum gastric function among patients with chronic atrophic gastritis (CAG) of different pathological types and to determine the value of combining these tests in assessing the extent and severity of CAG atrophy.

Methodology: We retrospectively analyzed 60 patients with CAG and 46 patients with chronic non-atrophic gastritis (CNAG) who underwent gastroscopy between July 2023 and June 2024. Endoscopic findings, histopathology, Hp status and serum gastric function indices were compared.

Results: Compared with the CNAG group, the CAG group showed a significantly higher Hp positivity rate, lower serum group I pepsinogen (PG I) and group II pepsinogen (PG II) levels and higher gastrin 17 (G-17) levels ($p < 0.01$). In patients with CAG, open-type cases had a significantly higher Hp positivity rate ($p < 0.05$), lower PG I and PG II levels and higher G-17 levels ($p < 0.01$) than closed-type cases. The positive Hp rate was significantly higher in the atrophic gastritis with intestinal metaplasia group than in the glandular reduction atrophic gastritis group ($p < 0.05$), with the contents of PG I and PG II significantly lower in the former than in the latter and the content of G-17 significantly higher in the former than in the latter ($p < 0.01$).

Conclusions: *Helicobacter pylori* infection is strongly associated with CAG, with marked differences in Hp rates and serum gastric function across pathological and microscopic types. Combined serum gastric function and Hp testing can help assess the extent and severity of atrophy.

Key words: Chronic atrophic gastritis; *Helicobacter pylori*; serum gastric function; pathological type; endoscopic classification.

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Introduction

Chronic atrophic gastritis (CAG) is a chronic gastric disease in which the gastric mucosal epithelium suffers repeated damage, resulting in the reduction of intrinsic glands and causing glandular atrophy of the gastric mucosa [1]. The disease can be divided into two major categories: multifocal atrophic gastritis and autoimmune gastritis, with the former dominated by the antrum and mostly developing from chronic non-atrophic gastritis (CNAG) caused by *Helicobacter pylori* (Hp) infection [2,3]. Autoimmune atrophic gastritis changes are mainly in the gastric body and mostly develop from autoimmune-induced gastric body gastritis [4]. Some patients may have no obvious symptoms, although most experience epigastric burning pain, dull pain or fullness and stuffiness (especially after eating), loss of appetite, nausea, belching, constipation or diarrhoea and other symptoms [5].

Serum gastric function status refers to a test that assesses gastric secretory function through blood samples and is primarily used to evaluate the health status of the gastric mucosa and the extent of gastric mucosal damage [6]. Serum gastric function tests typically include gastrin 17 (G-17), pepsinogen (PG) group I (PG I) and PG group II (PG II) [7]. Studies have shown that changes in PG and G-17 levels can reflect the functional and morphological status of the gastric mucosa [8]. The changes of these indicators are related to the promotion of Hp during gastric mucosal atrophy and intestinal metaplasia, which indirectly affect changes in the above indicators. Changes in the levels of PG I and PG II are related to the diagnosis of CAG and gastric cancer risk assessment [9,10]. Specifically, decreased levels of PG I and a decreased PG I/PG II ratio (PGR) are closely related to the presence of gastric mucosal atrophy [11]. Changes in G-17 levels are also associated with CAG [12]. In some studies, the difference in G-17 was not statistically significant, whereas in others, changes in G-17 levels were related to the status of the gastric mucosa [13,14].

To date, studies have examined the relationship between Hp infection and the development of CAG; however, most focus on the observation of patients with CAG as a whole or a single type. The systematic analysis of the relationship between pathological types (e.g. hypoglandular type and intestinal metaplasia type), endoscopic types (e.g. the Kimura–Takemoto type) and Hp infection remains relatively limited. Therefore, this study compares the Hp infection rate and serum gastric function status of patients with CAG with different microscopic and pathological types and explores the clinical reference value of the serum gastric function test combined with the Hp test for assessing the range and degree of CAG atrophy.

Methodology

Study participants

A total of 106 patients (57 men and 49 women) were included. Among them, 60 patients (aged 45 ± 4 years) were diagnosed with CAG, and 46 patients (aged 48 ± 5 years) were diagnosed with CNAG in Beijing Jishuitan Hospital between July 2023 and June 2024. The age range was 25–75 years. Gastroscopy findings, pathological biopsies, Hp infection status and serum gastric function test data of these patients were retrospectively analysed in detail. The study adopted different follow-up strategies for patients with various pathological types: (1) simple atrophic gastritis (without intestinal metaplasia/dysplasia) – gastroscopy every 2–3 years; (2) intestinal metaplasia – mild (re-examination every 2–3 years) and moderate-severe (re-examination every 1–2 years); and (3) intraepithelial neoplasia – low-grade (re-examination every 6–12 months) and high-grade (immediate endoscopic or surgical treatment). All patient data were obtained from the hospital's electronic medical record system and endoscopy centre database and were independently extracted and verified by two researchers to ensure their integrity and accuracy. In addition, the study was approved by Beijing Jishuitan Hospital (No. 201805-18-02). Written informed consent was obtained from all participants.

Inclusion and exclusion criteria

This study included patients who strictly followed the diagnostic criteria for CAG and CNAG in the Chinese Consensus on Chronic Gastritis report (Shanghai, 2017) [15] and who provided informed consent. The exclusion criteria included (1) patients with gastric and duodenal ulcers, gastric cancer or gastrinoma, (2) patients with severe heart, liver, kidney or other vital organ dysfunction and (3) patients who had used antibiotics, antacids or gastric mucosal protective agents in the past month.

Determination of serum gastric function

Five milliliters of venous blood was collected from all participants under fasting conditions. This step ensured the accuracy of the test results and avoided any effects on gastric function parameters during food digestion. Blood samples were collected early in the morning to reduce the impact of daily activities and diet on test results. Samples were centrifuged at 3,500 rpm for 15 minutes in an LD5-2A benchtop low-speed centrifuge (Jingli Co., Ltd., Beijing, China) following collection to separate serum. The centrifuged serum was used for subsequent experiments or aliquoted and stored in a freezer at -20°C until use. Serum gastric function was measured via enzyme-linked immunosorbent assay (ELISA). Specifically, PG I, PG II and G-17 were quantitatively detected in the patient's serum using this method. The reagents used for ELISA detection were provided by Biohan Biotechnology (Hefei) Co., Ltd. All operations were performed in strict accordance with the reagent instructions and standard operating procedures. All serum gastric function tests were performed on the day of the patient's admission. Blood sample collection and testing followed standard procedures, and the data were retrospectively extracted from the hospital's electronic medical record system and laboratory system.

Gastroscopy, biopsy and determination of Helicobacter pylori

All participants underwent gastroscopy on the day of admission, as well as gastric mucosal biopsy. Gastric mucosal tissue samples were obtained using endoscopic biopsy forceps, with the samples then sent to the laboratory for microscopic analysis to assess the degree and nature of inflammation. Biopsy sites included the antrum, angle and body and other suspicious lesions. *Helicobacter pylori* determination was performed using the carbon-13 urea breath test. All Hp tests were performed on the day the patients were admitted to the hospital for gastroscopy, and the data were retrospectively extracted from the hospital's electronic medical record system and laboratory system.

Statistical methods

Statistical analysis was performed using SPSS 22.0 software. Following a normality test, the measurement data were expressed as mean ± standard deviation ($\bar{x} \pm s$) and the independent sample *t*-test was used for inter-group comparison. The count data were expressed as

the number of cases (%), and the chi-squared test or Fisher's exact test was used for inter-group comparison. A p of < 0.05 was considered statistically significant.

Results

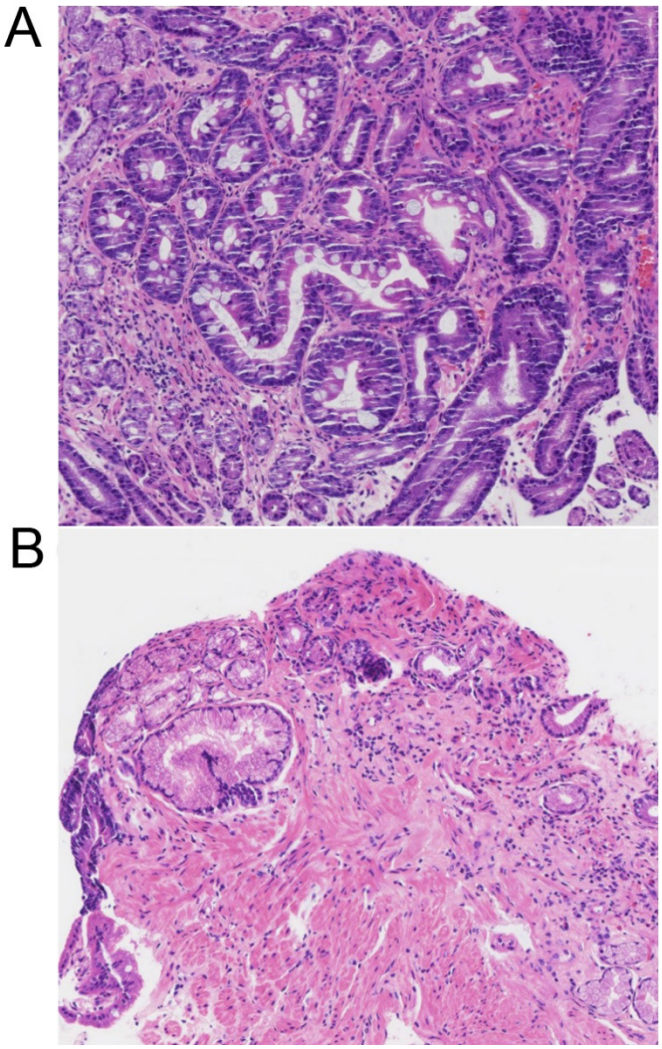
Comparison of Helicobacter pylori infection and serum gastric function between the two groups

A total of 106 patients were included in this study, comprising 60 patients with CAG and 46 with CNAG. A total of 41 individuals in the CAG group and 21 in the CNAG group tested Hp positive. The proportion of Hp-positive patients was significantly higher in the CAG group than in the CNAG group ($p < 0.01$). The serum gastric function test results showed that the PG I and PG II contents were significantly lower in the CAG group than in the CNAG group ($p < 0.01$), whereas the G-17 content was significantly higher in the former than in the latter ($p < 0.01$) (Table 1). The above results showed that serum gastric function status is closely related to CAG.

Helicobacter pylori infection and serum gastric function in patients with different endoscopic types of chronic atrophic gastritis

The Kimura–Takemoto classification divides atrophic gastritis into two categories: closed type and open type. The atretic (closed) type refers to the atrophic transitional zone that does not exceed the cardia on the lesser curvature of the gastric body, whereas the open type refers to the atrophic transitional zone that exceeds the cardia and progresses to the greater curvature. Each type is divided into three subtypes: C-1, C-2 and C-3 (C1: atrophy boundary confined to the antrum; C2: atrophy boundary exceeding the gastric angle; C3: atrophy boundary exceeding the gastric angle and close to the cardia) and into O-1, O-2 and O-3 (O-1 atrophy boundary just

Figure 1. Histopathological sections of gastric mucosa in atrophic gastritis with intestinal metaplasia and glandular reduction groups (×100 magnification).



A: Atrophic gastritis with intestinal metaplasia group; **B:** Atrophic gastritis with glandular reduction group.

Table 1. Hp infection and serum gastric function test results in CAG and CNAG groups.

Group	n	Hp		PG I (μg/L, $\bar{x} \pm s$)	PG II (μg/L, $\bar{x} \pm s$)	G-17 (pmol/L, $\bar{x} \pm s$)
		+	-			
CAG	60	41	19	58.5 ± 12.3	13.8 ± 3.9	14.9 ± 3.6
CNAG	46	21	25	118.8 ± 51.1	17.9 ± 5.8	5.6 ± 1.3
χ^2/t	-	4.58		-14.05	-7.89	24.49
p	-	< 0.01		< 0.01	< 0.01	< 0.01

Hp: *Helicobacter pylori*; CAG: chronic atrophic gastritis; CNAG: chronic non-atrophic gastritis; PG I: pepsinogen I; PGII: pepsinogen II; G-17: gastrin 17.

Table 2. Hp infection and serum gastric function test results of different Under-endoscope types of CAG.

Group	n	Hp		PG I (μg/L, $\bar{x} \pm s$)	PG II (μg/L, $\bar{x} \pm s$)	G17 (pmol/L, $\bar{x} \pm s$)
		+	-			
Closed type	34	20	14	69.3 ± 11.2	15.3 ± 3.7	12.9 ± 3.8
Open type	26	21	5	41.4 ± 9.8	12.6 ± 4.1	15.7 ± 2.9
χ^2/t	-	3.28		15.41	3.89	-2.96
p	-	< 0.05		< 0.01	< 0.01	< 0.01

Hp: *Helicobacter pylori*; CAG: chronic atrophic gastritis; PG I: pepsinogen I; PGII: pepsinogen II; G-17: gastrin 17.

passing through the cardia; O-2: atrophy boundary extending throughout the fundus; O-3: atrophy boundary extending to the gastric body) [16]. Based on the Kimura–Takemoto classification, 34 patients with closed type and 26 patients with open type were included in this study. Of the closed-type patients, 20 were Hp positive, whereas 21 of the open-type patients were Hp positive. The positive rate of Hp was significantly higher in open-type patients than in closed-type patients ($p < 0.05$). The serum gastric function test results showed that the PG I and PG II contents were significantly lower in open-type patients than in closed-type patients ($p < 0.01$). However, the G-17 content was significantly higher in the open group than in the closed group ($p < 0.01$) (Table 2).

Helicobacter pylori infection and serum gastric function in patients with different pathological types of chronic atrophic gastritis

Sixty patients with CAG were divided into the atrophic gastritis with intestinal metaplasia group (n = 28) and the atrophic gastritis of glandular reduction group (n = 32) according to their pathological types (Figure 1). In the atrophic gastritis with intestinal metaplasia group, 23 patients were Hp positive, whereas 19 patients were Hp positive in the glandular reduction group. The positive rate of Hp was significantly higher in the atrophic gastritis with intestinal metaplasia group than in the glandular reduction group ($p < 0.05$). The serum gastric function test results showed that the PG I and PG II contents were significantly lower in the atrophic gastritis with intestinal metaplasia group than in the glandular reduction group ($p < 0.01$). However, the G-17 content was significantly higher in the atrophic gastritis with intestinal metaplasia group than in the glandular reduction group ($p < 0.01$) (Table 3).

Discussion

This study found that the Hp infection rate was significantly higher in patients with CAG than in those with CNAG. There were also significant differences in the Hp infection rate and serum gastric function indicators (PG I, PG II, G-17) between different pathological and endoscopic types, suggesting that Hp infection is closely associated with gastric mucosal atrophy.

Helicobacter pylori infection affects the gastric mucosal epithelium through its virulence factors, promoting inflammation and glandular atrophy, and is an important cause of CAG and precancerous lesions [17-19]. Similar to previous findings, in this study, the Hp infection rate was higher in patients with CAG than in those with CNAG, and there was a significant difference in the Hp infection rate due to different sites of atrophy and whether it was accompanied by intestinal metaplasia. *Helicobacter pylori* infection predisposes to gastric mucosal atrophy, and asymptomatic Hp infection should be eradicated before gastric mucosal atrophy emerges. The diagnosis of CAG mainly relies on gastroscopy and histopathological examination of the gastric mucosa, with pathological examination being the gold standard [20]. Endoscopists make a judgment on the extent and degree of atrophy based on endoscopic findings combined with pathological findings. The Kimura–Takemoto classification helps to identify and monitor the mucosa in the high-risk background of gastric cancer and is key to reducing the mortality of this disease and improving the prognosis of patients. Reports have shown that Hp infection is an independent factor affecting the consistency between the Kimura–Takemoto classification and Operative Link on Gastritis Assessment system results [21]. In this study, the results showed that the proportion of Hp infection was significantly higher in open CAG than in closed CAG, and the effect of Hp infection on CAG progression was also further explained.

Pepsinogen is a precursor form of pepsin and is divided into two types: PG I and PG II, with the former mainly derived from specific cells of the fundus and corpus and the latter mainly derived from the inlet region of the stomach and specific glands of the duodenum [22]. Under the influence of Hp infection, gastric mucosal cells may undergo degeneration and necrosis, resulting in gastric gland damage and atrophy of the gastric body mucosa. This atrophy decreases the number of cells in the fundus and corpus, which are replaced by pyloric glands or intestinal epithelial cells, thereby decreasing PG I production. In contrast, PG II

Table 3. Hp infection and serum gastric function test results of different pathological types of CAG.

Group	n	Hp		PG I (μg/L, $\bar{x} \pm s$)	PG II (μg/L, $\bar{x} \pm s$)	G17 (pmol/L, $\bar{x} \pm s$)
		+	-			
CAG with intestinal metaplasia group	28	23	5	49.4 ± 12.8	13.5 ± 3.5	15.3 ± 4.6
CAG with Glandular reduction group	32	19	13	63.5 ± 14.6	16.8 ± 3.2	10.9 ± 3.2
χ^2/t	-	3.36		-7.32	-6.52	6.555
p	-	< 0.05		< 0.01	< 0.01	< 0.01

Hp: *Helicobacter pylori*; CAG: chronic atrophic gastritis; PG I: pepsinogen I; PGII: pepsinogen II; G-17: gastrin 17.

is less affected by corpus atrophy due to its wider distribution of secretory cells, meaning its levels change little, which leads to a decrease in the PGR [23]. Furthermore, G-17 is secreted by specific cells of the antrum and duodenum, and it mainly promotes the secretion of gastric acid and PG. When atrophy occurs in the antrum, the number of G-17-secreting cells decreases, resulting in decreased G-17 levels. However, when atrophy occurs in the gastric body, gastric acid secretion decreases and intragastric pH increases, which stimulates increased secretion by G-cells, thereby increasing serum levels of G-17 [24]. If atrophy of the gastric mucosa affects the entire stomach, the G-17 level becomes higher than in antral atrophy alone, and the G-17 level gradually rises as the degree of atrophy increases [25]. Previous studies have reported that changes in serum PG I and PG II levels are associated with the diagnosis of CAG and gastric cancer risk assessment. Specifically, decreased levels of PG II and increased PGR are closely associated with the presence of gastric mucosal atrophy. Changes in the levels of G-17 are also associated with CAG, where G-17 decreases as a result of decreased G-cells during antral atrophy; gastric acid secretion decreases during corpus atrophy, and negative feedback promotes the hypersecretion of G-cells, resulting in increased serum G-17 levels [11,12].

The results of this study showed that serum gastric function status is closely associated with the development of CAG. Compared with the CNAG group, serum PG I and PG II levels were significantly lower in the CAG group, whereas G-17 levels were significantly higher than those in the CNAG group. In patients with CAG, PG I and PG II levels were significantly lower and G-17 levels significantly higher in open-type patients than in closed-type patients. The PG I and PG II contents were significantly lower in the atrophic gastritis with intestinal metaplasia group than in the atrophic gastritis group, with the content of G-17 significantly higher in the former than in the latter.

This study has some limitations. First, the study included only 106 patients, and the sample size was relatively small, potentially limiting the generalisability and statistical power of the results. Second, more classification methods should be used in future studies using the Kimura–Takemoto taxonomy for endoscopic classification. Finally, patients were not followed up in this study, meaning disease progression and prognosis over time and their influencing factors in patients with CAG could not be assessed.

Conclusions

This study found that the Hp infection rate was significantly higher in patients with CAG than in those with CNAG. Furthermore, the serum gastric function status of patients with CAG was closely related to the presence of CAG. Specifically, the Hp infection rate and serum gastric function parameters in the atrophic gastritis with intestinal metaplasia group were significantly different from those in the glandular reduction atrophic gastritis group. This indicates that the serum gastric function test combined with Hp detection has potential reference value in CAG assessment. However, further prospective studies are needed to verify the effectiveness of the combined method.

Ethics approval and consent to participate

This study was conducted in accordance with the declaration of Helsinki. This study was approved by Beijing Jishuitan Hospital (No. 201805-18-02). Written informed consent was obtained from all participants.

Data Availability Statement

All data generated or analyzed during this study are included in this article.

Author contributions

Conceptualization: Z YX; Methodology: F YP; Formal analysis and investigation: Y CL & Z LY; Writing - original draft preparation: Z YX; Writing - review and editing: Y CL & Z LY.

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Conflict of interest

No conflict of interest is declared.

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