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ORIGINAL RESEARCH

Effects of Infection with Different Types of *Helicobacter pylori* on Gastric Secretion Function: A Cross-Sectional Clinical Study

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Purpose: Helicobacter pylori (Hp)-related gastropathies are accompanied by alterations in gastric secretion function, but the effects of infection of different Hp strains on gastric function are not yet well-elucidated. Our cross-sectional clinical study aim to research the effects of infection with different Hp types on gastric function.

Patients and Methods: We analyzed 525 patients' serum cytotoxin-associated protein gene A (CagA), vacuolating cytotoxin-associated protein gene A (VacA), urease (Ure), Gastrin-17 (G-17), Pepsinogen I (PGI), Pepsinogen II (PGII) and PGI/PGII ratio (PGR).

Results: The PGII levels (8.19 ± 5.44 *vs* 5.98 ± 10.75, $P = 0.013$) were higher in the Hp infected group than in the uninfected, while the PGR levels (16.81 ± 8.22 *vs* 23.23 ± 8.36, $P < 0.001$) were lower. The PGR levels were higher in the uninfected group (23.23 ± 8.36, $P < 0.001$) than in Hp-I (16.47 ± 7.45) and Hp-II infected groups (17.39 ± 8.98). In the uninfected group, the G-17 level was positively correlated with the levels of PGI (Pearson coefficient = 0.177, $P = 0.001$), PGII (Pearson coefficient = 0.140, $P = 0.008$) and age (Pearson coefficient = 0.121, $P = 0.022$), negatively with the PGR levels (Pearson coefficient = −0.201, $P < 0.001$). In the Hp-I (Pearson coefficient = −0.003, $P = 0.975$) and Hp-II (Pearson coefficient = 0.018, $P = 0.161$) infected groups, the G-17 levels were not correlated with age.

Conclusion: Hp-I with CagA and/or VacA positive and Hp-II without cytotoxicity can reduce gastric secretion function regardless of age and sex. Gastric function in patients with Hp eradication was similar to that in those without Hp infection. G-17 rises physiologically with age, but infection with Hp will affect it.

Keywords: Helicobacter pylori, Pepsinogen I, Pepsinogen II, Gastrin-17, cytotoxin-associated protein gene A, vacuolating cytotoxin-associated protein gene A

Introduction

Gastrin is a gastrointestinal hormone secreted by the gastric antrum G cells, of which G-17 is the most active, accounting for about 80–90% of the life-giving gastrin in the human body.[1,2] G-17 has the effect of promoting the secretion of other digestive juices and enzymes, such as bile, pancreatic juice, pepsin and insulin.[2,3] Pepsinogen (PG) is a proteinase secreted in human gastric mucosa cells and classified as PGI and PGII depending on the secretion site.[4] PGI is secreted mainly by the chief cells and mucous neck cells of the gastric fundus, while PGII is secreted by the gastric cardia, fundus, antrum and proximal duodenum. PG becomes pepsin activated upon release into the stomach cavity, and only about 1% of the PG enters the blood circulation, which is stable Serum PGI, PGII and PGR indirectly reflect the status and secretory function of gastric mucosa at different sites.[1,5–7]

Hp is a gram-negative bacillus that has been proven to be closely associated with many diseases such as atrophic gastritis, gastric ulcer, and gastric cancer.[8–10] Hp infection rates exceed 50% in natural populations worldwide. Hp infection in China is

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widely distributed. The infection rates ranging from 40% to 90%, with an average of 59%. At present, the methods used to detect Hp infection include rapid urease test, urea breath test, fecal antigen test and serum antibody test. The pathogenicity of Hp is attributed to a large extent to a variety of virulence components in which VacA and CagA play a major role.[11–13] VacA can cause eukaryotic vacuolar degeneration and mitochondrial dysfunction, trigger molecular changes in gastric epithelial cells, disrupt mucosal barriers, and promote the release of inflammatory mediators from epithelial cells.[14] CagA plays an important role in Hp-induced gastric mucosal inflammation to carcinoma transformation through the activation of multiple key pathways such as NF-κB, β-catenin, hepatic PI3K/AKT, releasing inflammation factors and IL-8.[15,16] Hp strains were genotyped according to the expression or non-expression of CagA and VacA, the former called Hp-I, the latter called Hp-II. Hp-I strains dominate disease-associated infections with strong virulence. Hp-I strains are virulent and predominate in disease-associated infections.[17,18]

Hp-related gastropathies are accompanied by alterations in gastric secretion function,[9,19,20] but so far there is less detailed evidence on the effects of infection of different Hp strains on gastric secretion function in patients. This study conducted a cross-sectional clinical study of Hp antibody typing and gastric secretion function in 525 patients seen in our hospital (Figure 1). Our data will provide new evidences for the relationship between gastric secretion function and infection of different Hp strains and provide new insights into the diagnosis of Hp-related gastropathies and the eradication treatment of Hp.

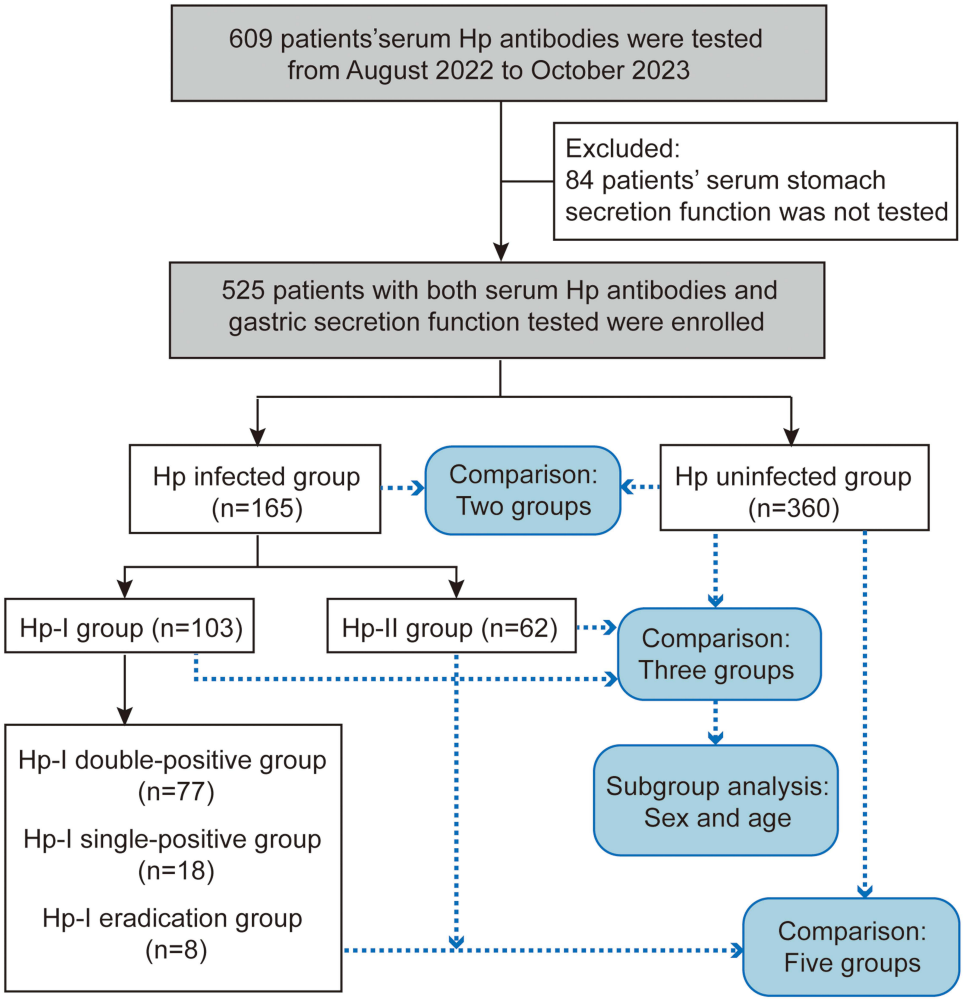

Figure 1 Study design schematic. Flow chart illustrating the study design.

Materials and Methods

Patient Enrolment

609 patients' serum Hp antibodies were tested from August 2022 to October 2023 in Zhejiang Rongjun Hospital. 84 of them were excluded because their serum gastric secretion function had not been tested. 525 patients (304 males and 221 females), aged between 14–92, who had tested both serum Hp antibodies and gastric function, were enrolled in this study (Figure 1).

The inclusion criteria were below: (i) Patients were tested for concurrent serum Hp antibody typing and serum gastric secretion function outpatient or inpatient. (ii) Not taking antibiotics or gastroprotective medications for one month. (iii) Gender and age are not limited. The exclusion criteria were below: (i) Patients with missing clinical data. (ii) Patients with partial or total gastrectomy. (iii) Suffering from severe systemic diseases.

According to the results of serum Hp antibody typing, 525 cases were classified into three groups:[18] (i)The Hp-I infected group of 103 cases with CagA and/or VacA positive. (ii) The Hp-II infected group of 62 cases with negative CagA, negative VacA and positive Ure. (iii) The Hp uninfected group of 360 cases with negative CagA, VacA and Ure. The Hp-I infected group was further divided into three subgroups: (i) Hp-I-double-positive infected group, patients with positive CagA, VacA and Ure, meaning virulent Hp infection. (ii) Hp-I-single-positive infected group, patients with positive Ure, one of CagA and VacA positive, meaning moderate cytotoxic Hp infection. (iii) Hp-I-eradication group, Hp-I patients with negative Ure, meaning eradication after Hp-I infection (Table 1, Figure 1).

Biochemical Examinations

We documented the patients' age and sex, draw venous blood when they had an empty stomach in the morning. We tested their serum Hp antibody typing with Helicobacter pylori IgG antibody test kit (protein-chip) manufactured by Taizhou syno bio technology Co., Ltd, including CagA, VacA and Ure. We tested their serum gastric secretion function indicators with PGI, PGII, G-17 combined detection kit (quantum dot fluorescence immunoassay) manufactured by Nanjing Vazyme Medical Technology Co., Ltd, including G-17, PGI, PGII and PGR.

Statistical Analysis

We used SPSS 25.0 software for statistical analysis. Data were presented using mean and standard deviation. The independent T tests were used to analyze the differences in serum gastric function indicators between Hp-infected and Hp-uninfected patients, as well as the differences in serum gastric function indicators of the same Hp antibody typing group but of different sexes or ages. One-way ANOVA was used to analyze the differences in serum gastric function indicators among groups with different Hp antibody typing groups. Pearson linear correlation analysis was used to study

Table 1 Grouping of Patients with Different Hp Antibody Types

Group	Hp-I			Hp-II	Hp (-)
N (%)	103 (19.6)			62 (11.8)	360 (68.6)
Subgroup Serum Hp antibodies	Hp-I-double-positive n=77 CagA+VacA+Ure+	Hp-I-single-positive n=18 CagA+VacA-Ure+ CagA-VacA+Ure+	Hp-I-eradication n=8 CagA+VacA+Ure- CagA+VacA-Ure- CagA-VacA+Ure-	/ CagA-VacA-Ure+	/ CagA-VacA-Ure-
Gender					
Male	59 (57.3)			44 (71.0)	201 (55.8)
Female	44 (42.7)			18 (29.0)	159 (44.2)
Age					
≤60	58 (56.3)			27 (43.5)	165 (45.8)
>60	45 (43.7)			35 (56.5)	195 (54.2)

Abbreviations: Hp, Helicobacter pylori; CagA, cytotoxin-associated protein gene A; VacA, vacuolating cytotoxin-associated protein gene A; Ure, urease.

the relationship between serum G-17, PGI, PGII and PGR. *$P < 0.05$ was considered statistically significant and **$P < 0.01$ or ***$P < 0.001$ were considered greatly significant.

Results

Serum Gastric Function Analysis of Hp Infection or Not

The serum PGII levels (8.19 ± 5.44 vs 5.98 ± 10.75, $P = 0.013$) were significantly higher in the Hp infected group than in the Hp uninfected group, while the PGR (16.81 ± 8.22 vs 23.23 ± 8.36, $P < 0.001$) was significantly lower in the Hp infected group than in the Hp uninfected group (Figure 2A and B). The levels of G-17 (44.92 ± 58.35 vs 44.65 ± 125.18, $P = 0.980$) and PGI (117.62 ± 59.81 vs 112.89 ± 108.97, $P = 0.523$) were not statistically different between the two groups (Table 2).

Serum Gastric Function Analysis of Different Hp Antibody Typings

There were statistical differences in serum PGII levels (F = 3.094, $P = 0.046$) and PGR levels (F = 33.878, $P < 0.001$) among Hp-I, Hp-II infected groups and the HP uninfected group. There was no statistical difference in the level of G-17 ($P = 0.527$), and PGI ($P = 0.804$) among the three groups. The serum PGII levels (8.19 ± 5.44 vs 5.98 ± 10.75, $P = 0.037$) were significantly higher in Hp-I infected group than in the Hp uninfected group (Figure 2C). The serum PGR levels were significantly higher in the Hp uninfected group (23.23 ± 8.36) than in Hp-I (16.47 ± 7.45, $P < 0.001$) and Hp-II infected groups (17.39 ± 8.98, $P < 0.001$) (Figure 2D).

The serum PGR levels were statistically different among Hp-I-double-positive infected group, Hp-I-single-positive infected group, Hp-I-eradication group, Hp-II infected group and Hp uninfected group (F = 19.446, $P < 0.001$). There were statistical differences in the level of PGR between Hp-I-double-positive (15.44 ± 7.03, $P = 0.003$), Hp-I-single-positive (17.26 ± 6.24, $P = 0.037$), Hp-II infected groups (17.39 ± 8.98, $P = 0.021$) and Hp-I-eradication group (24.60 ± 12.45). There were also statistical differences in the level of PGR between Hp-I-double-positive ($P < 0.001$), Hp-I-single-positive ($P < 0.001$), Hp-II infected group ($P = 0.003$) and Hp uninfected group (23.23 ± 8.36). There was no statistical difference in the levels of G-17 (F = 0.327, $P = 0.860$), PGI (F = 0.402, $P = 0.807$) and PGII (F = 1.985, $P = 0.096$) among the five groups (Figure 2E).

Serum Gastric Function Analysis of Different Sexes

Serum PGI levels were higher in male patients than in females both in Hp-I infected group (124.76 ± 56.27 vs 102.53 ± 55.26, $P = 0.048$) and the Hp uninfected group (126.43 ± 129.96 vs 95.87 ± 71.47, $P = 0.008$) (Figure 2F). Serum PGII levels were higher in males than in females in the Hp uninfected group (6.97 ± 13.58 vs 4.74 ± 5.19, $P = 0.034$) (Table 3, Figure 2G).

In males, there was statistical difference in serum PGR levels (F = 23.192, $P < 0.001$) among the Hp-I, Hp-II infected groups and Hp uninfected group. There was no statistical difference in the level of G-17 (F = 0.297, $P = 0.743$), PGI (F = 0.334, $P = 0.717$) and PGII (F = 0.690, $P = 0.502$) among the three groups. The serum PGR levels were significantly higher in the Hp uninfected group (22.99 ± 7.82) than in Hp-I (16.21 ± 7.21, $P < 0.001$) and Hp-II infected groups (17.03 ± 8.48, $P < 0.001$) (Figure 2H).

In females, there were statistical differences in serum PGI levels (F = 4.195, $P = 0.016$), PGII levels (F = 9.594, $P < 0.001$) and PGR levels (F = 33.878, $P < 0.001$) among the Hp-I, Hp-II infected groups and the Hp uninfected group. The serum PGI levels were significantly higher in Hp-II infected group (146.18 ± 86.43) than in Hp-I infected group (102.53 ± 55.26, $P = 0.027$) and the Hp uninfected group (96.87 ± 71.47, $P = 0.004$). The serum PGII levels were significantly lower in the Hp uninfected group (4.74 ± 5.19) than in Hp-I (7.15 ± 4.81, $P = 0.008$) and Hp-II infected groups (9.74 ± 6.80, $P < 0.001$). The serum PGR levels were significantly higher in the Hp uninfected group (23.54 ± 9.01) than in Hp-I (16.81 ± 8.49, $P < 0.001$) and Hp-II infected groups (18.29 ± 10.31, $P = 0.020$) (Figure 2I–K).

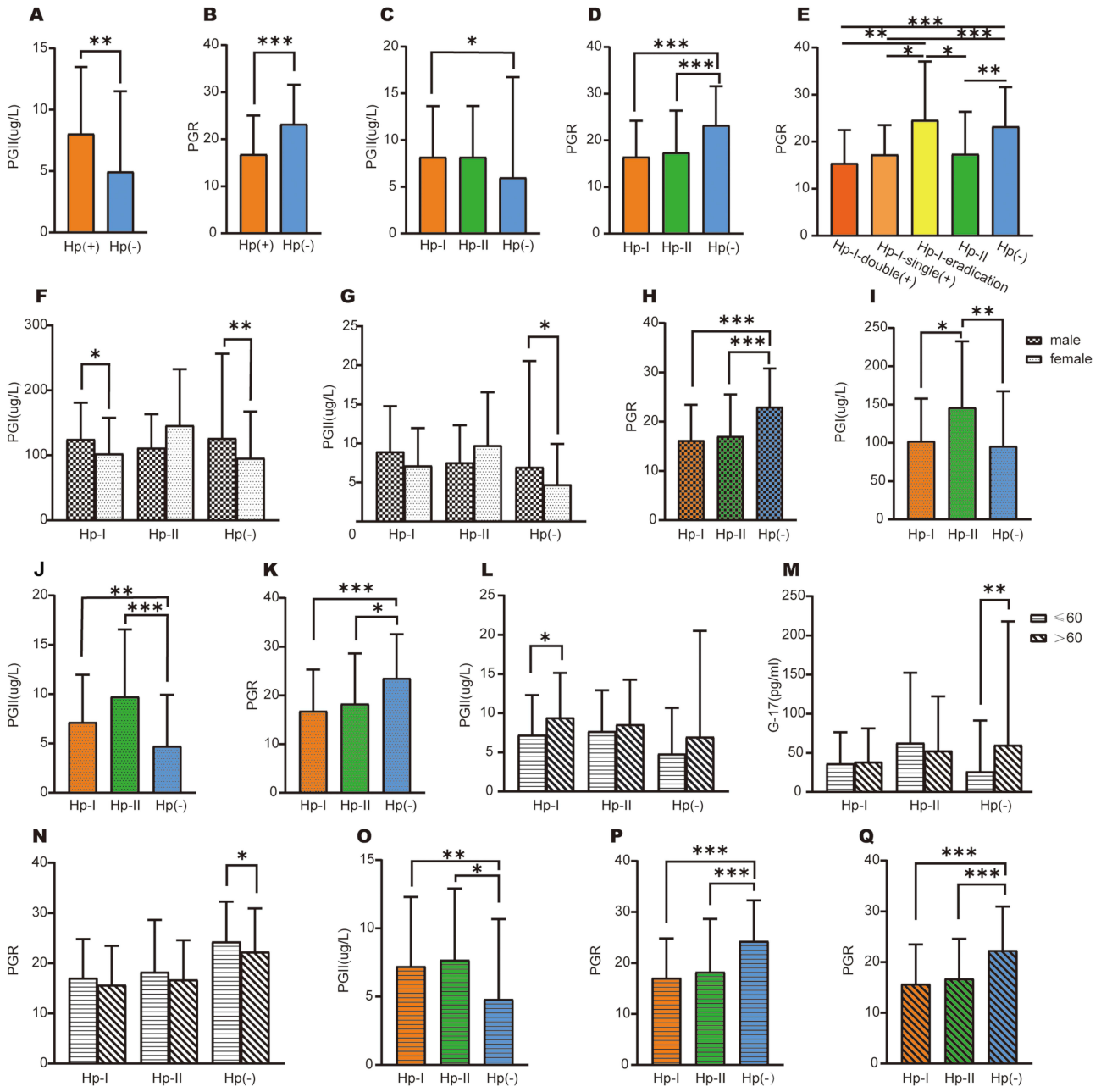

Figure 2 Analysis of gastric secretion function in patients with different Hp classifications. (**A** and **B**) PGII and PGR levels in the Hp infected and Hp uninfected groups. (**C** and **D**) PGII and PGR levels in Hp-I, Hp-II infected groups and the uninfected group. (**E**) PGR levels in the Hp-I-double-positive infected group, Hp-I-single-positive infected group, Hp-I-eradication group, Hp-II infected group and Hp uninfected group. (**F** and **G**) Comparison of PGI and PGII levels between men and women in Hp-I, Hp-II infected groups and the uninfected group. (**H**) Comparison of male PGR levels in Hp-I, Hp-II infected groups and the uninfected group. (**I** and **K**) Comparison of female PGI, PGII, PGR levels in Hp-I, Hp-II infected groups and the uninfected group. (**L** and **N**) Comparison of G-17, PGII and PGR levels between the younger and the older in Hp-I, Hp-II infected groups and the uninfected group. (**O** and **P**) Comparison of the younger's PGII and PGR levels in Hp-I, Hp-II infected groups and the uninfected group. (**Q**) Comparison of the older's PGR levels in Hp-I, Hp-II infected groups and the uninfected group. *$P < 0.05$; **$P < 0.01$; ***$P < 0.001$.

Serum Gastric Function Analysis of Different Ages

Serum PGII levels (9.43 ± 5.70 vs 7.23 ± 5.08, $P = 0.041$) were higher in patients over 60 years old in Hp-I infected group than in those less than or equal to 60 years old. In the Hp uninfected group, the G-17 levels (38.61 ± 42.64 vs 36.60 ± 39.81, $P = 0.007$) were higher in patients over 60 years old than in those less than or equal to 60 years old, while the serum PGR levels were lower (22.29 ± 8.62 vs 24.31 ± 7.95, $P = 0.022$) (Table 3, Figure 2L–N).

Table 2 Analysis of Serum Gastric Function in Hp Infected and Uninfected Groups

Gastric Function Indicators	Normal Range	Hp (+)N=165	Hp (-) n=360	T	95% CI		P
		Mean ± SD	Mean ± SD		Lower	Upper	
G-17 (pg/mL)	13–115	44.92±58.35	44.65±125.18	0.026	−19.82	20.35	0.980
PGI (ug/L)	67–200	117.62±59.81	112.89±108.97	0.638	−9.81	19.26	0.523
PGII (ug/L)	0–14.99	8.19±5.44	5.98±10.75	2.490	0.47	3.94	0.013*
PGR	>7.5	16.81±8.22	23.23±8.36	−8.206	−7.95	−4.88	0.000***

Notes: *$P < 0.05$; ***$P < 0.001$.
Abbreviations: Hp, Helicobacter pylori; G-17, Gastrin-17; PGI, Pepsinogen I; PGII, Pepsinogen II; PGR, PGI/PGII ratio.

In the patients less than or equal to 60 years old, there were statistical differences in serum PGII levels (F = 5.868, $P = 0.003$) and PGR levels (F = 19.927, $P < 0.001$) among the Hp-I infected group, Hp-II infected group and Hp uninfected group. The serum PGII levels were significantly lower in the Hp uninfected group (4.74 ± 5.19) than in Hp-I (7.15 ± 4.81, $P = 0.005$) and Hp-II infected groups (9.74 ± 6.80, $P = 0.014$). The serum PGR levels were significantly higher in the Hp uninfected group (24.31 ± 7.95) than in Hp-I (17.06 ± 7.75, $P < 0.001$) and Hp-II infected groups (18.25 ± 10.36, $P < 0.001$) (Figure 2O and P).

In the patients over 60 years old, there were statistical differences in serum PGR levels (F = 15.292, $P < 0.001$) among the Hp-I infected group, Hp-II infected group and Hp uninfected group. The serum PGR levels were significantly higher in the Hp uninfected group (22.29 ± 8.62) than in Hp-I (15.70 ± 7.77, $P < 0.001$) and Hp-II infected groups (16.74 ± 7.84, $P < 0.001$) (Figure 2Q).

Correlation Analysis of PG and G-17

In the Hp uninfected group, the level of G-17 was positively correlated with the levels of PGI (Pearson coefficient = 0.177, $P = 0.001$), PGII (Pearson coefficient = 0.140, $P = 0.008$) and age (Pearson coefficient = 0.121, $P = 0.022$), negatively with the levels of PGR (Pearson coefficient = −0.201, $P < 0.001$) (Figure 3A–D).

In Hp-I infected group, the level of G-17 was positively correlated with the levels of PGI (Pearson coefficient = 0.239, $P < 0.05$) and PGII (Pearson coefficient = 0.306, $P < 0.01$), negatively with the levels of PGR (Pearson coefficient = −0.220, $P < 0.05$). The level of G-17 was not correlated with age (Pearson coefficient = −0.003, $P = 0.975$) (Figure 3A–D).

In Hp-II infected group, the level of G-17 was positively correlated with the level of PGII (Pearson coefficient = 0.386, $P = 0.002$), negatively with the level of PGR (Pearson coefficient = −0.300, $P = 0.018$). The level of G-17 was not correlated with the level of PGI (Pearson coefficient = 0.236, $P = 0.065$) and age (Pearson coefficient = 0.018, $P = 0.161$) (Figure 3A–D).

Discussion

It is well known that PGI reflects the function of fundus gland cells. The elevated PGI indicates the increased acid secretion, while the decreased PGI indicates the decreased acid synthesis and secretion, even the atrophy of the gastric mucosa.[21] PGII is associated with the diseases of fundus and the gastric body. Its relative increase indicates atrophy or intestinalization of the fundus and the gastric body. The decreased PGR is associated with gastric mucosal atrophy.[5,22–24]

In our study, the serum PGII levels were higher in the Hp infected group than in the Hp uninfected group, especially in Hp-I infected patients, while the PGR levels were lower than in the Hp uninfected group. This result was consistent with the Zhou's study.[25] Infection with Hp causes decreased secretion of serum PGI and a relative increase in secretion of serum PGII, which reduces gastric secretion function. The effect of the cytotoxic Hp-I with CagA and/or VacA was stronger than the noncytotoxic Hp-II.[26,27] Wang's study of the diagnostic value of combined gastric function testing and Hp typing in chronic gastritis and gastric cancer found that PGI and PGR levels were reduced in Hp-I infected patients, while PG II and G-17 levels were increased compared with the Hp-II infected patients.[28] There was little difference in serological gastric function between the Hp-I and Hp-II infected groups in this study, which may be related to the Hp-I

Table 3 Analysis of Gastric Function at Different Ages and Sexes in Hp-I, Hp-II Infected Groups and the Uninfected Group

Group	Sex/ Age	n	G-17 (pg/mL)			PGI (ug/L)			PGII (ug/L)			PGR		
			Mean ± SD	t	*P*	Mean ± SD	t	*P*	Mean ± SD	t	*P*	Mean ± SD	t	*P*
Hp-I N=103	Male	59	40.24 ± 45.67	0.792	0.430	124.76 ± 56.27	1.998	0.048*	8.97 ± 5.79	1.695	0.093	16.21 ± 7.21	−0.383	0.702
	Female	44	33.76 ± 33.56			102.53 ± 55.26			7.15 ± 4.81			16.81 ± 8.49		
	≤60	58	36.60 ± 39.81	−0.246	0.806	107.90 ± 57.83	−1.507	0.135	7.23 ± 5.08	−2.075	0.041*	17.06 ± 7.75	0.887	0.377
	>60	45	38.61 ± 42.64			124.76 ± 54.27			9.43 ± 5.70			15.70 ± 7.77		
Hp-II N=62	Male	44	53.49 ± 72.96	−0.594	0.555	111.45 ± 51.79	−1.953	0.055	7.54 ± 4.78	−1.449	0.153	17.03 ± 8.48	−0.501	0.618
	Female	18	66.54 ± 91.31			146.18 ± 86.43			9.74 ± 6.80			18.29 ± 10.30		
	≤60	27	63.07 ± 89.28	0.509	0.612	117.45 ± 74.58	−0.431	0.668	7.69 ± 5.23	−0.615	0.541	18.25 ± 10.36	0.654	0.515
	>60	35	52.81 ± 69.44			124.68 ± 57.48			8.56 ± 5.71			16.74 ± 7.84		
Hp (-) N=360	Male	201	41.64 ± 112.20	−0.512	0.609	126.43 ± 129.96	2.661	0.008**	6.97 ± 13.58	2.136	0.034*	22.99 ± 7.82	−0.623	0.534
	Female	159	48.46 ± 140.15			95.87 ± 71.47			4.74 ± 5.19			23.54 ± 9.01		
	≤60	165	26.26 ± 65.14	−2.742	0.007**	101.70 ± 93.18	−1.801	0.073	4.81 ± 5.87	−1.908	0.057	24.31 ± 7.95	2.292	0.022*
	>60	195	60.21 ± 157.73			122.41 ± 120.20			6.98 ± 13.53			22.29 ± 8.61		

Notes: *$P < 0.05$; **$P < 0.01$.

Abbreviations: Hp, Helicobacter pylori; G-17, Gastrin-17; PGI, Pepsinogen I; PGII, Pepsinogen II; PGR, PGI/PGII ratio.

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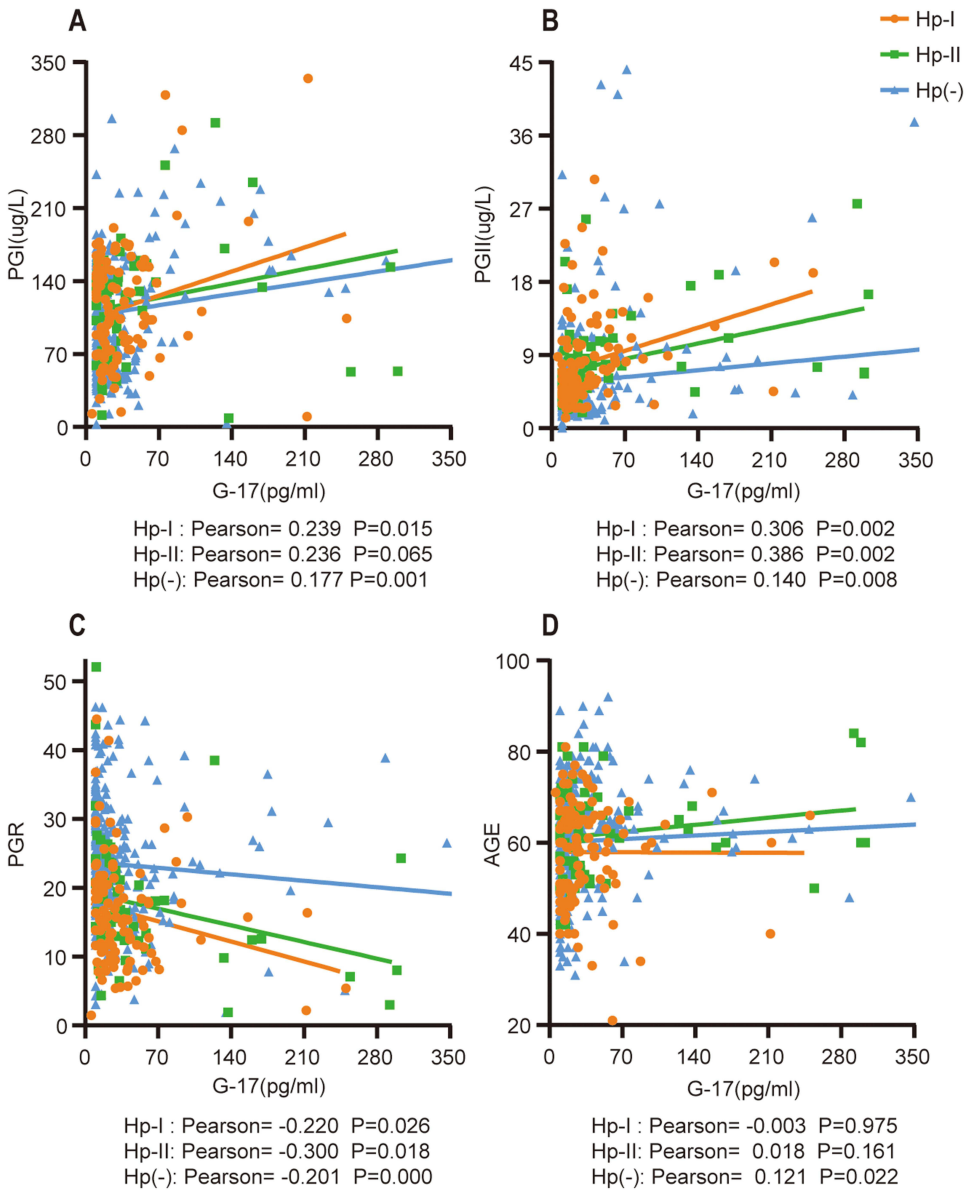

Figure 3 Pearson linear correlation analysis of gastric secretion function indicators, age and G-17 in Hp-I, Hp-II infected groups and the uninfected group. (**A**) G-17 and PGI. (**B**) G-17 and PGII. (**C**) G-17 and PGR. (**D**) G-17 and age.

infected group contained Hp-I-eradication patients. We found that PGR levels were significantly lower in Hp-I-double-positive, Hp-I-single-positive and Hp-II infected groups than in those not infected or eradicated after infection. Fukuda's research of long-term changes after Hp eradication in 5268 patients showed that patients with long term after eradication reached the uninfected condition serologically and histologically.[29] Consistent results were obtained for this study. PGR levels in patients who were eradicated after Hp infection did not differ from those who were not infected. Eradication of Hp can restore the declining gastric secretion function.

In Tsang's study, the Hp seroprevalence in the older age, male sex, smoking, lower education groups showed higher.[30] Hp seropositivity is strongly associated with poor socioeconomic conditions.[31,32] In this study, the serum levels of PGI and PGII were higher in males than in females. However, infection with either Hp-I or Hp-II causes a decrease in gastric secretory function in both male and female populations. We found the level of PGI was higher in patients with non-cytotoxic Hp infections than in those uninfected or cytotoxic Hp infections in females. Thus, we suggest that infection with non-cytotoxic Hp, whose CagA and VacA are negative, may promote the secretion of serum PGI. Di found that

PGII serology is inconsistent for the purposes of distinguishing patients whose Hp eradication therapy is successful from those who remain infected.[33] We found differences in PGII levels across groups only in females. Differences in findings among the different sex groups may be related to their different lifestyle habits and the insufficient number of patients included in the study.

Liu's study found that Serum G-17 level was higher in the Hp-positive group than in the negative group, higher in the Hp-I infected group than in the Hp-II infected group.[34] Moreover, serum G-17 level showed changes in trend proportional to the age.[35] Our study had similar results. We found age-disparate gastric secretion function such that older patients without Hp infection had higher G-17 levels and decreased PGR compared with younger patients. When G-17 secretion increased, PGI, PGII also physiologically increased, but PGR decreased. In patients with Hp-I infection, the older patients had higher PGII levels than the youngers. Infection with Hp-I or Hp-II may cause a decrease in PGR levels in patients of all ages. The G-17 levels increase with age, but if the patients are infected with Hp, this physiological change in G-17 will be affected. When the patients are infected with Hp-II, the secretion changes in PGI will not synchronize with G-17. From this we sensed the pathological effect of Hp infection on gastric secretion function.

Although a large number of patients were included in this study, the number of patients in each group is unbalanced. Our assessment of Hp infection based solely on the Hp antibody test, which may affect the results. Therefore, we should keep the number of cases in each group balanced and use other detection methods in the future research.

Conclusion

In conclusion, our study found that the Hp-I and Hp-II have a negative impact on the gastric secretion function. After Hp eradication, the patients' gastric function returns to physiological state without Hp infection. This study provides valuable information for the eradication treatment of different Hp types and has important implications for the diagnosis and treatment of Hp-associated diseases.

Data Sharing Statement

The data supporting the findings of this study are available from the corresponding author upon reasonable request.

Ethics Approval and Informed Consent

The study was approved by the Ethics Committee of Zhejiang Rongjun Hospital. Since this study used the clinical data obtained in previous clinical treatment, the exemption from informed consent had been approved. All patient data was confidential and we complied with the Declaration of Helsinki.

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Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

The author(s) report no conflicts of interest in this work.

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