ISSN 2307-8960 (online)

World Journal of *Clinical Cases*

World J Clin Cases 2021 November 16; 9(32): 9699-10051

Published by Baishideng Publishing Group Inc

World Journal of
Clinical Cases

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Thrice Monthly Volume 9 Number 32 November 16, 2021

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World Journal of Clinical Cases

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Thrice Monthly Volume 9 Number 32 November 16, 2021

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NAME OF JOURNAL
World Journal of Clinical Cases

ISSN
ISSN 2307-8960 (online)

LAUNCH DATE
April 16, 2013

FREQUENCY
Thrice Monthly

EDITORS-IN-CHIEF
Dennis A Bloomfield, Sandro Vento, Bao-Gan Peng

EDITORIAL BOARD MEMBERS
https://www.wjgnet.com/2307-8960/editorialboard.htm

PUBLICATION DATE
November 16, 2021

INSTRUCTIONS TO AUTHORS
https://www.wjgnet.com/bpg/gerinfo/204

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https://www.wjgnet.com/bpg/GerInfo/288

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https://www.wjgnet.com/bpg/gerinfo/242

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ONLINE SUBMISSION
https://www.f6publishing.com

World Journal of
Clinical Cases

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World J Clin Cases 2021 November 16; 9(32): 9815-9824

DOI: 10.12998/wjcc.v9.i32.9815

ISSN 2307-8960 (online)

ORIGINAL ARTICLE

Retrospective Study

Association between *Helicobacter pylori* infection and food-specific immunoglobulin G in Southwest China

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Author contributions: Liu Y and Li DY contributed to the conception and design of the work; Liu Y and Shuai P contributed to the acquisition, analysis and interpretation of data for the study; Liu Y contributed to drafting the work; Li DY and Liu YP revised the manuscript; Li DY was accountable for all aspects of the work.

Supported by Key Research and Development Projects of the Ministry of Science and Technology, China, No. 2017YFC0113901.

Institutional review board statement: The study was reviewed and approved by the Ethical Committee of Sichuan Academy of Medical Sciences and Sichuan Provincial People's Hospital [Approval No. 408(2020)].

Conflict-of-interest statement: The authors declare that they have no conflicts of interest.

Data sharing statement: No additional data are available.

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Abstract

BACKGROUND

Helicobacter pylori (*H. pylori*) has been found to be associated with extragastrointestinal diseases, possibly including adverse food reactions (such as food allergy or intolerance). However, there are few studies on *H. pylori* and food allergy or intolerance, and the results are inconsistent. Food-specific immunoglobulin (Ig) G has been revealed to be associated with food allergy or intolerance and can be used as a marker to explore the correlation between *H. pylori* infection and food allergy or intolerance.

AIM

To explore the relationship between *H. pylori* infection and food-specific IgG

METHODS

We retrospectively analyzed the physical examination data of 21822 subjects from February 2014 to December 2018 in this study. *H. pylori* infection was detected using the ^{13}C urea breath test. Food-specific IgG of eggs, milk and wheat in serum was assessed. Subjects were grouped according to *H. pylori* positivity, and the positive rates of three kinds of food-specific IgG were compared between the two groups. Multivariable logistic regression analysis was performed to elucidate the association between *H. pylori* infection and food-specific IgG.

RESULTS

The total infection rate of *H. pylori* was 39.3%, and the total food-specific IgG-positive rates of eggs, milk and wheat were 25.2%, 9.0% and 4.9%, respectively.

The infection rate of *H. pylori* was higher in males than in females, while the positive rates of food-specific IgG were lower in males than in females. The positive rates of food-specific IgG decreased with age in both males and females. In the *H. pylori*-positive groups, the positive rates of food-specific IgG of eggs, milk and wheat were all lower than those in the *H. pylori*-negative groups. Multivariate logistic regression analysis revealed that *H. pylori* infection was negatively correlated with the food-specific IgG-positive rates of eggs, milk and wheat (odds ratio value of eggs 0.844-0.873, milk 0.741-0.751 and wheat 0.755-0.788, in different models).

CONCLUSION

H. pylori infection was found to be negatively associated with the food-specific IgG of eggs, milk and wheat in Southwest China.

Key Words: Food-specific IgG; *Helicobacter pylori*; Adverse food reaction; Food allergy; Food intolerance; Humoral immunity

Core Tip: This is a retrospective study to evaluate the association of *Helicobacter pylori* (*H. pylori*) infection and food-specific immunoglobulin G. We analyzed the data of 21822 subjects who underwent *H. pylori* infection assessment by the urea breath test and testing for food-specific immunoglobulin G of eggs, milk and wheat. The key finding was that *H. pylori* infection was associated with lower positivity for food-specific immunoglobulin G. If the negative correlation could be further confirmed and the mechanism could be clarified, it would provide some advisable suggestions for medical decisions regarding asymptomatic *H. pylori* infection.

Citation: Liu Y, Shuai P, Liu YP, Li DY. Association between *Helicobacter pylori* infection and food-specific immunoglobulin G in Southwest China. *World J Clin Cases* 2021; 9(32): 9815-9824

URL: https://www.wjgnet.com/2307-8960/full/v9/i32/9815.htm

DOI: https://dx.doi.org/10.12998/wjcc.v9.i32.9815

Specialty type: Medicine, research and experimental

Country/Territory of origin: China

Peer-review report's scientific quality classification

Grade A (Excellent): 0
Grade B (Very good): B
Grade C (Good): C
Grade D (Fair): 0
Grade E (Poor): 0

Received: June 27, 2021
Peer-review started: June 27, 2021
First decision: July 14, 2021
Revised: July 19, 2021
Accepted: September 1, 2021
Article in press: September 1, 2021
Published online: November 16, 2021

P-Reviewer: Reshetnyak VI, Toyoshima O
S-Editor: Chang KL
L-Editor: Filipodia
P-Editor: Guo X

INTRODUCTION

Helicobacter pylori (*H. pylori*) infection, which is considered a major pathogenic factor in chronic gastritis, gastric ulcer and gastric cancer, is an important public health issue worldwide[1]. However, growing evidence suggests that *H. pylori* infection affects not only the gastrointestinal tract but also extragastrointestinal function, which has become a research hotspot. In contrast to the traditional view that *H. pylori* is a risk factor for disease, some studies have found a negative correlation between *H. pylori* infection and the development of certain diseases. For example, *H. pylori* infection showed a negative correlation with the development of some allergic diseases, such as asthma and eosinophilic esophagitis, especially in children and young people with early allergic reactions[2].

Notably, the relationship between food and chronic diseases has received increasing attention. Specific epitopes of food can be used as specific antigens to induce the immune response of the body, thus producing food-specific antibodies. Food allergy related to the classic pathway, which can be mediated by food-specific immunoglobulin (Ig)E, is well known by scholars. Few studies have researched the relationship between *H. pylori* infection and food allergy, and the results remain controversial[3]. In recent years, the correlation between food-specific IgG and a variety of allergic diseases or symptoms has attracted the attention of scholars and has been found to be related to irritable bowel syndrome[4], inflammatory bowel disease[5], eosinophilic esophagitis[6] and other autoimmune diseases[7]. The role of food-specific IgG in food allergy has also been discussed, and its application value in non-IgE-mediated detection of food adverse reactions has been affirmed by international authoritative guidelines[8].

Food intolerance is another common adverse food reaction. Although the pathogenesis of food intolerance is not directly related to immunity, some scholars indicate increased gut permeability in patients with food intolerance, which permits food substances to gain access to the circulation and trigger food-specific IgG production; thus, a correlation may also exist between food intolerance and food-specific IgG. Fewer studies have directly discussed the relationship between *H. pylori* infection and food intolerance. A study of 12765 people in North China by Sai *et al*[9] suggested that crab intolerance may be related to *H. pylori* infection.

In China, adverse reactions to food may be affected by various socioeconomic factors, eating habits, food types, geographical climates and so on[10]. Our study focused on food types and serum food-specific IgG. The three types of food—egg, milk and wheat—are widely consumed in Southwest China, where there is a relatively high positive rate of serum food-specific IgG. In this study, we used these three foods to explore the association between *H. pylori* infection and serum food-specific IgG in Southwest China.

MATERIALS AND METHODS

Participants

The physical examination data of the subjects were obtained from the Health Management Center, Sichuan Provincial People's Hospital (Chengdu, Sichuan Province). All the subjects completed the medical history questionnaire. Physical examinations, which included height, body weight and blood pressure, were performed by trained nurses. All subjects underwent laboratory examinations (routine blood tests and measurement of alanine aminotransferase, aspartate aminotransferase, gamma-glutamyl transpeptidase, serum creatinine, fasting blood glucose, hemoglobin A1c, total cholesterol, triglycerides, low-density lipoprotein cholesterol, high-density lipoprotein cholesterol, and uric acid), abdominal ultrasonography, chest imaging (X-ray or computed tomography), ^{13}C urea breath tests and testing for food-specific IgG of eggs, milk and wheat.

Subjects were excluded if they had: (1) A history of gastrectomy or subtotal gastrectomy; (2) Organic diseases that have been identified to affect gastrointestinal digestion and absorption; (3) An inability to perform the ^{13}C urea breath tests due to pregnancy, lactation or other reasons; (4) Immune system diseases, severe heart, liver or kidney dysfunction or tumors; or (5) A history of anti-*H. pylori* therapy in the past 6 mo.

All methods were carried out based on relevant guidelines and regulations. Ethics approval was obtained from the Ethical Committee of Sichuan Academy of Medical Sciences and Sichuan Provincial People's Hospital. Approval No. 408(2020).

H. pylori infection test

H. pylori infection was detected using ^{13}C urea breath testing (Beijing Boran Pharmaceutical Co., Ltd. Beijing, China), according to the recommendation of the Fifth Chinese National Consensus Report on the management of *H. pylori* infection[11]. All subjects fasted overnight for more than 8 h, maintained normal breathing, inserted a straw into the bottom of one sample tube, and exhaled slowly into the sample tube through the straw for 4 to 5 s. Thereafter, they pulled the straw out and tightened the cap immediately; this was considered a sample of zero points. Then, the subjects took another bottle with urea ^{13}C granules and 80 mL to 100 mL cold drinking water, rested for 30 min, and then collected another breath sample. The two collected gas samples were tested for $^{13}CO_2$, and δ‰ was used to represent the result: δ‰ = (isotopic abundance of ^{13}C for the test sample - isotopic abundance of ^{13}C for the reference sample) × 1000/isotopic abundance of ^{13}C for the reference sample. The detection value was defined as the δ‰ measured at 30 min subtracted from that measured at 0 min. *H. pylori* infection was considered positive when the detection value was ≥ 4.0.

Food-specific IgG test

A food-specific IgG screening enzyme-linked immunosorbent assay kit (HOB Biotech Co., Ltd. Jiangsu, China) was used. Serum samples were collected from the subjects, the amount used was 5 μL, and the test was carried out according to the operation manual. A blank well was used to calibrate the zero value of the enzyme analyzer [Thermo Fisher Scientific (China) Co., Ltd. Shanghai, China] at a wavelength of 450 nm, and the absorbance value Y of each tested sample was read. The standardized activity value X (U/mL) was obtained with the formula $Y = AX^3 + BX^2 + CX + D$

calculated from the standard curve. An activity value of X ≥ 50 U/mL was defined as food-specific IgG positive.

Statistical analysis

Statistical analysis was performed using IBM SPSS 21.0 (IBM Corp., Armonk, NY, United States). Continuous data were expressed as the mean ± standard deviation for normally distributed data or the median with 25th and 75th percentiles for non-normally distributed data. Categorical data were described as percentages. Student's *t*-test was used to analyze continuous variables, and the χ^2 test was used to analyze categorical variables. Univariable and multivariable regression models were performed using logistic regression analysis to identify the association between *H. pylori* infection and food-specific IgG. Various covariates, such as age, sex, body mass index, hemoglobin A1c, total cholesterol, triglycerides, alanine aminotransferase, aspartate aminotransferase, gamma-glutamyl transpeptidase, serum creatinine, uric acid, blood pressure, smoking and drinking, were used to adjust the confounding factors, with the results expressed as odds ratios (ORs) and 95% confidence intervals. A P value < 0.05 was considered statistically significant.

RESULTS

Baseline of the study population characteristics

The demographic and laboratory baseline characteristics of 21822 subjects (12396 males and 9426 females) are shown in Table 1. The average age was 43.82 ± 10.98 years (range: 18-89 years). The total infection rate of *H. pylori* was 39.3%, and the food-specific IgG-positive rates of eggs, milk and wheat were 25.2%, 9.0% and 4.9%, respectively. The infection rate of *H. pylori* was higher in males than in females (39.9% *vs* 38.6%, P = 0.043). The food-specific IgG-positive rates of the three foods in males were all significantly lower than those in females (20.4% *vs* 31.5% for eggs, 7.9% *vs* 10.5% for milk and 4.0% *vs* 6.2% for wheat, all P < 0.001).

The subjects were further stratified by age to investigate the prevalence of *H. pylori* infection and positive rates of food-specific IgG. The results revealed that the positive rates of the three food-specific IgG antibodies all decreased with age in both males and females (Table 2).

Comparison of the positive rates of food-specific IgG between different H. pylori infection status groups

Whether in the general population or between sexes, the positive rates of the three food-specific IgG antibodies in the *H. pylori*-positive group were all significantly lower than those in the *H. pylori*-negative group (22.8% *vs* 26.7% for eggs, 7.4% *vs* 10.1% for milk and 3.9% *vs* 5.3% for wheat) (Figure 1).

Logistic regression analysis of H. pylori infection and food-specific IgG positivity

Logistic regression analysis was performed to explore the independent association between *H. pylori* infection and food-specific IgG. In univariate analysis, the results revealed that *H. pylori* infection was associated with a lower risk of food-specific IgG (OR = 0.814, P < 0.001 for eggs; OR = 0.714, P < 0.001 for milk; and OR = 0.720, P < 0.001 for wheat). After adjusting for confounding factors in different models, the results remained significant (OR value of egg 0.844-0.873, milk 0.741-0.751 and wheat 0.755-0.788, P < 0.001) (Table 3).

DISCUSSION

The infection rate of *H. pylori* is high worldwide and is 50% in China[12]. However, compared with the high *H. pylori* infection rate, only 15%-20% of infected subjects have peptic ulcers, 5%-10% have *H. pylori*-related dyspepsia, and approximately 1% have gastric cancer, mucosa-associated lymphoid tissue lymphoma and other gastric malignant tumors[13-15]. Most of the infected subjects are asymptomatic and do not receive drug treatment. Scholars have focused on exploring the chronic process in such a large number of asymptomatic carriers. Moreover, the influence of *H. pylori* infection is not limited to the gastrointestinal tract itself. In 1994, Mendall *et al*[16] first reported the relationship between *H. pylori* infection and extragastric diseases. Subsequently, neurological, cardiovascular, hematologic, dermatological, ocular, metabolic and

Table 1 Demographic and clinical characteristics of the participants

Variables	Total, *n* = 21822	*H. pylori* negative, *n* = 13239	*H. pylori* positive, *n* = 8583	*P* value
Demographic data				
Sex (female), *n* (%)	9426 (43.2)	5791 (43.7)	3635 (42.4)	0.043
Age (yr)	43.82 ± 10.98	43.49 ± 11.10	44.32 ± 10.76	< 0.001
Drinking, *n* (%)	2295 (10.5)	1304 (9.8)	991 (11.5)	< 0.001
Smoking, *n* (%)	4578 (21.0)	2661 (20.1)	1917 (22.3)	< 0.001
Anthropometric data				
Body weight (kg)	64.08 ± 12.02	63.52 ± 11.83	64.94 ± 12.26	< 0.001
Height (cm)	163.65 ± 8.23	163.48 ± 8.26	163.90 ± 8.18	< 0.001
BMI (kg/m^2)	23.81 ± 3.37	23.65 ± 3.33	24.05 ± 3.41	< 0.001
SBP (mmHg)	117.43 ± 17.08	117.28 ± 16.92	117.67 ± 17.31	0.099
DBP (mmHg)	72.86 ± 11.39	72.76 ± 11.24	73.01 ± 11.62	0.109
Laboratory data				
ALT (U/L)	23 (16, 36)	23 (16, 36)	24 (16, 38)	< 0.001
AST (U/L)	27.20 ± 19.03	27.26 ± 21.89	27.10 ± 13.47	0.530
GGT (U/L)	24 (15, 42)	23 (15, 41)	24 (15, 45)	< 0.001
Creatinine (μmol/L)	67.24 ± 21.41	67.01 ± 23.54	67.58 ± 17.62	0.058
Fasting glucose (mmol/L)	5.11 ± 1.33	5.07 ± 1.24	5.16 ± 1.46	< 0.001
HbA1c (%)	5.54 ± 0.79	5.51 ± 0.74	5.58 ± 0.87	< 0.001
Total cholesterol (mmol/L)	4.86 ± 0.95	4.84 ± 0.94	4.89 ± 0.96	< 0.001
Triglycerides (mmol/L)	1.38 (0.95, 2.08)	1.67 (1.15, 2.45)	1.09 (0.80, 1.56)	< 0.001
LDL-C (mmol/L)	2.87 ± 0.81	2.86 ± 0.81	2.90 ± 0.83	< 0.001
HDL-C (mmol/L)	1.33 ± 0.33	1.33 ± 0.33	1.31 ± 0.33	< 0.001
Uric acid (μmol/L)	345.40 ± 90.58	343.64 ± 90.40	348.11 ± 90.80	< 0.001

H. pylori: *Helicobacter pylori*; BMI: Body mass index; SBP: Systolic pressure; DBP: Diastolic pressure; ALT: Alanine aminotransferase; AST: Aspartate aminotransferase; GGT: Gamma-glutamyl transpeptidase; HbA1c: Hemoglobin A1c; LDL-C: Low-density lipoprotein cholesterol; HDL-C: High-density lipoprotein cholesterol.

allergic diseases were found to be associated with *H. pylori* infection[17]. Immune mechanisms may play an important role in the relationship between *H. pylori* infection and extragastrointestinal diseases[18]. In consideration of the high *H. pylori* infection rate, the relationship between *H. pylori* and many other extragastrointestinal diseases cannot be ignored.

Over the years, adverse food reactions, which can be classified as food allergy or intolerance, have been increasing and have received more attention. Immune factors are very important in the pathogenesis of adverse food reactions. As an immune-based disease, food allergy is estimated to affect 5% of children under the age of 5 years and 4% of teens and adults in the United States[19]. Classic food allergy is usually identified as IgE-mediated immediate hypersensitivity reactions. However, with the development of research, delayed non-IgE-mediated reactions have also been included in the mechanism of food allergy[20]. IgG is the immunoglobulin with the highest serum content, accounting for 70%-75%; IgG can be divided into IgG1, IgG2, IgG3 and IgG4 subtypes, and the normal body content is approximately 66%, 23%, 7% and 4%, respectively[21]. Unlike IgE-mediated type I hypersensitivity (immediate hypersensitivity), IgG is mainly involved in type II (cytotoxic hypersensitivity) and type III hypersensitivity (immune complex-mediated hypersensitivity)[22]. The immune system can identify certain food molecules as harmful substances and produce an excessive protective immune response against these substances, generating food-specific IgG. Through antigen-antibody reactions, IgG antibodies form circulating

Table 2 Prevalence of *Helicobacter pylori* infection and food-specific immunoglobulin G positivity in different age groups

Age in yr	Number	*H. pylori* infection, *n* (%)	Food-specific IgG positivity, *n* (%)		
			Egg	Milk	Wheat
Male					
18-29	903	296 (32.8)	409 (45.3)	188 (20.8)	126 (14.0)
30-39	3258	1292 (39.7)	858 (26.3)	350 (10.7)	167 (5.1)
40-49	4714	1835 (38.9)	746 (15.8)	256 (5.4)	126 (2.7)
≥ 50	3521	1525 (43.3)	512 (14.5)	188 (5.3)	71 (2.0)
Total	12396	4948 (39.9)	2525 (20.4)	982 (7.9)	490 (4.0)
Linear by linear association value		26.981	436.396	260.529	212.714
P value		< 0.001	< 0.001	< 0.001	< 0.001
Female					
18-29	978	311 (31.8)	535 (54.7)	199 (20.3)	100 (10.2)
30-39	2747	1031 (37.5)	1102 (40.1)	361 (13.1)	197 (7.2)
40-49	3263	1355 (41.5)	795 (24.4)	250 (7.7)	198 (6.1)
≥ 50	1587	938 (38.5)	535 (21.9)	181 (7.4)	91 (3.7)
Total	9426	3635 (38.6)	2967 (31.5)	991 (10.5)	586 (6.2)
Linear by linear association value		12.391	462.821	143.539	54.716
P value		< 0.001	< 0.001	< 0.001	< 0.001

H. pylori: *Helicobacter pylori*; IgG: Immunoglobulin G.

Table 3 The risk of food-specific immunoglobulin G positivity according to *Helicobacter pylori* infection

	Non-adjusted	Model 1	Model 2	Model 3
Eggs	0.814 (0.764-0.867)	0.844 (0.791-0.901)	0.873 (0.812-0.938)	0.871 (0.810-0.936)
Milk	0.714 (0.647-0.788)	0.741 (0.671-0.818)	0.751 (0.669-0.842)	0.746 (0.665-0.838)
Wheat	0.720 (0.631-0.820)	0.755 (0.662-0.861)	0.787 (0.681-0.909)	0.788 (0.682-0.910)

Model 1: adjusted for sex and age; Model 2: adjusted for Model 1 plus body mass index, hemoglobulin A1c, total cholesterol, triglycerides, drinking and smoking; Model 3: adjusted for Model 2 plus alanine aminotransferase, aspartate aminotransferase, gamma-glutamyl transpeptidase, creatinine, uric acid, systolic pressure and diastolic pressure.

immune complexes with food particles that are deposited in various organs or systems *via* blood circulation. Therefore, food-specific-IgG may participate in the mechanism of non-IgE-mediated adverse food reactions, which is related to food allergy[8].

Food intolerance is another common chronic disease with many extragastrointestinal clinical manifestations and affects 15%-20% of the population[10]. The mechanism of food intolerance is multifactorial and is related to digestive system factors such as food composition, metabolic enzyme activity, transport mechanisms and intestinal permeability changes. Although food intolerance is defined as not directly related to the immune response, considering the mechanism of food intolerance mentioned above, more allergenic food components may enter the circulation through digestion in individuals with food intolerance, thus inducing the production of food-specific IgG[23]. Specific IgG antibodies that corresponding to certain foods could be detected in the serum of food-intolerant patients[24]. Therefore, food-specific IgG can also be indirectly or secondarily correlated with food intolerance. The high prevalence and chronic process of food allergy or intolerance as well as its relationship with the digestive system and immune system are similar to those of *H. pylori* infection, which made us interested in exploring the association between *H. pylori* infection and food-specific IgG.

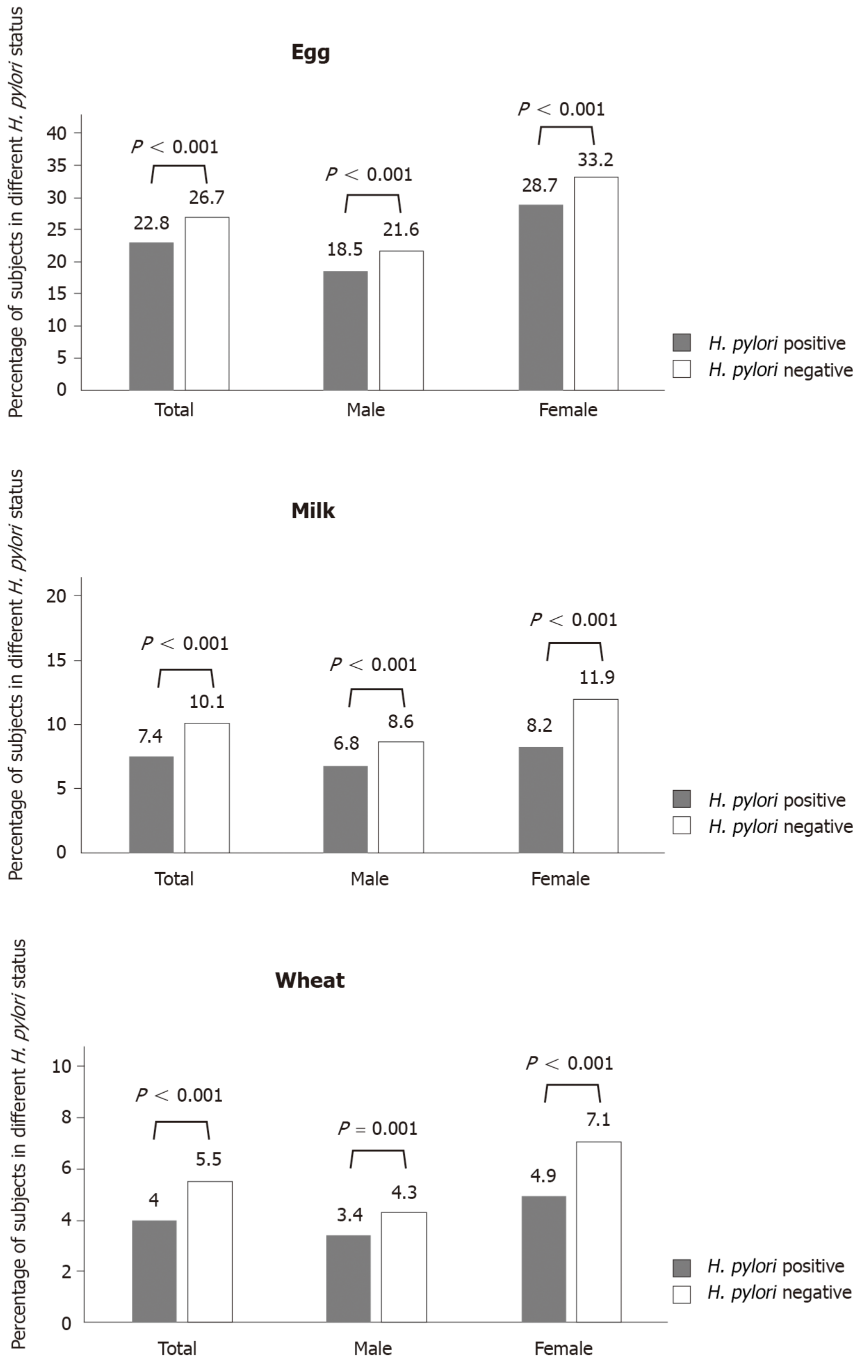

Figure 1 Positive rates of the three food-specific immunoglobulin G antibodies. *H. pylori*: *Helicobacter pylori*.

To date, studies on the relationship between food allergy or intolerance and *H. pylori* infection in large samples are limited. In our study, we analyzed the physical examination data of more than 20000 subjects. We selected eggs, milk and wheat as the research objects, as they are commonly consumed in Southwest China where there is a relatively high positive rate of food-specific IgG. The results suggested that *H. pylori* infection seemed to help the body achieve a lower rate of food-specific IgG positivity. Interestingly, our results were in contrast to those of a similar study in China[9]. The differences might be related to the sample size, food types and geographical differences, which need to be further studied.

Previous studies have found that *H. pylori* infection affects immune regulation so that *H. pylori* can avoid immune surveillance to establish long-term colonization. This may also be the cause of its association with some extragastrointestinal diseases[25]. For example, asthma has been reported to be inversely associated with *H. pylori* infection. The protective effects of *H. pylori* depend on Foxp3+ regulatory T cells[26]. Regulatory T cells are a potently immunosuppressive CD4+ T cell subset and play a key role in immune tolerance by controlling the extent of the response to self- and nonself-antigens. These cells can promote the rapid recovery of immune homeostasis[27]. *H. pylori* also upregulates the expression of CD80 and interleukin 10 *via* toll-like receptors on B lymphocytes and then promotes regulatory T cell differentiation[28]. Idiopathic thrombocytopenic purpura, an autoimmune disorder, was found to be associated with *H. pylori* infection in 1999[29]. One of the mechanisms involves an enhanced phagocytic capacity and low levels of inhibitory FcγRIIB in monocytes from *H. pylori*-infected patients, leading to increased monocyte autoreactivity with B and T lymphocytes. This may cause B lymphocytes to produce autoantibodies against circulating platelets[18]. Therefore, *H. pylori* may be related to some extragastrointestinal diseases through the regulation of both cellular and humoral immunity. The symptoms of both food allergy and intolerance are related to humoral immunity mediated by IgG. Future research on humoral immunity may be helpful for understanding the correlation between *H. pylori* infection and food allergy or intolerance.

A limitation of our study was that the subjects were from the health examination population rather than from a random sampling of the community, which led to sample deviation. Furthermore, our study lacked sociological data. Previous studies have revealed that the *H. pylori* infection rate is higher in developing countries[30]. Poor health conditions, low socioeconomic status and associated unhealthy dietary hygiene habits may facilitate exposure to more bacteria or antigens, which will promote immune tolerance to the corresponding antigens in the body and reduce the risk of adverse food reactions. Therefore, the two flowers—higher *H. pylori* infection rates and lower rates of food-specific IgG positivity—may both grow in the common soil of poor socioeconomic conditions mentioned above. Our study found that there may be a correlation between *H. pylori* infection and food-specific IgG, and whether there is a causal relationship and the mechanism between them require further study.

H. pylori is considered an important risk factor for gastric ulcer and gastric cancer. Aggressive drug therapy is recommended for patients who meet the indications[31]. However, our study found a negative correlation between *H. pylori* infection and food-specific IgG, which was not consistent with the commonly held perception of *H. pylori*. Considering the “beneficial protective effect” of *H. pylori* in some diseases as well as its high infection rate and the relatively limited proportion of symptomatic infected individuals in a population, some researchers have reassessed the role of such bacteria in the human body and proposed the question of whether *H. pylori* is a “commensal, symbiont or pathogen”[32]. Our results seem to provide a positive evaluation of *H. pylori* in discussing this issue and suggest that we need more individualized understanding of the effect of *H. pylori* on the body’s immunity. Further confirmation of the negative correlation found in our study and clarification of the mechanism in future studies would provide some advisable suggestions for medical decisions.

CONCLUSION

In conclusion, *H. pylori* infection was found to be negatively associated with the food-specific IgG of eggs, milk and wheat in Southwest China.

ARTICLE HIGHLIGHTS

Research background

Helicobacter pylori (*H. pylori*) has been found to be associated with extragastrointestinal diseases, possibly including adverse food reactions (such as food allergy or intolerance). However, there are few studies on *H. pylori* and food allergy or intolerance, and the results are inconsistent.

Research motivation

Food-specific immunoglobulin (Ig) G has been revealed to be associated with food allergy or intolerance and can be used as a marker to explore the correlation between *H. pylori* infection and food allergy or intolerance.

Research objectives

To explore the relationship between *H. pylori* infection and food-specific IgG.

Research methods

H. pylori infection was detected with the ^{13}C urea breath test. Food-specific IgG of eggs, milk and wheat was detected in serum. Subjects were grouped according to *H. pylori* positivity, and the positive rates of three kinds of food-specific IgG were compared between the two groups. Multivariable logistic regression analysis was performed to identify the association between *H. pylori* infection and food-specific IgG.

Research results

In the *H. pylori*-positive groups, the positive rates of food-specific IgG of eggs, milk and wheat were all lower than those in the *H. pylori*-negative groups. Multivariate logistic regression analysis showed that *H. pylori* infection was negatively correlated with the food-specific IgG-positive rates of eggs, milk, and wheat.

Research conclusions

H. pylori infection was negatively correlated with the food-specific IgG of eggs, milk and wheat in Southwest China.

Research perspectives

Our study might reflect only a negative association between *H. pylori* infection and food-specific IgG rather than causality. Establishing relevant animal models and exploring the underlying mechanism based on immunity or a well-designed clinical intervention study may help to verify our findings. Moreover, finding additional similar "protective" effects in asymptomatic patients with *H. pylori* infection may help us reassess the role of *H. pylori* in the body and provide advisable suggestions for medical decisions.

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