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Original Article

Helicobacter pylori infection and its risk factors in subjects receiving physical examination

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Abstract: Aim: This study aimed to analyze helicobacter pylori (Hp) risk factors, which may provide evidence for the prevention and treatment of Hp infection. Methods: 8115 subjects were recruited. Questionnaire surgery was done in 487subjects who received gastroscopy. Results: The incidence of Hp infection was 35.2% in males and 30.3% in females showing significant difference (P<0.05). The mean age was 48.65±13.55 years in Hp positive subjects and 45.77±14.10 years in Hp negative subjects, showing marked difference (P<0.05). The body mass index, uric acid, blood glucose, triglycerides and low-density lipoprotein cholesterol in Hp infection subjects were significantly higher than in Hp negative subjects (P<0.05), but the high-density lipoprotein cholesterol in Hp positive subjects was markedly lower than in Hp negative subjects (P<0.05). In 487 subjects, the incidence of Hp infection was 53.6%, which was significantly higher than in healthy subjects (P<0.05). The incidence of Hp infection in subjects living in city, with family members having Hp infection, drinking running water, smoking or drinking alcohol was significantly higher than in controls (P<0.05). The incidence of Hp infection was different between subjects with different gastric diseases (P<0.05). The incidence of Hp infection was the highest in subjects with peptic ulcer and the lowest in those with esophagitis. Conclusion: Hp infection is related to gender, age, body weight, living environments and living habits, which provide evidence for the prevention of Hp infection. Hp infection is closely related to the pathogenesis of diabetes and cardiovascular diseases.

Keywords: Helicobacter pylori, gastric disease, risk factors

Introduction

Helicobacter pylori (Hp) was identified in 1982 by Australian scientists Barry Marshall and Robin Warren who were awarded with the 2005 Nobel Prize in Physiology or Medicine. It is a Gram-negative, microaerophilic bacterium found usually in the mucus layer of the stomach and duodenum. The prevalence of Hp infection is higher than 50% worldwide, and Hp infection is mainly found in developing countries. However, 90% of subjects with Hp infection have no clinical symptoms, which might be related to the toxicity of Hp strains. The transmission route of Hp infection is still unclear, oral-oral route is widely accepted one, and Hp infection usually occurs in childhood. Hp infection has been one of the most common chronic gastric diseases and may initiate a series of pathogenic events, resulting in atrophic gastritis, metaplasia, dysplasia or even gastric cancer. Thus, to eradicate Hp may prevent the occurrence and development of gastric precancerous lesions and significantly reduce the risk for gastric precancerous lesions and gastric cancer. Hp not only causes gastric cancer, but is closely related to the pathogenesis of some chronic diseases including ulcerative colitis, rheumatoid arthritis, atherosclerosis, hypertension, diabetes, anemia and idiopathic thrombocytopenic purpura. As said by the Nobel Committee, "the discovery of Hp deepens the understanding of the relationship among chronic infection, inflammation and cancer in humans". Hp infection is a refractory disease, regular therapy is usually needed once it is present, and the spontaneous disappearance of Hp infection is almost impossible. Thus, it is necessary to detect Hp infection in high risk population.

Table 1. Gender and age of subjects recruited into present study

Group	Hp positive (%)	Hp negative (%)	*P* value
Total	2628 (32.4)	5487 (67.6)	
Gender			P=0.000
M	1302 (35.2)	2395 (64.8)	
F	1326 (30.0)	3092 (70.0)	
Mean age	48.65±13.55	45.77±14.10	P=0.000
Age group			P=0.000
≤29	181 (20.5)	700 (79.5)	
30~39	544 (28.1)	1395 (71.9)	
40~49	742 (35.0)	1380 (65.0)	
50~59	527 (36.4)	921 (63.6)	
60~69	435 (36.7)	751 (63.3)	
≥70	199 (36.9)	340 (63.1)	

Table 2. Logistic regression of risk factor of HP infection

Indicator	*P* value	OR value	95% CI	
Gender	0.019	1.144	1.022	1.281
Age	0.000	1.014	1.011	1.019
BMI	0.127	1.011	0.997	1.027
Glucose	0.406	1.015	0.980	1.051
TG	0.012	1.083	1.017	1.152
HDL-C	0.467	0.935	0.779	1.122
LDL-C	0.000	1.377	1.210	1.565

Notes: BMI: body mass index; TG: triglyceride; HDL-C: high-density lipoprotein cholesterol; LDL-C: low-density lipoprotein cholesterol.

Materials and methods

General characteristics

A total of 8115 subjects who received physical examination were recruited from Weihai and Nanchang between January 2015 and December 2015. There were 2628 males and 5487 females with the male to female ratio of 1:2.09. The mean age was 46.70±13.99 years (range: 16-92 years). Of these subjects, 487 received gastroscopy due to gastrointestinal symptoms.

Methods

^{13}C breath test was performed to detect Hp infection. A total of 8115 subjects were recruited, and general characteristics such as gender and age were recorded. The results from ^{13}C breath test, body mass index (BMI), uric acid, blood glucose, total cholesterol (TC), triglycerides (TG), low-density lipoprotein cholesterol (LDL-C) and high-density lipoprotein cholesterol (HDL-C) were also recorded. Questionnaire surgery was conducted in 487 subjects above, and general information, residence, and living habits were recorded for further analysis.

Statistical analysis

Statistical analysis was performed with SPSS version 17.0. Incidence of Hp infection is expressed as percentage. Quantitative data are expressed as mean±standard deviation (x±s) and were compared with independent sample t test. Qualitative data were compared with Chi square test. A value of $P<0.05$ was considered statistically significant.

Results

The overall incidence of Hp infection was 32.4%, which was lower than the national level. The incidence of Hp infection was 35.2% in males and 30.3% in females, showing marked difference (P<0.05). The overall mean age was 46.70±13.99 years. The mean age was 48.65±13.55 years in Hp positive subjects and 45.77±14.10 years in Hp negative ones, showing significant difference (P<0.05). In addition, the incidence of Hp infection varied significantly among different age groups and it increased with the increase in age (**Table 1**). Results of logistic regression showed that gender, age, TG, and LDL-C were risk factors of Hp infection (**Table 2**).

In 487 subjects with gastrointestinal symptoms, the incidence of Hp infection was 53.6%, which was significantly higher than in healthy subjects (P<0.05). The incidence of Hp infection in subjects living in city, with family members having Hp infection, drinking running water, smoking, or drinking alcohol was markedly higher than in controls (P<0.05). In subjects with dining utensils disinfection, hand washing before each meal, using antibiotics or proton pump inhibitor (PPI) within prior 1 month, the incidence of Hp infection was similar to that in controls (P>0.05) (**Table 3**).

Of the recruited subjects, 487 received gastroscopy. Results showed superficial gastritis

Table 3. Questionnaire surgery in gastric disease subjects

Group	Hp positive (%)	Hp negative (%)	*P* value
Total	261 (53.6)	226 (46.4)	
Residence			P=0.000
City	158 (61.0)	101 (39.0)	
Countryside	103 (45.2)	125 (54.8)	
Hp positive family members			P=0.000
Yes	29 (69.0)	13 (31.0)	
No	232 (52.1)	213 (47.9)	
Drinking water			P=0.039
Running water	229 (55.6)	183 (44.4)	
Well water	32 (42.7)	43 (57.3)	
Smoking			P=0.040
Yes	65 (62.5)	39 (37.5)	
No	196 (51.2)	187 (48.8)	
Drinking			P=0.002
Yes	61 (68.5)	28 (31.5)	
No	200 (50.3)	198 (49.7)	
Dining utensils disinfection			P=0.096
Yes	61 (61.0)	39 (39.0)	
No	200 (51. 7)	187 (48.3)	
Hand washing before each meal			P=0.115
Yes	186 (56.0)	146 (44.0)	
No	75 (48.4)	80 (51.6)	
PPI within prior 1 month			P=0.408
Yes	82 (50.9)	79 (49.1)	
No	179 (54.9)	147 (45.1)	
Antibiotics			P=0.377
Yes	68 (50.4)	67 (49.6)	
No	193 (54.8)	159 (45.2)	

had the highest incidence and gastric polyps had the lowest incidence. The incidence of Hp infection was the highest in peptic ulcer subjects and the lowest in esophagitis subjects. Significant difference was observed in the incidence of Hp infection among subjects with different gastric diseases (**Table 4**).

Discussion

Hp infection has a high incidence and easy to transmit in general population. China is a developing country and has a large, dense population. The Chinese living style and environments lead to a higher incidence of Hp infection in China as compared to Western developed countries [1]. Our results showed the incidence of Hp infection in both areas was lower than the national level, but still at a high level. On the basis of our findings, the incidence of Hp infection in males was significantly higher than in females, which may be related to the use of irritating diet, rapid food intake, smoking, drinking alcohol, work pressure and irregular sleeping males [2]. The incidence of Hp infection increased with the increase in age, which may be ascribed to more underlying diseases and poor immune function in old individuals [3]. The higher incidence of Hp infection in subjects with family members having Hp infection indicates that Hp infection may transmit between family members, but whether Hp infection is related to genetic factors is required to be further studied. The incidence of Hp infection in subjects with smoking, drinking alcohol or drinking running water was significantly higher than in controls, which may be ascribed to the smoking and/or drinking induced damage to the gastric mucosa or to more chronic diseases caused by long lasting smoking and/or drinking [4]. The incidence of Hp infection in subjects with dining utensils disinfection and hand washing before each meal was comparable to that in controls, which may be related to more subjects living in city because the incidence of Hp infection in subjects living in city was significantly higher than in countryside. The incidence of Hp infection in subjects with use of antibiotics or PPI within prior 1 month was also similar to that in controls. Generally, only Hp infection is confirmed can antibiotics and/or PPI be administered for therapy, but the therapy has risk for resistance, its efficacy is dependent on the patients' compliance, and it may not completely eradicate Hp [5]. Thus, above findings indicate that Hp infection is closely related to the age, gender, living environment, sanitary condition and living habits, which may provide evidence for the prevention and therapy of Hp infection. In addition, it is necessary to educate healthy subjects and those with Hp infection the information about Hp infection.

The incidence of Hp infection in subjects with gastric diseases was significantly higher than in healthy controls, suggesting that Hp is involved in the pathogenesis of gastric diseases. The urease produced by Hp may degrade urea in

Table 4. Findings from gastroscopy in subjects with gastric diseases

Gastric diseases	N	Hp positive (%)	Hp negative (%)	*P* value
Peptic ulcer	105	71 (67.6)	34 (32.4)	P=0.007
Erosive gastritis	19	10 (52.6)	9 (47.4)	
Superficial gastritis	329	168 (51.1)	161 (48.9)	
Gastric polyps	9	4 (44.4)	5 (55.6)	
Esophagitis	25	8 (32.0)	17 (68.0)	
Total	487	261 (53.6)	226 (46.4)	

the stomach into ammonia and carbonic acid, forming a protective layer to resist the antibacterial effect of gastric acid. Hp is closely related to the pathogenesis of gastric diseases. Hp may up-regulate the expression of cyclin-1 and PCNA and reduce nitrate to nitrite. Thus, the World Health Organization (WHO) has classified Hp as grade I carcinogen. In the present study, results showed superficial gastritis had the highest incidence, indicating that Hp infection mainly causes chronic gastritis. In peptic ulcer subjects, Hp infection had the highest incidence, indicating the close relationship between peptic ulcer and Hp infection, which was consistent with findings in a majority of studies [6]. Subjects with reflux esophagitis had the lowest incidence of Hp infection, which was slightly lower than mean level. This indicates that Hp infection might be a protective factor, and Hp may induce the secretion of gastrin to inhibit the acid reflux [7]. After Hp eradication therapy, the incidence of reflux esophagitis increases, which may be ascribed to the functional recovery of gastric parietal cells and subsequent increase in gastrin secretion as well as the neutralization of gastric acid by the ammonia produced by Hp. In recent years, studies have confirmed the association of Hp infection with chronic diseases including diabetes mellitus and cardiovascular diseases. Our findings indicated that the blood glucose, lipid profile, BMI and uric acid in subjects with Hp infection were markedly higher than in Hp negative subjects, which was consistent with previous findings [8]. Hp infection may induce the production of oxygen free radicals, which may damage the vascular endothelial cells and cause vascular sclerosis and formation of atheromatous plaques. In addition, free radicals may also affect the immune system to increase tumor necrosis factor, interleukin-6, elevate neutrophils and hsC-reaction protein and promote the production of fibrinogen, which facilitate the damage to the vascular endothelial cells. The heat shock protein produced secondary to endothelial injury may interact with the bacterial heat shock protein of Hp, further promoting the thrombosis [9, 10]. Moreover, Hp induces gastroenteritis, which may cause vitamin and folic acid deficiency, resulting in elevation of homocysteine and promoting arteriosclerosis. Thus, Hp infection is closely related to the pathogenesis of cardiovascular diseases and cerebrovascular diseases. Hp is contagious, and subjects with compromised immunity due to diabetes mellitus are susceptible to Hp infection. In addition, there are microvessel lesions, basement membrane thickening, reduced blood flow and reduced oxygen interchange in the blood of gastrointestinal tract in diabetes mellitus patients. The autonomic dysfunction of the gastrointestinal tract may cause motility disorder and delayed empty. Thus, food retention may induce the secretion of gastrin by G cells. The increased secretion of gastric acid may damage the gastric mucosa. The nerve injury may cause pyloric relaxation and bile reflux, further damaging the gastric mucosa. These environments are suitable for the growth of Hp. Therefore, there may be interaction between Hp infection and diabetes mellitus. In addition, there is evidence showing that Hp infection is also related to metabolic syndrome [11, 12]. Once Hp infection is present, the spontaneous disappearance of Hp infection is almost impossible. Hp infection will be life-long if regular treatment is not performed. Thus, for patients with diabetes mellitus, cerebrovascular diseases or cardiovascular diseases accompanied by gastrointestinal symptoms, Hp infection detection is necessary, and standardized therapy should be administered once Hp infection is confirmed.

Disclosure of conflict of interest

None.

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