

Prevalence of *Helicobacter pylori* and parasites in symptomatic children examined for *Helicobacter pylori* antibodies, antigens, and parasites in Yemen

Mabrook A. Bin Mohanna, DCH, FICMS,
Lutf M. Al-Zubairi, MSc, MD,
Abdul K. Sallam, DCH, FMCPS.

ABSTRACT

Objectives: To estimate the prevalence of *Helicobacter pylori* (*H. pylori*) and parasites in symptomatic children examined for *H. pylori* antibodies, antigens, and parasites in Yemen.

Methods: A record-based study was carried out at Specialized Sam Pediatric Center in Sana'a, Yemen for 3 years between 2011-2013. Out of the 43,200 patients seen for different causes through that period, 1008 (2.3%) (females: 675 [67%]; males: 333 [33%]) had gastric complaints, and were subjected to an examination of blood and stool for *H. pylori* and parasites. Data regarding age and gender was also collected.

Results: The age of the patients ranged from 3-15 years. The prevalence of *H. pylori* among children examined for *H. pylori* was 65%, 30% of them were males, and 35% were females (chi square [I^2]=142 p =<0.01). The prevalence in the 6-8 years age group was 83%, and it was 52% in the age group of 12-15 years. The prevalence of giardiasis was 10%, and amoebiasis was 25%.

Conclusion: Prevalence of *H. pylori* infection among children was high, and was more prevalent in the age group of 6-8 years than in the other age groups. Females were more affected than males. Parasites (amoebiasis and giardiasis) infestation was less prevalent.

In countries with low socioeconomic status, co-infections involving several different pathogens commonly occur. Several studies from different locations have reported a possible connection between *Giardia intestinalis* and *Helicobacter pylori* (*H. pylori*).^{1,2} Both organisms inhabit the gastrointestinal tract (GI) in their human hosts within a close proximity, and both organisms are recognized to infect children at a high rate in low-income countries.^{3,4} The *H. pylori* is a common bacterial infectious disease whose manifestations

predominately have an effect on the GI tract, a gram negative, spiral-shaped pathogenic bacterium residing in the mucosa overlying the epithelium of the gastric antrum was first isolated by Warren and Marshall in 1982.³ At first, this bacterium was classified as *Campylobacter pylori* but in 1989 it was included in a new genus, *Helicobacter*, and renamed *Helicobacter pylori*. It is among the most common bacterial infections in the world, and thousands of articles have been written regarding *H. pylori*.⁵

The *H. pylori* is capable to live in the stomach acid because it releases enzymes that neutralize the acid; this allows *H. pylori* to make its way to the "safe" area - the protective mucous lining, which permits acid to get through to the sensitive layer below. Both the acid and bacteria irritate the lining, and cause abdominal pain, or ulcer.⁶ The method of acquisition and transmission of *H. pylori* is uncertain, even though the most probable method of transmission is fecal-oral, or oral-oral. Risk factors, such as low-income in childhood, or affected family members also influence the prevalence. All children infected with *H. pylori* develop histologic chronic active gastritis but often asymptomatic. Manifestation of *H. pylori* infection in children is abdominal pain or vomiting, less often, refractory iron deficiency anemia, or growth retardation. Chronic colonization with *H. pylori* will prompt children to a notably augmented risk of developing duodenal ulcer, or gastric cancer. The *H. pylori* is classified as a group 1 carcinogen by the World Health Organization (WHO).⁷ The prevalence and rate of acquisition of *H. pylori* infection in children from developing countries is higher than in developed countries.⁸ The prevalence is 3-10% of the population each year in developing countries, while in developed countries it is 0.5%.⁸ In a very attractive longitudinal study from the US-Mexican border, Cervantes et al⁹ demonstrated that a younger sibling was 4 times more liable to become infected with *H. pylori* if the mother was infected with *H. pylori* in contrast with an uninfected mother. Younger siblings were 8 times more liable to become infected if their older index sibling had persistent *H. pylori* infection.⁹ Crowding, poor living conditions, and poor personal hygiene may play a role as well.¹⁰ In Saudi Arabia, they found that there was a significant relation between *H. pylori* infection, and recurrent abdominal pain among students.¹¹ Similarly, in Oman they found that *H. pylori* associated with active chronic gastritis is the most common form of stomach diseases. Particularly females, young, and middle ages group had the highest frequency

of *H. pylori* organisms in gastric antrum.¹² Regardless of the success in numerous diagnostic methods for the detection of *H. pylori*, such as endoscopy, urea breath test, stool and blood samples, and the enhancement in socioeconomic status, infection with *H. pylori* is still on rise, and physicians in many developing countries are facing the issues of availability and cost, to establish the diagnosis of *H. pylori* infection. Worldwide, non-invasive tests for active infection are preferred (such as, urea breath test, stool antigen test, and blood antibody test).¹³

The results from many researchers depend on one or 2 tests only for the diagnosis of *H. pylori*, and any test that would give positive result for *H. pylori* was regarded positive for final diagnosis. However, every diagnostic method has a percentage of false positive or negative result, therefore, if at least 2 methods for *H. pylori* indicates positive result at the same time for the same patient, this indicates that the patient has really been infected with *H. pylori*.^{14,15} The aim of this study is to determine the prevalence of *H. pylori*, and parasites among symptomatic Yemeni children, and to identify the possible cause of the acquisition and transmission of *H. pylori*.

Methods. A record-based study was carried out at the Specialized Sam Pediatric Center (SPC) in Sana'a, Yemen between January 2011 to December 2013. The SPC provide services to community through the outpatient clinic and receive patients from the city of Sana'a, its surrounding areas, and at times, from other governorates. Out of the 43,200 patients seen for different causes through this 3-year period, 1,008 (2.3%) was included in the study. There were 675 (67%) females, and 333 (33%) males, and had different types of gastric complaints (recurrent, chronic abdominal pain, gastritis, dyspepsia, nausea, vomiting, and chronic diarrhea) and were subjected to blood examination for *H. pylori* antibody and stool examination for *H. pylori*

antigen. For rapid chromatographic immunoassay for the qualitative detection of antibodies to *H. pylori* in serum or plasma, one step *H. pylori* test device (serum/plasma) (EUGENE®, Shanghai Eugene Biotech Co., Ltd, Shanghai, China) was used, and Chemtrue® One-Step *H. pylori* test (Shanghai Chemtron Biotech Co., Ltd., Shanghai, China), a rapid, visual immunochromatographic test for the qualitative detection of *H. pylori* antigen in fecal samples was used. Formalin ether concentration methods were used to test the prevalence of intestinal parasites (amoebiasis, giardiasis, intestinal roundworms, and tapeworms). Patients were considered to be infected with *H. pylori* if they were positive in both blood and stool, or one of the 2, either blood or stool test if there is a relevant complaint related to the abdomen. Also data regarding age and gender were collected and processed manually. Chi square test was performed using the Statistical Package for Social Sciences program version 20 for Windows (IBM Corp, Armonk, NY, USA). The study was approved by the Specialized Sam Pediatric Center Corporation.

Results. The total number of patients seen during the 3 years was 43,200. A total of 1,008 patients (2.3%) (females: 675 [67%], and males: 333 [33%]) with age ranging from 3-15 years, and had gastric symptoms were subjected to *H. pylori* and parasites examination.

Table 2 - Prevalence of abdominal pain and *Helicobacter pylori* (*H. pylori*) according to age in a study among Yemeni children.

Age group, years	Abdominal complaints n (%)	<i>H. pylori</i> positive	<i>H. pylori</i> negative
3-5	184 (18.3)	111 (60.0)	73 (40.0)
>5-8	214 (21.2)	178 (83.0)	36 (17.0)
>8-11	396 (39.3)	252 (64.0)	144 (36.0)
>11-15	214 (21.2)	111 (52.0)	103 (48.0)
Total	1008 (2.3)	652 (65.0)	356 (35.0)

Table 1 - Prevalence of *Helicobacter pylori* (*H. pylori*) and parasite in children according to gender in a study among Yemeni children.

Gender	Abdominal complaints	<i>H. pylori</i> positive*	<i>H. pylori</i> negative	<i>Giardia</i>	<i>Amoeba</i>
		n (%)			
Male	333 (33.0)	300 (30.0)	33 (3.0)	10 (1.0)	70 (7.0)
Female	675 (67.0)	352 (35.0)	323 (32.0)	90 (9.0)	182 (18.0)
Total	1008 (100)	652 (65.0)	356 (35.0)	100 (10.0)	252 (25.0)

*Chi square for *H. pylori* by gender = 142, $p < 0.01$

Of the 652 (65%) examined patients with *H. pylori*, 30% of them were males, and 35% were females. Chi square for *H. pylori* by gender was 142 ($p < 0.01$) (Table 1). There were 83% who had *H. pylori* in the age group from 6-8 years, 64% from the 9-11 years, and 52% from the 12-15 years age range (Table 2).

Discussion. In general, the prevalence of *H. pylori* is considered to be the most prevalent infectious disease known to occur in humans. Individuals living in countries with low-income had high incidence rates of *H. pylori* acquired at an early age.⁸ It is generally accepted that *H. pylori* infection is the major etiological factor for gastritis and peptic ulcer. Its eradication is connected with curing of these diseases, and important decrease of ulcer recurrence, and rebleeding. Some studies¹⁶ have verified that inflammations caused by *H. pylori* infection may lead to the development of adenocarcinoma of the stomach. The current study found that the prevalence of *H. pylori* infection was high among children complaining of chronic, or recurrent abdominal pain compared with other causes of abdominal pain. This agrees with other studies in many countries, such as Saudi Arabia,¹¹ Oman,¹² and Iraq.¹⁷

In this study, the prevalence of *H. pylori* was higher in the age group 6-8, and 9-11 years than in the age group 12-15 years. This finding is in agreement with other studies that revealed a higher incidence of *H. pylori* in a younger age than an older age.^{8,17} This may be attributed to low socioeconomic condition of the families, and some patients of the age group (12-15 years) consulted the adult clinics, instead of the pediatric clinics.

The findings in this study revealed that there was a significant difference between genders in the incidence of abdominal complaints and *H. pylori* infection; females (67%) were more affected than males (33%). This result agrees with a study conducted in Oman,¹² which showed that females, young, and middle age group had the highest frequency of *H. pylori* organisms, but it does not agree with many other studies, which found that there was no significant difference between genders regarding infection by *H. pylori*.¹⁷ In the present study, we found that the parasites (amoebiasis and giardiasis) infestation was less prevalent compared with *H. Pylori* infection. This result disagrees with other studies that found a significantly higher frequency of giardial infection in cases where infected children also harbored the bacterial pathogen *H. pylori*.² The results of this finding shows that *H. pylori* infection is more prevalent in the older than the younger age group (in our study, the age ranged from 3-15 years), while in

giardial infection the prevalence is more in younger age group (<5 years).¹⁸

In conclusion, the prevalence of *H. pylori* infection among children examined for *H. pylori* antibody and antigen was high. Females were more affected than males. The prevalence of *H. pylori* in the age group 6-8 and 9-12 years was higher than the other age groups. Parasites (amoebiasis and giardiasis) infestation were less prevalent compared with *H. pylori* infection. A possible way of eliminating *H. pylori* from the population would be via early treatment of the infected mothers and children, and health measures, such as sanitation and the standard of living should be improved.

Acknowledgement. The authors would like to thank Ameen Bin Mohana and Mohammed Bin Mohana for their technical assistance in preparing this paper, and the Specialized Sam Pediatric Center team.

Received 9th June 2014. Accepted 26th August 2014.

From the Department of Pediatrics, Faculty of Medicine and Health Sciences Sana'a University, Sana'a, Yemen. Address correspondence and reprints request to: Dr. Mabrook A. Bin Mohanna, Associate Professor, Department of Pediatrics, Faculty of Medicine and Health Sciences, Sana'a University, PO Box 18660, Sana'a, Yemen. Tel. +96 (7) 1245492. E-mail: mabrookmohanna@yahoo.com

References

1. Zeyrek D, Zeyrek F, Cakmak A, Cekin A. Association of *Helicobacter pylori* and giardiasis in children with recurrent abdominal pain. *Turkiye Parazitoloj Derg* 2008; 32: 4-7.
2. Isaeva GSh, Efimova NG. [Gastrointestinal giardiasis associated with *Helicobacter pylori*]. *Ekspr Klin Gastroenterol* 2010; 6: 30-34. Russian
3. Hestvik E, Tylleskar T, Kaddu-Mulindwa DH, Ndezi G, Grahniquist L, Olafsdottir E, et al. *Helicobacter pylori* in apparently healthy children aged 0-12 years in urban Kampala, Uganda: a community-based cross sectional survey. *BMC Gastroenterol* 2010; 10: 62.
4. Schwarz S, Morelli G, Kusecek B, Manica A, Balloux F, Owen RJ, et al. Horizontal versus familial transmission of *Helicobacter pylori*. *PLoS Pathog* 2008; 4: 1000180.
5. Shanks AM, El-Omar EM. *Helicobacter pylori* infection, host genetics and gastric cancer. *J Dig Dis* 2009; 10: 157-164.
6. Lin YH, Chang CH, Wu YS, Hsu YM, Chiou SF, Chen YJ. Development of pH-responsive chitosan/heparin nanoparticles for stomach-specific anti-*Helicobacter pylori* therapy. *Biomaterials* 2009; 30: 3332-3342.
7. Kliegman RM, Stanton BMD, St. Geme J, Schor, NF, Behrman RE. Nelson Textbook of Pediatrics: Expert Consult Premium Edition - Enhanced Online Features and Print. 19 ed. Philadelphia (PA): Saunders; 2011. p. 1292-1293.
8. Rosenberg JJ. *Helicobacter pylori*. *Pediatr Rev* 2010; 31: 85-86.
9. Cervantes DT, Fischbach LA, Goodman KJ, Phillips CV, Chen S, Broussard CS. Exposure to *Helicobacter pylori*-positive siblings and persistence of *Helicobacter pylori* infection in early childhood. *J Pediatr Gastroenterol Nutr* 2010; 50: 481-485.

10. Jones N, Chiba N, Fallone C, Thompson A, Hunt R, Jacobson K, et al. *Helicobacter pylori* in First Nations and recent immigrant populations in Canada. *Can J Gastroenterol* 2012; 26: 97-103.
11. Telmesani AM. *Helicobacter pylori*: prevalence and relationship with abdominal pain in school children in Makkah City, western Saudi Arabia. *Saudi J Gastroenterol* 2009; 15: 100-103.
12. Alwahaibi NY, Almahrooqi BM, Alrawahi SA. The prevalence of *helicobacter pylori* and gastritis in Oman. *J Dig Endosc* 2013; 4: 29-32.
13. Malfertheiner P, Megraud F, O'Morain CA, Atherton J, Axon AT, Bazzoli F, et al. Management of *Helicobacter pylori* infection-the Maastricht IV/ Florence Consensus Report. *Gut* 2012; 61: 646-664.
14. Zhou X, Su J, Xu G, Zhang G. Accuracy of stool antigen test for the diagnosis of *Helicobacter pylori* infection in children: A meta-analysis. *Clin Res Hepatol Gastroenterol* 2014; 38: 629-638.
15. Calvet X, Sánchez-Delgado J, Montserrat A, Lario S, Ramírez-Lázaro MJ, Quesada M, et al. Accuracy of diagnostic tests for *Helicobacter pylori*: a reappraisal. *Clin Infect Dis* 2009; 48: 1385-1391.
16. Konturek PC, Konturek SJ, Brzozowski T. *Helicobacter pylori* infection in gastric cancerogenesis. *J Physiol Pharmacol* 2009; 60: 3-21.
17. Alsaimary I, Al-Sadoon M, Jassim A, Hamadi S. Clinical findings and prevalence of *helicobacter pylori* in patients with gastritis B in Al-basrah governorate. *Oman Med J* 2009; 24: 208-211.
18. Ankarklev J, Hestvik E, Lebbad M, Lindh J, Kaddu-Mulindwa DH, Andersson JO, et al. Common coinfections of *Giardia intestinalis* and *Helicobacter pylori* in non-symptomatic Ugandan children. *PLoS Negl Trop Dis* 2012; 6: 1780.

Related Articles

Pandya HB, Patel JS, Agravat HH, Patel SB, Thakkar MC. Identification of *Helicobacter pylori* by different conventional staining techniques and its comparison with polymerase chain reaction. *Saudi Med J* 2013; 34: 942-948.

Al-Khattaf AS. *Helicobacter pylori* virulence markers in gastroduodenal disorders. Detection of cytotoxin-associated gene A and vacuolating cytotoxin-associated gene A genes in Saudi patients. *Saudi Med J* 2012; 33: 716-721.

Ye XW, Xiao J, Qiu T, Tang YJ, Feng YL, Wang K, et al. *Helicobacter pylori* seroprevalence in patients with obstructive sleep apnea syndrome among a Chinese population. *Saudi Med J* 2009; 30: 693-697.