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Positive Association between *Helicobacter pylori* and Gastroesophageal Reflux Disease in Children

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Abstract

Objectives—The role of *Helicobacter pylori* (*H. pylori*) in gastroesophageal reflux disease (GERD) remains controversial, particularly in children, since there are limited published data. Adult studies suggested that *H. pylori* infection may protect against GERD by causing atrophic gastritis, which leads to reduced gastric acid secretion. The objective of our study was to determine the role of *H. pylori* infection in the development of GERD in a pediatric population.

Methods—A retrospective analysis of 420 patients (M:F = 214:206) who underwent esophagogastroduodenoscopy (EGD) with biopsies between January 2000 and April 2006 was conducted. Patient demographics, clinical indications for EGD and the prevalence of reflux esophagitis (RE), the biomarker for GERD, in two groups, *H. pylori* positive and *H. pylori* negative, were reviewed. The prevalence of RE in the *H. pylori* positive and *H. pylori* negative groups was further analyzed based on gender and age (< 1 yr, 1 – 10 yrs, > 10 yrs). The mean age of the study population was 8.2 years (range 0 – 20 yrs). The clinical indications for EGD were as follows: recurrent abdominal pain (n = 186, 44%), malabsorption (n = 80, 19%), persistent vomiting (n = 80, 19%), suspected eosinophilic gastrointestinal disorders (n = 63, 15%) and others such as upper GI bleeding or IBD surveillance (n = 11, 3%). Statistical analysis was performed by using Chi-square test, Fisher's exact test and multivariate logistical regression analysis.

Results—Among the 420 patients, 16 patients (3.8%) were positive for *H. pylori* and 167 patients (39.8%) were found to have RE. Thirteen patients with *H. pylori* were found to have histologic evidence of RE. The prevalence of RE in the *H. pylori* positive population was 81.3% compared to 38.1% in the *H. pylori* negative population ($p \leq 0.05$). There were no patients with *H. pylori* in the youngest age group. In the second age group (1–10 yrs), 100% of the *H. pylori* positive patients had RE while 44.6% of the *H. pylori* negative patients had RE ($p \leq 0.05$). Both male and female patients with *H. pylori* had a higher prevalence of RE, 77.8% and 85.7% respectively. On a multivariate logistical regression, for the overall study cohort, *H. pylori* positive patients had an odds ratio of 5.79 of developing RE compared to *H. pylori* negative patients ($p \leq 0.05$).

Conclusions—Our study results indicate that there is a significantly higher prevalence of RE in an *H. pylori*- infected cohort independent of age or gender. The findings suggest that *H. pylori* infection in children is positively associated with RE.

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Keywords

Helicobacter pylori; gastroesophageal reflux disease; reflux esophagitis

Introduction

In 1983, Warren and Marshall reported an association between the presence of *H. pylori* in the gastric mucosa and antral gastritis in adults (1). *H. pylori* is a gram-negative, spiral, flagellated bacterium that colonizes the gastric mucosa in the infected host. *H. pylori* infection has been implicated in the development of gastritis and peptic ulcer disease, as well as chronic atrophic gastritis (2). *H. pylori* infection increases the risk of gastric malignancies such as adenocarcinoma and mucosal-associated lymphoid tissue (MALT) lymphoma (3). Antral gastritis and duodenal ulcer have been described as common presenting features of *H. pylori* infection in the pediatric population (4,5). It is well known that *H. pylori* infection is acquired in childhood, often before the age of 5 years, through fecal-oral, oral-oral or gastro-oral transmission (6). The infection may be life-long in the absence of treatment. Children with symptomatic *H. pylori* infection associated with duodenal and gastric ulcers, lymphoma or atrophic gastritis with intestinal metaplasia require treatment to eradicate the bacterium (7).

The relationship between *H. pylori* and gastroesophageal reflux disease (GERD) has been controversial in published literature in the past decade (8–20). *H. pylori* infection has been associated with a significantly reduced risk of developing GERD, Barrett's esophagus and esophageal adenocarcinoma in contrast to its increased risk for peptic ulcer disease and gastric cancer (8–10). Labenz *et al*. (11,12) who first reported in 1997 that there was an increased risk of development of GERD after *H. pylori* eradication, postulated a possible protective role of *H. pylori* infection. Subsequently, McColl and others (13–17) suggested that *H. pylori* infection might protect against GERD in infected patients who had atrophic gastritis and reduced gastric acid secretion. The regional distribution and severity of gastritis appeared to be a more important risk factor than the mere presence of *H. pylori* in the gastric mucosa (14). However, several papers contradicted Labenz's findings showing that *H. pylori* eradication may not induce GERD symptoms in adults and in fact the infection may have no association (18–20).

There are limited data regarding the relationship between *H. pylori* infection and GERD in the pediatric population (21–24). Levine *et al*. (21) reported that eradication of *H. pylori* was not associated with increased symptoms of GERD in children. Pollet *et al*. (22) found that *H. pylori* eradication did not provoke or worsen GERD in neurologically impaired children. Özçay *et al*. (23) found no significant difference in reflux esophgitis (RE) between children with and without *H. pylori* infection while Daugule *et al*. (24) showed a significantly higher prevalence of *H. pylori* in children with RE compared to children with hyperemic gastropathy.

The aim of this study was to evaluate potential relationships between *H. pylori* infection and GERD in pediatric patients based on a retrospective examination of biopsy specimens in conjunction with clinical and demographic characteristics.

Patients and Methods

Patients

A retrospective chart review of 562 patients who underwent EGD between January 2000 and April 2006 was carried out. One hundred forty-two patients were excluded from the study: 87 patients were eliminated for the absence of either an esophageal or gastric biopsy; 11 were eliminated because their biopsies were unavailable for review by the pathologist; and 44 were eliminated because they had had more than one procedure during the time period. A detailed

characterization and analysis of the remaining 420 patients (M:F = 214:206) was conducted. The mean age of the study population was 8.2 years (range 0 – 20 yrs). The prevalence of GERD in *H. pylori* positive and *H. pylori* negative groups was further analyzed based on gender and age (<1 yr, 1 – 10 yrs, > 10 yrs). In addition, the histologic severity of GERD in both groups was compared using Chi-square test. The indication for the procedures in order of prevalence was as follows: recurrent abdominal pain (n = 186, 44%), malabsorption (n = 80, 19%), persistent vomiting (n = 80, 19%), suspected eosinophilic gastrointestinal disorders (n = 63, 15%) and others such as upper GI bleeding or IBD surveillance (n =11, 3%) (Table 1).

Endoscopy and Biopsy

All endoscopies were performed using videogastroscopes (Pentax, Japan) EG1840, EG2430 or EG2731. Biopsy specimens were taken from the gastric antrum and distal esophagus during the procedure. The specimens were immediately fixed in 10% buffered formalin solution, embedded in paraffin, cut at 4–6 microns thick and stained with hematoxylin-eosin (H&E) for histologic evaluation. All biopsies with inflammation were also stained with a modified Diff-Quik stain to identify *H. pylori. H. pylori* infection was defined as histologic identification of bacillary forms and gastritis on H&E stain and/or modified Diff-Quik stain. A diagnosis of chronic active gastritis was made on the basis of a stromal lymphoplasmacytic infiltrate in combination with neutrophilic infiltration of the glandular epithelium. The updated Sydney system was used to assess the severity of *H. pylori* gastritis (25). A diagnosis of RE was based on histologic findings of basal cell hyperplasia with eosinophilic exocytosis (in the absence of such findings in the upper esophagus). All esophageal biopsy slides were re-evaluated by a single pathologist, for grading of the esophagitis using a modification of the scale suggested by Zentilin *et al*. (26). Biopsy specimens were graded on a 4 point scale; 0/3=normal, no basal cell hyperplasia, minimal (less than 3 eosinophils/high power field (HPF) exocytosis; 1/3=mild (up to 1/3 of epithelial thickness) basal cell hyperplasia, minimal to mild (between 2–8 eosinophils/HPF) exocytosis (mild active esophagitis); 2/3=moderate (from 1/3 to 2/3 of epithelial thickness) basal cell hyperplasia, moderate (between 10–15 eosinophils/HPF) exocytosis (moderate active esophagitis); 3/3=marked (2/3 to full-thickness) basal cell hyperplasia, marked (over 20 eosinophils/HPF) exocytosis (severe active esophagitis) (see the Supplementary Material 1–4). The study was approved by the Institutional Review Board of Weill Medical College of Cornell University.

Statistics

The Chi-square test and Fisher's exact test were used to evaluate the association between patients' demographic characteristics, *H. pylori* status, and RE status. Multivariate logistical regression analysis predicting RE status was used to estimate the odds ratios, 95% confidence intervals, and p-value of *H. pylori* status and demographic characteristics. All statistical tests are two-sided and $p < 0.05$ was considered statistically significant. Analyses were performed in SAS Version 9.1 (SAS Institute, Inc., Cary, N.C.).

Results

Among the 420 patients (M:F = 214:206), 16 patients had *H. pylori* infection and 167 patients had histologic evidence of RE. The prevalence of *H. pylori* in the entire patient population was 3.8%, and the prevalence of RE was 39.8% (Table 1).

All 16 patients with *H. pylori* had chronic active gastritis in the antrum. The biopsies were graded based on cell infiltration and *H. pylori* density using the updated Sydney system. One patient had mild neutrophilic/mononuclear cell infiltration (grade 1 of 3), ten had moderate cellular infiltration (grade 2 of 3), and five had marked cell infiltration (grade 3 of 3). Nine patients had a mild density of *H. pylori* (grade 1 of 3), four had a moderate density (grade 2 of

3) and three showed a marked density (grade 3 of 3) (see the Supplementary Material 5). No patients had intestinal metaplasia or atrophy in either the antrum or corpus. There was no correlation between the Updated Sydney grade of gastritis and the severity of esophagitis.

A total of 13 patients with *H. pylori* had histologic evidence of RE (Table 2). The prevalence of RE in the *H. pylori* positive patients was 81.3% compared to 38.1% in the *H. pylori* negative patients ($p \leq 0.05$) (Table 3). Among 13 patients with *H. pylori*, 76.9% had grade 1 RE, 15.4% had grade 2 RE and 7.7% had grade 3 RE (Table 4). Among 154 patients without *H. pylori*, 76.6% had grade 1 RE, 15.6% had grade 2 RE and 7.8% had grade 3 RE. Thus, most of the patients in both groups were found to have grade 1 RE (Table 4).

Nine male patients were positive for *H. pylori* while 7 female patients were positive for *H. pylori* with a prevalence of 4.2% and 3.4% respectively (Table 2). Ninety-nine male patients had RE (prevalence of 46.3%) while 68 female patients had RE (prevalence of 33.0%) ($p \leq 0.05$) (Table 3). Both male and female patients with *H. pylori* had a higher prevalence of RE, 77.8% and 85.7% respectively, compared to those without *H. pylori* (Table 5).

There were no patients with *H. pylori* in the youngest age group (< 1yr). In the next age group (1–10 yrs), the prevalence of *H. pylori* was 3.8%, equal to the prevalence in the entire study population; whereas, in the oldest age group (>10 yrs), the prevalence of *H. pylori* was 5.2% (Table 2). The prevalence of RE in both the second (1 – 10 yrs) and the third (>10 yrs) age groups was 46.7% and 43% respectively, compared to a lower prevalence of 10.9% in the youngest age group (< 1yr) ($p \leq 0.05$) (Table 3).

In the second age group (1 – 10 yrs), 100% of *H. pylori* positive patients had RE while 44.6% of *H. pylori* negative patients had RE ($p \leq 0.05$). In the third age group (>10 yrs), 66.7% of *H. pylori* positive patients had RE while 41.7% of *H. pylori* negative patients had RE (Table 5).

On a multivariate logistical regression analysis, *H. pylori* positive patients had an odds ratio of 5.79 (95% confidence interval 1.6–20.1) for having RE compared to *H. pylori* negative patients ($p \leq 0.05$). Patients between 1–10 years and patients older than 10 years had odds ratios of 7.00 (95% confidence interval 3.0–16.3) and 5.99 (95% confidence interval 2.6–14.0) respectively of having RE compared to patients less than 1 year ($p \leq 0.05$). Furthermore, male patients had an odds ratio of 1.84 (95% confidence interval 1.2–2.8) of having RE compared to female patients ($p \leq 0.05$) (Table 6).

Abdominal pain was the most common indication for EGD, 44.3% of all the indications. Among 186 patients with abdominal pain, only 5.9% of patients had *H. pylori* ($p \leq 0.05$) while 43% had biopsy proven RE (Table 1–3).

Discussion

In this study, we report that the prevalence of RE, the biomarker for GERD, among *H. pylori* positive patients regardless of their age and gender was twice as high as among *H. pylori* negative patients (81.3% vs. 38.1%). Based upon a multivariate logistical regression analysis, *H. pylori* positive patients were six times more likely to have RE compared to *H. pylori* negative patients. There was no difference in the apparent severity of RE between *H. pylori* positive and *H. pylori* negative patients.

The overall prevalence of *H. pylori* infection in our cohort was 3.8%. While this is lower than that of some previously published studies (27–31), it is consistent with the reported prevalence of less than 10% in children in developed countries (5,32–35). For example, Mourad-Baars *et al*. (34) reported a low seroprevalence (1.2%) of *H. pylori* infection in young Dutch children. The overall prevalence of *H. pylori* is declining in developed countries (5,32–35) and the rate

of infection is largely influenced by several factors, such as socioeconomic status and living conditions (27,36). The low prevalence of *H. pylori* in our study may be attributed to demographic factors in our population. *H. pylori* infection rates among 200 adults in New York City were highest in African-Americans (43%), followed by Hispanics (20%) and Caucasians (11%) (37). Our hospital serves a predominantly upper middle socioeconomic class population. Our pediatric patient population consists of approximately 50% Caucasian, 39% African-American/Hispanic and 17% Asian; 70% have private insurance. Furthermore, we speculate that the low prevalence of *H. pylori*, in part, may be explained by the frequent use of antibiotic treatment for other types of infection in children. Monotherapies with common antibiotics such as Amoxicillin and Clarithromycin can suppress or potentially eradicate *H. pylori* in 10–50% of treated subjects (38,39).

Similar to our findings of an increased prevalence of RE in *H. pylori* positive patients, Daugule *et al.* (24) showed that the prevalence of *H. pylori* was significantly higher among children with RE than in children with hyperemic gastropathy. We also found that *H. pylori* infection was more closely associated with RE in younger children (0–10 years) than in older children (> 10 years), although this may be related to an age specific distribution of *H. pylori* gastritis. Antral predominant nonatrophic gastritis is more common in children while corpus predominant gastritis is more common in adults. Antral predominant nonatrophic gastritis causes increased acid secretion and in turn increases the risk of developing GERD and related complications including RE. All sixteen patients with *H. pylori* in our study had antral nonatrophic gastritis. Because this retrospective study did not include biopsy specimens from the corpus, an age specific distribution of *H. pylori* gastritis was not evaluated.

The limitations of our study include the fact that the data reflect a single clinical center with a low prevalence of *H. pylori*. We did not take account of possible effects of antisecretory agents at the time of endoscopy in evaluating the degree of RE and chronic active gastritis in our study population. In addition, we did not address cytotoxin associated gene (cagA) status in *H. pylori* positive patients in the study.

In conclusion, our study showed that *H. pylori* infection was positively associated with RE, thus, in the study cohort, *H. pylori* appeared to be a risk factor for this biomarker of GERD. In light of these results, *H. pylori* eradication therapy may be beneficial, especially in younger children.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Table 1

Characteristics of Study Population

Characteristics	Number of Patients
Study Population	**420**
Age	
< 1 year	64 (15.2%)
1–10 years	184 (43.8%)
> 10 years	172 (41.0%)
Gender	
Female	206 (49.1%)
Male	214 (50.9%)
Indication	
Abdominal Pain	186 (44.3%)
Malabsorption	80 (19.1%)
Eosinophilic GI Disorders	63 (15.0%)
Persistent Vomiting	80 (19.1%)
Others	11 (2.6%)
H. pylori	
Negative	404 (96.2%)
Positive	16 (3.8%)
RE Grade	
0	253 (60.2%)
1	128 (30.5%)
2	26 (6.2%)
3	13 (3.1%)

Table 2

Comparison of Study Population *H. pylori* Positive versus Negative

	H. pylori		
	Negative N (%)	**Positive N (%)**	**P-value**
Age			
< 1 year	64 (100.0)	0 (0.0)	0.18†
1 – 10 years	177 (96.2)	7 (3.8)	
> 10 years	163 (94.8)	9 (5.2)	
Gender			
Female	199 (96.6)	7 (3.4)	0.67*
Male	205 (95.8)	9 (4.2)	
Indication			
Abdominal Pain	175 (94.1)	11 (5.9)	0.03†
Malabsorption	76 (95.0)	4 (5.0)	
Eosinophilic GI Disorders	63 (100.0)	0 (0.0)	
Persistent Vomiting	80 (100.0)	0 (0.0)	
Others	10 (90.9)	1 (6.3)	

*Chi-Square test

†Fisher's exact test

Table 3

Comparison of Study Population RE Positive versus Negative

	RE		
	Negative N (%)	**Positive N (%)**	**P-value**
Age			
< 1 year	57 (89.1)	7 (10.9)	<0.0001*
1 – 10 years	98 (53.3)	86 (46.7)	
> 10 years	98 (57.0)	74 (43.0)	
Gender			
Female	138 (67.0)	68 (33.0)	0.006*
Male	115 (53.7)	99 (46.3)	
Indication			
Abdominal Pain	106 (57.0)	80 (43.0)	0.20*
Malabsorption	50 (62.5)	30 (37.5)	
Eosinophilic GI Disorders	46 (73.0)	17 (27.0)	
Persistent Vomiting	45 (56.3)	35 (43.8)	
Others	6 (54.6)	5 (45.5)	
H. pylori			
Negative	250 (61.9)	154 (38.1)	0.0005†
Positive	3 (18.7)	13 (81.3)	

*Chi-Square test

†Fisher's exact test

Table 4

RE Grading in *H. pylori* Positive and Negative Patients

RE Grade	*H. pylori*	
	Negative	**Positive**
1	118 (76.6%)	10 (76.9%)
2	24 (15.6%)	2 (15.4%)
3	12 (7.8%)	1 (7.7%)

Table 5

H. pylori and RE by age and gender

	RE positive N (%)	RE negative N (%)	p-value*
Age (1–10 yrs)			
H. pylori positive	7 (100)	0 (0)	0.004
H. pylori negative	79 (44.6)	98 (55.4)	
Age (> 10 yrs)			
H. pylori positive	6 (66.7)	3 (33.3)	0.18
H. pylori negative	68 (41.7)	95 (58.3)	
Male			
H. pylori positive	7 (77.8)	2 (22.2)	0.08
H. pylori negative	92 (44.9)	113 (55.1)	
Female			
H. pylori positive	6 (85.7)	1 (14.3)	0.006
H. pylori negative	62 (31.2)	137 (68.8)	

*Fisher's Exact Test

Table 6

Logistical Regression Analysis Predicting RE Positive

	Odds Ratio	95% CI	P-value
H. pylori			
Negative	1 (reference)		
Positive	5.79	(1.60, 20.96)	0.007
Age			
< 1 year	1 (reference)		
1 – 10 years	7.00	(3.01, 16.27)	<0.0001
> 10 years	5.99	(2.56, 14.02)	<0.0001
Gender			
Female	1 (reference)		
Male	1.84	(1.22, 2.79)	0.004