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Significance and Related Factors of *Helicobacter pylori* Infection in Children with Dyspepsia

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Conflict of Interest
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ABSTRACT

Purpose: *Helicobacter pylori* is a Gram-negative bacterium that is associated with peptic ulcer disease (PUD) and gastric cancer. However, studies on the endoscopic finding and factors related to *H. Pylori* infection in children are lacking. This study aimed to evaluate the prevalence and factors associated with *H. pylori* infection in children with dyspepsia.
Methods: We retrospectively analyzed the medical records of patients aged <18 years who underwent upper endoscopy for dyspepsia between January 2015 and December 2022. *H. pylori* was diagnosed using a rapid urease test, and the dyspeptic symptoms included postprandial fullness, early satiation, epigastric pain, and nausea.
Results: Among 185 patients, the prevalence of *H. pylori* infection was 16.2%. Obesity and a family history of *H. pylori* infection were more frequently observed in patients with *H. pylori* infection than in those without. Anemia was also more common in patients with *H. pylori* infection than in those without. *H. pylori* was detected in six (18.8%) patients with PUD (n=32). All six patients had duodenal ulcers and anemia, and complications such as obstruction and bleeding were significantly more common among patients with PUD who were positive for *H. pylori* infection than among those without.
Conclusion: We observed a low prevalence of *H. pylori* infection in children with dyspepsia; however, these children exhibited a more severe clinical course. Therefore, caution should be exercised in detecting *H. pylori* infection in children, especially, who had obesity or family history of *H. pylori* infection.

Keywords: Children; *Helicobacter pylori*; Dyspepsia; Peptic ulcer

INTRODUCTION

Helicobacter pylori is a Gam-negative microaerophilic bacterium that generally infects children and persists for life if left untreated [1,2]. A meta-analysis study demonstrated that the overall seroprevalence rate of *H. pylori* in healthy children was 33% [3,4], whereas a study from Korea reported that the prevalence was 6–11% in children with recurrent abdominal pain [5]. Another study performing from eastern china said 18.6% of *H. pylori* infection rates in children with gastrointestinal (GI) symptoms [6]. *H. pylori* colonizes the gastric mucosa and may lead to chronic active gastritis, peptic ulcer disease (PUD), and gastric cancer [7]. *H. pylori* infection can be diagnosed using invasive tests, which involve taking biopsy samples

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during upper GI endoscopy [8]. During endoscopy, nodular surface was found to have excellent specificity for current *H. pylori* infection (95.8–98.8%) and diffuse redness had a good positive predictive value (65.6–91.5%) [9].

Dyspepsia is common indication for performing upper GI endoscopy and is known to be related to *H. pylori* infection. Children with *H. pylori* infection may present with recurrent abdominal pain and dyspepsia; however, some may be asymptomatic. One meta-analysis reported no correlation between *H. pylori* infection and GI symptoms except for epigastric pain [10]. In adults, dyspepsia symptoms improve after the eradication of *H. pylori* even in patients without endoscopic abnormalities [11]. In children, Correa et al. [12] identified *H. pylori* infection in 52% of children with non-ulcer dyspepsia and revealed that nausea symptoms were associated with *H. pylori* infection, with an odds ratio (OR) of 1.76. Another study by Ünlüsoy et al. [13] reported that 33% prevalence rate of *H. pylori* infection in children with functional dyspepsia as well as the dyspepsia symptom scores decreased after eradication of *H. pylori*. Detection of *H. pylori* infection is important in children with dyspepsia; however, on few studies have reported on their endoscopic findings and related factors.

Therefore, this study aimed to evaluate the prevalence and factors associated with *H. pylori* infection in children with dyspepsia. Furthermore, we aimed to evaluate the factors related to *H. pylori*-negative PUD.

MATERIALS AND METHODS

We retrospectively analyzed the medical records of patients aged <18 years who underwent upper GI endoscopy for dyspepsia between January 2015 and December 2022. All endoscopies were performed by three experienced pediatric gastroenterologists with more *than* 5 *years* of *experience* in endoscopic practice. Dyspepsia symptoms including postprandial fullness, early satiation, epigastric pain, and nausea were recorded. Patients who took medications that cause dyspepsia and who had pancreatic or biliary disorder were excluded.

Endoscopic and histological findings were collected from the medical records. Data on age, sex, body mass index (BMI), family history of *H. pylori* infection (who living in the same house), symptom duration, and weight loss and laboratory findings such as hemoglobin and platelet counts were also collected. A family history of *H. pylori* infection was considered as positive when any first-degree relative had *H. pylori* infection, and BMI was calculated by dividing the weight (in kilograms) by the square of the height (in meters). Obesity was defined as a BMI ≥95th percentile for sex and age [14].

The endoscopic aspect of antral nodularity is based on the irregular appearance of the mucosa, which is described as a cobblestone pavement appearance. Biopsies were obtained from the corpus and antrum, and a rapid urease test was performed simultaneously. Gastric biopsy specimens were stained with Giemsa. *H. pylori* infection was defined as *H. pylori* gastritis on histopathology with positive rapid urease test [15]. The endoscopic and histological findings were classified according to the Sydney classification [16]. Abnormal endoscopic findings included erythema gastritis, erosive gastritis, and PUD and abnormal histological findings include chronic gastritis, chronic atrophic gastritis, and chronic intestinal metaplasia. PUD complications included perforation, bleeding and gastric outlet obstruction [17]. We used 14 day first-line agent for *H. pylori* treatment. Standard

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dose of proton pump inhibitor-amoxicillin (50 mg/kg/day with a maximum of 2 g/day)-clarithromycin (25 mg/kg/day with a maximum of 1 g/day) based on patients' body weight were prescribed. For evaluating eradication after treatment, we performed non-invasive test such as urea breath test to all treated patients after 4 to 6 weeks after stopping standard triple therapy. When standard triple therapy was fail to achieve eradication of *H. pylori*, we used bismuth (BIS)-based quadruple therapy.

Statistical methods

Continuous data are expressed as means (±standard deviation). These data were further compared using the Mann–Whitney U-test or Student's *t*-test. Discrete data are expressed as numbers and percentages and were compared using Fisher's exact or chi-squared tests. To evaluate the factors associated with *H. pylori* infection, we calculated OR using logistic regression models. Statistical significance was set at $p<0.05$. Statistical analyses were performed using IBM SPSS Statistics for Windows, Version 24.0 (IBM Co.).

Ethical considerations

This retrospective analysis was approved by the Institutional Review Board of Chungnam National University Hospital and was conducted in accordance with the Declaration of Helsinki (IRB number: 2023-04-057). The requirement for informed consent was waived owing to the retrospective nature of the study.

RESULTS

Of the 185 patients with dyspepsia included in our study, 30 (16.2%) showed histopathological evidence of *H. pylori* infection. The median age of the patients were 12.8 years (range: 6–18 years), and the male to female ratio was 0.91:1. **Table 1** shows the clinical characteristics of the all patients according to the *H. pylori* infection status. The proportion of patients with obesity and/or overweight was higher in the *H. pylori*-positive group compared with the *H. pylori*-negative group (20.0 vs. 10.3%, $p=0.025$). A family history of *H. pylori* infection was also more frequent in the *H. pylori*-positive group than in the *H. pylori*-negative group (13.3 vs. 3.9%, $p=0.043$). Regarding laboratory findings, the hemoglobin level was significantly lower in the *H. pylori*-positive group than the *H. pylori*-negative group (10.48 vs. 12.89 g/dL, $p=0.002$). Out of the 30 patients who completed standard triple therapy of *H. pylori* infection,

Table 1. Clinical characteristics of all patients according to *H. pylori* infection status

Variable	Total (N=185)	*H. pylori* positive (n=30)	*H. pylori* negative (n=155)	*p*-value
Age (yr)	12.52±3.21	13.32±2.55	12.23±2.25	0.283
Male	88 (47.6)	16 (53.3)	72 (46.5)	0.312
BMI (kg/m^2)	20.22±2.52	21.94±2.32	17.21±2.86	0.027
Obesity and/or overweight	22 (11.9)	6 (20.0)	16 (10.3)	0.025
Family history of *H. pylori*	10 (5.4)	4 (13.3)	6 (3.9)	0.043
Presence of weight loss	61 (33.0)	10 (33.3)	51 (32.9)	0.518
Hb (g/dL)	12.21±1.97	10.48±2.20	12.89±1.51	0.002
Platelet (10^3/μL)	282.21±75.23	287.62±75.57	278.62±65.89	0.680
Presence of anemia	16 (8.6)	8 (26.7)	8 (5.2)	0.021
Presence of melena	7 (3.8)	2 (6.7)	5 (3.2)	0.025
Symptom duration (d)	131.52±76.23	133.8±90.38	128.53±52.27	0.906
Endoscopic abnormalities	143 (77.3)	26 (86.7)	117 (75.5)	0.140
Histologic abnormalities	173 (93.5)	28 (93.3)	145 (93.5)	0.357

Values are presented as mean±standard deviation or number (%).
BMI: body mass index, *H. pylori: Helicobacter pylori*, Hb: hemoglobin.

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24 (80.0%) had eradication of *H. pylori* infection. Also, among 8 patients who had anemia and *H. pylori* infection showed improvement of anemia after eradication of *H. pylori* infection (mean hemoglobin: 9.5 g/dL-> 13 g/dL, average days: 66 days). BIS-based quadruple therapy were applied to 4 patients who fail to achieve eradication of *H. pylori* with standard tripe therapy and they all showed complete eradication of *H. pylori*.

Table 2 shows the clinical characteristics of patients with PUD according to the *H. pylori* status. PUD was observed in 32 (17.3%) patients, and the frequency of *H. pylori* infection in patients with PUD was 18.8%. All patients with PUD in the H. pylori-positive group had duodenal ulcers, whereas duodenal ulcers were present in 46.2% of patients with PUD in the *H. pylori*-negative. Compared with patients with PUD in the *H. pylori*-negative group, those in the *H. pylori*-positive group had a higher rate of complications (33.3 vs. 23.1%, p=0.037), such as bleeding or obstruction. In addition, patients with PUD in the *H. pylori*-positive group showed a higher rate of anemia (33.3 vs. 0.0%, p=0.027) and melena (33.3 vs. 15.4%, p=0.027) compared with those in the *H. pylori*-negative group. However, sample size was too small to conclude its significance, so large-scale, well-designed studies will be needed to support these results.

Table 3 shows the clinical characteristics of patients with *H. pylori* infection according to the presence of nodularity on endoscopy. Among patients with *H. pylori* infection, 12 (40.0%) showed antral nodularity on endoscopy. Other endoscopic findings such as erosive gastritis and PUD were present in 26.7% and 20.0% of *H. pylori*-positive patients, respectively (data not shown). Younger patients had a greater tendency to have antral nodularity than older patients (11.33 vs. 15.11 years, p=0.020). No significant differences were observed in histological abnormalities or eradication rates between the two groups. The proportion of patients with anemia was higher in nodularity-positive patients than nodularity-negative patients (66.7 vs. 0.0%, p=0.021). *H. pylori* eradication rate after the first treatment was 73.3%.

In multivariate logistic regression analysis, family history of *H. pylori* infection (OR, 4.21; 95% confidence interval [CI], 1.78–7.23; p=0.043) and anemia (OR, 9.20; 95% CI, 2.89–10.21; p=0.015) were significant factors related to *H. pylori* infection. Factors associated with *H. pylori* infection are summarized in **Table 4**.

Table 2. Clinical characteristics of patients with peptic ulcer disease according to *H. pylori* infection status

Variable	Total (N=32)	*H. pylori* positive (n=6)	*H. pylori* negative (n=26)	*p*-value
Age (yr)	13.21±2.92	14.33±2.88	12.69±3.37	0.452
Male	16 (50.0)	4 (66.6)	12 (46.2)	0.500
BMI	21.10±2.21	24.12±1.21	18.80±2.76	0.087
Obesity and/or overweight	10 (31.3)	4 (66.6)	6 (23.1)	0.152
Family history of *H. pylori*	4 (12.5)	2 (33.3)	2 (7.7)	0.350
Presence of weight loss	6 (18.8)	2 (33.3)	4 (15.4)	0.506
Hb (g/dL)	12.00±2.52	11.90±3.55	12.40±1.37	0.007
Platelet (10^3/μL)	252.21±45.21	182.16±35.83	315.30±59.90	0.023
Presence of anemia	2 (6.3)	2 (33.3)	0 (0.0)	0.027
Presence of melena	6 (18.8)	2 (33.3)	4 (15.4)	0.027
Combined complication	8 (25.0)	2 (33.3)	6 (23.1)	0.037
Ulcer classification				0.046
Gastric ulcer	14 (43.8)	0 (0.0)	14 (53.8)	
Duodenal ulcer	18 (56.3)	6 (100.0)	12 (46.2)	
Histology (atrophy/IM)	18 (56.3)	6 (100.0)	12 (46.2)	0.049

Values are presented as mean±standard deviation or number (%).
BMI: body mass index, *H. pylori: Helicobacter pylori*, Hb: hemoglobin, IM: intestinal metaplasia.

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Table 3. Clinical characteristics of patients with *H. pylori* infection based on the presence of nodularity on endoscopy

Variable	Nodularity positive (n=12)	Nodularity negative (n=18)	*p*-value
Age (yr)	11.33±2.80	15.11±1.36	0.020
Male	6 (50.0)	10 (55.6)	0.622
BMI (kg/m^2)	20.96±5.65	20.93±4.06	0.522
Obesity and/or overweight	4 (33.3)	2 (11.1)	0.535
Family history of *H. pylori*	2 (16.7)	2 (11.1)	0.325
Presence of weight loss	2 (16.7)	8 (44.4)	0.294
Hb (g/dL)	10.00±1.89	12.46±1.87	0.031
Platelet (10^3/μL)	277.83±28.18	293.16±22.24	0.355
Presence of anemia	8 (66.7)	0 (0.0)	0.021
Eradication rate	8 (66.7)	14 (77.8)	0.348
Histologic abnormalities	12 (100.0)	16 (88.9)	0.545

Values are presented as mean±standard deviation or number (%).
BMI: body mass index, *H. pylori: Helicobacter pylori*, Hb: hemoglobin.

Table 4. Univariate and multivariate analyses of factors associated with of *H. pylori* infection in patients with dyspepsia

Variable	Univariate analysis	*p*-value	Multivariate analysis	*p*-value
	OR (95% CI)		OR (95% CI)	
Age (yr)	1.10 (0.91–1.43)	0.282		
Male	1.52 (0.51–4.51)	0.447		
Obesity and/or overweight	3.45 (1.34–7.03)	0.045	3.94 (1.67–8.08)	0.075
Family history of *H. pylori*	3.88 (1.64–6.53)	0.033	4.21 (1.78–7.23)	0.043
Presence of anemia	7.27 (2.42–9.31)	0.017	9.20 (2.89–10.21)	0.015
Presence of melena	6.46 (1.96–8.40)	0.045	6.55 (1.81–8.68)	0.065
Presence of endoscopic abnormalities	2.88 (0.60–5.82)	0.184		
Presence of histologic abnormalities	0.87 (0.09–4.56)	0.905		

OR: odds ratio, CI: confidence interval, *H. pylori: Helicobacter pylori*.

DISCUSSION

In this study that aimed to evaluate the prevalence and factors associated with *H. pylori* infection in children with dyspepsia, we found that the prevalence of *H. pylori* infection was 16.2% in patients with dyspepsia and 18.8% in those with PUD.

We also found a close association between *H. pylori* infection and obesity, which was similar to the results of a recent meta-analysis reporting that participants with obesity had a higher risk of *H. pylori* infection than lean participants (OR, 1.46) [18]. This may be explained by the role of gastric hormones. The levels of ghrelin, which increases appetite during hunger, and leptin, which decreases appetite and induces satiety, were lower in *H. pylori*-positive individuals than *H. pylori*-negative individuals [19,20]. Therefore, lower serum leptin delays the feeling of satiety during eating and leads to more energy intake and develops obesity. Another possible factor may be higher insulin resistance in *H. pylori* positive patients that leads to obesity development [21].

PUD prevalence was slightly higher in our study; however, the percentage of *H. pylori* infection in patients with duodenal ulcers was lower in our study than in a recent pediatric study [22]. Uğraş and Pehlivanoğlu [23] revealed that the prevalence of PUD was 13.2% in children with upper GI symptoms, whereas that of *H. pylori* infections was 76.9% in children with duodenal ulcers. Studies on *H. pylori*-negative PUD patients have reported that these patients are more likely to have bleeding or multiple and larger ulcers [24]; however, in our study, *H. pylori*-positive patients with PUD tended to have more severe endoscopic and laboratory findings. Studies on the association between PUD and *H. pylori* infection are rare, and more systematic studies are needed to draw definitive conclusions.

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H. pylori positivity is a risk factors for the transmission of *H. pylori* infection to the patient's family. Therefore, detecting *H. pylori* and eradicating it in an entire family is an important treatment strategy, which also includes preventing recurrence. A previous study claimed that parental infection and age ≥7 years were the major risk factors for *H. pylori* infection in children [25]. In our study, a family history of *H. pylori* infection was a significant risk factor associated with *H. pylori* infection in children. However, a family history of *H. pylori* infection had no effect on the presence of *H. pylori* infection in patients with PUD.

Regarding the relationship between *H. pylori* infection and anemia, Kibru et al. [26] have previously reported a higher prevalence of anemia in *H. pylori*-positive patients (30.95 vs. 22.5%, p=0.05) than in *H. pylori*-negative patients, which is similar to the results of our study. The relative risk of developing anemia and iron deficiency anemia was 1.29 and 1.56 times higher, respectively, in participants with *H. pylori* infection than in those without [27]. The association between anemia and *H. pylori* might explained by impairing iron absorption and GI blood loss due to *H. pylori* induced GI lesions and consumption of iron by the *H. pylori* itself [28].

Antral nodularity was the most common endoscopic finding (40%) in *H. pylori*-positive patients. Al Kirdy et al. [29] reported a similar prevalence of antral nodularity in patients with *H. pylori* infection to that in our study; however, they revealed that none of the patients with *H. pylori* infection developed ulceration. Luzza et al. [30] also presented a lower (1%) prevalence of erosion and ulcers than that observed in children with our study in. Because normal endoscopic findings were detected in 13.3% of *H. pylori*-positive patients, this finding does not preclude the diagnosis of *H. pylori*.

The present study had some limitations. First, the retrospective study design may have affected the analyzed variables. Second, the sample size was too small to draw definitive conclusions. Third, as for diagnosing *H. pylori*, we did not perform *H. pylori* culture and antimicrobial susceptibility test. However, diagnosis was done in accordance with the latest guideline. Further prospective design multicenter studies are needed to address these limitations. Despite these limitations, our study is valuable because we evaluated the factors related to *H. pylori* infection as well as the clinical characteristics of *H. pylori*-positive and -negative patients with PUD.

In conclusion, the prevalence of *H. pylori* infection was low in children with dyspepsia. However, children with obesity or who had family history of *H. pylori* infection had higher possibility of having *H. pylori* infection and *H. pylori* infected patients showed a more severe clinical course. So, investigation such as endoscopy and more caution is needed when evaluating children with dyspepsia who had these factors. Further multicenter studies with prospective design are needed to confirm our findings.

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