

## Peptic Ulcers and Erosions in Children at a Pediatric Unit in Turkey

TUGBA KOCA, FILIZ SERDAROGLU, SELIM DEREÇI AND MUSTAFA AKCAM

From the Department of Pediatric Gastroenterology, Hepatology and Nutrition, Suleyman Demirel University School of Medicine, Isparta, Turkey. [tgkoca@gmail.com](mailto:tgkoca@gmail.com)

### Correspondence to:

Dr Tugba Koca

Department of Pediatrics,  
Suleyman Demirel University School  
of Medicine, Cunur, Isparta, Turkey.  
[tgkoca@gmail.com](mailto:tgkoca@gmail.com)

Received: June 16, 2015;

Initial review: August 20, 2015;

Accepted: April 21, 2016.

**Objective:** To study the characteristics of peptic ulcer and erosion in pediatric patients.

**Methods:** Over a period of seven years, 1,026 children underwent upper gastrointestinal endoscopy in our pediatric gastroenterology unit.

**Results:** Peptic ulcers and erosions were found in 59 (7.2%) patients [ulcers in 42 (5.1%) and erosions in 17 (2.1%)]. Thirty (50.9%) children presented with acute upper gastrointestinal bleeding. *Helicobacter pylori* positivity was found in 27 patients (45.8%), and ulcerogenic medication use was found in 13 (22%) patients.

**Conclusion:** The main risk factors for childhood peptic ulcer and erosions were *H. pylori* infection and non-steroidal anti-inflammatory drug use.

**Keywords:** Endoscopy, *Helicobacter pylori*, Non-steroidal anti-inflammatory drugs.

Published online: June 01, 2016. PII:S097475591600006

Increasing availability of endoscopy for pediatric patients has resulted in increased diagnoses of peptic ulcer and erosions [1]. A limited number of studies in children have reported a range of 1.8%-19.5% in prevalence of peptic ulcer [2-4]. Peptic ulcer, together with erosions, has been reported in 10 - 20% of symptomatic children who underwent upper gastrointestinal (GI) endoscopy [3-5]. Generally, peptic ulcer occurs in association with *Helicobacter pylori*, corticosteroids or non-steroidal anti-inflammatory drugs (NSAIDs), systemic diseases or stress [3-6]. In adults, there has been a noticeable increase in the incidence of non *H.pylori*, non-NSAIDs related peptic ulcer [7-9], though not in the pediatric population.

The aim of this study is to describe the frequency and characteristics of peptic ulcer and erosions in pediatric patients who underwent upper GI endoscopy in our institution.

### METHODS

This retrospective study analyzed case records of 1,026 pediatric patients who underwent upper GI endoscopy over a period of seven years from January 2007 to January 2014. These children presented with various GI system complaints. We extracted sociodemographic data, complaints on presentation, medication use, family history of peptic ulcer or erosions, concomitant systemic disease and endoscopy findings. *H.pylori* infection was diagnosed using a rapid urease test, (Helident, RTA,

Kocaeli, Turkey) and a positive histology. At least two biopsies were performed from the gastric antrum, one for histological analysis (including direct visualization of the bacteria), and the other for rapid urease test.

Children with peptic ulcer/erosions with *H.pylori* positivity received triple therapy (lansoprazole, amoxicillin and clarithromycin) for 10 days; lansoprazole was continued for at least four weeks. Successful eradication was defined by follow-up endoscopy, or when urea breath test was negative for *H.pylori* six weeks after the completion of the drug therapy. Patients with peptic ulcers/erosions, who were negative for *H.pylori*, were treated with lansoprazole for four to six weeks. According to our clinical protocol for children diagnosed with peptic ulcers, the patients were re-assessed at four weeks, at two months, and thereafter only if the patient became symptomatic again.

**Statistical analysis:** Statistical analyses were performed using SPSS 15.0 (SPSS Inc., Chicago, Illinois, USA). The differences between patients with gastric ulcer and duodenal ulcer were assessed by the chi-square or dependent t-test for nominal or continuous variables, respectively. A *P* value of <0.05 was considered as statistically significant.

### RESULTS

During the study period, we diagnosed gastric and/or duodenal peptic ulcers/erosions in 59 children (31 girls

and 28 boys) with a mean (SD) age of 12.0 (4.3) years (range 1-18 years). Peptic ulcer was diagnosed in 42 (20 gastric, 20 duodenal and 1 both gastric and duodenal) and erosion in 17 (15 gastric, 1 duodenal and 1 both gastric and duodenal) cases. Thirty patients (50.9%) with peptic ulcers/erosions presented with gastrointestinal bleeding (hematemesis in 24, melena in 6). Other primary indications included epigastric pain ( $n=23$ ), pre-examination of percutaneous endoscopic gastrostomy placement ( $n=3$ ), iron deficiency anemia ( $n=1$ ), poor weight gains ( $n=1$ ), and vomiting ( $n=1$ ).

*Helicobacter pylori* positivity was seen in 27 (45.8%) patients. The rates of *H. pylori* infection were higher in children with duodenal ulcers compared to those with gastric ulcers (71.5% versus 40%,  $P=0.007$ ). Antral nodularity was found in 26 (44.1%) patients, which was higher in *H. pylori* positive patients ( $P<0.001$ ) (Table I).

Thirteen (22%) children had a history of ulcerogenic medication use. Of these, 12 had been given NSAID and one had been given corticosteroids. There was a history of NSAID use in 30% of the gastric ulcer patients and in 14.3% of the duodenal ulcer patients. There was a family history of ulcers in 16.9% of all the cases. In the gastric ulcer cases, 10% had a family history of ulcers, and in the duodenal ulcer cases, one-third had a family history of ulcers ( $P=0.04$ ). There was a concomitant systemic disease in seven patients (4 cerebral palsy, 3 portal hypertension), and two patients were active cigarette smokers. In 19 (32.2%) patients there was no evidence of *H. pylori* infection or history of ulcerogenic medications.

All *H. pylori* positive patients were administered the classic triple eradication therapy. Eight patients remained

*H. pylori* positive after initial treatment, and they were administered sequential therapy. After sequential therapy, these patients did not attend the follow-up appointments. In the other five, there were no symptoms and the UBT turned negative.

Over the mean (SD) follow-up period of 10.9 (13.3) months, ulcer recurred in five patients. Although the recurrence rate was higher in *H. pylori* positive ulcers, the difference was not statistically significant (14.8% vs. 3.1%,  $P=0.10$ ). The mean (SD) time of recurrence was 32 (7.6) months (range, 18-60 months). Of the five patients with recurrence, two presented with GI bleeding and three with epigastric pain.

## DISCUSSION

In this retrospective analysis of over 1000 children in whom upper GI endoscopies were performed, peptic ulcer or erosions were diagnosed in 7.2% of patients. We further documented association with *H. pylori* in nearly half of these patients, more so in those with duodenal ulcers/erosions.

In earlier studies, *H. pylori* infection has been reported at rates of 33-92% in children with duodenal ulcers and at 20-75% in those with gastric ulcers [3,10,11]. Several medications may cause mucosal inflammation and ulceration. Although NSAID are generally safe and are often used to control fever, even the use of a single dose in children may cause GI bleeding [12]. In a previous study, ulcerogenic medication use was found in 16.5% of peptic ulcer patients [2]. In recent studies in developed countries, there has been an increase in *H. pylori* - negative, non-NSAID-related peptic ulcer prevalence [7-9]. In the current study, the prevalence of peptic ulcers not related to *H. pylori* or NSAID was found to be 33.8%.

Characteristic antral nodularity has been reported more often in children with *H. pylori* infection than in adults [10,12]. Antral nodularity has been reported to have 98.5% specific and 91.5% positive predictive value in the determination of *H. pylori* infection [13]. In the current study, the specificity and the positive predictive value of antral nodularity for *H. pylori* detection were 79% and 74.1%, respectively.

The retrospective nature of the analysis constituted the major limitation of our study. Other limitation was lack of a control group for analyzing significant association with *H. pylori* positivity or NSAID use.

In conclusion, the results of this study show that peptic ulcers and erosions are not rare in children. These patients most commonly present with bleeding and/or

**TABLE I** CHARACTERISTICS OF PATIENTS ACCORDING TO *HELICOBACTER PYLORI* STATUS

	<i>H. pylori</i> positive, n(%)	<i>H. pylori</i> negative, n(%)	<i>P</i> value
Age, mean (SD)	12.8 (3.2)	11.3 (4.9)	0.1
Female gender, n (%)	13 (41.9)	18 (58.1)	0.5
Erosions, n (%)			
Gastric	3 (20)	12 (80)	
Duodenal	0	1 (100)	
Gastric+duodenal	0	1 (100)	
Ulcers, n (%)			
Gastric ulcers	8 (40)	12 (60)	
Duodenal ulcers	15 (71.5)	6 (28.5)	
Gastric+duodenal	1 (100)	0	
Antral nodularity, n (%)	20 (74.1)	6 (18.8)	<0.001

epigastric pain symptoms. *H. pylori* infection and NSAID are common associations found in children with peptic ulcers or erosions.

*Acknowledgement:* Caroline Walker for English language editing.

*Contributors:* MAK: had designed and conceptualized the study; FS and SD: reviewed the patients' records; TK: carried out the statistical analysis of the data; TK and FS: wrote the manuscript; MAK: revised the manuscript. All authors approved the final version.

*Funding:* None; *Competing interests:* None stated.

## REFERENCES

1. Suerbaum S, Michetti P. *Helicobacter pylori* infection. *N Engl J Med.* 2002;347: 1175-86.
2. Huang SC, Sheu BS, Lee SC, Yang HB, Yang YJ. Etiology and treatment of childhood peptic ulcer disease in Taiwan: A single center 9-year experience. *J Formos Med Assoc.* 2010;109:75-81.
3. Egbaria R, Levine A, Tamir A, Shaoul R. Peptic ulcers and erosions are common in Israeli children undergoing upper endoscopy. *Helicobacter.* 2008;13:62-8.
4. Tam YH, Lee KH, To KF, Chan KW, Cheung ST. *Helicobacter pylori*-positive versus *Helicobacter pylori* negative idiopathic peptic ulcers in children with their long-term outcomes. *J Pediatr Gastroenterol Nutr.* 2009;48:299-305.
5. Nijevitch AA, Sataev VU, Vakhitov VA, Loguinovskaya VV, Kotsenko TM. Childhood peptic ulcer in the Ural area of Russia: clinical status and *Helicobacter pylori*-associated immune response. *J Pediatr Gastroenterol Nutr.* 2001;33:558-64.
6. Moll Harboe K, Midtgaard H, Wewer V, Cortes D. Development of a perforated peptic ulcer in a child during high dose prednisolone treatment. *Ugeskr Laeger.* 2012;174:2308-10.
7. Arents NL, Thijs JC, vanZwet AA, Kleibeuker JH. Does the declining prevalence of *Helicobacter pylori* unmask patients with idiopathic peptic ulcer disease? Trends over an 8-year period. *Eur J Gastroenterol Hepatol.* 2004;16:779-83.
8. Arroyo MT, Forme M, de Argila CM, Feu F, Arenas J, De la Vega J, *et al.* The prevalence of peptic ulcer not related to *Helicobacter pylori* or non-steroidal anti-inflammatory drug use is negligible in Southern Europe. *Helicobacter.* 2004;9:240-54.
9. Quan C, Talley NJ. Management of peptic ulcer disease not related to *Helicobacter pylori* or NSAIDs. *Am J Gastroenterol.* 2002;97:2950-61.
10. Kalach N, Bontems P, Koletzko S, Mourad-Baars P, Shcherbakov P, Celinska-Cedro D, *et al.* Frequency and risk factors of gastric and duodenal ulcers or erosions in children: a prospective 1- month European multicenter study. *Eur J Gastroenterol Hepatol.* 2010;22:1174-81.
11. Ugras M, Pehlivanoglu E. *Helicobacter pylori* infection and peptic ulcer in eastern Turkish children: is it more common than known? *Turk J Pediatr.* 2011;53:632-7.
12. Berezin SH, Bostwick HE, Halata MS, Feerick J, Newman LJ, Medow MS. Gastrointestinal bleeding in children following ingestion of low-dose ibuprofen. *J Pediatr Gastroenterol Nutr.* 2007;44:506-8.
13. Bahú Mda G, da Silveira TR, Maguilnick I, Ulbrich-Kulczynski J. Endoscopic nodular gastritis: An endoscopic indicator of high-grade bacterial colonization and severe gastritis in children with *Helicobacter pylori*. *J Pediatr Gastroenterol Nutr.* 2003;36:217-22.

---

## Erratum

In the editorial entitled "Galactosemia – A Not to be Missed Inborn Error of Metabolism" published in January 2016 issue of *Indian Pediatrics*, on page no. 19, following correction is to be noted in first paragraph, lines 13-15:

"If we extrapolate the reported incidence from the western world (1:30,000) about 87 babies every year are born with galactosemia in India." This line is to be read as:

"If we extrapolate the reported incidence from the western world (1:30,000) about 870 babies every year are born with galactosemia in India."