Case report

**Helicobacter heilmannii** infection in a child after successful eradication of **Helicobacter pylori**: case report and review of literature

Seiichi Kato¹, Kyoko Ozawa¹, Hitoshi Sekine², Mikio Ohyauchi², Tooru Shimegawa², Takanori Minoura¹, and Kazuie Inuma¹

¹Department of Pediatrics, Tohoku University School of Medicine, 1-1 Seiryo-machi, Aoba-ku, Sendai 980-8574, Japan
²Department of Gastroenterology, Tohoku University School of Medicine, Sendai, Japan

An 11-year-old boy with **Helicobacter pylori**-associated duodenal ulcer was successfully treated with a combination of lansoprazole, amoxicillin, and clarithromycin. Endoscopy and gastric biopsies were repeated 2 and 12 months later, showing ulcer healing and eradication of **H. pylori**. However, a 3-year follow-up study demonstrated **H. heilmannii** in the antral mucosa based on its characteristic morphology and positive urease test and negative culture. The patient had no contact with domestic animals such as cats and dogs. A 7-day course with lansoprazole, amoxicillin, and clarithromycin was performed again, resulting in successful eradication of the organism. Pediatric cases with **H. heilmannii** infection reported are reviewed.

**Key words:** child, eradication, gastritis, **Helicobacter heilmannii**, **Helicobacter pylori**

Introduction

**Helicobacter pylori** infection is associated with the development of chronic gastritis, peptic ulcer disease, gastric adenocarcinoma, and mucosa-associated lymphoid tissue (MALT) lymphoma. **H. pylori** is also an important causal factor for gastritis and peptic ulcer disease in children.¹² Recently, an increasing number of **Helicobacter** species other than **H. pylori**, including **H. heilmannii** (formerly **Gastrospirillum hominis**), **H. felis**, and **H. cinaedi**, have been identified in human stomach or feces.³ Compared with **H. pylori**, **H. heilmannii** is longer (3.5–7.5 µm) with six to eight coils per cell and up to 12 sheathed flagella at each pole. It is thought that humans typically acquire **H. heilmannii** infection from domestic animals as a zoonosis.³ Similar to **H. pylori**, **H. heilmannii** has been implicated in the pathogenesis of chronic gastritis, peptic ulcer disease, gastric cancer, and gastric MALT lymphoma in adults.⁴⁻⁶ There are several reports in English of childhood **H. heilmannii** infection.⁹⁻¹⁸ We report here a pediatric case in which **H. heilmannii** infection was identified after **H. pylori** was successfully eradicated for duodenal ulcer.

Case report

In April 2000, an 11-year-old boy was referred to the University Hospital because of persistent epigastric pain. His father had a history of duodenal ulcer. Upper gastrointestinal endoscopy showed ulceration in the duodenal bulb and ulcer scar at the gastric angle. The **¹³C-urea breath test (UBT)** was positive with ∆**¹³C** value of 34.9‰ (cutoff value = 3.5‰).¹⁹ The urease test, histology, stool antigen test, and culture were all positive for **H. pylori** infection (Table 1). He was treated with a 10-day course of triple therapy consisting of lansoprazole, amoxicillin, and clarithromycin and became symptom free. Upper gastrointestinal endoscopy was repeated 2 months after the therapy was completed, showing complete healing of the duodenal ulcer. Gastric histology showed chronic inflammation without neutrophil infiltration (Table 1). **H. pylori** testing as already mentioned confirmed eradication of the organism. The 1-year follow-up endoscopy demonstrated healed ulcer and **H. pylori** tests including **¹³C-UBT** (0.7‰) were all negative. Histology showed mild chronic gastritis.

On February 2003, the patient and his parents agreed to undergo 3-year follow-up endoscopy. At that time, he had no gastrointestinal symptoms. Active gastroduodenal ulcer was not found and the gastric mucosa was endoscopically normal. **¹³C-UBT** was negative with ∆**¹³C** value of 1.2‰ and the stool antigen test was also negative (Table 1). Culture of the biopsy specimen was nega-
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**Table 1. Results of histology and Helicobacter pylori tests of the patient**

<table>
<thead>
<tr>
<th>Date</th>
<th>Inflammation</th>
<th>Activity</th>
<th>Urease test</th>
<th>(^{13}\text{C}-\text{UBT}\text{b} (%)</th>
<th>Culture</th>
<th>Stool ELISA</th>
</tr>
</thead>
<tbody>
<tr>
<td>July 2000</td>
<td>2/1</td>
<td>2/0</td>
<td>Positive/negative</td>
<td>34.9</td>
<td>Positive</td>
<td>Positive</td>
</tr>
<tr>
<td>Sept. 2000</td>
<td>2/1</td>
<td>0/0</td>
<td>Negative/negative</td>
<td>0.0</td>
<td>Negative</td>
<td>Negative</td>
</tr>
<tr>
<td>June 2001</td>
<td>1/1</td>
<td>0/0</td>
<td>Negative/negative</td>
<td>0.7</td>
<td>Negative</td>
<td>Negative</td>
</tr>
<tr>
<td>Feb. 2003</td>
<td>2/1</td>
<td>0/0</td>
<td>Positive/negative</td>
<td>1.2</td>
<td>Negative</td>
<td>Negative</td>
</tr>
<tr>
<td>Aug. 2003</td>
<td>1/0</td>
<td>0/0</td>
<td>Negative/negative</td>
<td>0.0</td>
<td>Negative</td>
<td>Negative</td>
</tr>
</tbody>
</table>

\(^{a}\)Gastric antrum/corpus. The grade is based on the Updated Sydney System

\(^{b}\)\(^{13}\text{C}\)-urea breath test; cutoff value, 3.5‰

HpSA, Premier Platinum HpSA

**Discussion**

The present case showed acquisition of *H. heilmannii* infection after successful eradication of *H. pylori*. Cases with simultaneous infection with *H. pylori* and *H. heilmannii* have been reported, although rare.\(^{21}\) In our case, detailed histological examination of the first and subsequent biopsy specimens did not show *H. heilmannii*-like organisms. Compared with *H. pylori* infection, *H. heilmannii* infection is less prevalent with the rate of 0.3%–0.4% of children\(^{11,14,16}\) and <0.5% of adults undergoing endoscopy.\(^{3}\) Because cats, dogs, and pigs are reservoirs of *H. heilmannii*, it is thought that close contact with these domestic animals is the major risk factor for acquisition of the infection.\(^{3,22}\) In one study of a child with *H. heilmannii* infection,\(^{12}\) the pet dogs were endoscoped, demonstrating the organism in the stomach. In addition, *ureAB* gene sequencing strongly supports the cat-to-human transmission of *H. heilmannii*.\(^{17}\) In most pediatric cases with *H. heilmannii* infection, dogs and/or cats were reported as domestic animals (Table 2). However, three infected cases including ours did not have known contact with such domestic animals.\(^{10,18}\) Because we used an endoscope disinfected with 0.3% peracetic acid and sterilized disposable biopsy forceps, it is not likely that *H. heilmannii* was transmitted via the endoscopic procedure. The exact transmission route of *H. heilmannii* remains to be established.

As described in Table 2, *H. heilmannii* infection causes chronic gastritis in children and is reported to be associated with gastric ulcer\(^{18}\) or anemia.\(^{15}\) *H. heilmannii* induces chronic inflammation in the gastric mucosa but the degree is generally milder than with *H. pylori*.\(^{7}\) In addition, *H. heilmannii* colonization is commonly focal and restricted to the antrum.\(^{7}\) In our case, previous *H. pylori* infection showed chronic active gastritis with grade 2 infiltration of neutrophils in the antrum, whereas the following *H. heilmannii* infection showed chronic gastritis without neutrophil infiltration.