Case report

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

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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.1,2 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.4–8 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 Δ13C value of 34.9‰ (cutoff value /H11005 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 Δ13C value of 1.2% and the stool antigen test was also negative (Table 1). Culture of the biopsy specimen was nega-
tive for *H. pylori*. However, urease test was positive for the antral specimen whereas it was negative for the corpus specimen. *H. heilmannii* with its characteristic morphology was found in the antral mucosa (Fig. 1). According to the Updated Sydney System, inflammation of the antrum increased up to grade 2 but that of the corpus was unchanged. No neutrophil infiltration was found. The patient and his family did not have contact with domestic animals, including cats and dogs. Because the parents desired eradication therapy, he was treated again with a 7-day course with lansoprazole (20 mg, b.i.d.), amoxicillin (750 mg, b.i.d.), and clarithromycin (400 mg, b.i.d.). Upper gastrointestinal endoscopy and biopsy were performed 6 months later. $^{13}$C-UBT, urease test, culture, and the stool antigen test showed negative results. *H. heilmannii* was not histologically detected. The degrees of inflammation in the antrum and corpus decreased to grades 1 and 0, respectively (see Table 1).

### 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. 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 and 0.5% of adults undergoing endoscopy. 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. In one study of a child with *H. heilmannii* infection, 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*. In most pediatric cases with *H. heilmannii* infection, dogs and/or cats were reported as domestic animals. However, three infected cases including ours did not have known contact with such domestic animals. 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 or anemia. *H. heilmannii* induces chronic inflammation in the gastric mucosa but the degree is generally milder than with *H. pylori*. In addition, *H. heilmannii* colonization is commonly focal and restricted to the antrum. 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.