Case report

Cap polyposis cured by *Helicobacter pylori* eradication therapy

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The pathogenesis of cap polyposis remains unknown. Here, we report a patient with cap polyposis that developed simultaneously in the colon and stomach, and which regressed after *Helicobacter pylori* eradication. A 63-year-old man was diagnosed as having cap polyposis with mucoid stool, diarrhea, and bleeding on defecation. Following 5 weeks of total parenteral nutrition, his symptoms and hypoproteinemia improved and he was discharged, although follow-up colonoscopic findings revealed no improvement. Subsequent gastroscopy revealed diffusely erosive polyps with cap-like “fur” from the angle to the antrum of the stomach, similar to the lesions observed in the colon. Because *H. pylori* infection was demonstrated in the stomach, eradication therapy was administered. After this treatment, his symptoms immediately disappeared, and the polyloid lesions in the colon and stomach had completely disappeared 8 months later. Because there have been no previous reports of a relationship between *H. pylori* and cap polyposis, this case is of great interest.

**Key words:** cap polyposis, eradication therapy, gastric lesion, *Helicobacter pylori*

Introduction

In 1985, cap polyposis was first reported, by Williams et al., as an idiopathic disease associated with hypoproteinemia. Cap polyposis has specific clinical, colonoscopic, and histological features, but its pathogenesis is unknown. We report here an unusual case of cap polyposis that was cured by the eradication of *Helicobacter pylori*.

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infiltration in the lamina propria: their surface was covered with caps of inflammatory granulation tissue (Fig. 2). Based on these findings, a diagnosis of cap polyposis was made.

The patient was discharged on June 4, 1998, due to decreased mucoid stool and improvement in total serum protein level. On December 18, 1998, barium enema revealed multiple varioliform elevated lesions from the rectum to the ascending colon (Fig. 3). Subsequently, he underwent follow-up colonoscopy every 6 months, with no improvement.

On October 7, 1998, gastroscopy was performed, revealing diffusely erosive polypoid lesions with cap-like “fur” from the angle to the antrum of the stomach (Fig. 4). These lesions were similar to those observed in the colon. Histopathological examination of biopsy specimens from polypoid lesions in the stomach revealed hyperplasia of glands, infiltration of neutrophils, and intestinal metaplasia (Fig. 5), as well as the presence of *H. pylori*. Although he initially refused *H. pylori* eradication therapy, triple therapy, consisting of 20mg of sodium rabeprazole, 800mg of clarithromycin, and 1500mg of amoxycillin daily, was administered for 2 weeks from March 31, 2000. After the treatment, the mucoid stool disappeared. On June 9, 2000, he was negative for *H. pylori* on urease test, culture, and histopathological examination. Colonoscopy 8 months after *H. pylori* eradication confirmed that varioliform elevated lesions and caps had completely disappeared from the rectum to the ascending colon. Gastroscopy

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Fig. 1. *a* Multiple flattened polypoid lesions were observed in the rectum. *b* Varioliform elevated lesions with caps were observed in the sigmoid colon

Fig. 2. Biopsy specimen of the sigmoid colon revealed elongated glands containing mucus, with inflammatory cellular infiltration. Inflammatory granulation tissue was observed in the surface layer. H&E, ×50

Fig. 3. Barium enema revealed multiple varioliform elevated lesions extending from the rectum to the upper ascending colon