

Clinical Remission of Rectal Cap Polyposis Using Eradication of H. Pylori- A Case Report

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Case Report

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Abstract: Cap polyposis is a rare disorder with characteristic endoscopic and histological features; its etiology is still unknown, and no specific treatment has been established (1, 2, 3). We report a case of rectal cap polyposis that improved remarkably after eradication of H. Pylori.

Keywords: Cap polyposis, H. Pylori.

INTRODUCTION

Cap polyposis is a rare but distinct disorder with characteristic endoscopic and histological features [2,3]. It is characterized by multiple distinctive erythematous, inflammatory colonic polyps located from the rectum to the distal colon [2, 3]. The polyps are covered with fibrin purulent mucus which appears like a 'cap.' The most common symptoms are mucous and bloody diarrhea with abdominal pain and tenesmu [1,2]. The etiology of this disease is still unknown, and no specific treatment has been established. There have been a few case reports about cap polyposis response to eradication of H. Pylori. (3) We report a cap polyposis case that dramatically improved after eradication of H. Pylori.

CASE PRESENTATION

A 19-year-old man was admitted to our hospital with bloody mucous stools, frequent defecation (6-7 times/day) and tenesmus for several weeks. On physical examination, abdomen was soft and with no tenderness or palpable mass.

Hemoglobin was 11,5 g/dL, white blood cell count was 6,380/mm³, platelet count was 319,000/mm³. Erythrocyte sedimentation rate was within normal. Stool occult blood test was positive, but, Clostridium difficile antigen assay of stool was negative.

Colonoscopy showed about 8-10 pedunculated polyps (10-15mm) in the rectum covered with white purulent exudates, and scattered hyperemia on rectum.

The polyps were located on apices of mucosal fold (Fig. 1). The histological findings revealed two polypoid fragments of colonic mucosa with ulcerated surface epithelium (Fig.2). The glands are cystically dilated and lined by mucin-secreting cells with increased goblet cells secretion. The lamina propria is markedly infiltrated by acute and chronic inflammatory cells along with dilated and congested blood vessels. No evidence of dysplasia or malignancy.

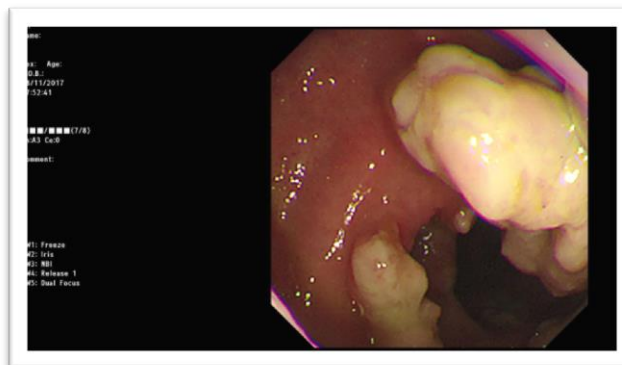


Fig-1: pedunculated polyps (10-15mm) in the rectum covered with white purulent exudates

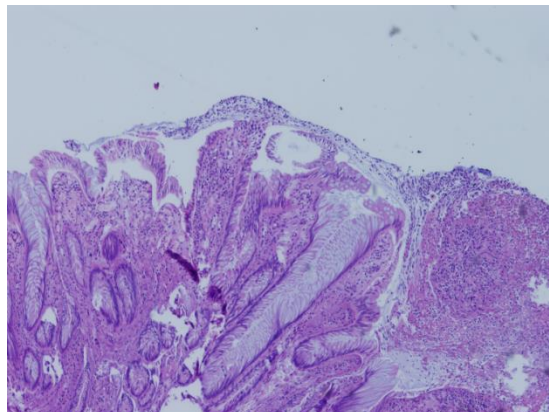


Fig-2: The microscopic finding revealed elongated hyperplastic glands with inflammatory infiltrate in the lamina propria and fibromuscular obliteration of lamina propria. The cap of polyp is formed by mucus, fibrin, and inflammatory cells

After discussion among the medical team, we initiated a regimen to eradicate *H.pylori* for 10 days (amoxicillin 1g twice daily, clarithromycin 500mg twice daily and pantoprazole 40mg twice daily). The patient experienced a dramatic clinical response in less than week in terms of very minimal rectal bleeding (1time in

2 weeks), absence of tenesmus and normal bowel habit. 4 months later, we repeated the colonoscopy which revealed reductions in the size and number of the pedunculated polyps (Fig.3). The patient continued to follow-up in our surgical clinic with the plan to do follow-up colonoscopy in 3-6 months.

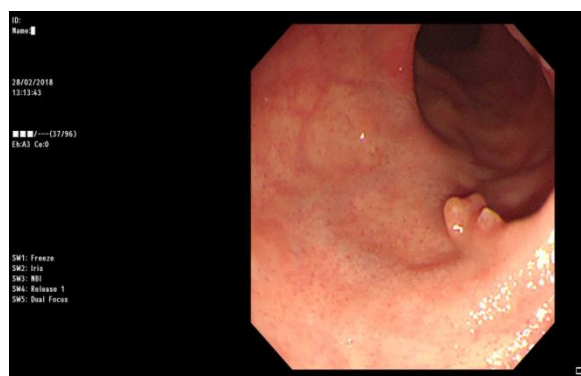


Fig-3: reductions in the size and number of the pedunculated polyps

DISCUSSION

Common clinical manifestation of cap polyposis is bloody mucous diarrhea lasting for weeks to months. It more prevalent in women. Tenesmus, rectal bleeding, abdominal pain, and altered bowel habits have also been reported. Epidemiology and etiology of cap polyposis are not well known. Several theories have been proposed to explain the pathogenesis, including a form of inflammatory bowel disease, an infectious origin such as *Helicobacter pylori* or *Escherichia coli* 018, improvement after antibiotics treatment, whereas other suggested on association with mucosal prolapse syndrome or abnormal colonic motility resulting in local ischemia and recurrent mucosal trauma. Diagnosis of this disease in the present case was established through colonoscopy finding, clinical manifestation and histological finding. The endoscopic finding showed multiple polyps with adherent fibrin purulent exudates like a cap, and this finding resembled inflammatory polyp or pseudomembranous colitis. The microscopic finding

revealed elongated hyperplastic glands with inflammatory infiltrate in the lamina propria and fibromuscular obliteration of lamina propriety. The cap of polyp is formed by mucus, fibrin, and inflammatory cells.

Several case reports have suggested a few treatment modalities, based on etiological hypothesis; anti-inflammatory agent, antibiotics, immunomodulatory, and endoscopic and surgical therapy. However optimal treatment has not yet been established. In our present case, the patient fortunately achieved remarkable clinical and endoscopic responses after eradication of *H. pylori*. Consequently, our present case might support the hypothesis that *H. pylori* play a role in the pathogenesis of cap polyposis. Of course, additional studies about the relationship between *H. pylori* and cap polyposis are needed. Nevertheless we suggest that eradication of *H. pylori* might be at least the first step in the treatment for cap polyposis patients to be considered...

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