Journal of Crohn's and Colitis (2013) 7, e108–e111

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SHORT REPORT

Cap polyposis mistaken for Crohn's disease: Case report and review of literature

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Received 12 April 2012; received in revised form 8 June 2012; accepted 8 June 2012

KEYWORDS
Cap polyposis;
Protein losing enteropathy;
Hypoalbuminemia;
Crohn's disease

Abstract

We report the case of a 19-year old male who presented with collapse and hypoglycemia associated with two weeks of frequent hard stools, abdominal pain relieved by defecation, postprandial vomiting and significant weight loss. Radiologically and endoscopically a diagnosis of Crohn's colitis was made and the patient was treated with steroids and immunosuppression. Following several hospital admissions treatment had to be escalated to include anti-TNF-α agents. Despite maximum therapy the patient continued to deteriorate symptomatically and biochemically with severe hypoalbuminemia and persistent anemia and a total colectomy was performed. Intra-operative finding was that of an inflamed large intestine and pseudo-polyposis but histology was reported as cap polyposis. The specimen was compared with the biopsies obtained from the earlier colonoscopies and it was felt that the previous samples were taken from areas of severely inflamed polypoid mucosa with histology mimicking colitis in inflammatory bowel disease.

1. Introduction

Cap polyposis is a very rare intestinal disease of unknown etiology which variably affects both sexes in their mid-fifties. Symptomatically however there can be significant overlap with Inflammatory Bowel Disease (IBD) such as Crohn's disease (CD) and ulcerative colitis (UC). These disorders are much more common, especially in New Zealand[1] and typically

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doi:10.1016/j.crohns.2012.06.005

affect young patients and those with a family history of IBD. Cap polyposis can easily be misdiagnosed as IBD as illustrated in our case.

2. Case

We report the case of a 19-year old Caucasian male who presented acutely with loss of consciousness and hypoglycemia of 2.3 mmol/L associated with two weeks of frequent hard stools containing fresh blood, abdominal pain relieved by defecation, post-prandial vomiting and weight loss of 10 kg. He had no significant past medical history, no recent travel history or infectious contacts and no non-steroidal anti-inflammatory drugs or illicit drug use. His maternal grandfather was diagnosed with colorectal cancer aged 62 years and a maternal cousin diagnosed with Crohn's disease aged 6 years. Physical examination found no abnormality aside from a tachycardia of 110–120 beats/min and some tenderness over the lower abdomen with no signs of peritonism. Blood test results at initial presentation are shown in Table 1. Stool samples were negative for *Clostridium difficile* toxin and other pathogens. Abdominal X-ray was normal but a CT scan showed diffuse circumferential wall thickening involving the distal ileum and the entire colon with widespread mesenteric vascular congestion associated with multiple prominent lymph nodes. Proctosigmoidoscopy was normal macroscopically. Based on these findings a diagnosis of Crohn's disease was considered and treatment with steroids initiated. The patient represented two weeks later with a further episode of loss of consciousness and hypoglycemia of 1.9 mmol/L. Blood tests showed a further increase in C-reactive protein (83 mg/L), mild leukocytosis ($11.1\times10^{6)}$ and a further drop in serum albumin (24 g/L). Ileocolonoscopy was performed which showed multiple discrete ulcers of variable sizes, cobblestoning and thickened bowel wall involving the entire colon with rectal sparing (Fig. 1). Examination of the biopsy series (terminal ileum to rectum) showed changes throughout the entire colon consistent with chronic active pancolitis with sparing of the terminal ileum (Figs. 2, 3).

Table 1 Laboratory results on initial presentation.

Full blood count	Hemoglobin 130 g/L (NR 120–145), platelet 368×10^{6} (NR 150–450), white cell 11.1×10^{6} (NR 3–10)
Electrolytes and renal function	Sodium 135 mmol/L (NR 135–145), potassium 3.9 mmol/L (NR 3.5–5.3), urea 3 mmol/L (NR 2.5–7), creatinine 79 μmol/L (NR 40–100)
Liver function test	Bilirubin 11 μmol/L (NR 2–21), ALP 73 U/L (NR 35–115), GGT 16 U/L (NR <61), ALT 9 U/L (NR 8–41), total protein 67 g/L (NR 60–80), albumin 33 g/L (NR 35–50), globulin 34 g/L (NR 18–36)
Iron studies	Serum iron 3 μmol/L (NR 10–30), ferritin 68 μg/L (NR 20–350), transferrin 1.5, transferrin saturation 8% (NR 16–50)
C-reactive protein	44 mg/L (NR<3)
Stool specimen	Negative culture for pathogens and *Clostridium difficile* toxin

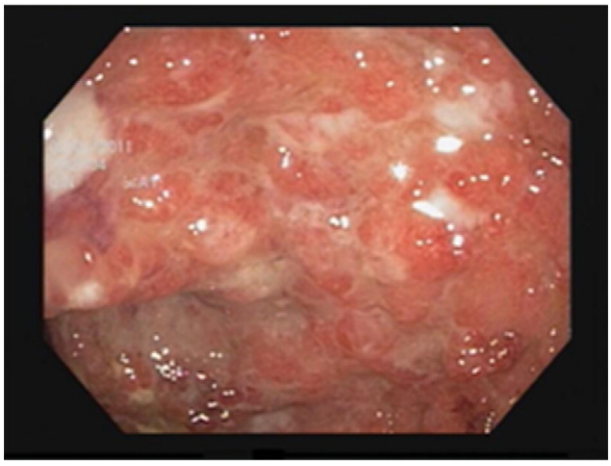

Figure 1 Markedly inflamed colonic mucosa with cobblestoning and fibrinopurulent exudates overlying multiple polyps.

In the following ten months the patient showed only partial clinical and biochemical improvement with less frequent bowel motions and improved appetite with some weight gain but developed pain in the large peripheral joints. However, he remained anemic (lowest hemoglobin 70 g/L) and hypoalbuminemic (lowest albumin 16 g/L), felt generally tired and fatigued, had pedal edema secondary to hypoalbuminemia but clinically no ascites. Symptomatically, he was treated with a blood transfusion, intravenous iron and a high protein diet. Adalimumab was commenced concurrent with prednisone but symptoms relapsed when the prednisone dose was tailed off. Treatment was switched to infliximab and azathioprine which resulted only in modest clinical and biochemical improvement. For further staging a wireless capsule endoscopy was performed which showed patchy erythematous areas in the proximal jejunum and multiple small polyps in the mid- and distal ileum. Gastroscopy and duodenal biopsies were unremarkable with no evidence of *Helicobacter pylori* infection. Finally, the patient was re-admitted with continuous weight loss and

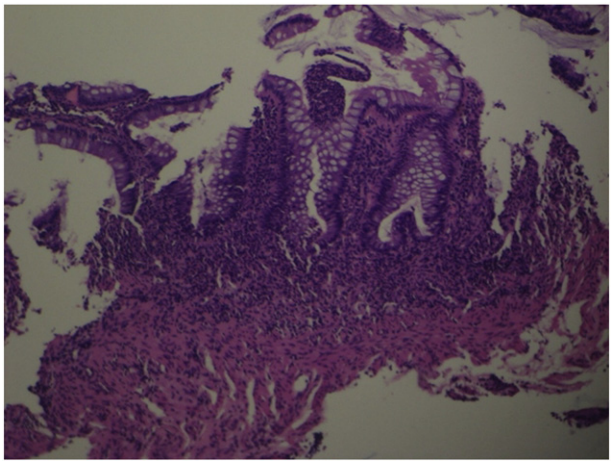

Figure 2 Crypt branching and shortening.