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CASE REPORT

# Solitary rectal cap polyp: Case report and review of the literature

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## Abstract

Rectal bleeding combined with the presence of a rectal mass has been traditionally associated with the presence of malignant disease. Cap polyposis is a relatively young and still undefined rare entity which mainly involves the rectosigmoid. It is characterized by the presence of inflammatory polyps. In this case report, we present a patient who was diagnosed with a solitary cap polyp of the rectum during the investigation of a bleeding rectal mass. The patient's age and the absence of family history were not in favor of malignancy, despite the strong initial clinical impression. After confirmation of the diagnosis, the patient underwent a snare excision and remains asymptomatic. Cap polyposis, although rare, should be suspected and, when diagnosed, should be treated according to location, number of polyps and severity of symptoms.



**Key words:** Cap polyposis; Inflammatory polyp; Rectal mass

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## INTRODUCTION

Cap polyposis was initially described by Williams *et al*[1] in 1985. From that initial report and before a review of the literature by Ng *et al*[2] in 2004, sporadic cases emerged in the literature in which constipation seemed to be closely associated with it, thus postulating colonic dysmotility and mucosal prolapse as the etiological factor. Diagnosis was usually set by exclusion of ulcerative colitis, after poor response to mesalamine and steroids, and surgical resection was considered the optimal treatment[3]. However, new cases of cap polyposis reported in the literature modified the initial belief concerning the pathogenesis and treatment of this entity. We herein present the case of a patient diagnosed with cap polyposis and review the literature, summarizing and analyzing the available data from cases reported in the past and proposing an algorithm for the treatment of cap polyposis.

## CASE REPORT

A 21-year-old Caucasian male presented in our clinic with intermittent rectal bleeding that had lasted for 10 mo. His past medical history was unremarkable. At admission, digital examination revealed a lobular mass located at

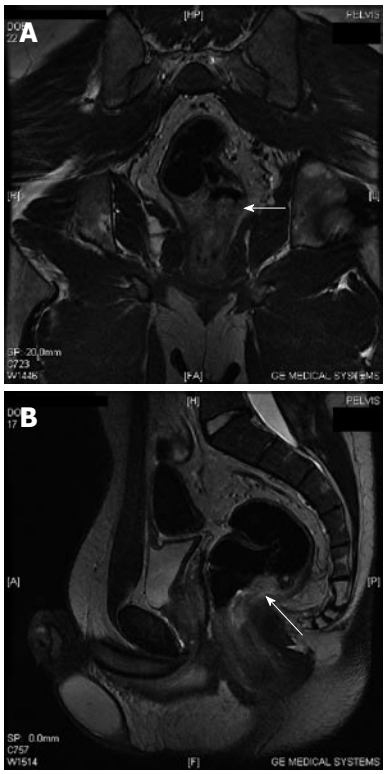


**Figure 1 Coronal (A) and sagittal (B) T2 sequence MRI demonstrating the polyp (arrows).**

the posterior wall of the rectum, 2 cm above the dentate line. Laboratory tests were unremarkable, with the exception of mild hypoproteinemia (protein: 5.7 g/dL, normal range 6-8 g/dL) and hypoalbuminemia (alb: 3.2 g/dL). CEA was within normal range (1.0 ng/mL). Colonoscopy revealed the presence of a large, lobular polyp, with fibrinopurulent exudate on its apical surface. The lesion was approximately 6 cm in diameter and occupied more than 50% of the lumen. Bioptic material was obtained and malignancy was excluded. The patient underwent Magnetic Resonance Imaging of the pelvis (Figure 1). The lesion present in the rectum was confined to the mucosa and did not breach the muscularis mucosa. Therefore, snare excision in piecemeal fashion was performed. The specimen consisted of multiple tissue fragments, 0.3-2.5 cm at maximum diameter, some with polypoid morphology, often with a reddish eroded surface, covered in areas by white mucus or purulent exudate. On a cut section, the tissue fragments were tan-brown or reddish in color and of soft, rubbery consistency, with small mucin-filled areas. Their total dimensions were estimated at 6 cm × 6 cm × 1 cm.

Histopathological examination showed polypoid tissue fragments exhibiting elongated, dilated or tortuous hyperplastic colonic crypts in their central parts and covered in most superficial regions by a "cap" of inflamed and ulcerated granulation tissue, fibrin and inflammatory exudate (Figures 2 and 3). The intervening lamina propria contained increased numbers of inflammatory cells. Mucin lakes of different sizes were present and areas of fibrosis were common in the central part of several fragments. Hemosiderin deposits were observed in places. A

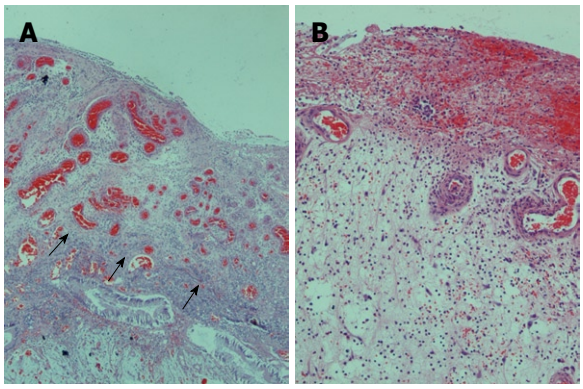


**Figure 2 A cap of inflamed and ulcerated granulation tissue was observed in most superficial regions (arrows) (A), covered by fibrin and inflammatory exudate, as presented in higher magnification in (B).**

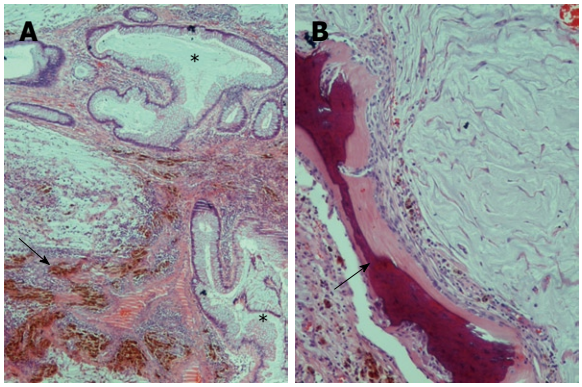


**Figure 3 Dilated or tortuous colonic crypts (asterisks) were observed in central parts, alternating with lakes of mucin and hemosiderin deposits (arrow) in fibrotic or inflammatory areas (A), and heterotopic bone formation (arrow) was observed focally, adjacent to mucin lakes (B).**

few small foci of osseous metaplasia were observed in the proximity of mucin lakes. The findings were considered consistent with the diagnosis of inflammatory cap polyp.

The patient was investigated for *Helicobacter pylori* (*H. pylori*) infection but gastroscopy and Campylobacter-like organism test were negative.The patient was readmitted 1 mo later for follow-up control, being asymptomatic. Serum albumin was within reference values (alb: 4.4 g/dL). Lower gastrointestinal endoscopy was repeated. At the site of polypectomy, a large ulcer with adenomatous tissue in its center was depicted. Subsequently, the patient was examined under anesthesia and the pathological tissue was removed and sent for histopathological examination, which was negative for recurrence. One year later, the patient remains asymptomatic.

## DISCUSSION

A PUBMED search using the key words "cap polyp" and "cap polyposis" was performed from 1993 to the present day (2011). The search retrieved 23 articles with 29 cases. The articles were reviewed, analyzing patient data concerning sex, age, clinical presentation, endoscopic findings, treatment and clinical outcome (Table 1).

**Table 1 Cases of cap polyposis reviewed**

| No. | Case | Age (yr) | Gender | Clinical presentation | Location | Solitary *vs* multiple lesions | Presumed or Initial diagnosis | Treatment | Outcome |
|---|---|---|---|---|---|---|---|---|---|
| 1 | Campbell *et al*[3], 1993 | 68 | M | Weight loss, diarrhea | Sigmoid | Multiple | UC | Total colectomy | Resolved |
| 2 | Campbell *et al*[3], 1993 | 65 | F | Diarrhea | Rectum | Multiple | Solitary rectal ulcer *vs* infection | Sigmoid diverting colostomy/ cleversal enema | Improved |
| 3 | Géhénot *et al*[4], 1994 | 42 | F | Bloody diarrhea | Recto sigmoid | Multiple | Non-specific colitis | Sigmoid colostomy | Resolved |
| 4 | Oshitani *et al*[5], 1998 | 54 | F | Hypoproteinemia, diarrhea | Desc. colon | Multiple | UC | Left hemicolectomy | Resolved |
| 5 | Peny *et al*[6], 1998 | 72 | M | Diarrhea, tenesmus | Rectum to ascending colon | Multiple | UC | Proctocolectomy | Resolved |
| 6 | Oriuchi *et al*[7], 2000 | 20 | F | Hypoproteinemia, mucousy diarrhea | Recto sigmoid | Multiple | | Avoidance of straining | Resolved |
| 7 | Oriuchi *et al*[7], 2000 | 52 | F | Hypoproteinemia, mucousy diarrhea | Rectum | Multiple | | Diversion transverse double barrel colostomy | Resolved |
| 8 | Kajihara *et al*[8], 2000 | 38 | F | Bloody diarrhea | Recto sigmoid | Multiple | CP | Metronidazole | Resolved |
| 9 | Isomoto *et al*[9], 2001 | 51 | F | Mucousy bloody diarrhea | Recto sigmoid | Multiple | Non-specific colitis | APR | Resolved |
| 10 | Esaki *et al*[10], 2001 | 21 | M | Weight loss, diarrhea | Recto sigmoid | Multiple | | Metronidazole | Resolved |
| 11 | Esaki *et al*[10], 2001 | 67 | F | Abdominal pain, mucousy diarrhea | Recto sigmoid | Multiple | | Anterior resection | Resolved |
| 12 | Esaki *et al*[10], 2001 | 21 | M | Abdominal pain | Rectum | Multiple | | Patient refused | |
| 13 | Esaki *et al*[10], 2001 | 76 | F | Diarrhea, tenesmus | Recto sigmoid | Multiple | | Patient refused | Resolved |
| 14 | Sadamoto *et al*[11], 2001 | 73 | M | Asymptomatic | Sigmoid to cecum | Multiple | CP | Observation | Resolved |
| 15 | Oiya *et al*[12], 2002 | 63 | M | Mucousy diarrhea | Rectum to ascending colon | Multiple | | *H. pylori* eradication therapy | Resolved |
| 16 | Shimizu *et al*[13], 2002 | 12 | F | Mucousy bloody diarrhea | Recto sigmoid | Multiple | UC | Metronidazole | Resolved |
| 17 | Park *et al*[14], 2002 | 60 | F | Tenesmus | Rectum | | | Low anterior resection | Resolved |
| 18 | Ohkawara *et al*[15], 2003 | 67 | F | Mucousy bloody diarrhea | Recto sigmoid | Multiple | Non-specific colitis | Patient refused | Resolved |
| 19 | Akamatsu *et al*[16], 2004 | 33 | F | Hypoproteinemia, mucousy bloody diarrhea | Rectum | Multiple | UC | *H. pylori* eradication therapy | Resolved |
| 20 | Akamatsu *et al*[16], 2004 | 50 | F | Weight loss, diarrhea | Recto sigmoid | Multiple | CP | *H. pylori* eradication therapy | Resolved |
| 21 | Akamatsu *et al*[16], 2004 | 53 | F | Mucousy bloody diarrhea | Recto sigmoid | Multiple | CP | *H. pylori* eradication therapy | Resolved |
| 22 | Bookman *et al*[17], 2004 | 36 | F | Mucousy bloody diarrhea | Recto sigmoid | Multiple | CP | Infliximab | Resolved |
| 23 | Maunoury *et al*[18], 2005 | 52 | F | Mucousy diarrhea | Rectum | Multiple | CP (recurrence) | Infliximab | Failed |
| 24 | Konishi *et al*[19], 2005 | 76 | F | Hypoproteinemia, Mucousy bloody diarrhea | Through out the colon | Multiple | CP | Sigmoidectomy for villous adenoma | Resolved |
| 25 | Ryu *et al*[20], 2006 | 64 | M | Weight loss, diarrhea | Recto sigmoid | Multiple | CP | Observation | Resolved |
| 26 | Nakagawa *et al*[21], 2009 | 52 | F | Weight loss, mucousy bloody diarrhea | Rectum to transverse colon | Multiple | CP | *H. pylori* eradication therapy | Resolved |
| 27 | Kim *et al*[22], 2009 | 53 | F | Mucousy bloody diarrhea, tenesmus | Recto sigmoid | Multiple | Pseudomem-branous colitis | Infliximab | Resolved |
| 28 | Obusez *et al*[23], 2010 | 70 | F | Diarrhea | Ileal pouch | Solitary | CP | Piecemeal snare polypectomy | Resolved |
| 29 | Yang *et al*[24], 2010 | 67 | F | Epigastric pain, nausea | Stomach | Multiple | | *H. pylori* eradication therapy | Resolved |
| 30 | Present case | 21 | M | Blood loss p.a. | Lower rectum | Solitary | Rectal cancer | Piecemeal snare polypectomy | Resolved |

CP: Cap polyposis; UC: Ulcerative colitis; *H. pylori*: *Helicobacter pylori*.

**Table 2 Symptoms of cap polyposis**

| Symptoms | % |
|---|---|
| Mucousy diarrhea | 86.7 |
| Bloody stool | 33.3 |
| Weight loss | 10 |
| Abdominal pain | 10 |
| Tenesmus | 10 |

Twenty-nine cases of histopathologically confirmed cap polyposis were reported from 1993 to 2010, with our patient being the 30th case. Twenty-two out of 30 patients (73.4%) were female, whereas the age of afflicted patients ranged from 12 to 76 years of age (mean age 51.6 years).

The main symptom at presentation was mucousy diarrhea (86.7%), followed by the presence of bloody stool (33.3%), abdominal pain, weight loss and tenesmus (10%). One patient was asymptomatic, 1 patient presented with epigastric pain and nausea and 1 (our patient) presented with rectal bleeding (Table 2). Clinical impression and, in some cases, initial diagnosis, included ulcerative colitis[3,5,6,13,14], pseudomembranous colitis[19], non-specific colitis[4,9], diverticular disease, solitary rectal ulcer, infection and rectal carcinoma.

Hypoproteinemia was present in most patients, probably as a result of protein-losing enteropathy, and was accompanied by edema of the lower extremities in some cases[5,11].

All patients underwent endoscopy. The rectosigmoid was most commonly affected (22 patients, 73.4%), whereas extension of cap polyposis to the proximal colon was found in 6 patients (20%). One of the aforementioned patients exhibited concurrent lesions in the stomach[12]. One was diagnosed with cap polyposis of the stomach solely during gastroscopy and finally, 1, who had been submitted to restorative proctocolectomy with ileal pouch anal anastomosis for ulcerative colitis, was diagnosed with a cap polyp in the ileal pouch[23].

Until the diagnosis of cap polyposis was established, several patients had been treated as patients suffering from ulcerative colitis or non specific colitis. Aminosalicylicates, broad-spectrum antibiotics and steroids had been administered without clinical response. Nine patients (30%) were treated with surgical resection, after which the problem resolved. In 3 patients, recurrence occurred and 2 were re-operated on, whereas one improved with cleversal enemas[3,5]. One patient was treated solely by the avoidance of straining at defecation. In 4 patients[10,11,15,20], the problem resolved spontaneously. Three patients were treated successfully with the administration of metronidazole[8,10,13]. Infliximab was administered in 3 patients[17,18,22], but in the case reported by Maunoury *et al*[18], the treatment was unsuccessful. After the first report of cap polyposis resolution after *H. pylori* eradication therapy by Oiya *et al*[12], 4 more cases[16,21] were treated accordingly, with complete remission of the symptoms and reversal of the endoscopic features.

In 1985, Williams *et al*[1] described 15 cases of what was a distinct endoscopic and pathological entity: inflammatory cap polyps of the large intestine. Therefore, the term cap polyposis was introduced to describe this new and rare entity. From the cases reviewed, cap polyposis seems to affect patients of any age (range 12-76 years of age, median age 51.6 years old), with a female predominance (22 out of 30, 73.4%). The main symptom of these patients at presentation was mucousy diarrhea, followed by bloody stool, weight loss, abdominal pain, tenesmus and bleeding per rectum. Constipation and straining at defecation were also reported[2].

Laboratory tests commonly included hypoproteinemia and hypoalbuminemia, as a result of protein-losing enteropathy. Oshitani *et al*[5] utilized scintigraphy with Tc-99m-labeled DTPA complexed with human serum albumin that showed protein loss from the descending colon in the case of a 54-year-old female patient. After cap polyposis is effectively treated, protein loss ceases and serum albumin returns to normal levels. Usually, there are no other remarkable findings in the laboratory tests performed.

At endoscopy, cap polyps are typically small, sessile and are covered by a "cap" of fibrinopurulent exudate, thus resembling pseudopolyps typically encountered in ulcerative colitis and pseudomembranous colitis[2,22]. They are usually located on the apical surface of transverse mucosal folds. A characteristic finding discriminating cap polyposis from ulcerative colitis is the presence of normal mucosa among the lesions. Magnifying colonoscopy with indigo carmine dye staining will reveal a type IIIL crypt pattern[8]. The polyps are multiple. In fact, only 2 cases of cap polyposis from those reviewed involved a solitary lesion, our patient and the patient reported by Obusez *et al*[23] in which the polyp was located in the ileal J-pouch. The size of these solitary polyps was also considerable compared to the small polyps usually encountered in cap polyposis. The polyp in our case was approximately 6 cm in diameter and 7 cm in the patient presented by Obusez *et al*[23]. The sites most commonly afflicted are the rectum and sigmoid colon. However, cap polyposis seems to extend to the proximal colon as well as the stomach, as reported by Oiya *et al*[12] and Yang *et al*[22].

Osseous metaplasia (heterotopic bone formation) is uncommonly observed in the gastrointestinal tract and occurs in association with benign or, in the majority of cases, malignant lesions. A recent review of the literature by Oono *et al*[25], who described a case of osseous metaplasia in a rectal inflammatory polyp, revealed only nine cases of osseous metaplasia reported in association with benign colorectal polyps, inflammatory, juvenile or adenomatous. Histologically, heterotopic bone formation is often associated with inflammation and/or ulceration, or with the presence of mucin production and extravasation. Repeated local trauma, factors released from adenoma cells or peculiar characteristics of the rectal mucosa were also considered as possible factors in the pathogenesis[25,26]. In our case, mucin lakes and inflammatory infiltrates were both in close proximity to the osse-

ous foci. Clinically, the presence of metaplastic bone in the reported cases seemed to be innocent.

The epidemiology and pathophysiology of cap polyposis have yet to be determined. Initially, cap polyposis was considered to be a manifestation of the mucosal prolapse syndrome, occurring as a consequence of colonic dysmotility[1-4]. This theory was supported by the frequent coexistence of chronic constipation and straining at defecation and by histopathological features such as disruption of the muscularis mucosa and presence of smooth muscle fibers and elastine in the mucosa. Intraluminal trauma has also been postulated as the initiating event, thus associated with mucosal prolapse[7]. However, colonic dysmotility or straining at defecation is not always present. Sadamoto *et al*[11] reported a case where the entire colon was afflicted in an asymptomatic patient. The presence of infection has also been investigated in the pathogenesis of cap polyposis[13,20] despite the fact that pathogens have not been isolated and broad-spectrum antibiotics such as levofloxacin were ineffective. The effectiveness of treatment with metronidazole seems to be the result of the anti-inflammatory rather than the antibacterial properties of the drug, functioning as a free radical scavenger[8,13,23]. The role of inflammatory response in the presence of cap polyposis has been implicated by additional data, such as the development of cap polyps on an anastomotic line, since the process of wound healing on the anastomosis is known to involve a complex network of numerous inflammatory cells and their secretory products, including TNF-a, which accelerates the wound healing process by inducing angiogenesis, fibroblast proliferation and production of several growth factors[19]. Moreover, complete clinical response to the administration of an anti-inflammatory agent (infliximab) has been reported[17,21] and the complete remission in patients undergoing *H. pylori* eradication therapy. It is known that *H. pylori* may cause indirect extragastric manifestations by the release of inflammatory modulators, molecular mimicry and systemic immune response[16,21]. This indirect implication of *H. pylori* is further supported by the absence of the microorganism in tissue obtained from the colonic polyps[16].

Treatment modalities include avoidance of straining at defecation with dietary modifications when constipation is present, *H. pylori* eradication therapy in patients suffering from *H. pylori* (+) gastritis, administration of metronidazole and, when these options prove to be ineffective, infusion of infliximab. Surgical excision should be reserved for patients who do not respond to conservative therapy or who recur. An interval of 3 mo has been advocated by Ng *et al*[2] before proceeding with surgery.

We propose the following algorithm: Once the diagnosis is set, patients should be evaluated according to the severity of symptoms, the number and location of the polyps. Endoscopic snare excision in piecemeal fashion is indicated in cases of solitary polyps, such as our patient, or when the total number of polyps renders snare excision feasible. In cases of multiple polyps, asymptomatic patients or patients with mild symptoms may be reassessed to allow for spontaneous resolution. If constipation is present, it should be assessed by defecography and the patient must be trained to avoid straining at defecation. Diagnostic workup for *H. pylori* infection should be undertaken and eradication therapy should be administered if infection is present. In the absence of *H. pylori* infection, administration of metronidazole may be utilized. The patient should be subsequently re-evaluated. If the symptoms persist and the endoscopic findings fail to demonstrate recess, the infusion of infliximab is a therapeutic option. Surgical resection is indicated if the drug is contraindicated or does not improve symptoms and endoscopic features, or if the patient recurs. Postoperative recurrence is possible and has been reported[3,5]. In such cases, repeat surgery is indicated. Endoscopic surveillance for colonic lesions or examination under anesthesia, with or without proctoscopy, for rectal lesions should be employed for surveillance and to obtain bioptic material in order to diagnose recurrence.

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