Case Report

Rectal Cap Polyposis Masquerading as Ulcerative Colitis with Pseudopolyposis and Presenting as Chronic Anemia: A Case Study with Review of Literature

Riti Aggarwal, Pallav Gupta, Prem Chopra, Samiran Nundy

Department of Pathology,
Sir Ganga Ram Hospital,
New Delhi, India

Address for correspondence:
Dr. Pallav Gupta,
Department of Pathology,
Sir Ganga Ram Hospital,
New Delhi - 110 060, India.
E-mail: pallavkmc1@gmail.com

ABSTRACT

Cap polyposis (CP) is an under recognized form of non-neoplastic colonic polyps, characterised by the presence of inflammatory polyps with a distinct "cap" of granulation tissue. CP is often seen masquerading as chronic inflammatory bowel disease. The most common symptoms are mucoid diarrhoea, bloody stools, abdominal pain, and tenesmus. In this case report, we present a patient who was diagnosed with CP during the investigation of unexplained chronic long standing anemia secondary to intermittent rectal bleeding. CP, although rare, should be considered in the differential diagnosis of patients presenting with intermittent rectal bleeding and mucoid diarrhoea.

Key Words: Anemia, bleeding, cap polyposis, mucosal prolapsed, rectum

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Cap polyposis (CP) is a rare benign colorectal condition, first described by William *et al*., in 1985.[1] It is characterized by the presence of inflammatory polyps that are covered by "caps" of granulation tissue. Earlier reports have described mucoid diarrhoea, bloody stools, abdominal pain, and tenesmus as the chief clinical presentations. We herein present a case of rectal CP in a 27-year-old man who presented with unexplained chronic long standing anemia secondary to intermittent rectal bleeding. He was initially diagnosed as ulcerative colitis and received medical therapy. As the patient was non-responsive to medical treatment he underwent hemicolectomy. Histopathological examination confirmed it to be a case of CP of the rectum.

CASE REPORT

A 27-year-old man visited another hospital with chief complaints of generalised weakness, pallor and intermittent rectal bleeding for 2 years prior to his presentation. Physical examination was unremarkable. His haematological profile showed low haemoglobin levels (8.5 g/dl). The rest of the laboratory parameters including total serum protein and erythrocyte sedimentation rate were within normal limits. Stool culture was negative for pathogenic organisms. Colonoscopy revealed pseudopolyps along with multiple erythematous and eroded mucosal lesions in the rectum [Figure 1]. He was diagnosed clinically as ulcerative colitis for which he received oral aminosalicylic acid followed by prednisolone for 6 months. However, the patient was non-responsive to this medical therapy. He was also transfused blood on two separate occasions along with hematenimics because of persistently low hemoglobin. One year after the initial treatment he was admitted to our hospital because of worsening of symptoms, which were now accompanied by mucous diarrhoea, straining at defecation and rectal prolapse (feeling of mass protruding out from rectum). Laboratory data at the time of present admission also revealed low hemoglobin (8.0 g/dl) however, remaining hematological investigations were within normal limits. On digital rectal examination, multiple soft elevated lesions were palpable. Contrast enhanced computed tomography of the lower abdomen and pelvis showed multiple rectal

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polyps and thickening of the rectal wall with a maximum wall thickness of 12 mm along with its abnormal inferior descent [Figure 2].

As the patient was non-responsive to medical therapy, low anterior resection of the rectum was done. Intra-operatively, multiple sessile polyps were noted in the rectum along with erythema of the surrounding mucosa. An intra-operative diagnosis of inflammatory polyposis was considered. The post operative course of the patient was uneventful.

Gross examination of the resected specimen revealed multiple sessile rectal polyps, ranging in size from 0.3 cm to 3.0 cm in maximum diameter. The surface of these polyps was ulcerated and was covered with a yellow white fibrinous exudate. The surrounding colonic mucosa showed mild erythema while the intervening colonic mucosa was unremarkable [Figure 3a]. Histologically, the surface of these polyps had "caps" of inflammatory granulation tissue and exudate while the base had hyperplastic and cystically dilated crypts. The lamina propria contained a large number of lymphomononuclear cells as well as mucin lakes. Hemosiderin laden macrophages were also observed at places. There was no evidence of adenomatous change or malignancy [Figure 3b]. The histopathological findings were consistent with the diagnosis of inflammatory CP.

Three months post-operatively the hemoglobin levels had improved to 11.3 g/dl.

DISCUSSION

CP usually affects people of age > 50 years (range: 12-76 years) with a female predominance.[1,2] In our case, the patient was a young male who presented with history of rectal bleeding and long standing anemia. The common symptoms of CP are mucous and bloody diarrhoea followed by abdominal pain and tenesmus.[3] Hypoproteinemia has also been reported as a result of protein losing enteropathy in patients with CP.[4] CP as a cause of unexplained anemia secondary to intermittent rectal bleeding deserves recognition.

Apart from the rectosigmoid, CP can involve the proximal colon and stomach.[5] The number of polyps may vary from 1 to more than 100.[6] Obusez *et al.*,[7] reported a case of a solitary cap polyp in the ileal pouch of a patient who had been treated by restorative procto-colectomy with ileal pouch-anal anastamosis.

Endoscopically, cap polyps are typically small, red, sessile and covered by a "cap" of fibrinopurulent exudate. These are often misdiagnosed as pseudopolyps seen in cases of ulcerative colitis. However, the presence of cap polyps at the apices of transverse mucosal folds with intervening normal mucosa differentiates these from ulcerative colitis. These polyps are not adenomatous, thereby familial adenomatous

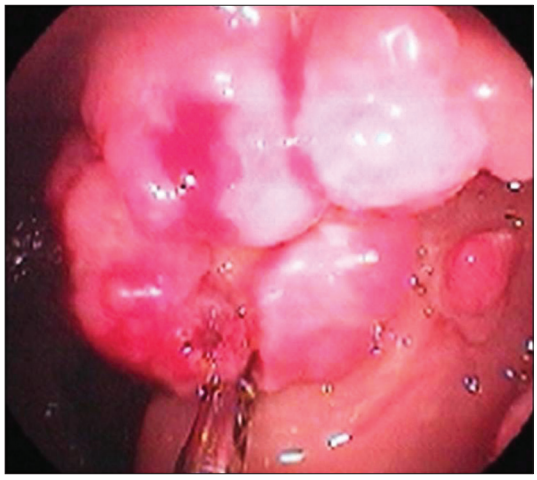

Figure 1: Endoscopy showing multiple pseudopolyps in the rectum some of which are ulcerated

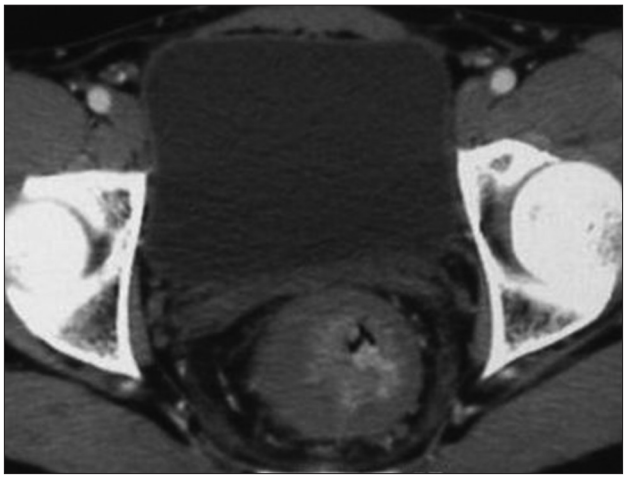

Figure 2: Coronal sections of contrast enhanced computed tomography shows circumferential thickening of rectum with irregularity and narrowing of lumen. Few enhancing polypoidal mass lesions are seen projecting into lumen of rectum

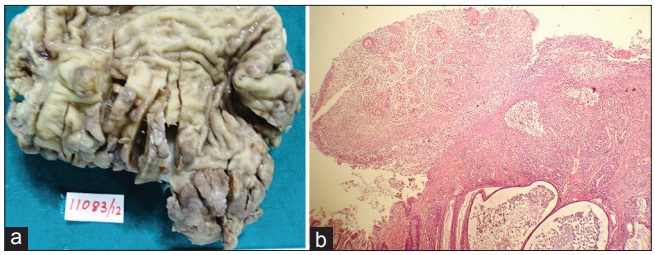

Figure 3: (a) Gross photograph showing multiple sessile rectal polyps, ranging in size from 0.3 cm to 3.0 cm in diameter. Surface of these polyps appears ulcerated and is covered with yellow-white fibrinous exudates, (b) microphotograph showing surface of polyps covered by "caps" of inflammatory granulation tissue and exudate while the base shows hyperplastic and cystically dilated crypts (H and E, ×100)

polyposis can be excluded. At times, small or early juvenile rectal polyps may be histologically indistinguishable from cap polyps but juvenile rectal polyps are usually pedunculated and have a characteristic cut surface of cystic fluid-filled spaces on gross examination.[8]

The exact aetiology of CP is yet to be elucidated. Abnormal colonic motility and repeated trauma to the colonic mucosa caused by straining at defecation have been postulated as the initiating events.[1,4,9] This is supported by the histologic features such as disruption of the muscularis mucosae and the presence of smooth muscle fibers and elastin in the mucosa. Conditions such as prolapsing mucosa, solitary rectal ulcer syndrome, inflammatory cloacogenic polyps, and gastric antral vascular ectasia together with CP comprise "mucosal prolapse syndrome" with straining at defecation as the common etiology.[10] Infection has been investigated as a possible cause in the absence of abnormal colonic motility; despite the fact that no pathogen has been isolated until date. The role of local inflammatory factors such as TNF-alpha and IL-10 involved in the wound healing process in cases of intestinal anastomosis has also been suggested as the initiating events in cases of CP.[11] However, the co-existence of CP and rectal prolapse in our case supports the theory of mucosal prolapse as the underlying mechanism of CP rather than a local inflammatory process.[12]

A systematic and multidisciplinary approach is necessary in the management of patients with CP as this condition holds good long-term prognosis. Conservative treatment modalities like avoidance of straining at defecation, use of metronidazole, infliximab, *Helicobacter pylori* eradication therapy and endoscopic polypectomy have been shown to be beneficial in some cases.[13,14] Surgical resection should be reserved for patients with recurrence or for those who don't respond to conservative therapy. To conclude, rectal CP should be considered in the differential diagnosis of patients presenting clinically with intermittent rectal bleeding and rectal polyposis on endoscopic examination.

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Image Quiz

An Intriguing Cause of Intractable Nausea and Vomiting

Mohit Girotra, Hemendra R. Shah[1], Rayburn F. Rego

Departments of Medicine, Division of Gastroenterology and Hepatology, [1]Radiology, University of Arkansas for Medical Sciences, Little Rock, AR, USA

Address for correspondence:
Dr. Mohit Girotra, Department of Medicine, Division of Gastroenterology and Hepatology, University of Arkansas for Medical Sciences, 4301 W. Markham Street, Shorey S8/68, Mail Slot # 567 Little Rock, AR, USA.
E-mail: mgirotra@uams.edu

A 60-year-old lady with history of reflux disease and breast cancer status post-lumpectomy and lymph node dissection followed by chemotherapy and now in remission, was admitted with progressively increasing nausea and vomiting of 2 months duration. She also described episodic epigastric abdominal pain, which increased on food intake and led to vomiting, which brought her instant relief. She had lost about 80 lbs of weight during her chemotherapy, which stabilized for few months but was now again falling because of her inability to eat. There was no alteration in bowel habits, fever or any other associated symptoms. She had two previous admissions for similar problems at a local hospital where symptomatic relief was achieved with dobhoff tube placement. She had been tolerating the tube feeds well, however, for last 2 days her symptoms reappeared raising concern of obstructed dobhoff and hence a gastroenterologist was consulted for endoscopic evaluation. Her blood-work was normal. Barium swallow showed extrinsic compression of the 3rd portion of duodenum, at the level of superior mesenteric artery (SMA) crossing, causing partial obstruction as barium passes through the 3rd to 4th portion [Figure 1]. Computed tomography (CT) scan was obtained which was diagnostic [Figure 2]. Esophagogastroduodenoscopy revealed a patent 3rd part of duodenum.

QUESTION

Q1. What is the diagnosis?

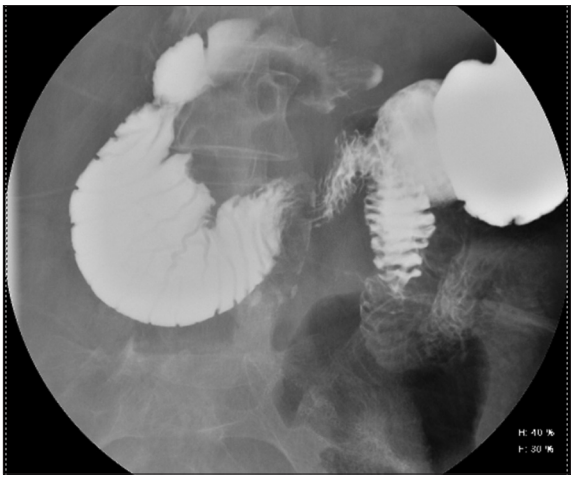

Figure 1: Barium swallow: Extrinsic compression of the 3rd portion of duodenum, at the level of superior mesenteric artery crossing, causing partial obstruction as barium passes through the 3rd to 4th portion

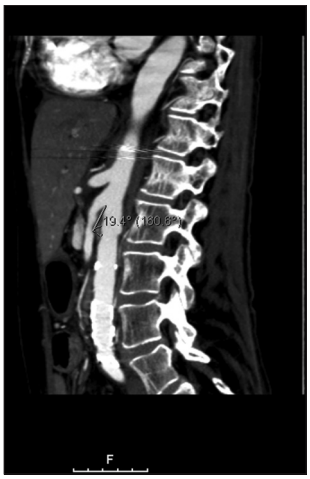

Figure 2: An aorta-SMA angle of less than 25° is considered diagnostic of SMA syndrome

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ANSWER

This CT image is the classical presentation of SMA syndrome. The normal angle between the aorta and SMA is 38-65°, but in SMA syndrome the angle is narrowed which results in compression of 3rd part of duodenum as it traverses between aorta and SMA causing functional obstruction.[1] An aorta-SMA angle of less than 25° is considered diagnostic, in adjunct to shortened aorto-mesenteric distance (2-8 mm).[2]

There may be several causes for decrease in this angle, including congenital abnormalities, but the most noteworthy is loss of intra-abdominal adipose tissue, which in healthy individuals prevents duodenal compression by maintaining distance between aorta and SMA. Patients with severe weight loss, irrespective of the cause, suffer from loss of this intra-abdominal fat and hence narrowing of the aorta-SMA angle.[3] This is commonly seen in patients with malignancies, chronic medical conditions like malabsorption, and wasting syndromes like AIDS.[3] It has also been reported with profound weight loss after bariatric surgery.[4] A high-index of suspicion is the key to diagnosis of this relatively rare but important condition.

Treatment approach is usually conservative and includes nutritional support via naso-jejunal tube or total parenteral nutrition,[5] with a hope of increasing body weight to restore the aorta-SMA angle. This approach is advocated in pediatric patients but often fails in adults with chronic conditions, who need surgical intervention eventually.

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