CASE REPORT

Endoscopic removal of a bleeding colonic polypoid angiodysplasia: case report

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Abstract

Arteriovenous malformations are common causes of lower gastrointestinal bleeding in the elderly. Among them, angiodysplasia is one subtype that appears on endoscopy as red, flat superficial lesions, and sometimes slightly elevated. Colonic angiodysplasia is very rarely seen as a polypoid lesion. The present case describes a bleeding large polypoid colonic angiodysplasia in a 60 year-old man. It was removed endoscopically using a PolyLoop® ligature device without complications. (Acta gastroenterol. belg., 2010, 73, 406-408).

Key word: Arteriovenous malformation, polypoid angiodysplasia, polypectomy

Introduction

Arteriovenous malformations (AVM) are among common causes of acute and chronic lower gastrointestinal (GI) bleeding. The GI vascular lesions are classified according to the most affected vascular structures (artery, vein, arteriovenous, capillary) and their location within the bowel wall. AVM enclose three groups: angiodysplasia where the arteriovenous communications are present in the mucosa and submucosa, hereditary hemorrhagic telangiectasia where the vessels are distributed in all the layers of the bowel, and gastric antral vascular ectasia - watermelon stomach - where the vessels (tortuous capillaries and veins) are found in the mucosa and submucosa (1). We report an extremely rare presentation of a colonic angiodysplasia as a large pedunculated polyp.

Case report

A 60 year-old man, previously healthy, presented with a two-month history of intermittent hematochezia without other GI symptoms. His physical exam was normal. Laboratory tests showed no evidence of anemia in addition to normal liver function tests. Colonoscopy revealed a large (around 4 cm) pedunculated polypoid lesion in the sigmoid (Fig. 1) with minimal oozing of blood from its tip. After the stalk was injected with epinephrine (1:10000 dilution), a PolyLoop® (Olympus Medical Systems Corp., Japan) was deployed and the polyp was then snared and removed. Histologic evaluation showed a polypoid structure lined by hyperplastic colonic mucosa. The muscularis mucosae was thickened. There were numerous dilated thin walled blood vessels in the lamina propria and numerous large tor-

Fig. 1. — a) Colonoscopic view of a large, sigmoid polypoid lesion with a thick stalk. b) The PolyLoop® deployed at the stalk after epinephrine injection. c) The base of the stalk after snare removal of the polyp.

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Fig. 2. — Histopathologic findings of the excised polypoid lesion. a) Multiple large congested arterial and venous vascular channels (see arrows) in the lamina propria extending into the submucosa (b) and mucosa (c). (hematoxylin and eosin stain, original magnification × 20, 200, 400 respectively, scale = 50 μm).
tuous thick walled arteries and veins in the submucosa (Fig. 2). In multiple areas the abnormal vessels disrupted the *muscularis mucosae*. The findings were consistent with polypoid angiodysplasia. After the removal of the lesion, the patient had no recurrence of hematochezia over a follow up period of six months.

**Discussion**

Colonic angiodysplasia, also known as vascular ectasia, is defined as the presence of dilated blood vessels in the mucosa and the submucosa (2) without any involvement of the muscularis propria or serosa. Up to one third of cases of lower GI bleeding are due to colonic angiodysplasias (3). They are usually multiple and mainly seen in the elderly (4). The most common location of these lesions is the right colon. Endoscopically, colonic angiodysplasias are described as red ectatic vascular mucosal lesions with a diameter ranging between few millimeters to few centimeters. They are usually flat, but may be slightly elevated in some cases. Histologically, the capillaries, veins, and arteries are abnormally thin-walled, dilated, and are usually seen in the mucosa and the submucosa (5). Thick-walled arteries are typically present in congenital AVM.

The etiology of angiodysplasia is still unknown. Roskell et al. (4) suggested the presence of a deficiency in the collagen type IV in the vessels of the mucosa that predispose the development of angiodysplasia. Another study conducted by Junquera et al. (6) found that angiogenic factors such as basic fibroblast growth factor (bFGF) and vascular endothelial growth factor (VEGF) are highly expressed in patients with angiodysplasia and thus play a very important role in their pathogenesis. Colonic angiodysplasias have been associated with chronic renal failure, aortic stenosis, liver cirrhosis, and von Willebrand’s disease (7,8,9,10,11).

Polypoid angiodysplasias are extremely rare in the colon. Only a few cases have been described in the literature over the past 50 years (Table 1) (12-21). The age at presentation ranged from 24 to 84 years. Their sizes ranged from 0.5 to 6.0 cm and they were evenly distributed throughout the entire colon.

Colonic polypoid AVM’s have been mistaken for adenomatous polyps and their diagnosis was made after polypectomy was performed. Due to their hypervascular structures, it is assumed that they would carry a significant potential risk for bleeding upon removal, especially if the vessels within are more than 1 mm in diameter (22). The size below which such lesions can be safely endoscopically transected is yet to be determined. However, it would be prudent to use a detachable snare (PolyLoop®) prior to the removal of large lesions.

Based on our literature review, the current case report the largest (base diameter about 4 cm) colonic polypoid angiodysplasia that has been successfully and safely removed by endoscopy using a PolyLoop® ligature device.

**References**

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| Table I. — *Summary of reported colonic polypoid angiodysplasia* |
|-----------------|--------------------|-----------------|-----------------|-----------------|-----------------|
| Case (year)     | Age/Gender | Presentation | Location | Maximal Diameter (cm) | Gross finding | Treatment |
| Koziara et al. (11) (1996) | 84/F | Hematochezia | Sigmoid | 3.5 | Pedunculated | Snare polypectomy |
| Park et al. (12) (2000) | 58/M | Iron deficiency anemia | Transverse colon | 1.5 | Pedunculated | Snare polypectomy |
| Park et al. (12) (2000) | 41/M | Hematochezia | Descending colon | 1.0 | Pedunculated | Snare polypectomy |
| D’Arienzo et al. (13) (2001) | 53/M | Hematochezia | Sigmoid | 2.0 | Pedunculated | Snare polypectomy |
| McKevitt et al. (14) (2002) | 24/M | Hematochezia | Rectum | 0.7 | Pedunculated | Snare polypectomy |
| Nasseri-Moghaddam et al. (15) (2004) | 26/M | Hematochezia | Sigmoid | 3.0 | Pedunculated | Snare polypectomy |
| Kakushima et al. (16) (2004) | 59/M | Hematochezia | Colon - not specified | 1.5 | Pedunculated | Snare polypectomy |
| Maeng et al. (17) (2004) | 59/F | Hematochezia | Transverse colon | 6.0 | Pedunculated | Surgery |
| Ji et al. (18) (2005) | 81/M | Hematochezia | Transverse colon | 3.5 | Semi-Pedunculated | Snare polypectomy |
| Lin et al. (19) (2007) | 69/M | Abdominal discomfort | Ascending colon | 2.5 | Pedunculated | Snare polypectomy |
| Kim et al. (20) (2009) | 66/F | Screening | Cecum | 0.5 | Semi-Pedunculated | Cold biopsy |
| Present case | 60/M | Hematochezia | Sigmoid | 4.0 | Pedunculated | Snare polypectomy |


