Colonic Polypoid Arteriovenous Malformation Causing Symptomatic Anemia

Allison Rzepczynski, MD1, Jason Kramer, MD2, Shriram Jakate, MD3, Lin Cheng, MD3, and Ajaypal Singh, MD2

1Department of Internal Medicine, Rush University Medical Center, Chicago, IL
2Department of Gastroenterology, Rush University Medical Center, Chicago, IL
3Department of Pathology, Rush University Medical Center, Chicago, IL

ABSTRACT
Vascular ectasias, which can be classified as angiodysplasias and arteriovenous malformations (AVMs), accounts for approximately 3% of lower gastrointestinal bleeding. Typically, colonic AVMs are solitary, large, and flat or elevated red lesions on endoscopy. We present an interesting case of a polypoid ulcerated AVM in the transverse colon causing symptomatic anemia, which was resected endoscopically with a resolution of symptoms. Polypoid colonic AVMs are rather rare with only 15 other cases described in the literature. This case highlights the approach to endoscopic management of these lesions.

INTRODUCTION
Gastrointestinal (GI) bleeding from the colon is a common reason for clinic visits and hospitalizations. Vascular ectasias accounts for about 3% of lower GI bleeding. Vascular ectasias can be classified into angiodysplasias and arteriovenous malformations (AVMs), which are abnormal shunts between arteries and veins without a capillary bed. Although AVMs are typically thought of as flat lesions, there are rare cases of polypoid AVMs, which are commonly mistaken for inflammatory fibroid polyps or atheroemboli-associated polyps. We describe an interesting case of an incidentally found polypoid ulcerated AVM in the transverse colon, which was successfully endoscopically resected.

CASE REPORT
A 65-year-old man with a significant medical history of HIV infection, end-stage renal disease, hepatitis C, and coronary artery disease was evaluated in a gastroenterology clinic for symptomatic anemia and screened for colon cancer as part of pretransplant evaluation. He had a colonoscopy 15 years ago for age-appropriate cancer screening, which was reportedly normal. He denied any personal or family history of colonic polyps or colon cancer. During his consultation appointment, he reported several episodes of hematochezia over the last few months which he thought were secondary to hemorrhoidal bleeding. His laboratory results from a month before the visit were notable for hemoglobin 12.4 g/dL with mean corpuscular volume 101.0. The colonoscopy revealed a 2.5-cm ulcerated, semipedunculated mass in the proximal transverse colon (Figure 1). The mass was biopsied and tattooed distally. There was brisk oozing noted after the biopsy; the site was injected with dilute epinephrine and hemostasis was achieved. Pathology of the mass showed colonic mucosa with vascular ectasia, fibrin thrombi, and overlying severely inflamed hyperplastic epithelium.

The pathology results were discussed with the pathologist and thought to be consistent with a hyperplastic or inflammatory polyp. He was admitted to the observation unit after his colonoscopy for postoperative monitoring. His laboratory tests were notable for hemoglobin 5.9 g/dL, which improved to 7.2 g/dL after 2 units of packed red blood cells. After discharge, he followed up with colorectal surgery and a decision was made to pursue endoscopic resection of the mass. He continued to have anemia after his initial colonoscopy with hemoglobin 9.0 g/dL 2 months later. He did not report any further episodes of hematochezia, but his anemia was thought to be secondary to slow bleed from the unresected mass. Repeat colonoscopy showed a slow oozing 25-mL polypoid lesion in...
the proximal transverse colon. A through-the-scope ultrasound probe was advanced to the lesion which revealed a hyperechoic mucosal mass with an intact muscularis propria (Figure 2). The lesion was injected with dilute methylene blue solution and resected en bloc using forced coagulation current. After the mass was successfully removed, slow oozing was noted and the area was injected with epinephrine for hemostasis. The resection site was closed with the placement of 3 hemostatic clips. The pathology was significant for a polypoid fragment of inflamed colonic mucosa and submucosa with vascular ectasia in the submucosa, consistent with AVMs (Figure 3). The patient’s hemoglobin improved from 8.2 g/dL to 13.0 g/dL 8 weeks after removal of the AVM. He did not report any further episodes of hematochezia or dizziness after removal of the lesion.

**DISCUSSION**

AVMs are a common cause of lower GI bleeding. Unlike other vascular abnormalities, AVMs can be found in all age groups, rather than just the elderly population. Typically, AVMs appear as bright red, flat lesions on colonoscopy. AVMs are usually solitary, are not restricted to the right side of the colon, and are larger. The pathophysiology of these lesions is not well defined, but intermittent, low-grade obstruction of the submucosal veins penetrating the muscularis is thought to lead to the development of these arteriovenous communications. The most common chief concern in patients with colonic AVMs is hematochezia (56%); however, 25% of the lesions are asymptomatic and diagnosed on screening colonoscopy. AVMs can be classified into 3 types: type 1 AVMs are solitary, localized lesions of the right side of the colon and are typically seen in older patients, type 2 AVMs are thought to be congenital in nature and are typically larger lesions seen in the small intestine and can be a source of obscure GI bleeding, and type 3 AVMs are punctate angiomas that commonly cause GI hemorrhage. To our knowledge, there have only been 15 other cases describing polypoid appearing AVMs in the literature. It has been suggested that polypoid AVMs should be in a separate classification from the 3 established types because these lesions can cause significant bleeding and the endoscopic management requires a different approach. Typically, histology is not required in diagnosing AVMs; however, polypoid AVMs can be difficult to accurately identify on initial endoscopic examination, as in our case. These lesions should be considered when an unusual, stalk-like lesion is visualized endoscopically without an apparent polyp. Of the 15 described cases, 14 were treated endoscopically. Only one case of polypoid AVM required surgical resection because of AVM causing intussusception. In 11 of the cases, hematochezia or iron deficiency anemia

![Figure 1. An endoscopic view of the 3-cm semipedunculated ulcerated mass in the proximal transverse colon.](image1)

![Figure 2. Biopsy of the excised polyp showing distended submucosal vascular channels. The overlying mucosa shows no adenomatous changes (hematoxylin and eosin stain 20× magnification).](image2)

![Figure 3. Biopsy of the excised polyp showing details of the distended submucosal vascular channels, which includes both larger thin-walled veins and smaller thick-walled arterial branches (hematoxylin and eosin stain 40× magnification).](image3)
resolved after resection. The remaining 4 cases did not have evidence of bleeding before resection but were instead found incidentally on screening colonoscopy. Endoscopists need to be aware of the different types and presentations of AVMs, as resection inherently involves higher risk of bleeding but is still warranted given their high propensity to cause GI bleeding.

DISCLOSURES

Author contributions: A. Rzepczynski and J. Kramer wrote the manuscript. S. Jakate and L. Cheng provided the pathology images. A. Singh edited the manuscript. A. Rzepczynski is the article guarantor.

Financial disclosure: None to report.

Previous presentation: This case was presented at the American College of Gastroenterology Annual Scientific Meeting; October 5–10, 2018; Philadelphia, Pennsylvania.

Informed consent was obtained for this case report.

Received March 29, 2019; Accepted September 6, 2019

REFERENCES

1. Suzuki K, Tanaka N, Ebine T, Momma T. Pelvic congenital arteriovenous malformation diagnosed by transrectal ultrasonography: A case report. Can Urol Assoc J. 2012;6:E61–3.
2. Lee HH, Kwon HM, Gil S, et al. Endoscopic resection of asymptomatic, colonic, polypoid arteriovenous malformations: Two case reports and a literature review. Saudi J Gastroenterol. 2017;23(1):67–70.
3. Choi HK, Park CI, Shin JA, et al. Hematochezia with colonic polypoid angiodyplasia in a young female patient. Gut Liver. 2008;2:126–9.
4. Kim BK, Han HS, Lee SY, Kim CH, Jin CJ. Cecal polypoid arteriovenous malformations removed by endoscopic biopsy. J Korean Med Sci. 2009;24(2):342–5.
5. Molina AL, Jester T, Nogueira J, CaJacob N. Small intestine polypoid arteriovenous malformation: A stepwise approach to diagnosis in a paediatric case. BMJ Case Rep. 2018;2018.
6. Cullen NR, Yfantis HG, von Rosenvinge EC. “Stalk”ing the etiology of rectal bleeding. Gastroenterology. 2013;144:1355.
7. Lim DH, Ahn JY, Seo M, et al. Polypoid arteriovenous malformation presenting with jejunojejunal intussusceptions in an adult. Clin Endosc. 2014;47:575–8.
8. Andrisani G, Petruzzello L, Ricci R, Spada C, Costamagna G. Giant arteriovenous malformation of the colon mimicking lipoma. Dig Liver Dis. 2014;46:89–90.

Copyright: © 2019 The Author(s). Published by Wolters Kluwer Health, Inc. on behalf of The American College of Gastroenterology. This is an open-access article distributed under the terms of the Creative Commons Attribution-Non Commercial-No Derivatives License 4.0 (CCBY-NC-ND), where it is permissible to download and share the work provided it is properly cited. The work cannot be changed in any way or used commercially without permission from the journal.