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Rectal Arteriovenous Malformation Masquerading As Rectal Varices

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Jenna L. Phillips [1], Simon Cooper [1], Keshav Iyer [1], Arravintha Gobinathan [1], Jeffrey P. Snow [2], Jordan Ditchek [3], Gary Schwartz [4]

1. Department of Education, Nova Southeastern University Dr. Kiran C. Patel College of Allopathic Medicine, Davie, USA 2. Department of General Surgery, Memorial Healthcare Sytem, Hollywood, USA 3. Department of Radiology, Nova Southeastern University Dr. Kiran C. Patel College of Allopathic Medicine, Davie, USA 4. Department of Medical Education, Nova Southeastern University Dr. Kiran C. Patel College of Allopathic Medicine, Davie, USA

Corresponding author: Jenna L. Phillips, jp4141@mynsu.nova.edu

Abstract

We report a case of a bleeding rectal arteriovenous malformation (AVM), initially diagnosed as a rectal varix, which was ultimately treated with staged transarterial embolization.

A 67-year-old man with a past medical history significant for hypertension, type 2 diabetes mellitus, and rectal varices presented with progressive hematochezia resulting in a drop in his hemoglobin from 11 g/dL to 8 g/dL. His medication history was notable for chronic ibuprofen use, ginkgo biloba, and ginseng supplementation. Flexible sigmoidoscopy revealed active bleeding from a lesion that was initially thought to be a rectal varix at the rectosigmoid junction and was subsequently treated with an endoscopic clip. Repeated episodes of bleeding led to computed tomography angiography (CTA), which demonstrated tortuous perirectal vasculature concerning for an AVM. The diagnosis of a complex rectal AVM was made, and imaging showed that this vascular lesion was supplied by the inferior mesenteric artery (IMA) with early venous drainage into the internal iliac venous systems bilaterally. Following an initial embolization, the patient experienced recurrent bleeding 38 days later and underwent repeat embolization to achieve hemostasis. The targeted vessels included the superior rectal arterial branches, hemorrhoidal arteries, and AVM nidus. The patient was discharged without complications the following day.

In this patient, a rectal AVM was an important diagnostic consideration after recurrent hematochezia despite presumed rectal varices in the absence of portal hypertension. In this case, staged transarterial embolization achieved hemostasis after endoscopic management was unsuccessful.

Categories: Other, General Surgery
Keywords: arteriovenous malformation, hemorroids, recurrent hematochezia, staged transarterial embolization, superior rectal artery

Introduction

Rectal AVMs are rare high-flow vascular lesions characterized by direct arteriovenous shunting through an abnormal nidus, bypassing the capillary bed [1-3]. The most common presenting symptom of rectal AVMs is painless hematochezia [4-6], which overlaps with far more prevalent conditions such as hemorrhoids, angiodysplasia, and inflammatory bowel disease [4,7-8]. Therefore, rectal AVMs are frequently misdiagnosed or subject to significant diagnostic delay [4,7-9]. Accurate diagnosis requires stepwise imaging progressing from luminal endoscopy to cross-sectional angiography and, ultimately, catheter-directed digital subtraction angiography, which remains the gold standard for definitive diagnosis and can simultaneously serve as a therapeutic modality [1,4,10]. No well-established treatment guidelines exist for rectal AVMs, and the evidence base is limited to case reports and small series [9,11].

Importantly, rectal AVMs should be distinguished from rectal varices, as the two entities have different underlying pathophysiology despite potentially overlapping clinical presentations. Rectal AVMs consist of abnormal arterial and venous connections, whereas rectal varices are dilated portosystemic collateral veins that most commonly develop in the setting of portal hypertension. This distinction is clinically relevant because both conditions may present with recurrent hematochezia but require different diagnostic and therapeutic approaches.

We report the case of a 67-year-old man without cirrhosis or portal hypertension who presented with progressive hematochezia, initially diagnosed as a rectal varix. A staged diagnostic evaluation revealed a complex rectal AVM with arterial supply from the superior rectal artery (SRA), which was ultimately managed with selective embolization of the superior rectal arterial branches, the hemorrhoidal arteries, and the AVM nidus. We review the diagnostic imaging approach, endovascular management, relevant genetic associations, and the pathophysiologic relationship between high-flow arteriovenous shunting and local venous hypertension within the rectal vascular territory.

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Case Presentation

A 67-year-old man with a past medical history of hypertension, type 2 diabetes, and rectal varices presented to the emergency department with progressively worsening rectal bleeding. In 2023, he had intermittent hematochezia. At that time, he underwent a colonoscopy that demonstrated rectal varices. The patient was advised to undergo variceal ligation. After losing his health insurance, the patient did not undergo the procedure. He was treated nonoperatively, after which his symptoms improved. He subsequently was lost to follow-up for two years. Over the month prior to presentation, the patient experienced progressively worsening rectal bleeding. He described this as episodes of bright red blood and dark blood mixed with the stool. This bleeding was persistent throughout this period. There was no known history of cirrhosis. The patient drank alcohol extensively when he was younger, but stopped drinking many years ago. There was no history of nausea, vomiting, diarrhea, constipation, fever, chills, or heartburn. The patient was taking ginkgo biloba, ginseng, and ibuprofen at a dose of 600 mg three times per day. The patient was a non-smoker. There was no history of liver disease.

The patient stated that his hemoglobin (Hb) was 11 g/dL one month ago. When he presented to the emergency room, his hemoglobin level was 8 g/dL. At that time, he received one unit of blood.

On examination, the patient was normotensive with no evidence of orthostatic hypotension. He was anicteric. The abdomen was protuberant but non-distended. Bowel sounds were active in all four quadrants. There were no masses or evidence of organomegaly. Rectal examination demonstrated normal tone, without any masses or tenderness. Hemorrhoids were present. There was no evidence of blood at the anal verge.

Hemoglobin was 8.0 g/dL, and hematocrit (Hct) was 26.3%. White blood cell (WBC) count was 8200 cells/µL. Prothrombin time was 10.5 seconds with an international normalized ratio (INR) of 1.0. Fecal occult blood was positive. The platelet count was 324,000/µL.

A flexible sigmoidoscopy was performed, which demonstrated internal hemorrhoids during retroflexion. The hemorrhoids were grade II, meaning that they prolapsed but reduced spontaneously. A medium-sized rectal varix at the rectosigmoid junction was identified with active bleeding. A clip was placed to achieve hemostasis. The distal sigmoid colon appeared to be normal.

Five days later, the patient presented to the emergency department with recurrent rectal bleeding. At that time, his blood pressure was 134/66, with no evidence of orthostatic hypotension. His abdomen was non-tender, and bowel sounds were normal. There were no masses or evidence of organomegaly. Laboratory findings were as follows: Hemoglobin 7.9 g/dL, hematocrit 25.6%, and the platelet count was 439,000 /µL. Alkaline phosphatase was 64 U/L, aspartate aminotransferase (AST) was 23 U/L, and alanine aminotransferase (ALT) was 21 U/L. All labs are reported in Table *1*, with normal biological values for reference.

Test	1 month before presentation	Initial presentation	Day 5: recurrent bleeding	Reference range	Units
Hb	11.0	8.0	7.9	13.5-17.5	g/dL
Hct	Not reported	26.3	25.6	38.0-50.0	%
WBC count	Not reported	8200	None reported	4500-11,000	µL
Platelet count	Not reported	324,000	439,000	150,000-400,000	µL
Prothrombin time	Not reported	10.5	Not reported	9.5-11.5	Seconds
INR	Not reported	1.0	Not reported	0.8-1.2	Ratio
Fecal occult blood	Not reported	Positive	Not reported	Negative	Qualitative
Alkaline phosphatase	Not reported	Not reported	64	38-126	U/L
AST	Not reported	Not reported	23	15-46	U/L
ALT	Not reported	Not reported	21	<50	U/L

TABLE 1: Timeline of patient labs

Hb - hemoglobin; Hct - hematocrit; WBC - white blood cell; INR - international normalized ratio; AST - aspartate aminotransferase; ALT - alanine aminotransferase

CTA of the pelvis demonstrated hemorrhoids projecting into the lumen of the rectum. There was a cluster of prominent and tortuous vessels in and adjacent to the rectal wall (Figure *1*).

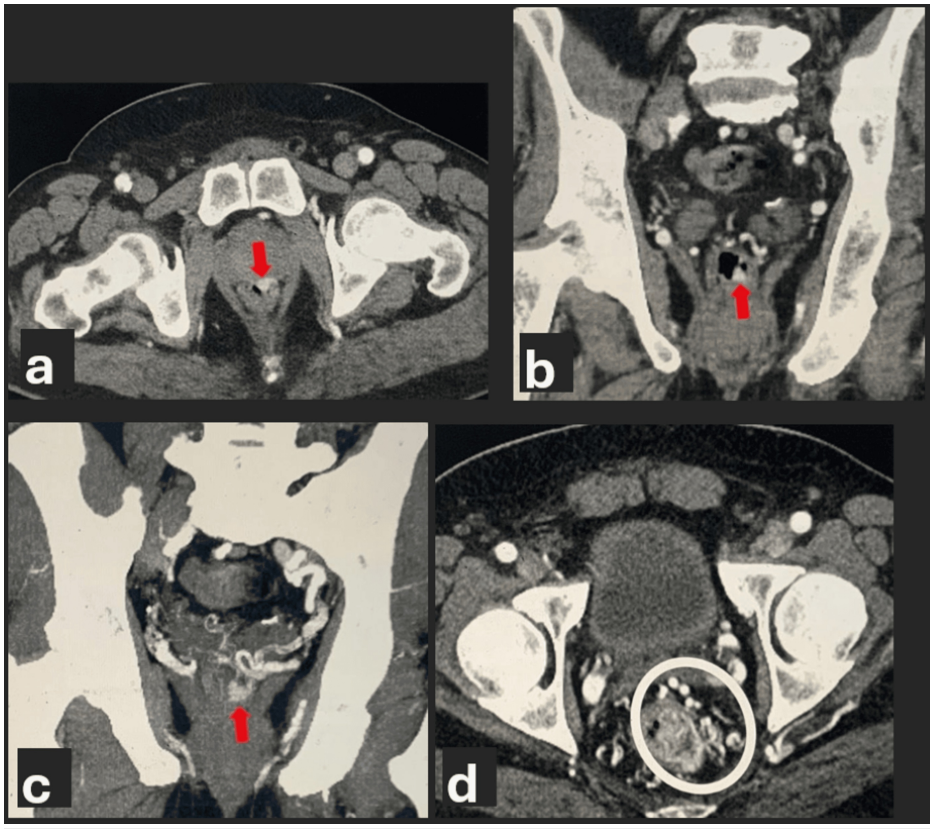


FIGURE 1: Contrast-enhanced CT angiogram of the pelvis

Axial (a and d), coronal, (b) and coronal maximum intensity projection (MIP) (c) images from a contrast-enhanced CT angiogram of the pelvis demonstrate an opacified hemorrhoid projecting into the lumen of the rectum (red arrows) and a cluster of prominent and tortuous vessels in and adjacent to the rectal wall, consistent with components of the AVM (white circle).

An inferior mesenteric artery angiogram with selective and subselective arterial embolization demonstrated contrast-opacified tortuous arteries near the midline, and early draining veins in the internal iliac venous system bilaterally (Figure *2*). The embolization was performed with the Liquid Embolic System-18 (LAVA-18) followed by coil embolization of the distal branches.



FIGURE 2: Inferior mesenteric artery angiogram

An inferior mesenteric artery angiogram demonstrates the catheter positioned within a distal branch of the superior rectal artery. Contrast opacifies tortuous arteries near the midline (red arrow) and early draining veins in the internal iliac venous system bilaterally (black arrows). Note the clip placed in the rectum at endoscopy (white arrow).

The patient presented to the emergency department on day 38 with recurrent rectal bleeding. An inferior mesenteric arteriogram was performed. It demonstrated markedly abnormal vascularity involving the rectum with dilated veins, multiple areas of blush, and tortuous arteries. The superior rectal artery angiogram demonstrated abnormal appearance of the rectal mucosa and enhancement with multiple areas of abnormal blush, including hemorrhoidal blush and proximal blush consistent with previous findings of a complex AVM. Embolization of the superior rectal arterial branches, hemorrhoidal arteries, and the arteriovenous malformation nidus was performed on day 49 with the LAVA-18 system. The patient was discharged from the hospital on day 50. The patient was followed up and had no further bleeding. He was discharged from active care on day 208. A timeline of the patient's hospital course is shown in Figure *3*. This timeline shows the sequence of events from the initial presentation to the colorectal surgeon through the time of hospital discharge.

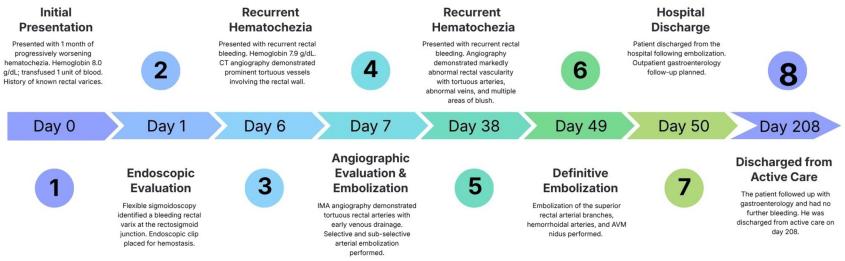


FIGURE 3: Timeline of the patient's clinical course

Created by the authors using Canva (Canva Pty Ltd., Sydney, Australia).

Discussion

Diagnosis

AVMs in the anorectal region represent a small proportion of high-flow vascular lesions, contributing to missed or delayed diagnoses. Because the most common presenting symptom is painless hematochezia, rectal AVMs are often initially evaluated for more common causes of bleeding, such as hemorrhoids, angiodysplasia, inflammatory bowel disease, and malignancy before an AVM is considered. A high index of suspicion and a deliberate staged imaging strategy is warranted to accurately diagnose a rectal AVM [1,4]. Our patient illustrates this pattern: his lesion was characterized as an isolated rectal varix at colonoscopy two years prior to presentation and the diagnosis was not revised until cross-sectional and catheter-based imaging were obtained. Proper characterization is paramount for identifying feeding and draining vessels and the central nidus, classifying the lesion, and guiding management [1,10,12].

Consistent with guidelines for lower gastrointestinal (GI) bleeding, luminal endoscopy was the first-line diagnostic test and was performed on our patient soon after initial presentation [1]. In the case of rectal AVM, colonoscopy can identify a raised submucosal lesion through the mucosal lining, and can detect hemorrhage into the luminal space, providing guidance for subsequent clinical decision-making, while helping to rule out more common causes of lower GI bleeding [4]. Submucosal and extraluminal networks cannot be fully characterized, however, so colonoscopy serves as a triage modality before targeted vascular imaging is employed.

Once a vascular lesion is suspected, cross-sectional imaging assumes a central role. CTA is a widely available, rapid imaging modality well suited for a bleeding patient. It can both localize the site of hemorrhage and characterize the angioarchitecture of the suspected lesion by identifying enlarged feeding arteries, an enhanced nidus, and early filling veins [1].

Magnetic resonance imaging (MRI), specifically dynamic-time-resolved magnetic resonance angiography (MRA), serves to complement CTA through superior soft tissue contrast, enhanced detail in the angioarchitecture, and critically, assessment of flow dynamics [10]. Assessment of arterial inflow, venous drainage, and early venous shunting allows the lesions to be identified as high-flow lesions, where as a high rate of misdiagnosis has traditionally occurred when characterization is based on morphology alone [12]. Without the flow characteristics provided by MRA, rectal AVMs have been misdiagnosed as inflammatory bowel disease when evaluated using conventional T1- and T2-weighted images [4].

Catheter-directed digital subtraction angiography (DSA) remains the gold standard for definitive diagnosis of high-flow AVMs while simultaneously being capable of acting as a treatment modality. DSA provides the most precise characterization of the angioarchitecture, feeding arteries, nidus configuration, and venous drainage [1,4,10]. Superselective catheterization during DSA allows embolization to serve as both a means of confirming the diagnosis and a direct treatment modality [10,12]. In a previous case report, DSA demonstrated a feeding artery arising from the superior rectal artery and draining into the superior rectal vein, with superselective embolization achieving durable hemostasis [4], mirroring our patient's case.

Our patient's case followed the stepwise imaging pathway outlined above, a workflow that is required in part because the International Society for the Study of Vascular Anomalies (ISSVA) and Cho-Do classifications, guide lesion classification and embolic approach, respectively [10]. MRI/MRA was not warranted here but remains an adjunct to CTA in patients with contrast media allergy. The present case reinforces the importance of a stepwise imaging sequence in rectal AVM to reduce delays in care and unnecessary testing.

Treatment

No well-established guidelines exist for this condition, possibly reflecting its rarity. However, the

management of our patient's AVM highlights the value of adopting a stepwise escalation from endoscopic to endovascular intervention, a process that parallels recommendations by the World Society of Emergency Surgery regarding anorectal vascular emergencies, which specifically address bleeding anorectal varices [13].

The distinction between a rectal varix and an AVM is clinically relevant because the management of each differs. For bleeding rectal varices associated with portal hypertension, the 2024 American Association for the Study of Liver Diseases (AASLD) practice guidance identifies transjugular intrahepatic portosystemic shunt (TIPS) with concomitant embolization as a primary treatment option [14]. This approach differs fundamentally from the management of rectal AVMs, which involve direct arteriovenous shunting and require treatment directed toward the abnormal arterial inflow and AVM nidus. Endoscopic therapies, such as band ligation and injection sclerotherapy, have only been associated with modest success and are suggested as bridging therapies to more definitive treatment or as alternatives when interventional radiology (IR) is unavailable [14-15]. Selective arterial embolization has been described as a treatment approach, although the evidence base is limited to case reports and small series, given the rarity of this condition, with localized lesions potentially amenable to surgical resection through either transanal or formal bowel resection procedures [5].

The patient's medication history included ibuprofen 600 mg three times daily, ginkgo biloba, and ginseng, which were clinically relevant because of their potential bleeding considerations. A 2026 systematic review and meta-analysis by Tawfik et al. found that ibuprofen in particular was associated with a significant risk of gastrointestinal bleeding (odds ratio (OR) 2.28, 95% confidence interval (CI) 1.71-3.03) [16]. The Society for Perioperative Assessment and Quality Improvement consensus statement recommends holding both ginkgo biloba and ginseng for at least two weeks before any surgical procedure because of concerns regarding bleeding risk [17].

An endoscopic clip was placed at sigmoidoscopy, consistent with the American College of Gastroenterology (ACG) guideline recommendations for endoscopic hemostasis of actively bleeding lesions identified during endoscopy [18]. The clip failed to achieve durable hemostasis, and the patient returned five days later with recurrent bleeding. Following failure of endoscopic therapy, CTA demonstrated findings concerning for an AVM, prompting an IMA angiogram with findings consistent with an AVM, and selective and sub-selective arterial embolization was performed. Despite embolization, the patient returned on day 38, and repeat arteriography demonstrated markedly abnormal vascularity consistent with a complex AVM.

On day 49, definitive embolization was done targeting the superior rectal arterial branches, hemorrhoidal arteries, and the AVM nidus. This approach is similar to the "emborrhoid technique", which involves catheter-directed embolization of the superior rectal arteries via the IMA, originally developed for hemorrhoidal disease [19-20]. In this case, the technique was extended to target the AVM nidus directly, reducing arterial inflow to the anorectal vascular plexus. The strongest precedent for this application is in a case report by El-Sheikha et al. (2019), in which a rectal venous malformation was successfully treated with superior rectal artery embolization after three failed sclerotherapy attempts, which sustained improvements on follow-up MRI at one year [21]. However, that case involved a venous malformation, rather than an arteriovenous malformation. This case extends the potential application of SRA embolization to a high-flow rectal AVM with angiographically confirmed early venous drainage.

Recurrent bleeding has been reported after rectal artery embolization, occurring in 11 of 34 patients in the largest available retrospective series, although that cohort included heterogeneous etiologies rather than strictly AVMs. Continued surveillance imaging and assessment for rebleeding is essential to understanding the risks and benefits of extending SRA embolization to this population [22].

Genetics

The vast majority of arteriovenous malformations arise sporadically from somatic mutations. However, several hereditary conditions have been linked to gastrointestinal AVMs, including hereditary hemorrhagic telangiectasia (HHT) and Klippel-Trenaunay syndrome (KTS) [23-25].

HHT is an autosomal dominant disorder characterized by the formation of multiple AVMs due to mutations affecting transforming growth factor-beta (TGF-β) signaling, a pathway essential for establishing and maintaining vascular integrity [23,26]. HHT type I results from mutations in ENG, which encodes endoglin, a membrane glycoprotein involved in TGF-β signaling [23,26]. HHT type II is caused by mutations in ACVRL1, which encodes activin receptor-like kinase 1 (ALK1), a TGF-β receptor expressed on the endothelial lining of developing arteries [23,26]. A much smaller subset develops juvenile polyposis-associated HHT secondary to mutations in MADH4, encoding SMAD4, a critical downstream mediator of TGF-β signaling [23,26]. Approximately two-thirds of HHT cases are associated with ENG mutations and the majority of the remainder with ACVRL1 mutations, with SMAD4 mutations accounting for only a small minority [23,26].

KTS is another inherited disorder associated with gastrointestinal AVMs, characterized by the triad of capillary malformation, venous and lymphatic abnormalities, and hypertrophy of soft tissue and bone [24,27]. This disorder is recognized as part of the PIK3CA-related overgrowth spectrum, a group of disorders

caused by activating mutations in the PIK3CA gene [24,25]. These mutations result in hyperactivation of the phosphoinositide 3-kinase (PI3K)/protein kinase B (AKT)/mechanistic target of rapamycin (mTOR) signaling pathway, which regulates cellular proliferation, angiogenesis, and vascular development; dysregulation contributes to abnormal vascular architecture and AVM formation, including in the gastrointestinal tract [25]. Although our patient did not demonstrate any clinical findings suggestive of an inherited vascular syndrome, recognition of these genetic associations remains critical when evaluating patients with recurrent or multifocal vascular lesions [23-25].

Shared pathophysiology

The coexistence of rectal varices and a rectal AVM in this case highlights the anatomy of the SRA and superior rectal vein (SRV) system. The SRA provides the principal arterial supply to the rectum and contributes terminal branches to the corpus cavernosum recti, a specialized subepithelial vascular cushion composed of connective tissue surrounding direct arteriovenous communications between terminal arterial branches and rectal veins [20]. This structure contributes to continence and forms the anatomic basis of the hemorrhoidal cushions [19-20]. Hemorrhoidal disease reflects pathologic enlargement and displacement of these cushions [20]. Rectal varices are dilated portosystemic venous collaterals above the dentate line, most often associated with portal hypertension [15], and rectal AVMs are high-flow malformations characterized by direct arteriovenous shunting through a nidus [1-2]. Although pathologically distinct, these entities may overlap within the same rectal vascular territory [7-8]. In this case, angiography demonstrated that the SRA was the dominant arterial feeder to a complex AVM converging into a nidus within the mid-to-lower rectum.

A key pathophysiologic question in this case is whether the AVM served as the primary cause of variceal bleeding. Rectal AVMs transmit high-flow arterial inflow directly into the venous system, bypassing the capillary bed [1,10]. In this patient, shunting from the SRA into the SRV and adjacent pelvic venous pathways may have produced localized venous hypertension, resulting in pararectal venous engorgement and bleeding from fragile variceal channels. This is relevant because the patient lacked the conventional causes of rectal varices, such as cirrhosis or clinically significant portal hypertension [15]. The development of rectal varices here is therefore best interpreted as a mechanistic inference: high-flow shunting may have increased local venous pressure sufficiently to produce findings typically attributed to portal hypertension. The control of bleeding after SRA embolization is consistent with this proposed relationship.

In sporadic peripheral AVMs, somatic mutations affecting the mitogen-activated protein kinase (MAPK) pathway, including MAP2K1, KRAS, BRAF, and HRAS, have increasingly been recognized as molecular drivers [28-29]. The present case raises the possibility that shared angiogenic or vascular remodeling pathways contribute to the coexistence of a rectal AVM and adjacent hyperplastic venous networks. Future studies incorporating genetic or tissue-based molecular analysis may clarify whether such lesions reflect sporadic MAPK-driven malformations, hereditary syndromes, or secondary remodeling from chronic high-flow shunting.

The etiology of this lesion is best considered in terms of the distinction between congenital AVM and acquired angiodysplasia, a degenerative vascular ectasia associated with aging, intermittent venous obstruction, and increased wall tension [1,30]. Although the patient's age could suggest acquired ectasia, several features favor a congenital AVM that remained clinically silent for decades: a discrete nidus, high-flow arterial supply from the SRA, and early venous drainage into both the portal venous superior rectal system and systemic pelvic venous pathways [1,12]. This dual drainage is consistent with the rectum's role as a portosystemic junction and supports a complex pelvic vascular malformation rather than degenerative angiodysplasia [15,20]. The AVM may therefore have been congenital but clinically quiescent until later life, when progressive vascular remodeling, increased shunt flow, venous dilation, or age-related changes in the rectal vasculature reached a threshold for hemorrhage.

Although rectal AVMs have been described previously, they remain an uncommon cause of lower gastrointestinal bleeding and may be difficult to distinguish from more prevalent anorectal conditions. This case adds to the existing literature by illustrating the diagnostic challenge of recurrent hematochezia initially attributed to a rectal varix in a patient without cirrhosis or clinically significant portal hypertension. It also highlights the role of selective arterial embolization in managing a complex rectal AVM. Rather than describing a novel entity, this case emphasizes practical diagnostic and management considerations for clinicians evaluating patients with recurrent rectal bleeding of unclear etiology.

Literature review

A literature search was conducted using Google Scholar for articles published from database inception through 2026. The search strategy included the search terms "AVM rectal artery" and "arteriovenous malformation rectal artery". Relevant articles were identified and reviewed, with particular emphasis on published case reports and case series describing rectal arteriovenous malformations. A total of 13 articles were included in the final literature review. Articles were reviewed for information on clinical presentation, diagnostic evaluation, management, and patient outcomes. A summary of the reviewed literature is presented in Table *2*.

Ref #	Author(s) (Year)	Study type	Population	Clinical presentation	Diagnostic challenge	Outcome
[4]	Ma et al. (2024)	Case report	56-year-old man	Hematochezia progressed to repeated episodes of massive rectal bleeding despite initial treatment, ultimately requiring blood transfusion and supportive care.	Colonoscopy showed diffuse rectal inflammation and bleeding, while MRI demonstrated rectal wall thickening and tortuous vessels. DSA characterized the AVM, with the superior rectal artery and vein serving as the feeding and draining vessels, respectively.	Superior rectal artery embolization resulted in significant clinical improvement and complete resolution of rectal bleeding. No recurrence of bleeding or other symptoms at one-year follow-up.
[5]	Hayakawa et al. (1998)	Case report + literature review	39-year-old man	Persistent jelly-like anal bleeding followed by massive hemorrhage, syncope, and severe anemia.	Initial workup with MRI and barium enema revealed a nonspecific rectal lesion; selective superior rectal arteriography was required to identify and confirm AVM.	Underwent transanal resection without postoperative complications.
[6]	Meyer et al. (1981)	Case series + literature review	4 patients; 61–78 years	Recurrent rectal bleeding or melena resulting in chronic blood loss and anemia, often requiring repeated hospitalizations and/or blood transfusions.	Extensive GI workup was nondiagnostic; incorrectly attributed recurrent bleeding to diverticular disease. Selective mesenteric angiography ultimately localized the AVM. Most common sites were the cecum and right colon.	Most common treatment was right hemicolectomy, with the majority of patients achieving hemostasis with no recurrent symptoms, except one patient with persistent intermittent bleeding.
[7]	Komekami et al. (2016)	Case report + literature review	38-year-old man	One year of persistent anal bleeding, resulting in orthostatic dizziness and anemia	Bleeding internal hemorrhoids identified on colonoscopy, no AVM identified. Contrast-enhanced CT raised suspicion for AVM; suspicion confirmed following angiography.	Complete resolution of symptoms was achieved following selective embolization of AVM using a microcatheter.
[8]	Ishikawa et al. (2020)	Case report	86-year-old man	Increasing frequency of hematochezia despite prior treatment for presumed bleeding hemorrhoids. Active rectal bleeding on examination, and mild anemia.	Colonoscopy revealed a submucosal lesion just above the dentate line; extensive workup including endoscopic ultrasound, CT, MRI, and angiography was necessary to characterize and confirm the AVM.	Transcatheter arterial embolization of the left superior, middle, and inferior rectal arteries successfully resolved bleeding. No recurrence at one-year follow-up.
[9]	Mureb et al. (2025)	Case report	37-year-old man	Severe anemia in the setting of recurrent hematochezia and concurrent hematuria for one year. Initially suspected bladder mass.	Cystoscopy identified a bladder mass, while colonoscopy showed diffuse dilated rectal veins without a discrete mass; biopsy was avoided due to bleeding risk. CT and pelvic MRI demonstrated findings suggestive of multiple vascular malformations, with angiography further characterising the AVM.	Four embolization procedures improved the hematuria, but failed to control rectal bleeding. Laparoscopic low anterior resection with diverting ileostomy was subsequently performed, with pathology confirming AVM and resolution of symptoms at six-month follow-up.
				Frequent		

				hematochezia with severe anemia; colonoscopy revealed a pulsating submucosal lesion and enlarged, tortuous rectal vessels.	3D-reconstructed contrast-enhanced computed tomography (CECT) and selective angiography confirmed a rectal AVM supplied by the superior rectal artery with early venous return.	Combined retrograde transvenous and transarterial embolization with N-butyl-2-cyanoacrylate (NBCA)-Lipiodol resulted in complete cessation of bleeding and normalization of hemoglobin, with no recurrence at six-month follow-up.
[11]	Jubashi et al. (2021)	Case report	57-year-old man			
[21]	El-Sheikha et al. (2019)	Case report	25-year-old woman with Klippel-Trenaunay syndrome	Patient with KTS presented with predominant rectal involvement and extensive rectal/pelvic venous malformation.	MRI and angiography revealed extensive rectal/pelvic venous malformation with superior rectal artery supply and early venous filling.	Following three unsuccessful foam sclerotherapy attempts, continued bleeding necessitated superior rectal artery embolization. The procedure was successful, reducing bleeding within 10 days, and marked improvement on MRI at one-year follow-up.
[31]	Gao et al. (2024)	Case report + literature review	17-year-old girl	Multiple episodes of acute rectal bleeding with progressive tachycardia and severe anemia, ultimately requiring blood transfusion.	While initial CT angiography showed abnormal pelvic/rectal vessels, pelvic MRI and digital subtraction angiography were required to confirm the AVM and identify its feeding vessels.	Rectal vascular component was improved following selective embolization of the right superior rectal artery, although persistent pelvic venous abnormalities were present at follow-up.
[32]	Yoshimi et al. (2024)	Interventional radiology case report	72-year-old man	Gross hematochezia prompted imaging; CT identified a rectal AVM with a venous sac and multiple tortuous draining vessels.	Colonoscopy was avoided due to bleeding risk, while tortuous vessels complicated embolization access. Rectal balloon distension improved venous access and allowed catheter advancement into the venous sac.	Retrograde transvenous coil embolization successfully obliterated the AVM without complications. No recurrent bleeding or ischemia identified on colonoscopy at eight-month follow-up.
[33]	Muniandy et al. (2021)	Case report	41-year-old man	Rectal vascular malformation was incidentally discovered during evaluation of low back pain, with no symptoms of rectal bleeding or other related complaints.	Colonoscopy and multimodal imaging characterized the circumferential vascular lesion, with angiography showing vascular blush from the superior rectal artery without a nidus or early venous drainage.	Given that the patient was asymptomatic, the AVM was managed conservatively with observation and repeat MRI planned at six months.
[34]	Talman et al. (1979)	Retrospective review/case series	13 patients with unexplained lower GI bleeding; 9 with colonic AVMs	Repeated episodes of intermittent bright red or maroon rectal bleeding, frequently resulting in multiple hospitalizations and blood transfusions.	Routine GI workup often failed to identify the source of bleeding, requiring arteriography to localize the vascular lesion.	Right colectomy successfully controlled bleeding in the majority of AVM patients, with one case of rebleeding and one postoperative death following pulmonary embolism.
				Grade II-III hemorrhoids		Superselective superior rectal artery embolization with

[35]	Tradi et al. (2018)	Prospective clinical study	25 patients with grade II–III refractory hemorrhoids	refractory to medical management, with bleeding, pain, and reduced quality of life.	Angiography demonstrated AVM-like vascular features, with an angiographic nidus in 92% of patients and early venous opacification in 60%.	microcoils achieved 72% clinical success at one year, with significant improvements in bleeding, pain, and quality of life. No ischemia or incontinence was reported.

TABLE 2: Summary of reviewed literature

AVM - arteriovenous malformation; MRI - magnetic resonance imaging; GI - gastrointestinal; CT - computed tomography; DSA - digital subtraction angiography; KTS - Klippel-Trenaunay syndrome; SRA - superior rectal artery; 3D - three-dimensional; CECT - contrast-enhanced computed tomography; NBCA - N-butyl-2-cyanoacrylate.

A common finding across the reviewed literature was chronic or recurrent hematochezia, often associated with anemia [4-9,11,31-34]. In several reports, recurrent hematochezia progressively worsened over time, with patients experiencing numerous hospitalizations that ultimately culminated in massive hemorrhage and, in some cases, syncope, necessitating blood transfusions and more extensive evaluation [4-6,9,11,31,34]. Some patients had recurrent bleeding for months or even years before a definitive diagnosis was made, as reported by Hayakawa et al., Meyer et al., Gao et al., Mureb et al., Ma et al., Talman et al., and Jubashi et al. [4-6,9,11,31,34]. Only one case deviated from this typical presentation and progression. Muniandy et al. described a rectal AVM discovered incidentally during imaging for an unrelated condition [33]. The patient was asymptomatic and was therefore managed conservatively, in contrast to the patients in the other cases who were symptomatic and, in some instances, presented with severe or life-threatening bleeding. The age range across all articles was 17-86 years. Clinical presentation varied from entirely asymptomatic to massive hemorrhage and hemodynamic instability [4-9,11,31-34]. This broad age distribution suggests that AVMs can present across a wide age range without a clear predilection for a particular age group. Overall, while recurrent hematochezia and anemia were the most common presentations, the clinical spectrum ranged from incidental, asymptomatic lesions to severe and potentially life-threatening hemorrhage.

Another pattern identified across the literature was the overlap between rectal AVMs and other more common sources of lower GI bleeding, particularly hemorrhoids [7-8,35]. Some patients were initially diagnosed with or treated for hemorrhoids before the underlying vascular malformation was recognized. This was demonstrated in the case reported by Ishikawa et al., in which an elderly patient underwent rubber-band ligation for presumed bleeding hemorrhoids without resolution of his symptoms [8]. When he returned to the hospital for further evaluation, angiography ultimately identified the underlying rectal AVM. Similarly, Komekami et al. reported a patient with actively bleeding internal hemorrhoids that were initially believed to be the source of his bleeding [7]. However, persistent symptoms prompted further evaluation with contrast-enhanced CT and angiography, ultimately identifying the rectal AVM as the source of bleeding. Other reports further demonstrated the potential for rectal AVMs to coexist with hemorrhoidal disease [7-8,35]. This overlap was particularly evident in the study by Tradi et al.; in a group of 25 patients with grade II-III refractory hemorrhoids, the SRA was targeted for embolization, highlighting the involvement of the same arterial circulation that commonly supplies rectal AVMs [35]. This overlap further complicates diagnosis, as the presence of hemorrhoids may lead to the vascular malformation being overlooked as the underlying source of bleeding. Therefore, persistent or recurrent bleeding despite treatment for presumed hemorrhoids should prompt consideration for an underlying rectal AVM.

Despite the relatively consistent clinical presentation, establishing the underlying diagnosis was often challenging. Patients frequently underwent multiple diagnostic studies and imaging modalities before the AVM was identified. The most commonly reported studies included colonoscopy, barium enema, CT, MRI, and angiography [4-9,11,31-34]. In several cases, routine endoscopic evaluation was nondiagnostic, with findings ranging from normal-appearing mucosa to nonspecific changes such as edema, wall thickening, or erosion [4-9,11,31-33]. These findings highlight the difficulty of diagnosing rectal AVMs with conventional lower GI endoscopic evaluation alone, particularly when the lesion is located outside the bowel wall. In contrast to endoscopic evaluation, angiography was consistently reported as a critical and often definitive diagnostic modality [4-9,11,31-34]. Angiography identified the abnormal arterial feeders, vascular nidus, and early venous drainage associated with the AVM, confirming the diagnosis and providing important information about its vascular anatomy to guide treatment [4-9,11,31-32]. Several reports described enlarged and tortuous superior rectal arterial branches supplying the AVM, while other studies identified complex arterial supplies involving multiple branches. CT angiography, MRI, and digital subtraction angiography were also used in combination to further define the extent of the AVM and its vascular anatomy [4,7-9,11,31-32].

Management strategies varied widely across the reviewed literature, with both surgical and endovascular approaches described [4-9,11,31-35]. Earlier reports, such as Hayakawa et al. and Meyer et al., commonly

utilized surgical resection for symptomatic AVMs, ranging from transanal resection for localized rectal disease to right hemicolectomy for colonic lesions [5-6]. More recent reports tended to utilize less invasive approaches as alternatives to surgical resection [4,7-9,11,31-33,35]. Examples include selective and superselective arterial embolization, which were reported to control rectal bleeding in most cases [4,7-9,11,31-32,35]. For more complex cases, specialized endovascular approaches were implemented to achieve hemostasis, including retrograde transvenous embolization, combined transarterial and transvenous embolization, and balloon-assisted techniques [11,32]. These alternative approaches provided improved access to the vascular nidus in cases with challenging vascular anatomy. Although embolization was reported to resolve bleeding in the majority of cases, some patients experienced persistent or recurrent bleeding following the procedure [9,31]. In select cases, surgical resection was ultimately required following unsuccessful endovascular treatment, as seen in the case report published by Mureb et al. [9]. Overall, treatment appeared to be tailored to the individual patient based on the vascular anatomy and extent of the lesion, as well as the response to prior interventions.

Rectal AVMs are a rare but important cause of recurrent or severe lower gastrointestinal bleeding that can be difficult to diagnose and manage. The most common clinical presentations of rectal AVMs were hematochezia and anemia, with severity ranging from recurrent bleeding to massive hemorrhage and hemodynamic instability [4-9,11,31-34]. The coexistence of rectal AVMs and hemorrhoids was also frequently reported, with hemorrhoids often presumed to be the underlying source of bleeding [7-8,35]. This may contribute to delayed diagnosis, with recurrent bleeding and worsening anemia persisting in the interim. These findings underscore the importance of a thorough diagnostic evaluation when the source of bleeding remains unclear. Routine endoscopic evaluations, including colonoscopy, were often nondiagnostic or revealed only nonspecific findings, while angiography provided definitive vascular characterization of the AVM in reported cases [4-9,11,31-34]. More recent publications have increasingly described selective embolization as a minimally invasive treatment option, although persistent or recurrent bleeding following endovascular treatment may require additional intervention [4,7-9,11,31-32,35]. Overall, the reviewed literature highlights the diagnostic and therapeutic challenges associated with rectal AVMs and the importance of maintaining a high index of suspicion when bleeding persists without an adequate explanation.

This case report is limited by its single-patient nature, which restricts its generalizability. However, by highlighting the diagnostic challenges associated with arteriovenous malformations that are consistent with the current literature, this report may inform diagnostic decision-making in these rare cases and help reduce adverse outcomes associated with delayed diagnosis and treatment. The patient's long-term outcomes, including recurrence, symptom relief, and progression, also remain unknown.

Conclusions

This case highlights the potential for rectal AVMs to be misdiagnosed as rectal varices in patients with lower GI bleeding, particularly in the absence of portal hypertension and when endoscopic hemostasis fails. In this case, diagnosis was achieved through a stepwise imaging approach culminating in catheter angiography. Definitive treatment was achieved through extension of SRA embolization ("emborrhoid-type") to the high-flow AVM.

Beyond the diagnostic challenge, this case adds to the limited literature describing staged transarterial embolization as a minimally invasive treatment option for rectal AVMs. In this case, continued clinical and imaging surveillance is warranted to assess treatment durability and identify recurrent bleeding. This case suggests that rectal AVM should be considered when recurrent hematochezia is attributed to varices in the absence of portal hypertension.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Jenna L. Phillips, Simon Cooper, Keshav Iyer, Arravintha Gobinathan, Gary Schwartz

Acquisition, analysis, or interpretation of data: Jenna L. Phillips, Simon Cooper, Keshav Iyer, Arravintha Gobinathan, Jeffrey P. Snow, Jordan Ditchek, Gary Schwartz

Drafting of the manuscript: Jenna L. Phillips, Simon Cooper, Keshav Iyer, Arravintha Gobinathan, Jordan Ditchek, Gary Schwartz

Critical review of the manuscript for important intellectual content: Jenna L. Phillips, Jeffrey P. Snow, Gary Schwartz

Supervision: Gary Schwartz

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