

# Percutaneous Transhepatic Embolization of Bleeding Rectal Varices Using A New Embolic And Sclerotic Mixture Augmented By Amplatzer Vascular Plug 2

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Radiology Case. 2016 Sep; 10(9):44-51 :: DOI: 10.3941/jrcr.v10i9.2580

## ABSTRACT

We report a case of 59-year-old female with non-alcoholic-steato-hepatitis (NASH) induced cirrhosis, who presented with hematochezia. The patient had a history of bleeding esophageal varices treated with endoscopic variceal ligation (EVL). Colonoscopy showed large rectal varices which were the source of her lower gastrointestinal bleeding (LGIB). Since endoscopic treatment for LGIB are limited, and because the patient had portal vein thrombosis which contraindicated transjugular intrahepatic portosystemic shunt (TIPS), we performed percutaneous transhepatic embolization of her rectal varices using a new mixture of embolic and sclerotic agents, followed by Amplatzer plug 2 (AVP 2). To our knowledge, the use of this new mixture with the AVP 2 in the rectal varices treatment has not been previously published in literature. Our case provides an alternative treatment modality that can be used for rectal varices treatment, when TIPS and endoscopic management fails or is contraindicated.

## CASE REPORT

### CASE REPORT

A 59-year-old African American female with non-alcoholic-steato-hepatitis induced cirrhosis, portal hypertension and portal vein thrombosis with a history of esophageal endoscopic variceal ligation four years ago developed severe bloody bowel movement for 2 weeks that was progressively worsening to a point where the patient experienced blood clots in her stools. The patient presented to an outside hospital with hepatic encephalopathy (HE) and LGIB. She underwent an endoscopic evaluation which showed stable esophageal varices with evidence of prior banding, as well as, grade 2-3 gastric varices without any stigmata of active bleeding. Colonoscopy was also performed in the same setting and revealed large rectal varices. The patient was emergently

administered three units of packed red blood cells and one unit of platelets. The patient was then admitted and treated for HE and LGIB resulting from her rectal varices.

The patient was then transferred to our institution for assessment of possible TIPS placement. The patient continued to experience significant bloody bowel movements. Abdominal ultrasound (US), as well as, contrast enhanced computerized tomography (CT) and magnetic resonance imaging (MRI) of the abdomen was obtained for the patient. Abdominal US showed cirrhotic liver with large amount of thrombus partially occluding the cephalic portion of the superior mesenteric vein (SMV) as well as the portal vein and its main right and left branches, with small residual patent lumen. However, perfusion of some of the distal right and left portal vein

branches was maintained. Splenomegaly and moderate amount of ascites were also seen. Contrast enhanced CT of the abdomen and pelvis confirmed the US findings. In addition, the splenic vein (SV) was seen patent and there were multiple dilated vascular spaces in the rectal wall consistent with rectal varices draining into a dilated inferior mesenteric vein (IMV) (Fig. 1). MRI of the abdomen identified abnormal signal intensity and abnormal enhancement in the hepatic dome as well as the posterior inferior segment of the liver, which was not typical for HCC.

Since the endoscopic treatment options for rectal varices are limited, the patient was referred to interventional radiology to explore the possibility of performing TIPS. However, her high Model for End-stage Liver Disease (MELD) score, history of HE and portal vein thrombosis precluded her from this option [1]. An interdisciplinary consensus between the Interventional Radiology and Hepatology Services opted to perform percutaneous transhepatic embolization of her rectal varices. The patient gave consent to perform this non-standard procedure after being informed of its risks and potential benefits. Of note, the MELD score is used to assess the severity of chronic liver disease. It is used mainly in potential candidates for TIPS or liver transplant procedures. Three variables are used to calculate MELD score, serum bilirubin, serum creatinine, and international normalized ratio for prothrombin time (INR) [2].

Using ultrasound guidance, access to the patent peripheral anterior right portal branch was obtained. Venogram was performed to confirm location within the portal venous system (Fig. 2). Using a 0.018 in wire (Nitrex; Covidien, Plymouth, MN), an introducer set (Neff set; Cook, Bloomington, IN) was successfully advanced into the main portal vein, and portal venogram was then obtained that showed a large filling defect in the main portal vein. A guide wire (Glidewire; Terumo, Tokyo, Japan) and 4 French catheter (Berenstein; Cook, Bloomington, IN) were advanced through the partially occluded portal vein into the splenic vein, and then into the IMV. Inferior mesenteric venogram was then performed and demonstrated multiple large serpiginous rectal varices draining into a dilated IMV (Fig. 3).

The Neff set was then exchanged for a 6 French guiding sheath (Ansel; Cook, Bloomington IN), and pressures within the IMV were recorded and revealed systolic, diastolic and mean pressures of 38, 36 and 37 mmHg respectively. Pressure in the right atrium were also measured through a preexisting central venous line and revealed systolic, diastolic and mean pressures of 5, 2 and 3 mmHg respectively, giving a portosystemic gradient of 34 mmHg (normal value is up to 5 mmHg). The sheath and catheter were then advanced into the caudal most aspect of the IMV, as close as possible to the rectal varices. An embolization mixture of embolic and sclerotic agents consisting of 1 gm of Avitene (Bard, Tempe, AZ), 4 ml of 3% Sotradecol (Angiodynamics, Latham, NY), and 1 cc of shredded Gelfoam (Pfizer, New York, NY), mixed in 40 ml of non-ionic contrast medium (Omnipaque 350; GE Healthcare) [3] was prepared and injected through the catheter into the rectal varices while watching for reflux, until stasis was achieved. A follow-up inferior mesenteric venogram was

performed showing absent flow of contrast in the rectal varices and caudal IMV suggesting successful embolization and sclerosis.

The catheter was then removed and a 12 mm AVP 2 was introduced through the sheath into the caudal IMV. After confirming appropriate positioning of the device through a venogram done through the sheath, the device was released. A final venogram was then obtained which confirmed cessation of flow in the caudal IMV and rectal varices (Fig. 4). The sheath was then removed and the tract in the liver was secured using a 6 mm AVP 2 (Fig. 5).

The patient had no post-procedural complications and continued to show improvement of her peripheral blood cell count. The course of her hospital stay was otherwise uneventful. The patient was deemed stable for discharge home a week after the procedure. No clinical or radiological signs of recanalization were observed during routine follow-up for the background liver disease.

## DISCUSSION

### *Etiology & Demographics:*

Lower gastrointestinal bleeding is defined as blood loss from the gastrointestinal (GI) tract distal to the ligament of Treitz. It is suggested when patients present with hematochezia. In patients with portal hypertension, varices can develop at the portosystemic anastomoses along the gastrointestinal tract due to portosystemic shunting. Esophago-gastric varices are considered the most common varices to develop. Any varices that develop at any other location are considered "ectopic" [4-7].

Rectal varices develop at the anastomoses between portal and systemic venous systems in an attempt to reduce the high pressure in portal circulation by forming multiple collaterals that drain into the low-pressure systemic venous circulation. The portal venous system is represented by the superior rectal vein which drains into the IMV. The systemic venous system is represented by both middle and inferior rectal veins, which drain into internal iliac vein [8-10].

Rectal varices are one of the most common types of ectopic varices. The incidence of rectal varices among portal hypertension patients ranges from 3-75% [4-7]. Despite the fact that bleeding from rectal varices is considered rare with a frequency ranging from 0.5 to 3.6%, it can be fatal due to massive hemorrhage [11-14].

The occlusion of esophago-gastric varices as a treatment of upper gastrointestinal bleeding (UGIB) redirects the high pressure portal circulation blood to develop ectopic varices. Accordingly, the incidence of rectal variceal bleeding is found to increase after endoscopic treatment for esophageal varices [15-18].

### *Clinical & Imaging Findings:*

In our case, the patient underwent esophageal endoscopic variceal ligation four years prior to her presentation, which is known to increase the incidence of ectopic varices due to shift

of blood to other porto-systemic collaterals [15-18]. Moreover, the patient was suffering from partially thrombosed portal vein, which also contributed to progressive worsening of her pre-hepatic portal hypertension and the development of her rectal varices.

Trans-rectal endoscopy and ultrasonography are the main investigations for diagnosis of rectal varices [10,19]. Computed tomography angiography and magnetic resonance angiography may be used to detect the presence of rectal varices as well as the afferent and efferent vessels [19].

#### *Treatment & Prognosis:*

Until now, there is no standard treatment for rectal varices. However, many therapeutic strategies have been described including surgery [19], endoscopic sclerotherapy (ES), EVL and minimally invasive endovascular intervention [20-22]. The choice of treatment varies according to the different hemodynamics seen in each case, and depends mainly on the number of feeding and draining veins, presence of venous occlusion, and the presence of recanalized umbilical vein.

TIPS and embolotherapy are minimally invasive endovascular interventions and these techniques are preferred alternatives for the management of rectal varices that are resistant to surgical and endoscopic treatment [23]. Our patient had a history of HE and extensive portal venous thrombosis, which made placement of TIPS technically challenging and unfavorable. This was the reason we opted for a percutaneous transhepatic embolotherapy of her rectal varices.

Several reports have been published on the successful embolization of rectal varices via different endovascular approaches. Hidajat and his colleagues [24] described placement of TIPS and transjugular embolization using coils for the management of rectal varices. This technique helps prevent recurrent bleeding by lowering the portal pressure after the creation of TIPS. Other authors such as Minamiguchi and his colleagues [25] preferred to embolize the rectal varices by balloon-occluded antegrade transvenous sclerotherapy using 5% ethanolamine oleate-iopamidol mixture and microcoils through a transhepatic approach. Another technique was published by Kimura and his colleagues [26] who performed double balloon – occluded embolotherapy.

Other authors preferred to approach the rectal varices through catheterization of a recanalized umbilical vein or an enlarged-engorged paraumbilical vein, and used several embolic agents to occlude the varices [1,27]. Arai and his colleagues [28] reported ileocolic vein catheterization to obliterate rectal varices using coils and 5% EOI.

In our case, we performed embolization of the rectal varices through percutaneous transhepatic approach with injection of a new mixture of embolic and sclerotic agents, followed by placement of AVP 2 device. The new described embolic and sclerotic mixture offers immediate occlusion of the varices and appears to be safe, since we did not encounter any immediate migration into the systemic veins. The mixture also appears to be cost-effective. Another AVP 2 was introduced to seal the hepatic puncture site, thus avoiding

intraperitoneal bleeding which is reported to occur in 10.6% of patients treated with transhepatic approach [29] (Table 1, Fig. 6).

#### *Differential Diagnoses:*

The differential diagnoses of LGIB include several diseases such as bleeding hemorrhoids, anal fissure, which can be diagnosed clinically. Diverticular disease is one of the most common causes of LGIB and can be presented by pain at the left lower quadrant pain, change in bowel habits and bouts of bleeding per rectum which often stop spontaneously. It can be diagnosed by CT or lower gastrointestinal endoscopy as an out-pouching sac from the bowel wall [30].

It can be also due to vascular causes such as ischemic colitis and rectal wall arteriovenous malformations. In ischemic colitis, patients are usually presented with acute onset of abdominal pain with an urge for defecation. Passage of bright red blood is often observed mixed with stool. Abnormal bowel wall thickening due to submucosal oedema is a classic sign. Intramural, portal, and intraperitoneal gas can be also seen. Laboratory studies are usually normal except in severe cases, where leukocytosis, metabolic acidosis, and elevated lactate level can be noted. Patients with bowel wall arteriovenous malformations commonly presented with an intermittent, recurrent, chronic bleeding as the common presentation. However, massive bleeding can be sometimes observed. Lower GI bleeding due to arteriovenous malformations are usually seen in elderly patients. Radiologically, CT or MRI venography are important diagnostic tools to detect tufts of abnormal vascular networking between arterial and venous systems. Coagulopathy could be another cause of LGIB [30].

Inflammatory bowel disease (IBD) is another common cause of LGIB. Presented usually by bloody diarrhea, which can vary in severity. However, it is rarely presented with massive bleeding. Bowel wall thickening, target sign, fat halo sign, strictures and fistulae are different radiological signs can be spotted in patients with IBD [30].

Patient with GI malignancies can present with LGIB, which can vary between occult bleeding, bright red blood per rectum, or melena. A variety of radiological findings can be found by imaging modalities, such as apple-core appearance resulted from luminal narrowing by soft tissue masses, fungating mass inside the luminal cavity, or massive infiltrating scirrhous type of carcinoma [30] (Table 2, Fig.7).

#### TEACHING POINT

Transhepatic embolization of bleeding rectal varices can be an alternative treatment modality that can be used for rectal varices management, when TIPS and endoscopic management fails or is contraindicated. Embolization with the new embolic and sclerotic mixture consisting of Avitene, Sotradecol, shredded Gelfoam, mixed in non-ionic contrast medium and augmented by AVP 2 for transhepatic embolization of bleeding rectal varices, appears to be useful and cost-effective method, however, further studies on larger sample size are still needed to evaluate the efficacy and safety of this method.

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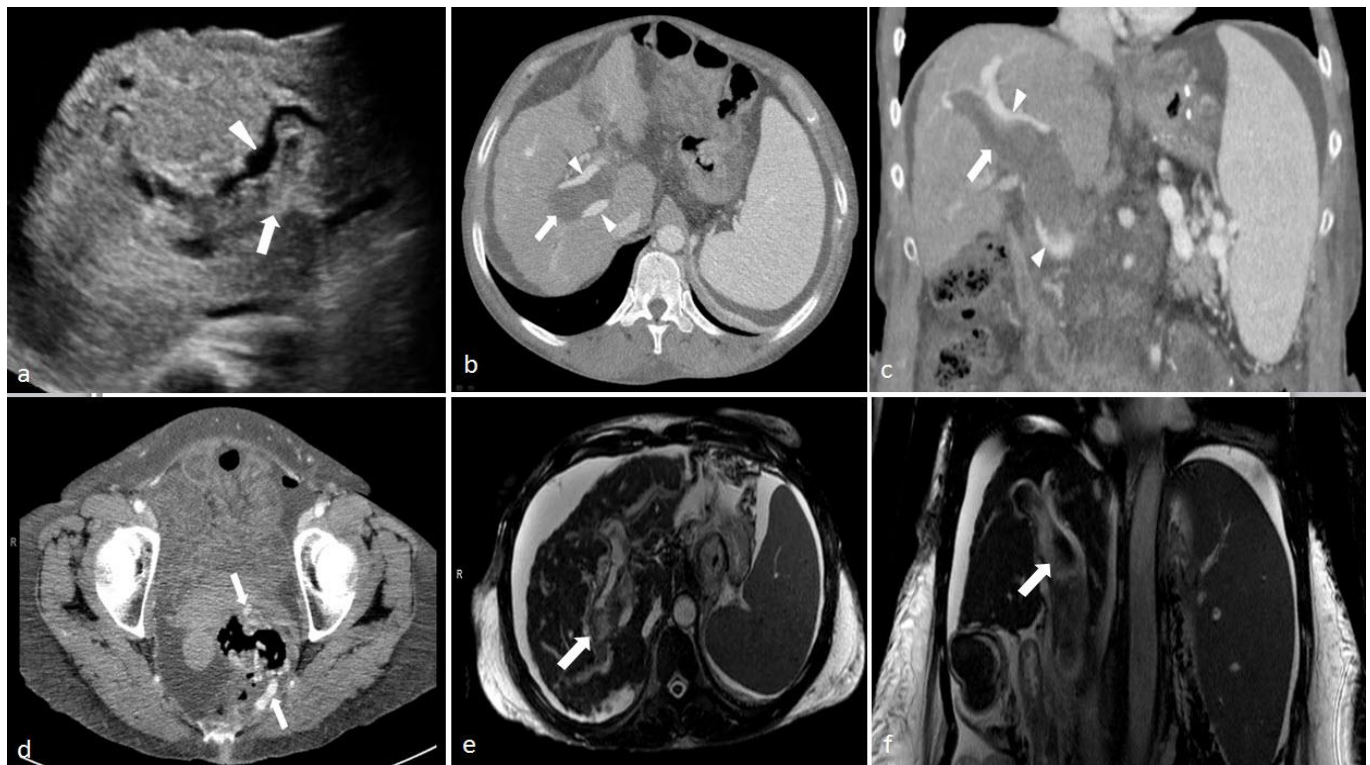
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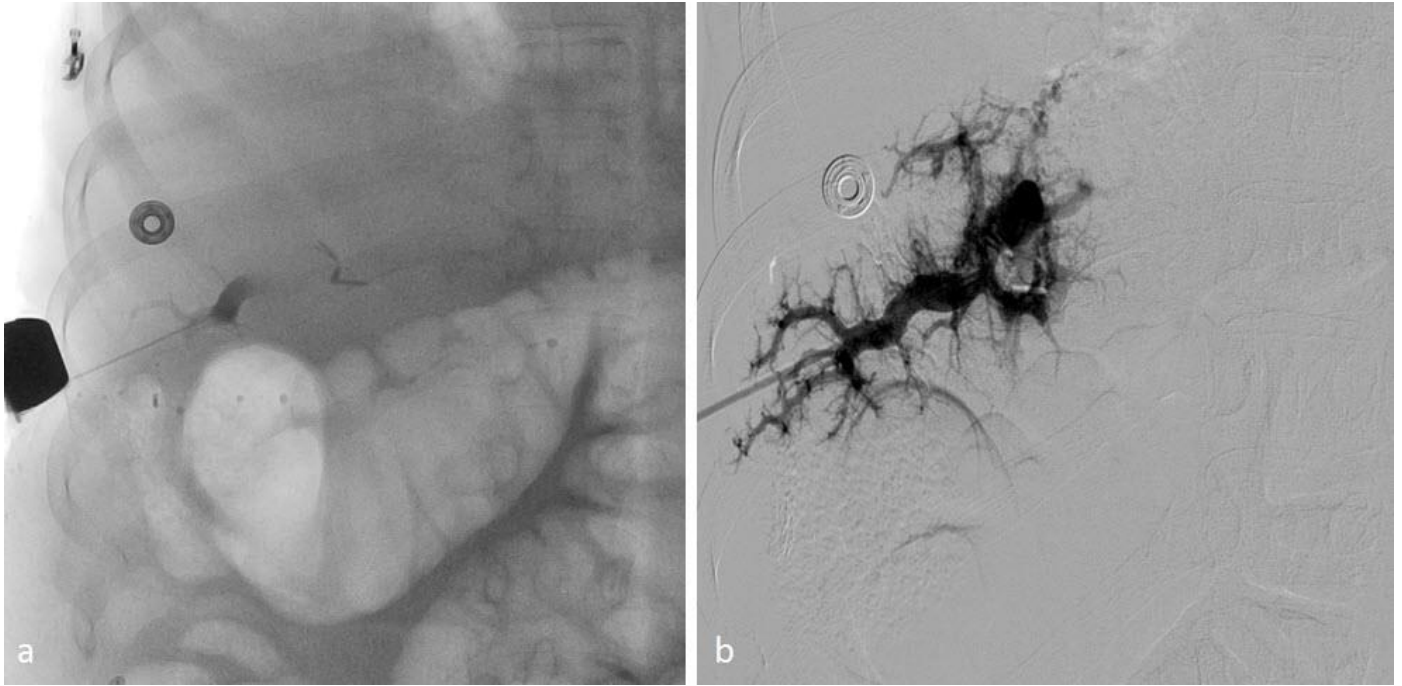
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FIGURES



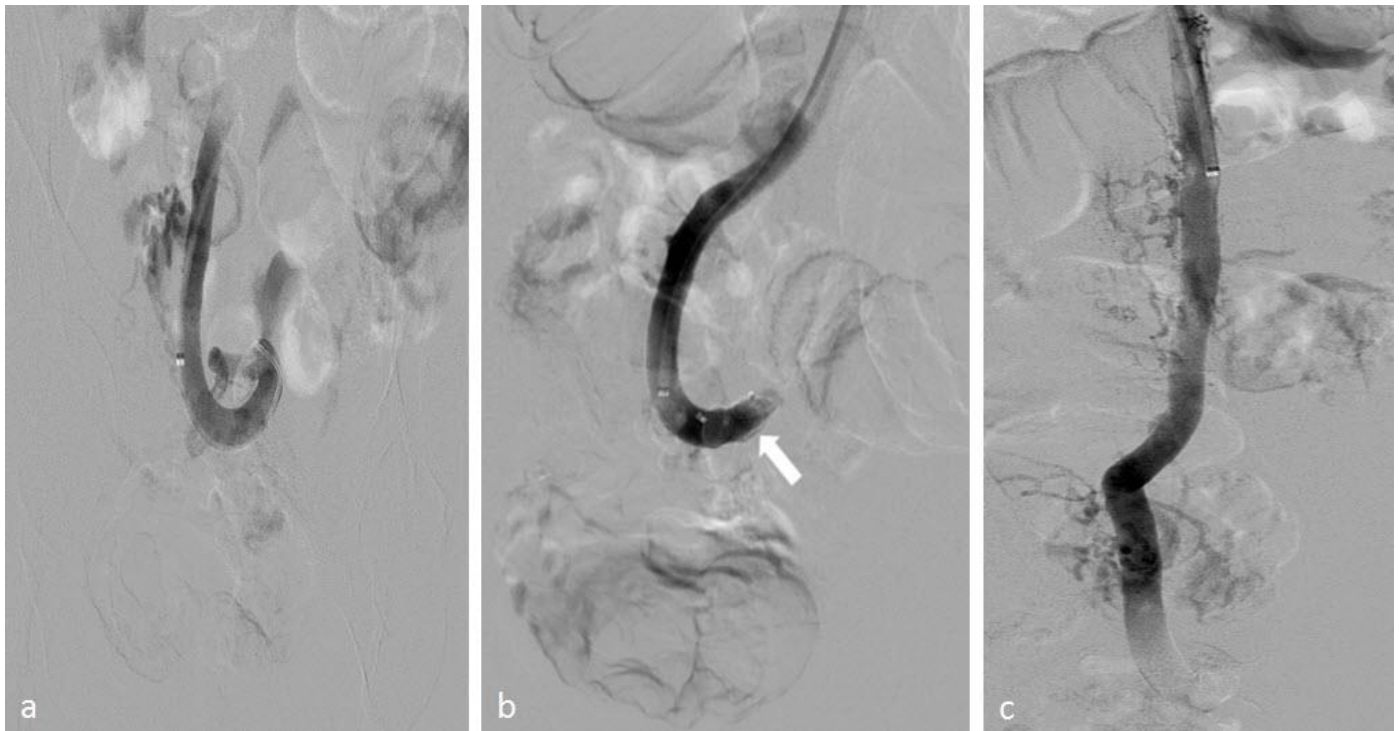
**Figure 1:** A 59-year-old female with non-alcoholic-steato-hepatitis (NASH) induced cirrhosis, who presented with hematochezia. FINDING : (a) Ultrasonography of the liver showing large echogenic thrombus (large arrow) partially occluding the portal vein with small residual patent lumen (arrowheads) in a patient with cirrhotic liver. Ascites is also noted. (b) and (c) Contrast-enhanced axial and reformatted coronal CT images showing large amount of thrombus (large arrow) partially occluding the portal vein and its main right and left branches, with small residual patent lumen (arrowheads). Perfusion of some of the distal right and left portal vein branches is seen. Splenomegaly and moderate amount of ascites were also noted. (d) Contrast-enhanced axial CT image showing multiple dilated vascular spaces (large arrows) in the rectal wall consistent with rectal varices. (e) and (f) Axial and coronal T1 MRI images showing mixed signal thrombus (large arrow) partially occluding the portal vein and its main right and left branches extending to the portosplenic confluence. Splenomegaly and moderate amount of ascites were also noted. TECHNIQUE: US: 3.5 MHz curvilinear transducer, gray scale image. CT: mAs 207, kVp 100, slice thickness 3 mm. MRI: 3.0T, 7mm slice thickness, TR 4.1. TE 2.0. FOV: 35 and 43 cm.



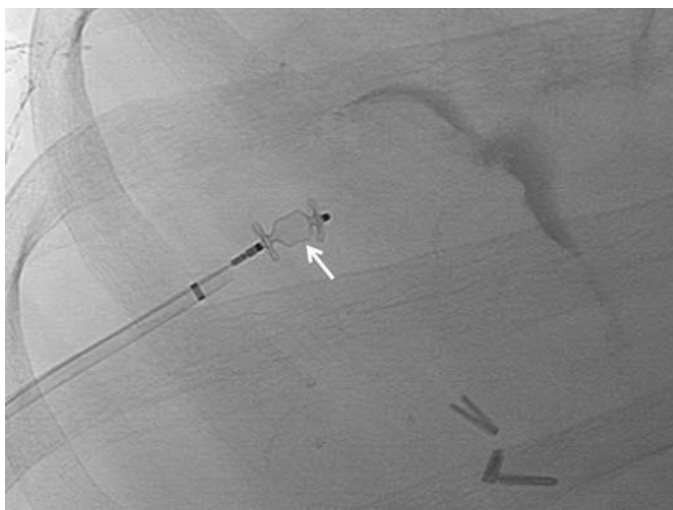
**Figure 2:** A 59-year-old female with non-alcoholic-steato-hepatitis (NASH) induced cirrhosis, who presented with hematochezia. (a) and (b) Steps of percutaneous access to a peripherally patent portal vein following ultrasound guidance. A venogram demonstrated contrast opacification of the patent peripheral anterior right portal branch.



**Figure 3:** A 59-year-old female with non-alcoholic-steato-hepatitis (NASH) induced cirrhosis, who presented with hematochezia. (a), (b) and (c) Inferior mesenteric venogram showing dilated inferior mesenteric vein (large arrows) with multiple dilated serpiginous tributaries in the rectal wall consistent with rectal varices (small arrows).



**Figure 4:** A 59-year-old female with non-alcoholic-steato-hepatitis (NASH) induced cirrhosis, who presented with hematochezia. (a) Venogram performed after embolization of the rectal varices by a new mixture of Avitene, Sotradecol and shredded Gelfoam, mixed in non-ionic contrast medium showing absent filling of the varices. (b) Embolization was augmented by deployment of a 12 mm AVP 2 device (large arrow). (c) Inferior mesenteric venogram performed after complete embolization of rectal varices were achieved. No residual rectal varices could be detected.



**Figure 5:** A 59-year-old female with non-alcoholic-steato-hepatitis (NASH) induced cirrhosis, who presented with hematochezia. A non-subtracted image showing a 6 mm AVP 2 device (small arrow) deployed to obliterate the tract in the liver created by the access sheath.

<b>Etiology</b>	Portal hypertension.
<b>Incidence</b>	One of the most common types of ectopic varices. The incidence of rectal varices among portal hypertension patients ranges from 3-75%.
<b>Gender ratio</b>	Occurs in both sex.
<b>Age predilection</b>	No specific age predilection.
<b>Risk Factors</b>	Occlusion of esophago-gastric varices as a treatment of upper gastrointestinal bleeding.
<b>Treatment</b>	No standard treatment for rectal varices. However, many therapeutic strategies have been tried including surgery, endoscopic sclerotherapy, EVL and minimally invasive endovascular intervention.
<b>Finding on CT imaging</b>	Multiple dilated vascular spaces in rectal wall that enhance at the portal phase.

**Table 1:** Summary table for rectal varices.

Differential diagnosis	Clinical data and laboratory results	Imaging (CT)
<b>Anal fissure</b>	Can be detected by clinical examination.	---
<b>Bleeding hemorrhoids</b>	Can be detected by clinical examination.	---
<b>Diverticular disease</b>	Left lower quadrant pain, change in bowel habits and bouts of bleeding per rectum.	Out-pouching of bowel result in blind-ended diverticula in communication with the lumen of the bowel.
<b>Ischemic colitis</b>	Acute onset of abdominal pain and tenderness with an urge to defecate. Bright red blood is often mixed with stool. Laboratory studies are usually normal. In severe cases, leukocytosis, metabolic acidosis, and elevated lactate level can be found.	Symmetrical or lobulated thickening of bowel wall. Irregularly narrowed lumen. Submucosal oedema may produce low-density ring bordering lumen (target sign). Intramural or portal venous gas.
<b>Rectal wall arteriovenous malformations</b>	More in patients over 50 years old. An intermittent, recurrent, chronic bleeding is the common presentation. Massive bleeding can be sometimes observed.	Detection of abnormal vascular networking between arterial and venous systems which demonstrate venous enhancement at the arterial phase.
<b>Coagulopathy</b>	Can be detected by laboratory findings.	---
<b>Inflammatory bowel disease</b>	Common cause of LGIB. Presented usually by bloody diarrhea. Rarely result in massive bleeding.	Can vary between abnormal bowel wall thickening, target sign, fat halo sign, strictures and fistulae.
<b>Colonic, rectal and anal malignancies</b>	Occult bleeding, bright red blood per rectum or melena can be a presenting symptom of GI malignancy.	Soft tissue mass that usually narrows the bowel lumen that might form apple-core appearance. Fungating mass inside the luminal cavity.

**Table 2:** Differential table of rectal varices.

**ABBREVIATIONS**

AVP 2: Amplatzer plug 2  
 CT: Computerized tomography  
 ES: Endoscopic sclerotherapy  
 EVL: Endoscopic variceal ligation  
 GI: Gastrointestinal  
 HE: Hepatic encephalopathy  
 IBD: Inflammatory bowel disease  
 IMV: Inferior mesenteric vein  
 INR: International normalized ration for prothrombin time.  
 LGIB: Lower gastrointestinal bleeding  
 MELD score: Model for end-stage liver disease  
 MRI: Magnetic resonance imaging.  
 NASH: Non-alcoholic-steato-hepatitis  
 SMV: Superior mesenteric vein  
 SV: Splenic vein  
 TIPS: Transjugular intrahepatic portosystemic shunt  
 UGIB: Upper gastrointestinal bleeding  
 US: Ultrasound

**KEYWORDS**

Rectal varices; embolization; portal hypertension; amplatzer vascular plug; Avitine; Sotradecol

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