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Portal Hypertensive Mayhem: A Case Report of Colonic Variceal Bleeding Treated by Antegrade Embolization

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Abstract

In patients with cirrhosis of the liver, acute variceal bleeding can be fatal. Bleeding colonic varices is a very rare cause of lower gastrointestinal bleeding that can result in massive hemorrhage in patients with cirrhosis. Here we present the case of a 50-year-old gentleman, having cirrhosis of the liver with chronic portal vein occlusion, who presented with massive bleeding per rectum. Contrast computed tomography (CT) revealed a large shunt arising from the superior mesenteric vein forming submucosal colonic varices and joining the right renal vein. He underwent successful obliteration of the varix by antegrade endovascular embolization via the trans-splenic access. Endovascular embolization could be an effective and life-saving treatment option for colonic variceal bleeding.

Keywords
- ectopic colonic varices
- antegrade embolisation
- portal hypertension

Introduction

Varices that occur at a site other than the esophagus and stomach are termed "ectopic varices" and account for less than 5% of all varix-related bleeding episodes.[1] When ectopic variceal bleeding occurs, the mortality rate reaches as high as 32.1%, including death in 25.0% of cases with colonic varices.[2] Colonic varices are generally associated with portal hypertension due to liver cirrhosis or other causes of portal venous obstruction.[3]

Case Report

A 50-year-old gentleman, known case of cryptogenic cirrhosis for past 15 years with chronic occlusion of the portal vein, was referred to us with complaints of bleeding per rectum. The index presentation was 3 days back when he was hospitalized with severe bleeding per rectum. He underwent endoscopic sclerotherapy for rectal varices and received multiple blood transfusions. However, he had recurrence of bleeding per rectum and was referred to our hospital for further management.

On admission, his blood pressure was 108/70 mm Hg, pulse rate 110 beats/min, SpO_2 98% on room air, and hemoglobin 5.3 g/dL. The patient was shifted to the intensive care unit and blood products were transfused. Colonoscopy revealed fresh blood around an ectopic colonic variceal nipple (►**Fig. 1**) besides rectal varices. Cyanoacrylate glue (1 mL) was injected into the colonic varix.

As part of further evaluation, contrast computed tomography (CT) was done, which revealed a large shunt (►**Fig. 2**) arising from the superior mesenteric vein (SMV). The shunt was seen traversing around the ascending colon, protruding into the colonic lumen, taking a tortuous course, and finally joining the right renal vein. In addition, there was occlusion of

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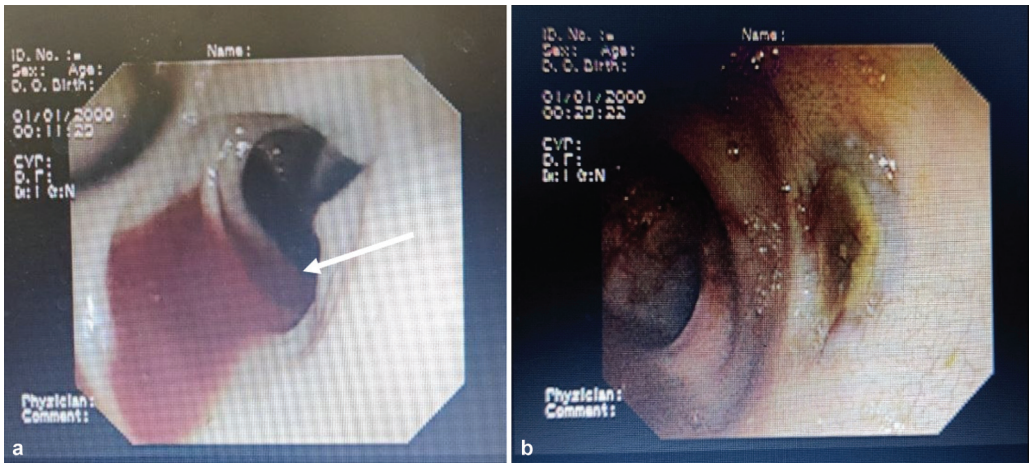

Fig. 1 (**a**) Fresh blood seen around a nipple (*arrow*) over the ectopic varix in the ascending colon. (**b**) Injected site demonstrating hemostasis after cyanoacrylate injection.

the main portal vein and nonvisualization of the intrahepatic portal vein branches (►**Fig. 2c**). Later that night, the patient had severe bleeding again and was immediately taken up for endovascular embolization of the abnormal shunt.

Due to its unusual opening in the right renal vein, distant location of the bleeding point from renal vein access, its tortuous course, and the ongoing bleeding, retrograde access of the shunt from renal vein was not chosen. A transhepatic or transjugular access was also not possible in view of chronic occlusion of the main portal vein and nonvisualization of its intrahepatic branches. Thus, the trans-splenic access was taken by direct percutaneous puncture of an intraparenchymal branch of the splenic vein. A venogram obtained showed the dilated tortuous shunt arising from the SMV and draining into the right renal vein. The shunt was embolized by deploying few coils followed by injecting a mixture of cyanoacrylate glue and Lipiodol (1:1 ratio). Repeat venogram showed closure of the main shunt, but few new afferent veins not seen previously draining into the varix were observed. These were also embolized using coils and glue/Lipiodol mixture. Check mesenteric venogram showed complete obliteration of the varix and filling of few thin perigastric/periesophageal varices (►**Fig. 3**).

Hemostasis was achieved by glue embolization of the splenic parenchymal track. Postprocedure, the hemodynamic parameters improved rapidly, but the patient developed

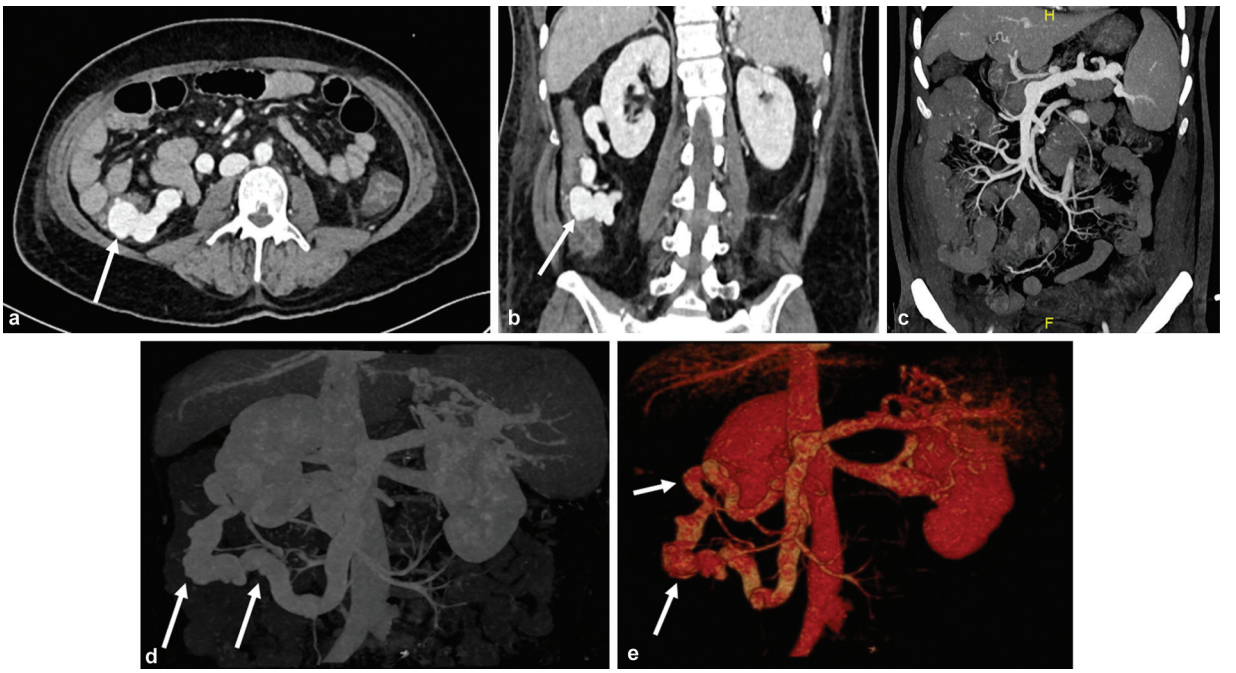

Fig. 2 (**a**) Axial and (**b**) coronal computed tomography (CT) of the abdomen showing the abnormal shunt protruding into the lumen of the ascending colonic (*arrow*). (**c**) Nonopacification of the portal vein and its branches. Note the round contour of the (**d**) superior mesenteric vein (SMV) and splenic vein confluence and (**e**) showing the entire shunt arising from SMV and joining right renal vein, on a 3D reconstructed CT angiography (*arrows*).

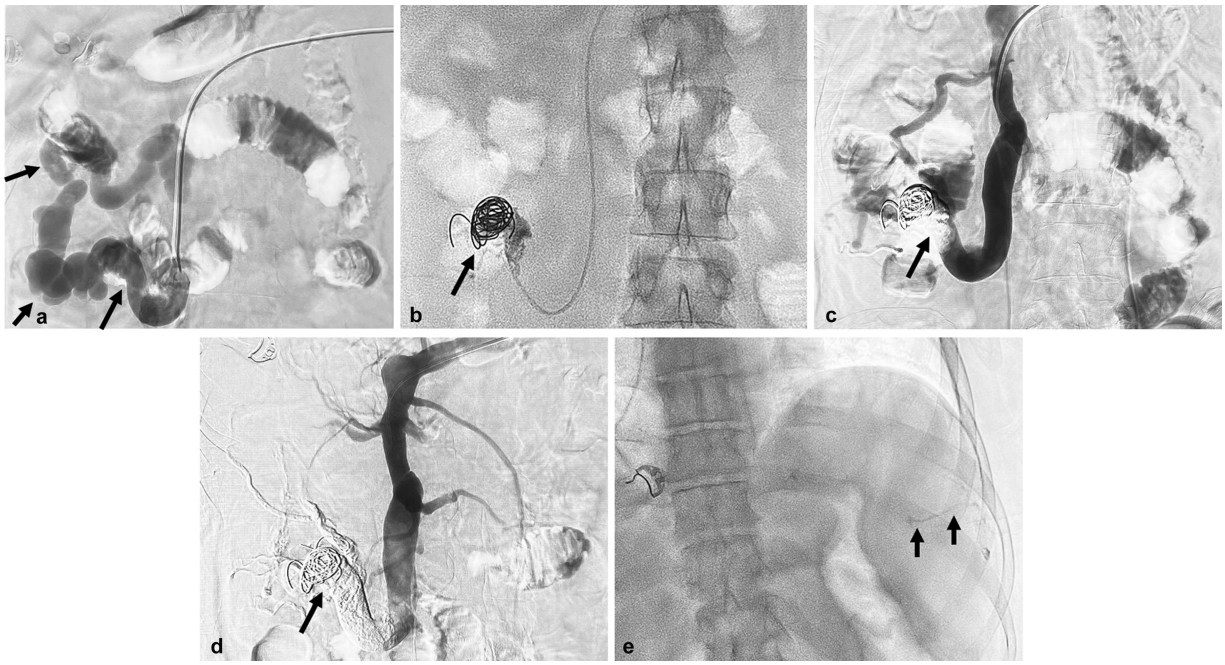

Fig. 3 Varix embolization. (**a**) Percutaneous trans-splenic access obtained and venogram shows dilated tortuous shunt arising from the superior mesenteric vein (SMV; *arrow*) and draining into the right renal vein. (**b, c**) The shunt was embolized by deploying multiple coils followed by injecting a mixture of glue and Lipiodol (*arrows*). (**d**) Postembolization angiography showing complete cessation of flow in the abnormal shunt. (**e**) Glue cast in the splenic parenchymal track.

acute kidney injury and decompensation of liver function with development of significant ascites, right pleural effusion, and mild hepatic encephalopathy. He was managed conservatively and gradually his condition improved. He was discharged after 15 days in a stable condition with the advice that he will need to undergo liver transplantation. Five months after this episode, he underwent a living donor liver transplant and is doing well 11 months after the transplant. His laboratory parameters at the time of admission and discharge are shown in ►**Table 1**.

Discussion

Colonic varices are a rare cause of lower gastrointestinal bleeding with an incidence of 0.07%. However, in patients with cirrhosis of the liver approximately 1 to 8% lower gastrointestinal bleeding is caused by colonic variceal bleeding.[4]

The principal method for the diagnosis of colon varices is colonoscopy; however, with massive bleeding where the varices may be obscured by blood, contrast CT and MRI are alternative diagnostic tools.[5]

Table 1 Lab parameters at the time of admission and discharge

Test	On admission	On discharge
Hb	5.3 g/dL	8.3 g/dL
TLC	11,000/mm^3	4,000/mm^3
Platelets	150,000/mm^3	15,0000/mm^3
Bilirubin	2.1 mg/dL	1.7 mg/dL
Direct B	0.56 mg/dL	0.5 mg/dL
INR	2.01	1.56
AST	33 U/L	36 U/L
ALT	27 U/L	29 U/L
ALP	57 U/L	60 U/L
Albumin	1.7 g/dL	2.2 g/dL
Creatinine	0.9 mg/dL	0.8 mg/dL
Sodium	131 mmol/L	134 mmol/L

Abbreviations: ALP, alkaline phosphatase; ALT, alanine aminotransferase; AST, aspartate aminotransferase; Hb, hemoglobin; INR, international normalized ratio; TLC, total leukocyte count.

There have been some reports of massive lower gastrointestinal bleeding caused by colonic varices treated by surgery.[2] However, the presence of cirrhosis in this patient precluded surgical options and endoscopic or interventional radiological therapies were preferred. On the basis of the distribution of the varices, the procedure chosen could be endoscopic variceal ligation, sclerotherapy or cyanoacrylate injection, transjugular intrahepatic portosystemic shunt (TIPS), balloon-occluded retrograde transvenous obliteration (BRTO), or coil embolization.[6]

There are multiple reports of antegrade embolization of the colonic varix (via transhepatic or trans-TIPS access).[2] However, this is the first reported case of antegrade embolization of the colonic varix via the trans-splenic access. Splenic access is usually considered to be riskier due to bleeding complications. We used glue embolization of the parenchymal track to minimize this risk.

In general, embolization of portosystemic shunts is associated with increase in the portal pressure and thus portal hypertension–related complications. These include ascites, varices in different locations, and rarely mesenteric venous thrombosis. In our case, the patient developed severe ascites, which was managed conservatively. No other major complication was noted.

In conclusion, we demonstrated an effective management of a massively bleeding colonic varix by antegrade embolization of the shunt via the splenic route by timely radiological intervention. Critical thinking and a multidisciplinary team approach helped us achieve a successful outcome in this case.

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Conflict of Interest
None declared.

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