

Colonic Varices as an Unsettling Cause of Lower Gastrointestinal Bleeding

Jayasree Ravilla¹, Du Doantrang¹

1. Internal Medicine, Monmouth Medical Center, Long Branch, USA

Corresponding author: Jayasree Ravilla, jayashreeravilla@gmail.com

Review began 05/02/2024

Review ended 05/11/2024

Published 05/17/2024

© Copyright 2024

Ravilla et al. This is an open access article distributed under the terms of the Creative Commons Attribution License CC-BY 4.0., which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

Abstract

Colonic varicose veins are very rare and are usually discovered incidentally during colonoscopy or when complications occur, such as lower gastrointestinal (GI) bleeding. The primary cause of colonic varices is usually portal hypertension secondary to liver disease or very rarely due to pancreatic disease (e.g., pancreatic adenocarcinoma). Varicose veins secondary to cirrhosis are often seen in the upper GI tract but rarely in the lower GI tract. Here, we report a 54-year-old woman who presented with colonic varices due to decompensated alcoholic cirrhosis. The main intention of this case report was to raise awareness of the possibility of developing colonic varices from liver cirrhosis and to promptly identify and manage its side effects due to the major complication which is lower GI bleeding.

Categories: Gastroenterology, Internal Medicine, Radiology

Keywords: tips, cirrhosis, gastrointestinal bleeding, varices, colon

Introduction

Colonic varices are a rare cause of gastrointestinal (GI) bleeding. Colon varices are usually caused by liver cirrhosis and lesser etiological causes include congestive heart failure, splenic vein and mesenteric vein thrombosis, and rarely due to idiopathic factors. We present a fatal case of colonic varices secondary to liver cirrhosis [1-3]. Dilated, circuitous portosystemic collateral veins that are situated outside of the gastro-esophageal region are referred to as 'ectopic varices'. It is estimated that only 3.4 % of the patients who develop portal hypertension secondary to liver cirrhosis develop colonic varices [4,5]. In this report, we present an unfortunate case of fatal colonic varices secondary to alcoholic liver cirrhosis.

Case Presentation

A 54-year-old woman with a history of alcoholic cirrhosis (graded child-pugh class C with a score of 15) and grade III esophageal varices (revised Japanese classification) presented to the emergency department complaining of bright red blood per rectum for one day. Additionally, she reported symptoms of dizziness and fatigue. She had a history of chronic alcoholism that spans nearly 30 years. She denied any hematemesis, any melena, change in her stool, recent antibiotic use, recent travel history, weight changes, and any illicit or recreational drug use. Her home medications included Lasix 40 mg and spironolactone 100 mg orally daily for ascites, which she complies without any side effects. She was worked up for liver transplant but was deferred transplantation due to the elevated phosphatidylethanol (Peth) value at > 200 ng/ml a week prior to admission.

Her initial vitals were stable and labs revealed a drop in her hemoglobin (Hb) from 11 g/dl (baseline) to 9 g/dl. Physical examination revealed a distended abdomen with signs of ascites. On physical examination, she had no signs of encephalopathy, but orthostatic vital signs were positive. A comprehensive metabolic panel (CMP) showed an albumin level of 2.8 g/dL. Esophagogastroduodenoscopy was performed which revealed grade III esophageal varices, grade A esophagitis (Los Angeles Classification), portal hypertensive gastropathy, and normal duodenum. However, flexible colonoscopy was deferred due to poor prep and large blood of blood in the rectum and was scheduled for a later date (Figure 1).

How to cite this article

Ravilla J, Doantrang D (May 17, 2024) Colonic Varices as an Unsettling Cause of Lower Gastrointestinal Bleeding. Cureus 16(5): e60490. DOI 10.7759/cureus.60490

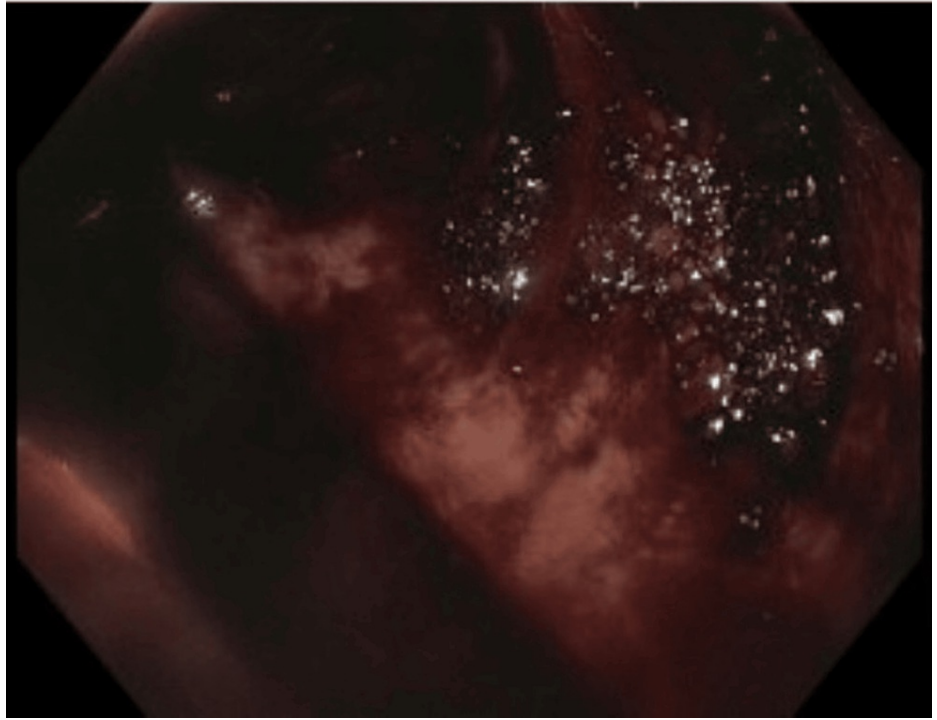


FIGURE 1: Colonoscopy image

Emergent colonoscopy showing poor bowel preparation and colonic lumen obscured with blood.

She was transferred to the Intensive Care Unit (ICU) due to persistent orthostatic vitals and for closer hemodynamic monitoring (Table [1](#)).

Labs	Hour 0	Hour 6
White blood cell (4,000–11,000 cells/mm ³)	4530 cells/mm ³	9000 cells/mm ³
Hemoglobin (11–15.1 g/dL)	10.8 g/dL	6.1 g/dL
Hematocrit (33.1%–44.5%)	30%	21.2%
Platelet (150,000–400,000 cells/mm ³)	200,000 cells/mm ³	190,000 cells/mm ³
Alkaline phosphatase (32–91 U/L)	40 U/L	80U/L
Aspartate aminotransferase (15–41 U/L)	20 U/L	22 U/L
Alanine aminotransferase (7–52 U/L)	45 U/L	40 U/L
Total bilirubin (0.3–1 mg/dL)	1 mg/dL	0.9 mg/dL
International normalized ratio (0.9–1.1)	1.5	1.9
Blood urea nitrogen (6–20 mg/dL)	29 mg/dL	34 mg/dL
Creatinine (0.44–1.03 mg/dL)	1 mg/dL	1.03 mg/dL
Serum albumin (3.5–5.1 g/dL)	3.2 g/dL	3 g/dL
Urine drug screen	negative	
Blood alcohol (<10 mg/dL)	normal	

TABLE 1: Lab values of the patient

Basic labs pertinent to the case.

A computed tomography (CT) scan with intravenous contrast of the abdomen and pelvis revealed liver cirrhosis, patent portal vein, splenic artery aneurysm, and multiple varices of the right colon, which were new findings compared with the abdominal scan she had performed a year earlier. It did not show any active extravasation. On the same day, within six hours of presentation to the emergency department, the patient developed hemodynamic instability with hypotension due to massive lower GI bleeding. Vital signs revealed a blood pressure of 60/50 mm Hg. The massive blood transfusion protocol was initiated and she required multiple blood transfusions, but subsequent CT enterography showed colonic varices but no extravasation (Figure 2).

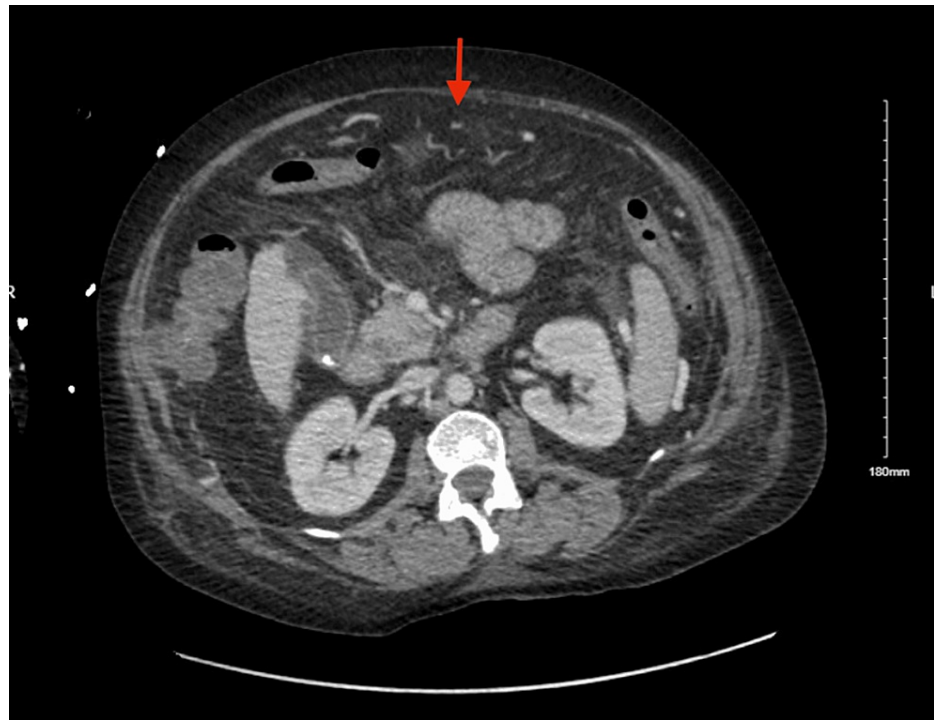


FIGURE 2: CT enterography

There was active extravasation of blood in the right colon.

Interventional radiology was consulted due to emergent situation and CT mesenteric angiography showed pancolonic varices but no active bleeding site was identified. As no active source of bleeding was identified, a transjugular intrahepatic portosystemic shunt (TIPS) was planned. During the procedure, a right colonic variceal connection was discovered near the superior mesenteric vein and successfully embolized with a coil (Figure 3).



FIGURE 3: IR-guided superior mesenteric vein coiling

Although the portal system was patented, the TIPS could not be created due to technical difficulties in accessing the system via the right hepatic vein. She became very hemodynamically unstable with blood pressure (50/32 mm Hg) not responding to blood transfusions and vasopressors. Unfortunately, she suffered cardiac arrest due to hypovolemic shock and died within a day of arrival.

Discussion

GI varices of the distal colon which can present as isolated colonic varices have not been fully explored. In patients with cirrhosis, upper GI varices, i.e., esophageal varices are seen in 50 % of the cases. The presence of upper GI varices in the esophagus or stomach appears to have nil effect on the occurrence of these lesions [6,7]. The risk of hemorrhage significantly increases with the prevalence of colonic varices at a rate of 12 % annually [6,7]. Though the common etiology is primarily portal hypertension, it can occur in idiopathic cases. GI bleeding ranging from mild to fatal cases were found in the literature [8]. Here, we focus on this case of pancolonic varices presenting with life-threatening rectal bleeding.

Varices in the GI tract arise from portal hypertension secondary to increased intrahepatic vascular resistance and elevated blood flow through the portal and collateral venous systems [4]. The sites commonly involved include jejunum or ileum (18%), duodenum (17%), rectum (8%), and peritoneum (9%). Rectal and cecal varices are commonly seen during colonoscopy in the lower GI tract but isolated colonic varices are extremely rare [9]. The pathophysiology of cirrhosis includes the advancement of portal hypertension leading to collateral vessel formation and arterial vasodilation resulting in significantly elevated blood flow in portal circulation. This event creates a hyperdynamic circulatory state leading to the formation of varices and although esophageal varices are the common variant, it can also lead to ectopic lesions like pancolonic varices. It is found that nitric oxide (NO) plays a vital role in increased vasodilation thus leading to portosystemic collateral vessels in response to angiogenesis [9,10].

The majority of patients are asymptomatic during the presentation but few can be present with detrimental lower GI bleeding. CT angiography or mesenteric angiography is the most accurate diagnostic tool, with

identification of varices during the venous phase [4]. Colonoscopy is the diagnostic option, although the findings are discovered incidentally during the procedure. During the endoscopy, colonic varices are identified by dilated, tortuous vascular tracts with a bluish tinge on the mucosal surface. Colonoscopy also helps to know the extent of the disease, thus helping the treatment course [10,11]. Our patient was diagnosed appropriately with colonic varices through CT enterography and colonoscopy with appropriate diagnostic modules.

In emergency situations, some successful treatment options include TIPS, balloon retrograde transvenous occlusion (BRTO), and venous coil embolization. Usual interventions like banding of varices and sclerotherapy are used when varices are limited to the rectum, but they are generally avoided in proximal involvement due to the risk of perforation. Alternatively, beta-blockers, which are commonly used for medical management of esophageal varices, can be used but most of the colonic varices are discovered with the complications of active GI bleeding [12]. In many cases, TIPS proved to be an effective way to stabilize patients with active bleeding. In patients with failed TIPS, venous coil embolization of colonic varices with tissue propylene injection can be performed. Complications of this procedure like vessel perforation, migration of coil, necrosis, and embolization of the non-target lesion should be considered [3,4]. In very few documented cases, BRTO, an endovascular procedure, is performed with some success. Subtotal colectomy has also been reported as the treatment option but regrettably due to the rarity of cases, there are no established treatment guidelines for management of colonic varices [13].

Conclusions

The prevalence of colonic varices is difficult to establish because this condition remains subclinical for most of the time. Unfortunately, due to the rarity of colonic varices, there are currently no adequate treatment guidelines. Although surgical intervention is the next treatment choice after IR intervention and banding through colonoscopy, it is often challenging due to the coagulopathy caused by cirrhosis. The aim of the treatment is to decrease portal hypertension, which is the primary cause of bleeding colonic varices. Delays in diagnosis and treatment can lead to serious consequences, including death.

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: Jayasree Ravilla, Du Doantrang

Acquisition, analysis, or interpretation of data: Jayasree Ravilla, Du Doantrang

Drafting of the manuscript: Jayasree Ravilla, Du Doantrang

Supervision: Jayasree Ravilla, Du Doantrang

Critical review of the manuscript for important intellectual content: Du Doantrang

Disclosures

Human subjects: Consent was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

References

1. Abdalla A, Veeramachaneni H, Smith K, Sharma S, Patel V: Isolated colonic varices in pancreatic adenocarcinoma. *ACG Case Rep J*. 2023, 10:e01184. [10.14309/crj.0000000000001184](https://doi.org/10.14309/crj.0000000000001184)
2. Sohn W, Lee HL, Lee KN: Variceal hemorrhage of ascending colon. *Clin Gastroenterol Hepatol*. 2012, 10:24.
3. Ko BS, Kim WT, Chang SS, et al.: A case of ascending colon variceal bleeding treated with venous coil embolization. *World J Gastroenterol*. 2013, 19:311-5. [10.3748/wjg.v19.i2.311](https://doi.org/10.3748/wjg.v19.i2.311)
4. Reddy S, Malik P, Gurell M: Colonic varices: an under-recognized but potentially deadly complication of cirrhosis. *Am J Gastroenterol*. 2018, 113:S1150.
5. Sunkara T, Caughey ME, Culliford A, Gaduputi V: Idiopathic isolated colonic varices: an extremely rare condition. *J Clin Med Res*. 2018, 10:63-5. [10.14740/jocmr3230w](https://doi.org/10.14740/jocmr3230w)
6. Ganguly S, Sarin SK, Bhatia V, Lahoti D: The prevalence and spectrum of colonic lesions in patients with cirrhotic and noncirrhotic portal hypertension. *Hepatology*. 1995, 21:1226-31.
7. Aliya FG, Patrick SK: The epidemiology and pathogenesis of gastrointestinal varices. *Tech Gastrointest*

- Endosc. 2017, 19:62-8. [10.1016/j.tgie.2017.03.005](https://doi.org/10.1016/j.tgie.2017.03.005)
8. Cervantes Millán G, Tejada Saenz RM, López Acosta ME, Perez Aguirre J: Lower gastrointestinal bleeding associated with variceal hemorrhoidal disease in a patient with pan-colonic varices. *Cureus*. 2023, 15:e42013. [10.7759/cureus.42013](https://doi.org/10.7759/cureus.42013)
 9. Sato T, Akaike J, Toyota J, Karino Y, Ohmura T: Clinicopathological features and treatment of ectopic varices with portal hypertension. *Int J Hepatol*. 2011, 2011:960720. [10.4061/2011/960720](https://doi.org/10.4061/2011/960720)
 10. Iwakiri Y: Pathophysiology of portal hypertension. *Clin Liver Dis*. 2014, 18:281-91. [10.1016%2Fj.cld.2013.12.001](https://doi.org/10.1016%2Fj.cld.2013.12.001)
 11. Dina I, Braticevici CF: Idiopathic colonic varices: case report and review of literature. *Hepat Mon*. 2014, 14:e18916. [10.5812%2Fhepatmon.18916](https://doi.org/10.5812%2Fhepatmon.18916)
 12. Gallagher J, Quach B, Sempokuya T, Sivaraman A: A unique presentation of familial idiopathic colonic varices. *ACG Case Rep J*. 2023, 10:e01185. [10.14309%2Fcrj.0000000000001185](https://doi.org/10.14309%2Fcrj.0000000000001185)
 13. Liu C, Srinivasan S, Babu SB, Chung R: Balloon-occluded retrograde transvenous obliteration of colonic varices: a case report. *CVIR Endovascular*. 2020, 3:17. [10.1186%2Fs42155-020-00108-3](https://doi.org/10.1186%2Fs42155-020-00108-3)