Colorectal Variceal Bleeding Managed by Endoscopic Therapy in Patients with Cirrhosis

KK Rawal

Abstract
Ectopic varices are an unusual cause of gastrointestinal bleeding accounting for up to 5% of all variceal bleeds. Colorectal variceal bleeding is even rarer. As the bleed is massive and has a poor prognosis, one must be aware of this condition in a patient of portal hypertension with life-threatening lower gastrointestinal bleeding. Two cases of colorectal variceal bleeding in cirrhotic patients treated successfully by endoscopic therapy are reported here.

Introduction
Significant varices can develop in the stomach, small bowel, colon, rectum, biliary tree and surgical ostomy sites. The most common sites for colorectal varices are the cecum and rectum. Due to paucity of data there are no guidelines to manage bleeding in these patients. Two cases of colorectal varices (one each of cecum and rectum) in patients with cirrhosis are described. They presented with massive lower gastrointestinal bleeding which was controlled by endoscopic therapy.

Case Reports

Case 1
A 42-year-old male presented in profound shock with history of passing large amount of fresh blood in the stool. His history was significant for alcoholism. Patient had history of hematemesis for which multiple sessions of band ligation for esophageal varices were done. Patient was diagnosed to have piles for occasional bleed per rectum in the past. His physical examination revealed icterus, pedal edema and moderate ascites. His blood pressure was 90/60 mm Hg and pulse rate was 124/min. His hemoglobin was 4.9 g/dl, platelet count 75,000/mm³ and MELD score was 14. Patient received 6 units of packed red blood cells during resuscitation. Upper GI scopy was negative for any active bleeding focus. Emergency colonoscopy without preparation showed fresh blood with clots from rectum to cecum. An urgent CT angiography demonstrated large vascular channels along the cecum extending up to superior mesenteric vein (Figure 1). Repeat colonoscopy after preparation revealed large varices on the lateral wall of cecum (Figure 2a). In absence of: facilities for transjugular intrahepatic portosystemic shunt (TIPS), negative consent for surgery and risk of vessels communicating directly to inferior vena cava, 1.5 ml glue (N-butyl-2-cyanoacrylate) was injected into the cecal varices and bleeding was controlled (Figure 2b). Patient is now being evaluated for liver transplantation.

Case 2
An 81-year-old female presented with history of passing fresh blood in stool in large quantity. She had history of hematemesis in the past from esophageal varices which was managed by repeated band ligation. She was diagnosed as a case of cryptogenic cirrhosis. Her blood pressure was 96/64 mm Hg and pulse rate was 104/min. Her Hb was 8.5 gm/dl and platelet count 47,000/mm³. Her MELD score was 10. She received 2 units of packed red blood cells. Upper GI endoscopy did not show any active bleeding. Emergency colonoscopy was notable for the presence of three tortuous tumor-like vascular structures located between 4 to 10 cm from anal verge (Figure 3a). A red plug sign was found on one of the varices (Figure 3b). Varices were ligated by applying 6 rubber bands. On third day of admission patient suffered severe cerebro-vascular ischemic stroke.
Discussion

Ectopic varices are large portosystemic venous collaterals found at sites in the abdomen except gastrointestinal region. They account for 5% of all variceal bleeding and have a poor prognosis. The frequency of bleeding as reported is 17% in the duodenum, 17% in the jejunum or ileum, 14% in the colon, 8% in the rectum and 9% in the peritoneum. 30% of all variceal bleeds may be extra-sophageal in origin.

Colorectal variceal bleeding is uncommon but life-threatening. They are usually found in portal hypertension but are also associated with biliary atresia, biliary sclerosis, mesenteric vein thrombosis, congestive heart failure and idiopathic factors. Portal hypertension was the cause of ectopic varices in both the present cases. Cecal varices are rare. Out of total 18 cases of cecal varices reported till date all presented with massive lower gastrointestinal bleeding expect one. This is the first reported case of cecal variceal bleeding treated by endoscopic glue injection. The frequency of rectal varices varies from 28 to 44% in patients with cirrhosis.

Rectal varices are differentiated from hemorrhoids by their location above dentate line and collapsible nature. Prevalence of hemorrhoids is same in the patients with or without liver diseases.

The literature on this subject comprises of few case series and case reports without any randomized trials. Colonoscopy should be the principal method for diagnosis of colorectal variceal bleeding. Care should be taken not to over distend the lumen with air which could cause collapse of varices in the setting of hypotension due to massive bleeding. Endoscopic ultrasound is better than endoscopy to identify colorectal varices. CT angiography is useful as a minimal invasive tool to diagnose these varices in short time. In case 1 it detected the varices early in the course. Color Doppler imaging can detect the direct communication between the varices and inferior vena cava, hence useful to minimize the risk if employed prior to glue injection or embolization of varices.

Vaso-active drugs and beta-blockers have been used in colorectal variceal bleeding. Endoscopic varical ligation and sclerotherapy can be used alone or in combination for rectal variceal bleeding. Endoscopic glue injection or embolization can be safe and effective if vessels communicating between varices and inferior vena cava were avoided as in case 1. But these are not the definitive treatment because no decompression of portal venous system occurs. TIPS with or without embolization has been used as a primary intervention or salvage modality in many cases. Other treatment options for colorectal variceal bleeding include portacaval shunts, colonic resection and balloon-occluded retrograde transvenous obliteration. It is still controversial whether obliteration of esophageal varices increases subsequent ectopic varices formation and bleeding. However both the present cases bled from ectopic varices after the obliteration of esophageal varices.

In conclusion, diagnosis and treatment of ectopic variceal bleeding may be difficult. A normal initial upper GI endoscopy in a cirrhotic patient with massive hematochezia should alert the clinician for an extra esophageal site of variceal bleeding. These patients need further evaluation. In both the present cases upper GI endoscopy did not reveal any source of bleeding. CT angiography showed cecal varices in case 1, which was subsequently confirmed by colonoscopy. In the absence of guidelines for management of colorectal variceal bleeding, the treatment should be governed by available local expertise and situation.

References