A Therapeutic Approach to Cecal Varices

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ABSTRACT

Ectopic varices account for 1%–5% of all variceal bleeding episodes. The most common presentation of cecal varices is an acute episode of a massive lower gastrointestinal hemorrhage. However, cecal varices can be found incidentally and can be silent for a prolonged period of time before presenting with a massive gastrointestinal hemorrhage. Through this case of a 63-year-old woman, we would like to highlight the paucity of literature in the treatment of nonbleeding cecal varices.

INTRODUCTION

Ectopic varices account for 1%–5% of all variceal bleeding episodes.¹ Fewer than 100 cases of colonic varices have been reported so far, and cecal varices are even rarer among them. The most common presentation of cecal varices is an acute episode of a massive lower gastrointestinal hemorrhage.² We present a 63-year-old woman with no significant medical history who presented with abdominal pain and chronic diarrhea. Isolated cecal varices were found on colonoscopy with no evidence of bleeding.

CASE REPORT

A 63-year-old woman with no significant medical history presented with intermittent abdominal pain over the past 1 year. The patient also reported chronic diarrhea with 10–15 bowel movements a day intermittently for many years. She had a cholecystectomy more than 30 years ago, and her diarrhea improves when she is compliant with cholestyramine. The patient stated that her diarrhea further improved after making changes to her diet (avoiding red meat, avoiding greasy food). The patient denied nausea, vomiting, fever/chills, and blood in bowel movements. Family medical history was negative for inflammatory bowel disease and colon carcinoma.

On examination, the patient appeared distressed in abdominal pain. She was oriented to time, place, and person and was hemodynamically stable. Abdominal, cardiovascular, and respiratory system examination were unremarkable. Laboratory findings showed a hemoglobin of 12.2 g/dL with a hematocrit of 35.9%, aspartate transaminase level of 23 units/L, alanine aminotransferase level of 28 units/L, alkaline phosphatase level of 75 units/L, and total bilirubin of 0.2 mg/dL. Abdominal computed tomography showed luminal narrowing of the cecum suggestive of a cecal mass and inflammatory changes with desmoplastic reaction anterior to inferior vena cava indicative of a carcinoid tumor (Figure 1). 5-hydroxyindoleacetic acid and chromogranin were within the normal limits at 7.6 mg/d and 3 nmol/L, respectively.

Colonoscopy with good bowel preparation showed large nonbleeding cecal and ascending colon varices and numerous polyps in the ascending and transverse colon (Figure 2). The polyps were biopsied, and the pathology was reported as benign tubular adenomas. Upper gastrointestinal endoscopy showed grade I varices in the lower esophagus. Abdominal and thoracic computed tomography (CT) with and without contrast sought as a part of malignancy workup was negative.

Because portal hypertension is a known cause of cecal varices, the FibroSure score was sought, which was F0 indicative of the absence of cirrhosis. Abdominal magnetic resonance showed patent hepatic and portal veins and hepatic artery. The patient was...
recommended to eliminate lactose from her diet in addition to other changes she has already made as a trial to improve her diarrhea. She was also counseled on compliance with cholestyramine. The patient was followed in an outpatient gastroenterology clinic, and her diarrhea and abdominal pain resolved, the symptoms likely being secondary to non-compliance with cholestyramine and bile acid diarrhea. Because there is no treatment algorithm for the treatment of nonbleeding cecal varices, it was decided to monitor the patient with a close outpatient follow-up.

DISCUSSION

Cecal varices are most commonly caused by portal hypertension. Some of the other causes are biliary atresia, biliary sclerosis, portal vein thrombosis, and familial. Cecal varices in our patient did not have a causal factor that made the approach to treatment challenging. On review of literature, we found no clear guidelines in the management of non-bleeding cecal varices. Cecal varices can be silent for a significant period of time without hemorrhage and have also shown to cause life-threatening gastrointestinal hemorrhage.

Cecal varices can be diagnosed with panendoscopy, barium enema, nuclear scintigraphy, CT angiography, enteroscopy, endoscopic ultrasound, wireless capsule endoscopy, diagnostic angiography, color Doppler flow imaging among others. Most clinicians prefer endoscopy as the preferred diagnostic tool for the identification of cecal varices, but over insufflation or hypotension associated with acute hemorrhage can cause the collapse of the varices and underdiagnosis of the varices. Blood during an acute hemorrhage can also obscure visualization during colonoscopy, hindering the identification of the cause of bleeding. CT angiography, along with endoscopy with adjuvant use of endoscopic ultrasound, has shown to be sensitive in the identification of cecal varices.

Endoscopic therapies are limited for the treatment of cecal varices, and band ligation is not considered a safe procedure. Injection sclerotherapy and embolization are considered as bridging to definitive therapy of portal hypertension because collaterals or progression of cecal varices is likely to develop. Liver transplant and TIPS are the treatment of choice when portal hypertension is causing cecal varices. Some cases reports have reported total or partial colectomy in bleeding cecal varices when the bleed cannot be managed conservatively. The use of vasoactive substances like octreotide, terlipressin, nitrates, and β-blockers has been tried but not studied.

There is a paucity of literature on the treatment of both bleeding and nonbleeding cecal varices except for a few case reports. Prospective and retrospective studies to formulate a treatment plan for nonbleeding cecal varices are needed because the most common presentation is life-threatening gastrointestinal hemorrhage.
DISCLOSURES

Author contributions: P. Kudaravalli wrote the manuscript and reviewed the literature. SA Saleem wrote the manuscript, revised the manuscript, and is the article guarantor. VS Pendela revised the manuscript. D. Manocha wrote and approved the final version.

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