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Vesical Varices: An Unusual Cause of Hematuria

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Abstract

Portal hypertension is a common complication of cirrhosis and may lead to the formation of venous collateral channels. Usually, varices due to portal hypertension develop in the lower esophagus, stomach, rectum, or other parts of the digestive tract. Extraintestinal ectopic varices are rare. Here, we present a patient with ectopic urinary bladder varices, who presented with hematuria and symptomatic anemia. The bleeding was managed with minimally invasive endovascular approach.

Keywords
- bladder
- ectopic
- schistosomiasis
- albumin
- cirrhosis
- glue

Introduction

Formation of portosystemic shunts and varices is a frequent consequence of cirrhosis and portal hypertension.[1] Usually, varices due to portal hypertension develop in the lower esophagus, stomach, rectum, or other parts of the digestive tract.[1,2] Vesical varices are an uncommon type of extra-intestinal ectopic varices, which are usually seen in patients who have undergone prior abdominal surgery or endoscopic interventions that alter the dynamics of the portal system.[1–3] Bleeding from these varices is potentially a life-threatening complication which often needs to be tackled with a multidisciplinary approach. Here, we present a case of bleeding from vesical varices which was managed with minimally invasive percutaneous variceal obliteration and placement of transjugular intrahepatic portosystemic shunt (TIPS).

Case

A 52-year-old gentleman was admitted with multiple episodes of painless gross hematuria requiring three units of packed red blood cells transfusion. There was no history of anorexia, fever, weight loss, or any surgery in the past. Physical examination was unremarkable except for pallor. Personal history was suggestive of significant alcohol use.

Laboratory tests revealed a low hemoglobin level of 6.2 g/dL (normal range: 11.0–14 g/dL) with normal leukocyte and platelet counts. Prothrombin and partial thromboplastin times were within normal limits. Liver and kidney function tests were essentially normal except for low serum albumin (2.8 g/dL, normal range: 3.5–5.2 g/dL).

Ultrasound examination ruled out any apparent genito-urinary tract malignancy, urinary tract calculi, or prostato-

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megaly but showed tortuous vascular channels abutting the anterior wall of the urinary bladder (►**Fig. 1**). Features of chronic liver disease (CLD) and minimal ascites were also noted. Possibility of vesical varices secondary to liver cirrhosis was raised based on the imaging findings. To delineate the anatomy of these vascular channels and plan a therapy, a computed tomography scan was performed which demonstrated enlargement of the inferior mesenteric vein (IMV) leading to multiple vesical varices (►**Fig. 2**). The vesical varices were draining via a common efferent channel into the right internal iliac vein.

Serological assays done to rule out other causes of CLD were negative for viral and autoimmune markers. A final diagnosis of alcohol-related liver cirrhosis and portal hypertension with ectopic vesical varices was made. The patient had a Model for End-Stage Liver Disease score of 7 and was classified as having Child–Pugh class A status (6 points).

The patient underwent indwelling catheterization and continuous bladder irrigation to prevent bladder outlet obstruction. Symptomatic treatment including β-blockers, vasopressors, and fluid replacement was administered. Despite these measures, the patient continued to have intermittent, transfusion-dependent hematuria for 48 hours. In a multidisciplinary meeting, it was decided to embolize the varices and place a TIPS to decompress the portal system.

Under general anesthesia, the IMV was accessed via the transjugular route in the catheterization laboratory. A mixture of 4 mL of N-butyl cyanoacrylate glue (Endocryl, Samarth Life Sciences, India) and Lipiodol (Guerbet, France) in a 1:3 ratio was injected to embolize the shunt and varices

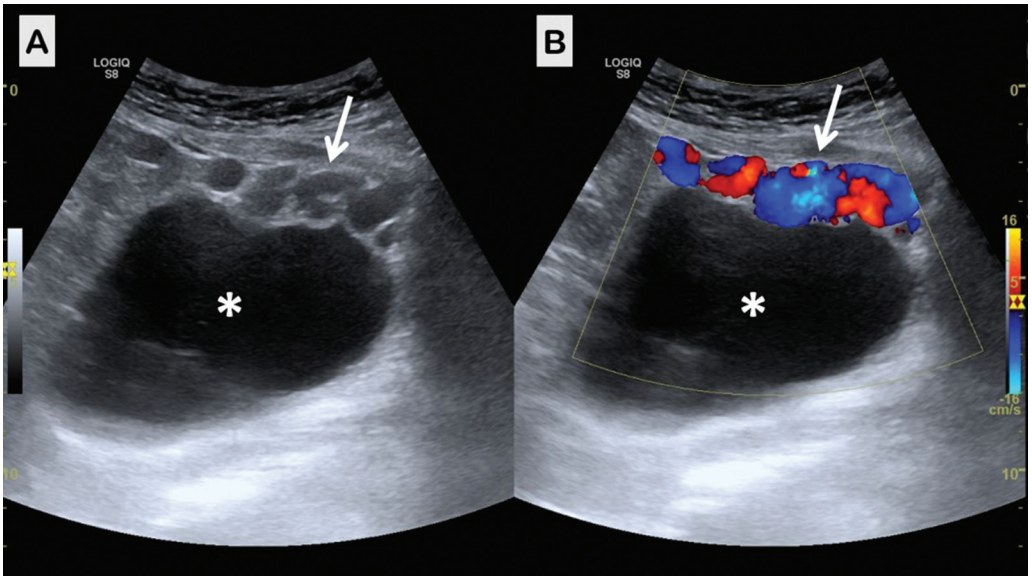

Fig. 1 Transabdominal grayscale ultrasound (**A**) and adjoining Doppler image (**B**) showing multiple vascular channels (arrows) abutting the anterior wall of the urinary bladder (asterisks).

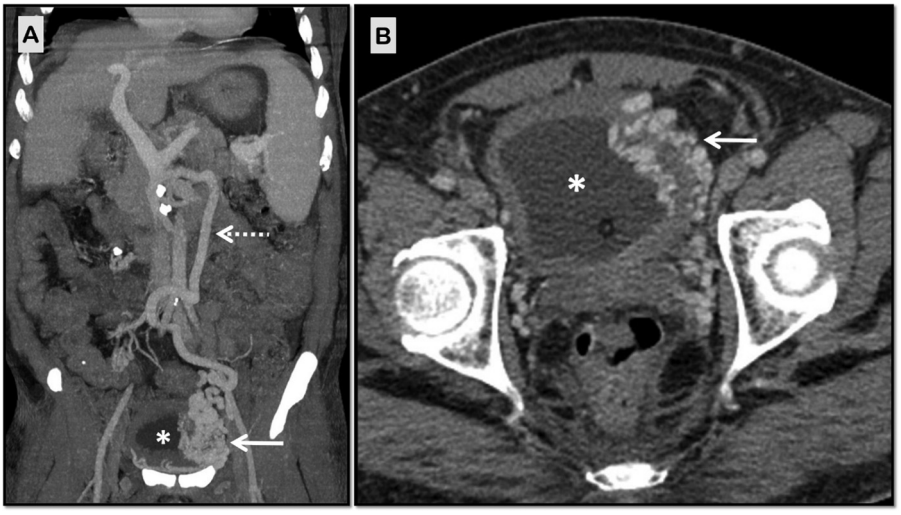

Fig. 2 Coronal maximum intensity projection (**A**) and axial (**B**) venous phase computed tomographic images of the abdomen of the patient demonstrating a dilated and tortuous inferior mesenteric vein (IMV; dashed arrow in **A**) supplying a bunch of vesical varices (solid arrows in **A** and **B**). Asterisk denotes the urinary bladder lumen.

(►**Fig. 3**). Subsequently, a 10 × 90 mm hybrid TIPS stent (Niti, Taewoong, South Korea) was placed to bridge the right hepatic and portal vein (►**Fig. 4**). The portal pressure gradient reduced from 22 mm Hg prior to the procedure to 8 mm Hg after placement of TIPS stent. Hematuria completely resolved and the patient was discharged on the fifth day following the procedure. The patient has completed 1 year of follow-up and continues to do well with no episodes of hematuria.

Discussion

Lower esophagus and stomach are the most common sites of formation of portosystemic collateral channels in patients with cirrhosis. Venous collaterals appearing elsewhere are referred to as ectopic varices.[3] Duodenum, jejunum, ileum, colon, rectum, surgical stoma, biliary tree, and peritoneum are the usual sites of development of ectopic varices.[2,3] They open up in response to increased intrahepatic vascular resistance and serve as pathways of low resistance outflow. Extraintestinal ectopic varices are rare with an incidence of less than 0.25%.[2–5] Most reported cases of extraintestinal ectopic varices had prior history of abdominal surgery or endoscopic sclerotherapy, which possibly resulted in disruption of usual splanchnic-bed collateral channels.[6] It has also been proposed that the surgical adhesions could appose abdominal structures drained by systemic veins with bowel segments drained by portal tributaries, thereby creating portosystemic collaterals at atypical locations.[3] They are often incidentally detected on imaging done for surveillance in patients with cirrhosis. Infrequently, patients can present with symptoms of bleeding from these varices.[7,8] Although ectopic varices account for only 5% of all variceal bleeding, they carry a high mortality rate of up to 40% after the initial hemorrhagic episode.[4,5] This is because unlike gastroesophageal varices, ectopic varices are true veins with larger diameters and hence they are under greater wall tension and increased risk of bleeding.

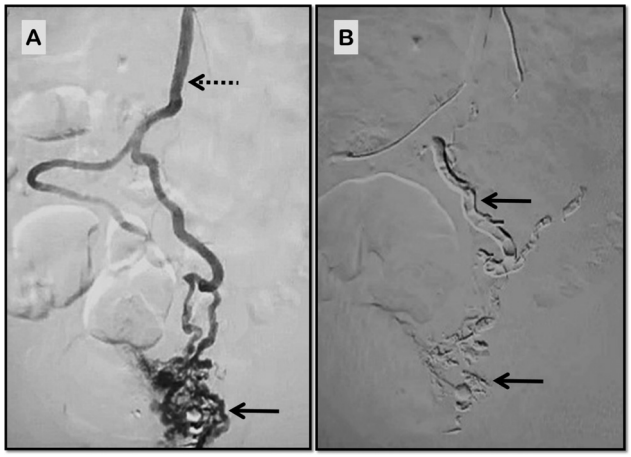

Fig. 3 Digital subtraction angiography spot images from selective cannulation of inferior mesenteric vein (IMV) demonstrating opacification of the IMV (dashed arrow in **A**) and vesical varices (solid arrow in **A**). Postembolization N-butyl cyanoacrylate (NBCA) glue cast (solid arrows in **B**) noted in the IMV and varices.

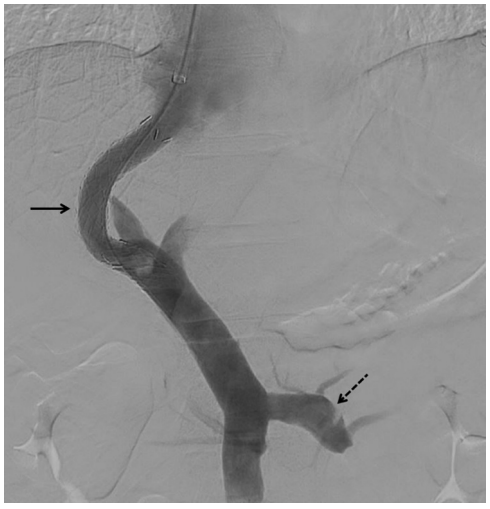

Fig. 4 Digital subtraction angiography (DSA) image after placement of transjugular intrahepatic portosystemic shunt (TIPS) stent (arrow) shows a patent stent with antegrade flow in the inferior mesenteric vein (IMV) (dashed arrow).

Hematuria is one of the most common clinical symptoms for urologists and is typically observed in urinary stone disease, benign prostatic hyperplasia, and urinary tract malignancies. However, CLD with ectopic vesical varices due to portal hypertension is an extremely rare cause of hematuria.[6,9–12] Other causes of vesical varices include schistosomiasis infection leading to portal vein thrombosis, retroperitoneal fibrosis causing narrowing of the inferior vena cava, and venous incompetency related to pregnancy.[13–15]

Currently, there are no uniformly accepted guidelines for the treatment of vesical varices. Management strategies vary depending on the clinical context and may include β-blockade, cystoscopic variceal obliteration using laser or N-butyl cyanoacrylate glue, percutaneous variceal embolization, and even surgical devascularization on rare occasions.[11,16–19] However, these treatments often provide only short-term relief as they do not address the underlying portal hypertension, leading to higher rates of rebleeding in a patient with CLD. Combining TIPS placement with variceal obliteration can potentially manage elevated portal pressures and prevent the formation of new portosystemic collaterals and recurrence of bleeding. Nevertheless, the existing evidence on this approach is limited,[20,21] underscoring the need for further prospective studies on this topic for better understanding.

Conclusion

Ectopic varices should be considered as a potential cause in patients experiencing hematuria with a history of CLD and portal hypertension. TIPS with variceal obliteration offers a minimally invasive endovascular approach to effectively manage the bleeding.

Conflict of Interest
None declared.

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