



# Percutaneous Antegrade Transvenous Variceal Obliteration for Portal Vesiculopathy-Related Gross Hematuria

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## Abstract

### Keywords

- vesical varices
- portal hypertension
- hematuria

Vesical varices secondary to portal hypertension are extremely rare cause of hematuria with only a few cases reported to date. We report a case of portal vesiculopathy in a patient of hepatitis B virus-related chronic liver disease presenting with gross hematuria, successfully treated with percutaneous antegrade transvenous obliteration using *n*-butyl cyanoacrylate glue and Amplatzer vascular plug.

## Introduction

A 46-year-old gentleman, with a known hepatitis B virus-related chronic liver disease (Child-Pugh score: A6), presented with gross hematuria, passage of clots, and dysuria for the last 2 days. The patient was on regular medical management for chronic liver disease.

Laboratory evaluation revealed an abnormal hemogram with low hemoglobin of 8.2 g/dL, international normalized ratio of 1.39, and partial thromboplastin time of 28 seconds. Urine analysis revealed numerous red blood cells in the urine without any significant pus cells.

He was initially managed with bladder catheterization and bladder irrigation. He underwent contrast-enhanced computed tomography (CECT) abdomen (Toshiba Aquilion, Minato, Tokyo, Japan, 160 slice) and CT urography to assess for urinary tract stone disease or urothelial neoplastic disease. CT abdomen showed features of cirrhosis and portal hypertension. A dilated recanalized paraumbilical vein was noted along the anterior abdominal wall from the left portal vein. Multiple dilated and tortuous venous channels were seen predominantly along the dome and lateral walls of the urinary bladder with probable afferent venous supply from

the recanalized paraumbilical vein (►Fig. 1) and drainage to the systemic venous system via bilateral internal iliac veins.

A multidisciplinary team discussion comprising a hepatologist, urologist, and interventional radiologists led to the decision for treating him with percutaneous antegrade transvenous obliteration of the vesical varices.

The patient was shifted to angiographic suite for further management.

Under aseptic precautions and monitored anesthesia care, the recanalized paraumbilical vein was accessed percutaneously, using a 5F micropuncture access kit with ultrasound guidance. A 7F vascular access sheath was placed in the paraumbilical vein. A venogram through the access sheath confirmed dilated recanalized paraumbilical vein as a tributary of the left portal vein with a hepatofugal flow pattern. Venogram also revealed rapid filling of the dilated and tortuous vesical varices and systemic drainage via internal iliac veins (►Fig. 2).

A 10 mm Amplatzer vascular plug II (AVP II, St Jude Medical, Saint Paul, Minnesota, United States) was deployed in the hepatic end of the paraumbilical vein to prevent antegrade flow from the portal vein to the varices and migration of embolic agents to the portal vein (►Fig. 3). A

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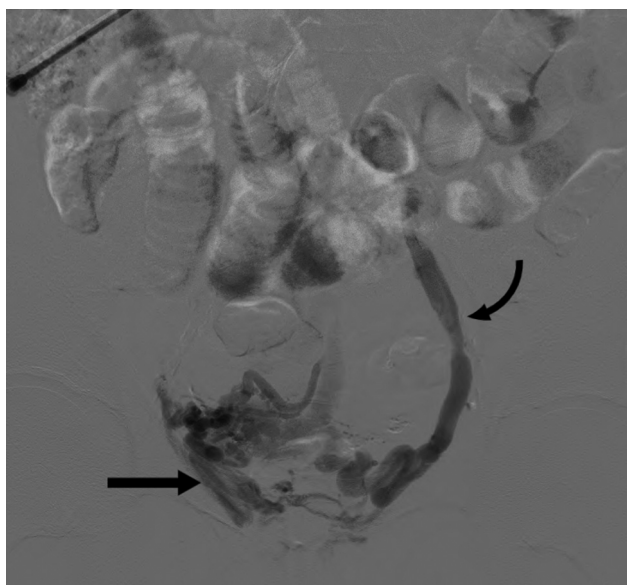
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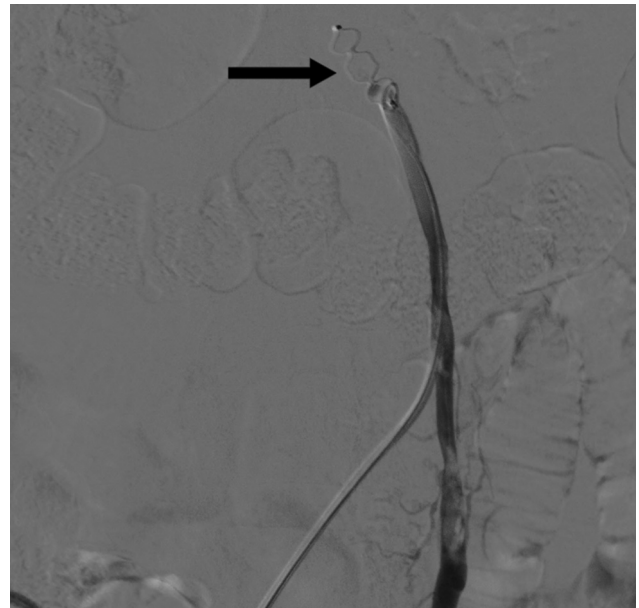


**Fig. 1** Postcontrast venous phase coronal section of the abdomen with maximum intensity projection shows recanalized paraumbilical vein from the left portal vein (curved arrow), dilated and tortuous vesical varices (black arrow). Hyperdense contents within the bladder lumen which was hyperdense in the plain computed tomography as well (not shown in figure) suggestive of blood clots (asterisk) around the hypodense inflated indwelling bladder catheter (dotted arrow).

5F K.M.P catheter (Cook Medical, Bloomington, Indiana, United States) was advanced caudally over a 0.035" hydrophilic guidewire using the "one-sheath inversion technique"

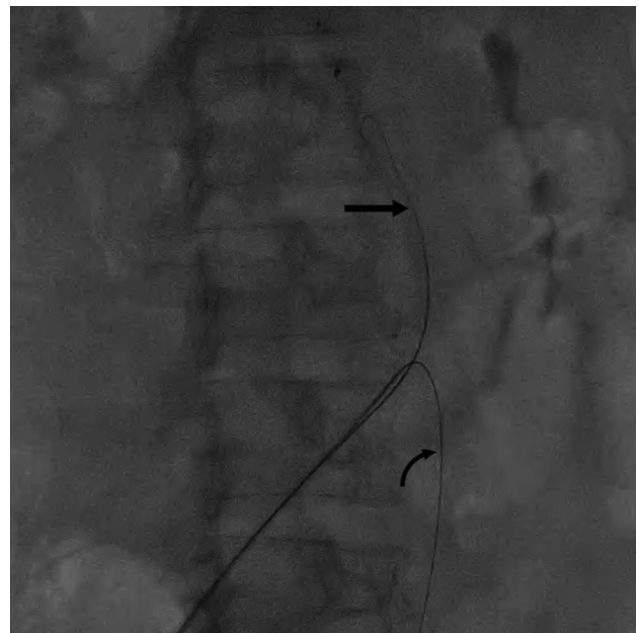


**Fig. 2** Venogram shows vesical varices seen predominantly along the dome and lateral aspect of the bladder wall (black arrow) and earlier systemic shunting via left internal iliac vein (curved arrow).



**Fig. 3** Venogram after 3 minutes of deployment of the Amplatzer vascular plug II confirmed cessation of flow across the plug (black arrow).

where in the same access was used to redirect the catheter caudally into the vesical end of the paraumbilical vein<sup>1</sup> (**Fig. 4**). Once the catheter was placed near the vesical end of the paraumbilical vein, 33% *n*-butyl cyanoacrylate (NBCA) glue was used to embolize the vesical varices under strict fluoroscopic control (**Fig. 5**). To even close the venous access point, the other operator withdrew the sheath while



**Fig. 4** Fluoroscopic spot image showing one sheath inversion technique. A safety guidewire (extra stiff guidewire) is parked in the preexisting direction of the sheath in the paraumbilical vein (straight arrow). Gradual withdrawal of sheath is done to allow passage of a hydrophilic guidewire to the opposite direction in the paraumbilical vein (curved arrow).



**Fig. 5** Fluoroscopic image showing injection of 33% *n*-butyl cyanoacrylate glue to the vesical varices and paraumbilical vein (black arrow).

the catheter was being gently withdrawn while administering glue. Postembolization fluoroscopic spot images showed glue cast in the varices and the recanalized paraumbilical vein, AVP II in the hepatic end of the paraumbilical vein (► Fig. 6).



**Fig. 6** Postembolization fluoroscopic spot image showing glue cast in the varices and the recanalized para umbilical vein (black arrow), Amplatzer vascular plug II in the hepatic end of the para umbilical vein (curved arrow).

Postprocedure period was uneventful with cessation of hematuria within 24 hours of intervention. The patient was discharged 2 days postprocedure. He was followed up for 6 months with no further episodes of hematuria or any other evidence of decompensation like ascites.

## Discussion

Hematuria is a common clinical symptom. Common etiologies of gross hematuria include urinary calculi, urinary tract infection, benign prostatic hyperplasia, urinary tract malignancy, and inflammatory disease of the urinary tract. Vesical varices are a rare cause of gross hematuria. Vesical varices are reported in association with portal hypertension,<sup>2</sup> schistosomiasis,<sup>3</sup> pregnancy,<sup>4</sup> ataxia,<sup>5</sup> Klippel Trenaunay syndrome,<sup>6</sup> retroperitoneal fibrosis,<sup>7</sup> and history of prior abdominal surgery.<sup>8</sup> Among the reported cases of vesical varices, portal hypertension was the most frequent cause. The first case of ectopic vesical variceal bleeding secondary to portal hypertension was reported by Sano et al in 1989.<sup>2</sup>

Esophageal and gastric varices are commonly seen in patients having cirrhosis with portal hypertension. Varices outside these anatomical locations are called ectopic varices and are rare. The presence of vesical ectopic varices is extremely rare because the bladder wall is not the usual pathway for portal decompression to the system circulation.<sup>4</sup>

The described patient developed portal hypertension secondary to hepatitis B-induced cirrhosis. Imaging with CT in this patient showed the vesical varices contiguous with recanalized paraumbilical vein and probable systemic venous drainage through the internal iliac veins on either side. Long-standing portal hypertension led to the recanalization of the paraumbilical vein with the formation of a portosystemic shunt in the bladder wall and resultant vesical varices. The vesical venous plexus formed the varices and was responsible for shunting the portal blood to the systemic circulation.

Cystoscopy is considered the gold standard for the diagnosis of vesical varices since the blood vessels are visualized directly in the bladder walls.<sup>9</sup> Conventional ultrasound, color Doppler, CECT, and magnetic resonance imaging (MRI) with MR angiography are used to diagnose the vesical varices either independently or in combination with cystoscopy. CT angiography and MR angiography are helpful tools to map the varices, anatomical details of the varices, and formulation of interventional/surgical planning.

Currently, no established treatment guidelines are available for the vesical varices secondary to portal hypertension. Surgical devascularization around the bladder,<sup>9</sup> cystoscopic-guided direct injection of NBCA glue to vesical varices,<sup>10</sup> and percutaneous transhepatic obliteration of varices<sup>11</sup> have been described.

We managed the present case successfully using plug-assisted antegrade transvenous obliteration for managing of gastric varices, primarily due to ease of access to the shunt through the recanalized paraumbilical vein. An alternative approach could be a percutaneous transhepatic obliteration technique as described by Sato et al.<sup>12</sup> Our technique was

similar to Kamada et al<sup>1</sup> including paraumbilical vein access and one sheath inversion technique for shunt occlusion in a patient with recurrent hepatic encephalopathy.

## Conclusion

Vesical varices secondary to portal hypertension are an extremely rare clinical entity. Interventional radiologists should be conversant with the diagnosis and treatment of this entity as it offers a minimally invasive treatment option to the patient.

### Conflict of Interest

None declared.

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