Endovascular Recanalization of a Short-Segment Occlusion of the Inferior Vena Cava Using Simultaneous Fluoroscopy and Ultrasound Guidance in a Patient With Budd-Chiari Syndrome and Chronic Kidney Disease (Stage 4): Technical Feasibility and Short-Term Outcomes

Kumar Muthukumar, MBBS, DMRD, DNB, FRCR, FNVIR[1]; Santhosh Babu Bharathy, MBBS, MD, FNVIR[1]; Vinu Moses, MBBS, MD[1]; Uday George Zachariah, MBBS, MD, DM, FRCP[2]; Shyamkumar Nidugala Keshava, MBBS, DMRD, DNB, FRCR, FRANZCR[1]

[1]Department of Interventional Radiology, Christian Medical College, Vellore, India
[2]Department of Hepatology, Division of Gastrointestinal Sciences, Christian Medical College, Vellore, India

Abstract: Budd-Chiari syndrome is caused by an obstruction to hepatic venous outflow, and endovascular treatment has a well-established role in its management. This case report describes an endovascular technique in the management of symptomatic inferior vena cava occlusion in a patient with chronic kidney disease (stage 4) not on dialysis. Considering the risk vs benefit, the procedure was performed without iodinated contrast. The details of the procedure, its outcome, and the relevant literature are discussed.

VASCULAR DISEASE MANAGEMENT 2022;19(2):E42-E45
Key words: Budd-Chiari syndrome, inferior vena cava, iodinated contrast, occlusion, recanalization, ultrasound guidance

Background

Budd–Chiari syndrome (BCS) is caused by an obstruction to hepatic venous outflow.[1] Endovascular treatment has a well-established role in its management.[2] This case report describes the case of a patient with BCS and chronic kidney disease (stage 4).

Case Report

A 29-year-old man presented with intermittent pedal edema and shortness of breath for 2 weeks duration. He also had a history of melena and hematemesis in the past 4 years. On examination, he had enlarged superficial veins on the anterior abdominal wall, hepatosplenomegaly, and bilateral pedal edema. Ultrasound (US) showed splenomegaly, heterogeneous echotexture of the liver, and minimal ascites. On Doppler evaluation, a short-segment (< 5 mm) occlusion of the suprahepatic inferior vena cava (IVC) was noted, with secondary conical dilation of the caudal IVC, resembling the appearance of a minaret.[3] The right hepatic vein (HV) was significantly attenuated and irreparable. Middle and left HVs were communicating with long-segment occlusion near the ostia (**Figure 1**), and intrahepatic collateral veins were seen. Based on the clinical and imaging findings, a diagnosis of Budd–Chiari Syndrome was made.

The patient's liver function tests were as follows: total bilirubin, 0.69 mg/dL (< 0.3 mg/dL); direct bilirubin, 0.56 mg (1.2 mg/dL); Total Protein, 7.2 g/dL (6.0-8.5); Albumin, 4.0 g/dL (3.5-5); aspartate aminotransferase, 40 U/L (< 40); alanine transaminase, 41 U/L (< 41); and alkaline phosphatase, 138 U/L (40-145). His serum creatinine was 5.2 mg/dL. He was not on renal replacement therapy at this time. In view of the renal dysfunction, a contrast computed tomography abdomen was not obtained.

Percutaneous endovascular treatment was planned for IVC occlusion without using iodinated contrast under fluoroscopy. Because the lesion was visible on transabdominal US, it was considered the first option for additional image guidance. Carbon dioxide, for use as a contrast medium, was kept on standby.

The procedure was performed in the angiography room under local anesthesia. The right common femoral vein was accessed under US guidance, and a 7 Fr sheath was placed. A 5 Fr pigtail catheter was placed in the infrahepatic IVC. Under fluoroscopy and transabdominal US guidance, a few attempts were made with a 5 Fr multipurpose catheter and stiff Glidewire (Terumo). Using real-time US guidance, the short-segment occluded IVC was traversed by the backend of a stiff Glidewire. After confirming the wire was well within the right atrium on US, the catheter was advanced under fluoroscopy a short distance into the right
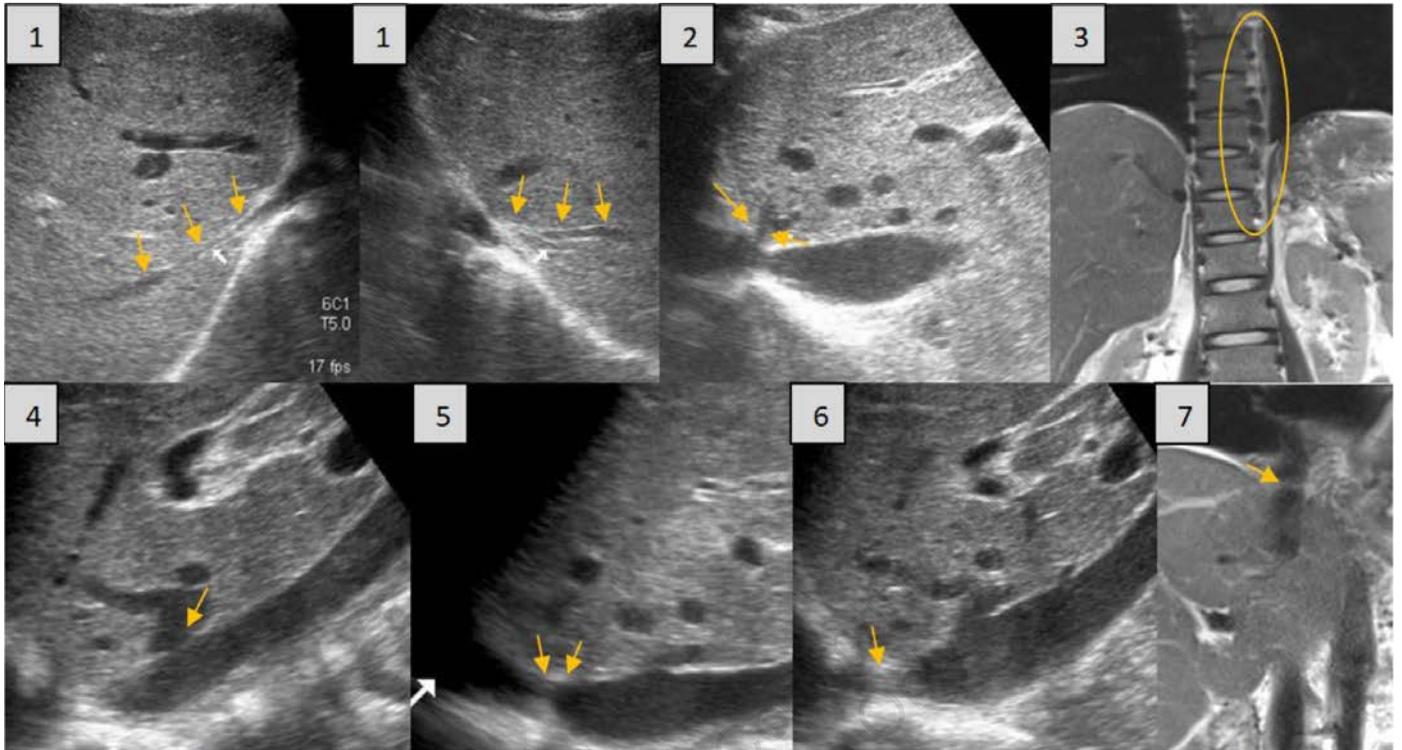
Figure 1. Ultrasound-guided images: The right hepatic vein (HV) is attenuated along its entire length **(1)**. The middle HV and left HV common ostia seen at a higher level **(2)**. Magnetic resonance imaging coronal image showing hemiazygos collaterals **(3)**. Accessory HV ostium draining into the inferior vena cava (IVC) **(4)**. Cavoatrial junction and right atrium **(5)**. Superhepatic IVC occlusion with a thick membranous structure and conical expansion of the caudal IVC **(6)**. IVC stenosis secondary to membranous obstruction at the cavoatrial junction **(7)**.
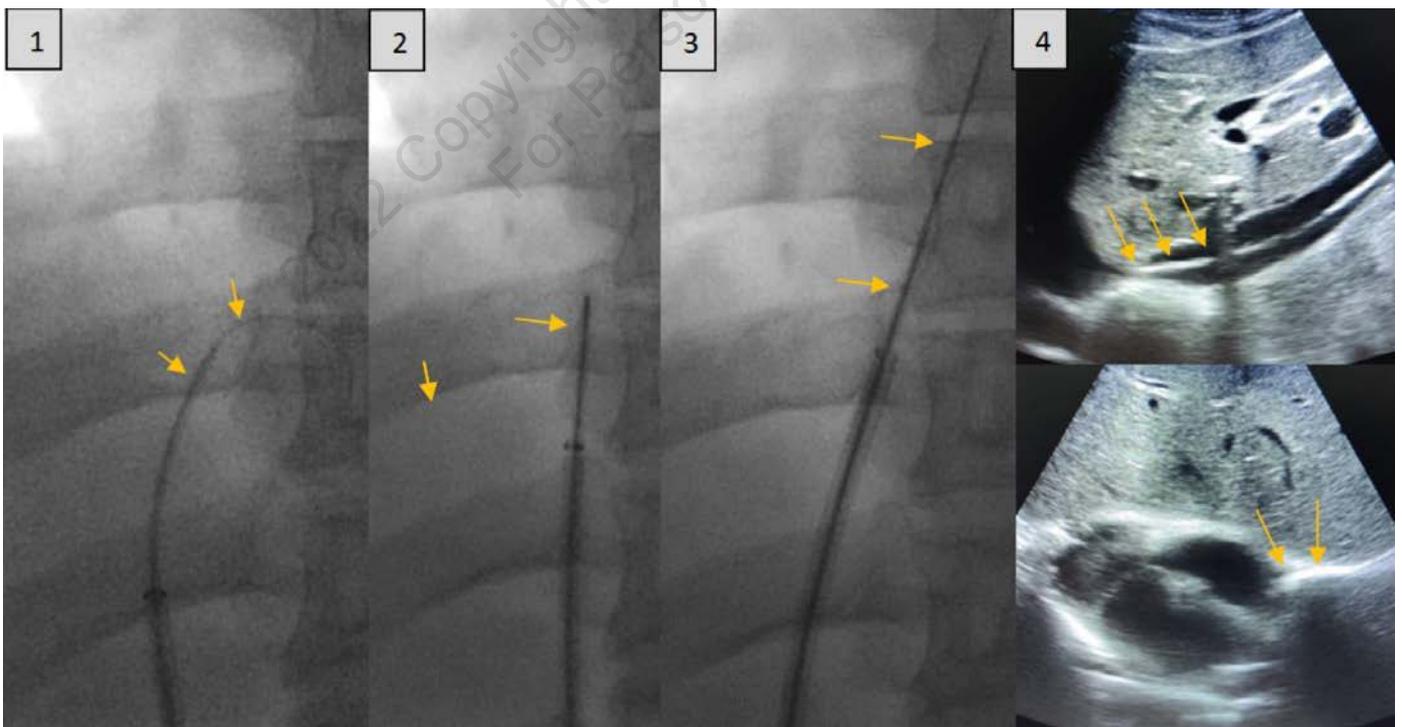
Figure 2. Fluoroscopic image depicting a 7 Fr long sheath positioned in the inferior vena cava (IVC) during the initial attempt of a multipurpose catheter/Glidewire combination to cross the stenosis **(1)**. Reverse end of the Glidewire with proper approximation of the long sheath during sharp recanalization **(2)** and **(3)**. Simultaneous ultrasound guidance to check the orientation and direction of the Glidewire in order to pierce the membrane and enter the right atrium **(4)**.
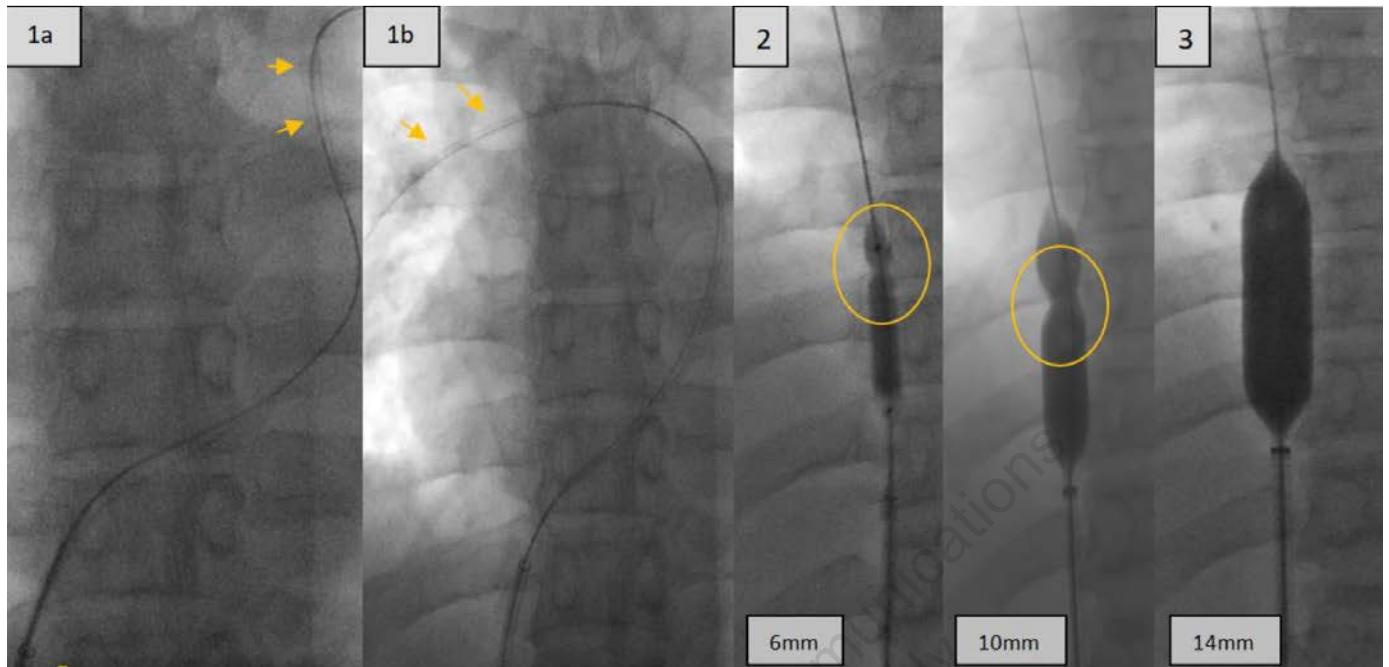
Figure 3. Fluoroscopic image showing Glidewire navigation after sharp recanalization, confirming the location to be in the left pulmonary artery **(1a)** and right pulmonary artery **(1b).** Balloon angioplasty using 6 mm and 10 mm diameter balloons, depicting the waist at the level of inferior vena cava (IVC) stenosis **(2)**. Fluoroscopic image of a stenotic IVC segment during 14 mm diameter balloon inflation with effacement of the waist **(3)**.

atrium. The Glidewire end was reversed, and the catheter was advanced into the right atrium (**Figure 2**). The Glidewire was exchanged for a 0.035" Amplatz exchange-length wire. Serial balloon angioplasty was performed using 8 mm, 12 mm, and 14 mm diameter balloons. Recanalization of the suprahepatic IVC occlusion was observed on US (**Figure 3**). The recanalization was also tested by a reduction in the pressure gradient, which reduced from 16 mm Hg to 7 mm Hg. In addition, the curved distal end of the pigtail catheter could be easily moved across the region of occlusion. There were no complications.

The patient had an uneventful postprocedure recovery and was discharged on the third day. On subsequent follow-up 3 months later, he remained asymptomataic for portal hypertension, although he was noted to have a worsening renal function with non–oliguric renal failure (serum creatinine, 12.3 mg/dL and 16.4 mg/dL), for which he remains under evaluation and treatment.

Discussion

BCS, a relatively uncommon but devastating disorder, comprises clinical manifestation of hepatic venous outflow obstruction.[4] Obstruction of the IVC at the diaphragmatic level was first described by Osler in 1878. Since then, membranous obstruction of the IVC has been well described in the literature, with most reports from Japan, India, and South Africa. Okuda et al demonstrated these lesions to be an aftereffect of thrombosis in the IVC or HV, which organize over time.[5]

Over the years, many prothrombotic states leading to BCS have been described in India.[6] Recanalization of stenotic or occluded HVs or IVC to restore venous outflow is the initial procedure of choice. Segments with focal stenosis or occlusion are best treated with angioplasty.

Various approaches may be used to cross the IVC stenosis or occlusion. A catheter-guidewire technique followed by balloon angioplasty is preferable.[7] A transjugular cannula–assisted technique facilitates recanalization of the IVC in patients with short segmental hepatic IVC occlusion. A transjugular liver biopsy cannula provides additional support to the catheter-wire combination, and transabdominal US helps in positioning the tip of the cannula at the stump adjacent to the stenosis.[8]

Iodinated contrast agents play a major role in IVC recanalization, as a routine venogram is performed using contrast. Utility of a blank road map obtained using iodinated contrast is helpful during balloon angioplasty. However, iodinated contrast use in patients with renal insufficiency, as in this case, has the inherent risk of further deterioration of renal function, sometimes requiring dialysis. The other alternative contrast agent is carbon dioxide.[9] Image guidance provided by US and fluoroscopy are complimentary to each other.[10] US is a real-time imaging of the IVC and the surrounding liver parenchyma. Fluoroscopy shows the movement of the catheter and the guidewire in a 2-dimensional projection.

A sharp recanalization should be considered only when regular attempts are not successful.[11,12] In addition, we recommend dual-image guidance to reduce the chance of complications such as inadvertent perforations or hemopericardium. The on-table technical outcome may be assessed by Doppler, free movement of a pigtail catheter across the segment postprocedure, and pressure gradient.

Conclusion

Nonutilization of iodinated contrast should be considered after assessing the risk vs benefit in patients who have chronic kidney disease (stage 4) and are not on dialysis. The novel technique described here would be suitable to recanalize short-segment occlusions of IVC. More studies are required for further assessment of the technique. ■

Address for correspondence: Shyamkumar Nidugala Keshava, MBBS, DMRD, DNB, FRCR, FRANZCR, Department of Interventional Radiology, Christian Medical College, W4GP+7CW, Vellore, Tamil Nadu 632004, India. Email: shyamkumar.n.keshava@gmail.com

REFERENCES

1. Sharma A, Keshava SN, Eapen A, Elias E, Eapen CE. An update on the management of Budd-Chiari syndrome. *Dig Dis Sci.* 2021;66(6):1780-1790. doi:10.1007/s10620-020-06485-y
2. Strozzi M, Besic KM, Ivana KS, Darko A. Endovascular treatment of an obstructive membrane between inferior vena cava and right atrium in an unrecognized Budd-Chiari syndrome. *CVIR Endovasc.* 2020;3(1):76. doi:10.1186/s42155-020-00168-5
3. Garge SS, Keshava SN. Intramural inferior vena cava occlusion: the minaret sign. *The Egyptian Journal of Internal Medicine.* 2016;28:131-132.
4. Okuda K. Membranous obstruction of the inferior vena cava (obliterative hepatocavopathy, Okuda). *J Gastroenterol Hepatol.* 2001;16(11):1179-1183. doi:10.1046/j.1440-1746.2001.02577.x
5. Ludwig J, Hashimoto E, McGill DB, van Heerden JA. Classification of hepatic venous outflow obstruction: ambiguous terminology of the Budd-Chiari syndrome. *Mayo Clin Proc.* 1990;85(1):51-55. doi:10.1016/s0025-6196(12)62109-0
6. Datta DV, Vashishta S, Samanta AK, Chhuttani PN. Diagnostic value of combined transhepatic venography and inferior vena cavography in chronic Budd–Chiari syndrome. *Am J Dig Dis.* 1978;23(11):1031-1041. doi:10.1007/BF01263104
7. Mohanty S, Saxena R, Acharya SK. Activated protein C resistance in Budd-Chiari syndrome. *Int J Hematol.* 2000;72(2):255.
8. Srinivas BC, Dattatreya PV, Srinivasa KH, Prabhavathi, Manjunath CN. Inferior vena cava obstruction: long-term results of endovascular management. *Indian Heart J.* 2012;64(2):162-169. doi:10.1016/S0019-4832(12)60054-6
9. Cho, KJ. Carbon dioxide angiography: scientific principles and practice. *Vasc Specialist Int.* 2015;31(3):67-80. doi:10.5758/vsi.2015.31.3.67
10. Keshava SN, Vinu M, Sharma M, et al. Technical and medium-term clinical outcomes of transjugular intrahepatic portosystemic shunt with fluoroscopy and additional trans–abdominal ultrasound guidance. *Indian J Radiol Imaging.* 2021;31(4):858-866. doi:10.1055/s-0041-1735928
11. Rivers–Bowerman MD, Lightfoot CB, Meagher RP, Carter MD, Berry RF. Percutaneous sharp recanalization of a membranous IVC occlusion with an occlusion balloon as a needle target. *Radiol Case Rep.* 2017;12(3):537-541. doi:10.1016/j.radcr.2017.04.021
12. Honnef D, Wingen M, Günther RW, Haage P. Sharp central venous recanalization by means of a TIPS needle. *Cardiovasc Intervent Radiol.* 2005;28(5):673-676. doi:10.1007/s00270-004-0323-y