Radiology Case Reports 18 (2023) 4172–4175

Available online at www.sciencedirect.com

ScienceDirect

journal homepage: www.elsevier.com/locate/radcr

Case Report

Transhepatic inferior vena cava recanalization in a case of Budd Chiari syndrome: A novel approach ☆

Saurabh Ashok Sujanyal, MBBS[a,*], *Priyansh Parag Shah*[b], *Joseph Gartrell Willis, MD, MBA* [c], *Husameddin El Khudari, MD*[a], *Rakesh K. Varma, MD, FSIR*[a]

[a] *Division of Interventional Radiology, University of Alabama, USA*
[b] *Department of Cardiology, Beth Israel Deaconess Medical Center, Boston, USA*
[c] *Department of Radiology, University of Alabama at Birmingham, Birmingham, Alabama, USA*

ARTICLE INFO

Article history:
Received 9 August 2023
Accepted 12 August 2023

Keywords:
Transhepatic endovascular recanalization
Budd Chiari syndrome
Inferior vena cava occlusion
Sharp recanalization
Portal Hypertension

ABSTRACT

Sharp recanalization for short-segment intravascular occlusion, using an endovascular route, has been described for inferior vena cava (IVC) occlusion. Often, the technical challenge to the endovascular management of Budd-Chiari syndrome (BCS) is the recanalization of the occluded hepatic vein or suprahepatic IVC. Presented here, the challenge was the level of occlusion of the suprahepatic IVC, with the resultant separation of both the patent IVC segments in a horizontal plane, making it technically challenging for sharp recanalization. We describe the use of percutaneous transhepatic access into the suprahepatic IVC via the middle hepatic vein under ultrasound guidance with eventual sharp recanalization of the occluded segment of the IVC, in a woman with BCS. This novel approach has not been described in the literature and can serve as an important addition to guide complex suprahepatic IVC recanalization.

Introduction

Budd-Chiari syndrome (BCS) is a rare disorder and is defined as hepatic venous outflow tract obstruction [1]. The hepatic venous outflow tract obstruction can be classified according to the location of obstruction: small hepatic veins (HV), large HV, suprahepatic inferior vena cava (sIVC), and any combination thereof [2]. Epidemiologically, pure IVC or combined IVC/HV block predominates in Asia, whereas pure HV block predominates in Western countries [3]. Chronic or subacute occlusion of sIVC is often asymptomatic or minimally symptomatic prior to disease progression. As the disease progresses, it usually presents with symptoms of abdominal distension, abdominal pain, upper gastrointestinal bleeding, jaundice, or encephalopathy [4]. Management includes medical therapy, interventional procedures, and liver transplants. Medical therapy is associated with steady improvement in 20% of pa-

☆ Competing Interests: The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

* Corresponding author.
E-mail address: saurabhsujanyal@gmail.com (S.A. Sujanyal).

https://doi.org/10.1016/j.radcr.2023.08.067

Fig. 1 – IVC venogram during attempted sharp recanalization shows complete occlusion of the iIVC and a patent RHV (arrow) draining into the IVC.

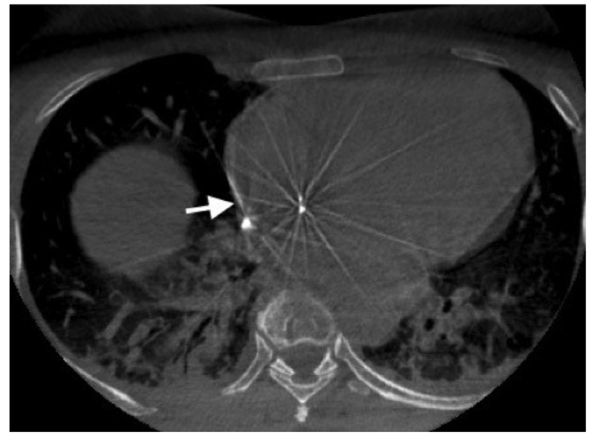

Fig. 2 – Cone beam CT during recanalization from the transfemoral approach shows contrast in the pericardial space (arrow).

tients without any need for additional therapy. Nevertheless, interventional procedures remain the mainstay and include transjugular intrahepatic portosystemic shunt (TIPS) creation and percutaneous recanalization of venous obstruction. For suprahepatic IVC block, percutaneous recanalization can be expected to achieve a complete response in 60% of patients whereas, for pure hepatic vein block, TIPS is successful in 85% of patients [1]. This case report describes a transhepatic approach for sIVC occlusion recanalization in a patient after a failed transfemoral approach.

Case presentation

A 51-year-old female with a past medical history of fibromyalgia, iron deficiency anemia, hypertension, and obesity presented to an outside hospital for evaluation of protruding veins on her abdomen and portal hypertension. Endoscopy revealed grade 1 esophageal varices and liver biopsy showed marked congestion and necrosis with increased fibrosis consistent with BCS. IVC venogram confirmed sIVC occlusion (Fig. 1) with multiple retroperitoneal and abdominal wall collaterals draining the suprarenal IVC. IVC pressure was 23 mm Hg and right atrial pressure was 12 mm Hg with a gradient of 11 mm Hg across the occlusion. She had undergone unsuccessful attempts at sharp recanalization of the sIVC through a transfemoral route at an outside hospital and was referred to our institution for recanalization of the sIVC for portal decompression.

An initial attempt at sharp recanalization through a transfemoral approach was pursued. A Rösch-Uchida transjugular liver access set (Cook, Inc., Bloomington, IN) was advanced into the cranial aspect of the infrahepatic IVC, at the level of occlusion, under continuous fluoroscopic guidance and confirmed by transabdominal ultrasound. Next, a 21-gauge Chiba needle was advanced through the access set and was used to puncture the sIVC.

Subsequently, a V-14 wire was advanced through the needle into the superior vena cava (SVC). Cone beam CT was performed demonstrating the wire coursing through the IVC, hepatic parenchyma, and into the right atrium. Using a 3 mm monorail balloon catheter, the tract was dilated to 3 mm. Subsequently, a 4-French glide catheter was advanced over the wire into the right atrium. The catheter was then retracted and an over-the-wire tractogram was performed demonstrating a trans-pericardial course into the right atrium. Contrast pooling into the pericardial space was confirmed on the cone beam CT (Fig. 2). This was attributed to the relative anatomy of the sIVC, which was short and separated in the same horizontal plane as the intrahepatic IVC (iIVC) segment, making it technically challenging for the Chiba needle to connect the 2 segments in the same horizontal plane.

In order to assist recanalization, the initial thought was to place a Fogarty balloon in the sIVC via a direct percutaneous transhepatic approach, which would serve as a target. Under continuous sonographic guidance, a 21-G needle was advanced percutaneously into the sIVC, through the hepatic parenchyma. The needle traversed the overlying middle hepatic vein (MHV) into the sIVC. A 0.018-inch microwire was advanced into the SVC and the needle was exchanged for a 6-French Neff set (Cook, Inc.). A 4-French glide catheter was then advanced coaxially into the right atrium and used to perform an over-the-wire tractogram to exclude an inadvertent transpericaridal course. Tractogram confirmed a nontranspericardial course. Additionally, there was opacification of the infrahepatic IVC on the tractogram. Given this additional finding, an attempt was made at snaring the wire in the sIVC through the transfemoral approach to establish recanalization. However, instead of a straighter course, the wire and the catheter looped around the hepatic dome, to ultimately grasp the wire in the sIVC. This was consistent with complete occlusion between the MHV and the right hepatic vein (RHV) with the intrahepatic collaterals connecting the

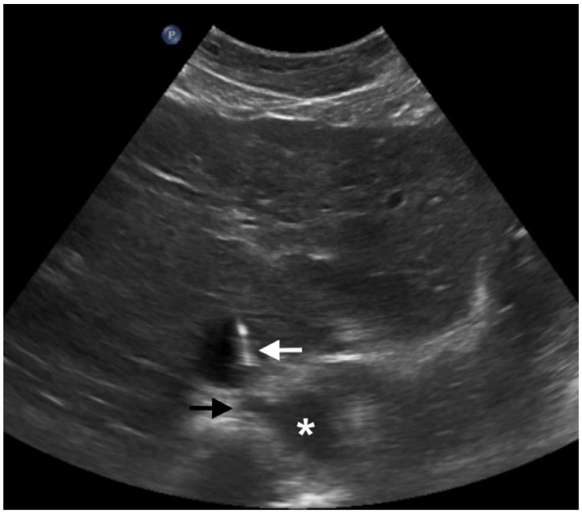

Fig. 3 – Ultrasound-guided percutaneous access into the sIVC (black arrow) and right atrium (asterisk) with a needle through the MHV (white arrow).

MHV and RHV. Also, with the relative anterior position of the sIVC as compared to the iIVC, it was deemed that a much caudal puncture through the infrahepatic IVC would be required, but would likely result in an extrahepatic course rather than through the caudate lobe. Considering these findings, recanalization from the transjugular approach was pursued in order to connect the MHV and the RHV, which drained directly into the infrahepatic IVC. A Kumpe catheter was advanced over the wire into the sIVC and into the MHV. Multiple attempts at crossing the occlusion between the MHV and the RHV using a catheter wire combination were performed but were unsuccessful. Finally, the Kumpe catheter was exchanged for a Rösch-Uchida TIPS cannula and the 21 G-Chiba needle was used for sharp recanalization between the MHV and the RHV. A glide wire was then advanced into the infrahepatic IVC. Thereafter, angioplasty of the suprahepatic and infrahepatic IVC was performed to 10 mm. No extravasation was identified. A 14 mm x 60 mm Zilver Vascular Self-Expanding stent (Cook Inc.) was deployed across the areas of occlusion. The stent was then angioplastied to 14 mm. Follow-up venography demonstrated a widely patent stent with the restoration of flow from the IVC into the right atrium and interval decompression of the collaterals. There was no gradient across the IVC, poststenting. The patient is clinically improving, showing the decreased size of her varices, is experiencing no symptoms postprocedure, and has had no complications secondary to the endovascular procedure. Her postprocedure CT 8 months later shows a patent IVC stent with interval decompression of the venous collaterals (Figs. 3–5).

Discussion

IVC thrombosis caused by webs necessitated thoracoabdominal surgery before the advent of percutaneous transluminal angioplasty. However, surgical management had considerable

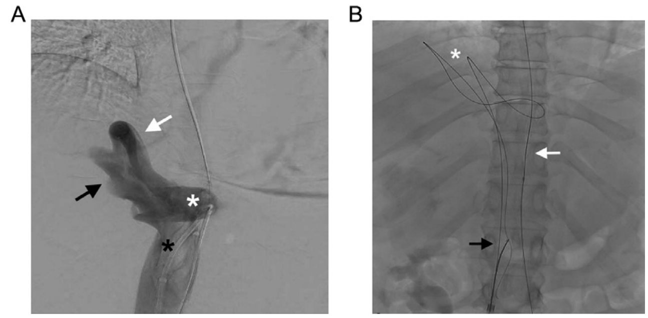

Fig. 4 – (A, B) Pull-back tractogram through the transhepatic catheter (white arrow) shows (A) MHV (white asterisk) draining through a large intrahepatic collateral (white arrow) into the RHV (black arrow) and finally to the iIVC (black asterisk); (B). Corresponding fluoroscopic image during snaring of the transhepatic wire (white arrow) in the MHV through the IVC (black arrow), demonstrates the course of the wire connecting the MHV and RHV through intrahepatic collaterals (asterisk).

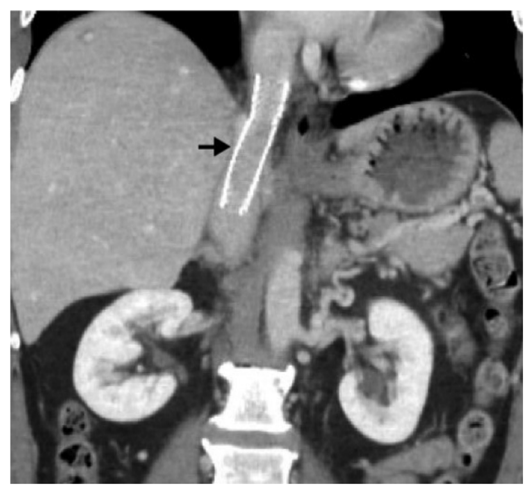

Fig. 5 – Follow-up CT shows a patent stent (arrow).

mortality and morbidity [5]. In recent years, TIPS and percutaneous recanalization of venous obstruction have remained the mainstays for the management of BCS.

Direct percutaneous transhepatic puncture of the sIVC, if visible under ultrasound, can be performed in order to aid complex sIVC recanalization. Our access to the sIVC through MHV was critical as it outlined the relative anatomy of sIVC as compared to the patent iIVC, requiring a possible extrahepatic course from iIVC to sIVC in order to avoid transpericardial access. Also, bridging the occlusion between the MHV and sIVC with the transhepatic access, aided in advancing the TIPS cannula into the MHV for sharp recanalization between the MHV and RHV, to eventually gaining access into the infrahepatic IVC. Once recanalization was established the transhepatic tract was embolized to diminish the risk of bleeding. In our case, with ultrasound guidance, we were able to advance the needle into the sIVC using a single pass. This also indirectly avoided making subsequent inadvertent punctures. An alternative route, such as the jugular or femoral vein approach can be used for recanalization. Nevertheless, without transhepatic outlining of the sIVC, can be associated with the risk of cardiac perforation or IVC rupture.

Conclusion

BCS can present with various levels of obstruction, sometimes causing symptomatic portal hypertension. Careful assessment of the level of IVC and intrahepatic obstruction, on cross-sectional imaging and preprocedural venograms, can aid the planning for recanalization. Transhepatic approach, in appropriate cases, can be used as a viable addition to a bidirectional approach for the recanalization of complex sIVC occlusion.

Patient consent

This is to inform you that written, informed consent was obtained from the patient for publication of their case for the case report entitled “Transhepatic Inferior Vena Cava Recanalization in a case of Budd Chiari syndrome: A novel approach.”

REFERENCES

[1] DeLeve LD, Valla DC, Garcia-Tsao GAmerican Association for the Study Liver Diseases. Vascular disorders of the liver. Hepatology 2009;49(5):1729–64. doi:10.1002/hep.22772.

[2] 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;65(1):51–5. doi:10.1016/s0025-6196(12)62109-0.

[3] Valla DC. Hepatic venous outflow tract obstruction etiopathogenesis: Asia versus the West. J Gastroenterol Hepatol 2004;19(s7):S204–11.

[4] Kohli V, Pande GK, Dev V, Reddy KS, Kaul U, Nundy S. Management of hepatic venous outflow obstruction. Lancet 1993;342(8873):718–22. doi:10.1016/0140-6736(93)91712-u.

[5] Lois JF, Hartzman S, McGlade CT, Gomes AS, Grant EC, Berquist W, et al. Budd-Chiari syndrome: treatment with percutaneous transhepatic recanalization and dilation. Radiology 1989;170(3):791–3.