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Case Report

Percutaneous sharp recanalization of a membranous IVC occlusion with an occlusion balloon as a needle target

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ARTICLE INFO

Article history:
Received 29 January 2017
Received in revised form
6 April 2017
Accepted 29 April 2017
Available online 7 June 2017

Keywords:
Sharp recanalization
Membranous IVC occlusion

ABSTRACT

A 50-year-old male with right upper quadrant symptoms and hepatic dysfunction was found to have multiple dilated hepatic veins (HVs) with intrahepatic collateralization and membranous occlusion of the intrahepatic inferior vena cava (IVC) consistent with primary Budd–Chiari syndrome. Venacavograms depicted drainage of the intrahepatic collaterals through a left-sided HV entering the IVC above the level of the occlusion. Sharp recanalization of the membranous IVC occlusion was performed with an occlusion balloon as a needle target under echocardiographic monitoring followed by balloon angioplasty with restoration of IVC patency. Clinical, laboratory, and venographic procedural success has been demonstrated to 9 months with minimal residual stenosis.

Introduction

Budd–Chiari syndrome (BCS) is a rare group of disorders which result from hepatic venous outflow tract obstruction [1]. The obstruction can occur from the small hepatic veins (HVs) to the junction of the inferior vena cava (IVC) and right atrium [1,2]. Depending on the vessels involved, BCS is classified as IVC type, HV type, or combined IVC–HV type. BCS is also classified as primary or secondary in etiology. Primary BCS is more frequently encountered and results from primary venous pathologies including thrombosis, stenosis, and occlusion [1,3]. Stenosis or occlusion of the IVC is defined as membranous or segmental depending on the length of the involved segment; a stenosis or occlusion measuring less than or equal to 1 cm in length is classified as membranous, whereas a stenosis or occlusion measuring greater than 1 cm in length is classified as segmental [3]. Secondary BCS results from extrinsic venous compression by adjacent structures or tumor invasion [1]. Predisposing factors for BCS are found in up to 75% of patients and include a number of hereditary and acquired

Competing Interests: The authors have declared that no competing interests exist.

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http://dx.doi.org/10.1016/j.radcr.2017.04.021

hypercoagulable states and other rarer causes [4]. The clinical presentation of BCS is varied and may be fulminant, acute, subacute, or chronic in nature [1]. Up to 75%-80% of patients with BCS present with signs and symptoms such as fever, ascites, abdominal pain, hepatic failure, gastrointestinal bleeding, and encephalopathy [1,2]. The remaining 15%-20% of patients have compensated subclinical disease [1]. Imaging investigation of BCS with ultrasound, computed tomography (CT), MRI and catheter venography plays an important role in evaluating disease severity, planning appropriate management, and assessing treatment response [5]. The aim of treatment for BCS is to relieve hepatic venous congestion, and different management options include medical, percutaneous/endovascular, and/or surgical approaches [5]. We present the case of a 50-year-old male with primary BCS resulting from membranous occlusion of the intrahepatic IVC treated successfully with sharp recanalization and angioplasty.

Case report

Institutional review board approval was obtained for this report. A 50-year-old male presented with a 2-month history of right upper quadrant pain, fever, night sweats, and weight loss. His personal medical history was unremarkable apart from prior heavy ethanol use. His father had a history of bilateral deep vein thromboses. The review of systems was negative for gastrointestinal bleeding, and there were no risk factors for viral hepatitis. On physical examination, there was right upper quadrant tenderness with bilateral leg and scrotal edema. Initial laboratory testing is listed in Table 1.

Table 1 – Initial laboratory data.

Parameter (units)	Value (normal range)
White blood cell count (counts/L)	9.9×10^9 ($4.5\text{-}11.0 \times 10^9$)
Hemoglobin (g/L)	125 (135-180)
Mean corpuscular volume (fL)	92 (76-96)
Platelet count (counts/L)	65×10^9 ($150\text{-}450 \times 10^9$)
INR	1.6 (0.8-1.2)
Urea (mmol/L)	6.7 (2.9-9.3)
Creatinine (μmol/L)	103 (54-113)
Sodium (mmol/L)	138 (136-144)
Potassium (mmol/L)	5.3 (3.4-5.0)
AST (U/L)	211 (17-59)
ALT (U/L)	194 (21-72)
Total protein (g/L)	69
Albumin (g/L)	26 (35-50)
Total bilirubin (μmol/L)	38 (0-24)
Direct bilirubin (μmol/L)	7 (0-5)
GGT (U/L)	204 (15-73)
ALP (U/L)	186 (38-126)
LDH (U/L)	865 (313-618)

INR, international normalized ratio; AST, aspartate aminotransferase; ALT, alanine aminotransferase; GGT, gamma-glutamyltransferase; ALP, alkaline phosphatase; LDH, lactate dehydrogenase.

A triphasic CT scan demonstrated hepatic parenchymal heterogeneity with borderline splenomegaly and mild ascites, multiple dilated HVs with intrahepatic collateralization, and a short, focal web-like obstruction of the IVC measuring less than 1 cm in length consistent with membranous occlusion, a form of IVC type primary BCS (Fig. 1A). The intrahepatic collaterals were noted to drain through the left HV which entered the IVC above the level of the membranous occlusion (Fig. 1B). The infrahepatic IVC and HVs were patent. Venacavography with C-arm CT was also performed for additional characterization of these findings (Fig. 2). The occlusion could not be crossed with multiple catheter and wire combinations at the time of venacavography.

A subsequent liver biopsy was consistent with hepatic outflow obstruction. Serology for autoimmune and viral hepatitis was unremarkable. Additional testing revealed the presence of antiphospholipid antibodies, including the anticardiolipin antibody and the lupus anticoagulant. The anticardiolipin antibody level was elevated, and testing for the lupus anticoagulant was strongly positive. Remaining tests for hypercoagulability, including factor V Leiden, protein C/S, and the JAK2 V617F mutation, were noncontributory.

The patient was started on therapeutic heparin. The case was jointly reviewed with hepatobiliary and vascular surgery, and a plan for definitive endovascular management with percutaneous sharp recanalization and angioplasty of the membranous IVC occlusion was devised. It was planned to serially dilate the membranous IVC occlusion to the diameter of the IVC below the occlusion (18 mm) over 2 sessions.

The initial procedure was carried out under general anesthesia with intermittent monitoring by transesophageal echocardiography to assess for potential complications such as hemopericardium and cardiac tamponade. Right internal jugular venous access was obtained, and a 10-French (Fr) sheath was advanced into the right atrium. Additional access to the right common femoral vein was obtained, and a 12-Fr sheath was positioned in the IVC. A 7-Fr 11.5-mm occlusion balloon catheter (Boston Scientific, Marlborough, MA) was inserted through this sheath and placed along the inferior margin of the membranous occlusion. A 65-cm 21-gauge Chiba needle (Cook Medical, Bloomington, IN) was delivered to the membranous IVC occlusion from a superior approach through the cannulated sheath of a liver access and biopsy set (Cook Medical) and used to cross the membrane, bursting the occlusion balloon immediately inferior to the membrane (Fig. 3). Following sharp recanalization, a 0.014" Mailman guidewire (Boston Scientific) was advanced into the IVC and snared into the caval sheath for through and through venous access. A 4-Fr Kumpe catheter (Cook Medical) was advanced through the femoral sheath, and venacavography showed no evidence of contrast extravasation or hemorrhage. Initial angioplasty in the first treatment session was carried out using a 4 mm × 60 mm Fox SV balloon (Abbott Vascular, Santa Clara, CA). The wire was then exchanged for a 0.035" exchange length Amplatz guidewire (Boston Scientific), and serial angioplasty was carried out using 7 mm × 40 mm, 10 mm × 40 mm, and 12 mm × 40 mm Armada 35 balloons (Abbott Vascular) and a high pressure 14 mm × 40 mm Atlas balloon (Bard Medical, Covington, GA). IVC patency was restored at the conclusion of the procedure.

The patient returned to the department 3 weeks later for the second and final scheduled treatment session. Through and through right common femoral vein to right internal jugular venous access was established, and the region of membranous IVC occlusion was angioplastied to a diameter of 18 mm with 16 mm × 40 mm and 18 mm × 40 mm Z-Med II

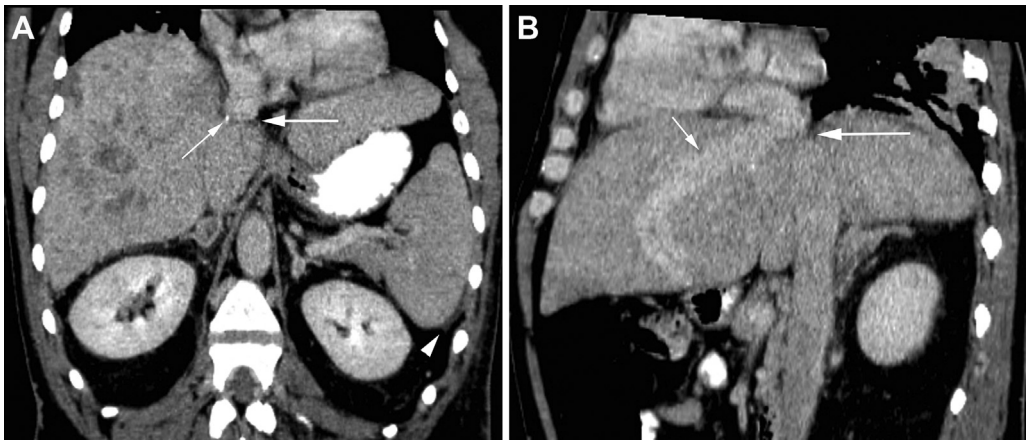

Fig. 1 – (A) Oblique coronal portal venous phase CT image demonstrates short, focal web-like obstruction of the intrahepatic IVC (large arrow) measuring less than 1 cm in length consistent with membranous occlusion. A small punctate focus of calcification suggests a component of chronicity to this finding (thin arrow). The hepatic parenchyma is mildly heterogeneous, and there is a trace amount of ascites at the hepatic and splenic tips (arrowhead). (B) Oblique sagittal CT image in the same phase shows the left hepatic vein (thin arrow) draining into the IVC above the level of the occlusion (large arrow). CT, computed tomography; IVC, inferior vena cava.

balloons (B. Braun Medical, Bethlehem, PA) with no residual balloon waisting and a satisfactory venographic result (Fig. 4).

The patient was then transitioned from therapeutic heparin to long-term oral warfarin anticoagulation after the procedure to minimize the risk of future thrombogenic events. There has been clinical, laboratory, and procedural success at up to 9 months with no significant venous restenosis on CT or MRI.

Discussion

Membranous IVC occlusion has been described in the setting of both hereditary and acquired hypercoagulability [6–10], with trauma reported to be a precipitating factor in 2 cases [7]. Histopathologic analysis of IVC specimens with membranous occlusion at autopsy reported that the membranes were composed of fresh and organized thrombus, fibrous tissues, and calcifications in keeping with thrombosis and its sequelae [11]. Our patient's antiphospholipid antibodies likely contributed to the development of the membranous IVC occlusion.

The aim of the treatment for all forms of BCS is to relieve hepatic venous congestion to maintain appropriate hepatic perfusion and preserve normal hepatocyte function [5]. Treatment strategies are tailored to multiple patient and disease-specific factors [5]. Available options for treatment of BCS with HV and/or IVC involvement include medical management with anticoagulants and diuretics [2]; endovascular management with balloon angioplasty, stenting, catheter-directed thrombolysis, transjugular intrahepatic portosystemic shunting, and retrieval stent filter placement [1,3,8,9,12–15]; and surgical management with surgical portosystemic shunting, orthotopic liver transplantation, and surgical membranectomy [1,2,16].

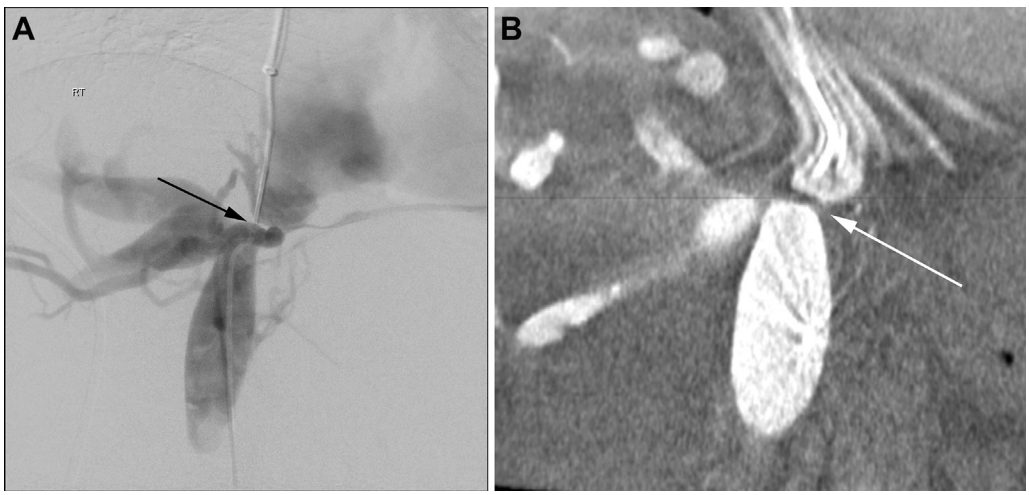

Fig. 2 – Membranous IVC occlusion (arrow) on venacavography (A) and coronal C-arm CT (B). CT, computed tomography; IVC, inferior vena cava.

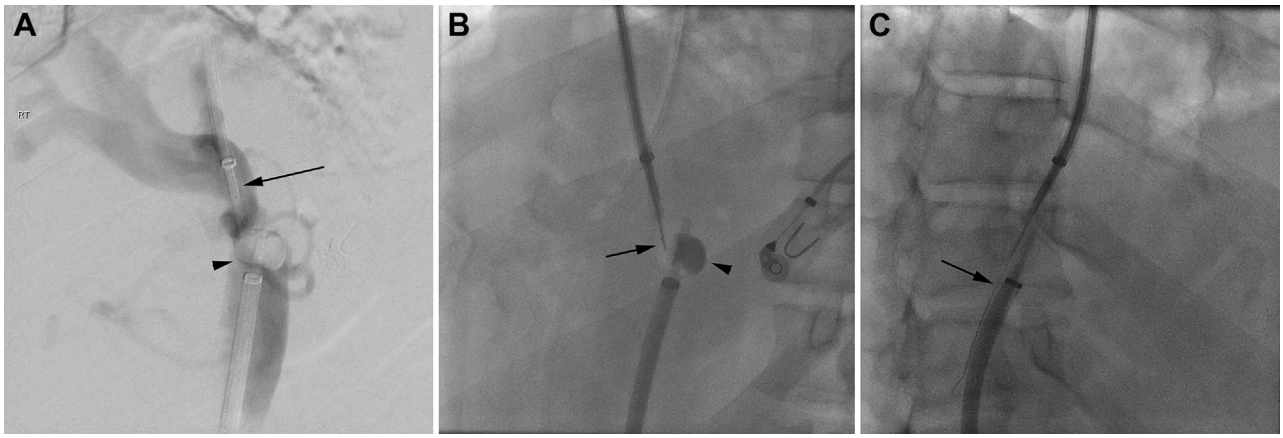

Fig. 3 – (A) Venogram shows a cannulated sheath (arrow) superior to the membranous IVC occlusion and a 7-French 11.5-mm occlusion balloon (arrowhead) (Boston Scientific) at the inferior margin of the web. (B) A 65-cm 21-gauge Chiba needle (arrow) (Cook Medical) is advanced into position immediately prior to puncture of the occlusion balloon (arrowhead). (C) A 0.014" Mailman guidewire (arrow) (Boston Scientific) is advanced across the membranous occlusion following sharp recanalization.

Our patient was initially managed with therapeutic dalteparin and spironolactone which significantly improved his ascites, leg edema, and liver enzymes. However, definitive management of the membranous IVC occlusion was indicated to prevent progression to chronic liver disease. We pursued an endovascular approach given the risks of surgery and opted to attempt percutaneous recanalization and angioplasty to restore IVC patency.

Sharp recanalization is a well-described technique for crossing chronic venous occlusions [17–19]. At the time we performed the procedure in early 2014, there had been numerous reports of sharp recanalization of membranous IVC occlusions using the stiff end of a 0.035" guidewire [20], straight needles [13], J-type transseptal needles [3,9,20–22], custom stainless steel needles with curved heads [23–25], and the Rosch-Uchida transjugular liver access set (Cook Medical) [26]. We were unsuccessful in crossing the occlusion with multiple guidewires at the time of initial venacavography and elected to use a 21-gauge Chiba needle (Cook Medical) based upon availability, cost, and perceived safety with the delivery method described above; however, other methods of sharp recanalization would have also been appropriate. Recanalization of chronic venous occlusions with a radiofrequency wire has also been described as a more minimally invasive alternative to sharp recanalization [27], but we did not have access to this equipment at our institution. During the procedure, we placed an occlusion balloon along the inferior margin of the IVC membrane to serve as a needle target for sharp recanalization as balloon rupture immediately confirms intravascular needle positioning. Sharp recanalization of venous occlusions with a balloon as a needle target has been reported previously [17,18]; however, to our knowledge, this is the first report to utilize this approach for recanalization of a membranous IVC occlusion. Alternate needle targets for sharp recanalization include guidewires, vascular snares, and catheters [17]; however, these devices occupy smaller cross-sectional areas within the target vessel relative to an appropriately sized inflated balloon catheter and may not reside centrally within the target vessel lumen. Accordingly, the balloon technique may lead to more accurate and precise needle placement during attempted sharp recanalization. It may also aid procedural efficiency by allowing the operator to rapidly confirm intravascular needle positioning with balloon rupture after the needle traverses the membrane.

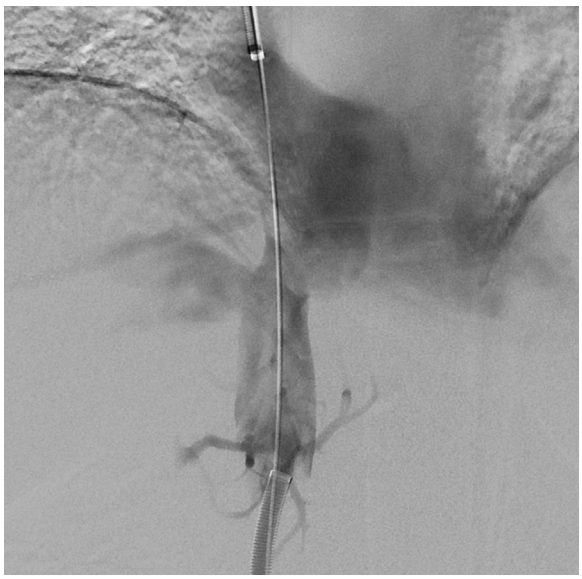

Fig. 4 – Venacavography shows restoration of IVC patency following angioplasty to 18 mm. IVC, inferior vena cava.

Following successful sharp recanalization, we performed IVC angioplasty to 18 mm with multiple balloons over 2 scheduled sessions to minimize the likelihood of IVC injury. Other operators may advocate for a more cost-efficient approach utilizing fewer balloons in a single treatment session; however, we elected to proceed cautiously.

A maximum balloon diameter of 18 mm was selected as this was the diameter of the IVC immediately inferior to the membranous occlusion.

Huang et al [25] evaluated long-term outcomes in patients with membranous and segmental IVC occlusion following angioplasty ± stenting. They reported overall cumulative 1-, 3-, and 5-year primary IVC patency rates of 98%, 91%, and 84%, respectively, in the membranous IVC occlusion group. For angioplasty alone, the overall cumulative 1-, 3-, and 5-year primary IVC patency rates were 98%, 91%, and 83%, respectively. Patients with postangioplasty IVC recoil greater than 50% or a residual IVC-right atrium pressure gradient greater than 15 cm H_20 were treated with self-expandable stents [25]. The overall cumulative 1-, 3-, and 5-year primary IVC patency rates for patients requiring stenting were 100%, 89%, and 89%, respectively [25]. We did not deploy a stent across the site of membranous IVC occlusion given the satisfactory result with angioplasty alone.

In summary, percutaneous sharp recanalization of a membranous IVC occlusion with balloon angioplasty led to successful restoration of IVC patency. Utilization of a balloon as a needle target is a helpful procedural adjunct and should be considered for similar future cases as it facilitates accurate and precise needle placement and rapidly confirms intravascular needle positioning following sharp recanalization.

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