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Case Report

Budd–Chiari syndrome after liver transplantation resulting from inferior vena cava occlusion at the suture line

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ABSTRACT

A 64-year-old male with Budd–Chiari syndrome (BCS) due to inferior vena cava (IVC) occlusion after liver transplant presented with massive ascites and lower extremity edema. He was found to have chronic total occlusion of the supra-hepatic IVC with thrombosis in the infra-hepatic IVC, hepatic, renal, and iliac veins. Attempts to recanalize the occlusion by multiple operators failed. He was not a surgical candidate. The patient underwent venography of the IVC, and placement of a McNamara catheter for catheter-directed thrombolysis on the first day. The second day, he underwent right internal jugular access with contrast injections to mark the superior aspect of the occlusion via a Multipurpose catheter. An adult transseptal needle (Bard Electrophysiology Division C. R. Bard, Inc., Lowell, MA, USA) was used to create a tract through a 6 French Raabe Sheath and traverse the occlusion. A 10-mm Snare (Cook, Bloomington, IN, USA) cranially retracted the guidewire. Intravascular ultrasound was performed to further delineate the diameter of the IVC at the lesion before dilation with a 6.0 mm × 40 mm PTA balloon and a 10 mm × 29 mm Palmaz Stent (Cordis Corporation, Bridgewater, NJ, USA) deployment. The patient lost 24.6 kg in 2 weeks with resolution of ascites and pedal edema.

<**Learning objective:** This case provides a unique approach to percutaneous intervention of inferior vena cava chronic total occlusion in the setting of Budd–Chiari syndrome post-liver transplant. There was use of an inferior and superior marker followed by use of transseptal needle to transverse the occlusion followed by balloon dilation and stent placement. While the disease and intervention have been described, the use of dual cranial/caudal markers and use of transseptal needle is unique to this particular case.>

Introduction

Inferior vena cava (IVC) occlusion complicating liver transplantation can cause severe morbidity in patients including portal hypertension leading to abdominal fullness, weakness, abdominal pain, lower extremity edema, hepatomegaly, splenomegaly, ascites, distended abdominal vein, leg ulcers, and more rarely gastrointestinal bleeding, jaundice, and hepatic encephalopathy [1].

Initial management of symptoms from IVC occlusion is medical therapy with diuretics and anticoagulants. In some cases fibrinolytic therapy for acute thrombosis might also be beneficial. IVC occlusion post-liver transplantation has been reported to be infrequent at 1–6% (especially with piggyback anastomosis), but can lead to portal hypertension, renal failure, pedal edema, graft loss, and even death depending on the level of occlusion [2–5]. Surgical porto-systemic shunt placement has been proposed, and liver transplantation is also an option. Balloon dilation of IVC occlusion is rarely successful because of rupture of new anastomosis, acute re-occlusion, and restenosis. IVC stent placement has been used for chronic occlusion in patients with Budd–Chiari syndrome (BCS) whose symptoms were refractory to conservative management [1–3,6–9]. Patients who underwent IVC stent placement were shown to have improvement in venous outflow [9]. IVC stenting with preceding catheter-directed thrombolysis has shown to be beneficial [9–11].

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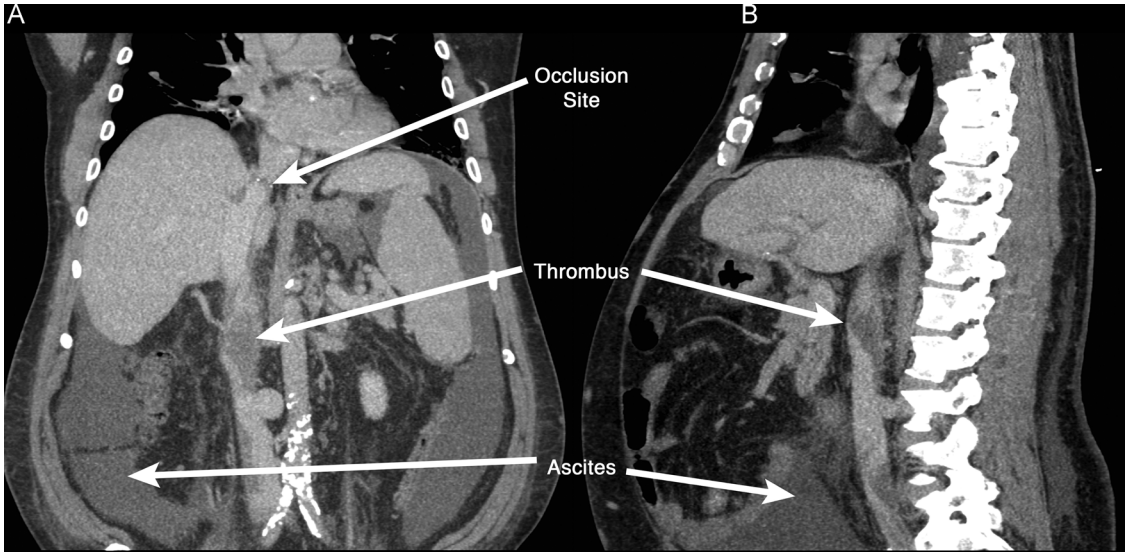

Fig. 1. Inferior vena cava occlusion below the hepatic transplant with multiple areas of non-occlusive thrombus is present on computed tomography venogram. Ascites can be visualized as well. Additionally, there is heavy calcification present on the iliac arteries.

Case report

A 64-year-old African American male 9 years post-orthotopic liver transplantation who had IVC occlusion and BCS with chronic pedal edema and ascites worsening over a 3-month period was referred to interventional cardiology for evaluation and treatment.

The patient had hepatitis C with cirrhosis and underwent liver transplant in 2003. He developed massive ascites, abdominal distention, and lower extremity edema over the 3 months that he could not ambulate and was restricted to his bed. He had gained approximately 27 kg. An ultrasound of the liver showed patent main portal vein with partially occlusive thrombus in the IVC and renal veins bilaterally (as seen in Fig. 1), hepatomegaly, and a large volume of ascites. Along with diuretics, he was put on anticoagulation as an outpatient. A venogram showed supra-hepatic IVC occlusion and attempts to cross the occlusion were unsuccessful by several operators. A vascular surgeon then saw him but no surgical intervention was possible because the supra-hepatic portion of his IVC had multiple collaterals to the superior vena cava and right atrium (RA). He was then referred to interventional cardiology.

On presentation, the patient weighed 128 kg. He had no jugular venous distention. His abdomen was markedly distended with fluid wave, ascites, marked pitting edema involving his entire lower extremity, back, and abdomen up to his chest wall. He also had venous stasis dermatitis of his lower extremity. A computed tomography venogram of his abdomen was performed as shown in Fig. 1. The imaging showed ascites, right pleural effusion, and supra-hepatic IVC stenosis 1 cm in diameter axially and 7 mm in length. There were multiple areas of large clots in the infra-hepatic IVC right around his anastamotic site with thrombus extending into the left renal vein and at the bifurcation of the iliac veins. His medications on presentation included losartan 50 mg daily, felodipine 10 mg daily, furosemide 40 mg twice a day, insulin formulations for his diabetes mellitus, tamsulosin for his benign prostate hypertrophy, levothyroxine for hypothyroidism, and pantoprazole for his gastroesophageal acid reflux.

Day 1: On 1/9/2013, the patient was brought to the catheterization laboratory and a 6 French sheath was inserted in right common femoral vein. An IVC venogram showed complete occlusion of the hepatic IVC at the liver transplant anastomosis site as well as partially thrombosed bilateral common iliac veins and infra-renal IVC as shown in Fig. 2A. Left renal vein was subtotally occluded. A McNamara catheter was inserted into the left renal vein and recombinant tissue plasminogen activator infusion begun through the McNamara catheter at 1 mg/h and the sheath at 1 mg/h. The 6 French sheath was sutured in place and low-dose heparin was also infused intravenously. The patient was transferred to the cardiac intensive care unit.

Day 2: Repeat venography showed no improvement on 1/10/2013, with persistent supra-hepatic IVC occlusion around the surgical anastomotic site, persistent thrombus in the infra-hepatic IVC and left renal vein thrombosis. Multiple attempts to traverse the IVC occlusion with numerous wires (such as .035 mm × 180 mm Stiff Angled Glidewire, .035 mm × 190 mm followed by a .035 mm × 300 mm Supracore Wire, .014 mm × 182 mm Choice Xs Guidewire, 14 mm × 190 mm BMW Guidewire) and catheters failed (including 6 French IMA catheter, 4.1 French Multipurpose catheter, 6 French DVX CUS Catheter, 6 French 125 cm MP Vista Guide Catheter). A 6 French sheath was inserted into the right internal jugular (IJ). A multipurpose catheter was inserted in the IJ and positioned in the RA just above the IVC occlusion as shown in Fig. 2B.

The femoral sheath was exchanged for an 8FR 55 cm Raabe Sheath (Cook, Bloomington, IN, USA) followed by insertion of 8FR Mullins Sheath (Bard, Louisville, CO, USA). A transseptal needle was introduced through the sheath and was used to transverse the supra-hepatic IVC occlusion with fluoroscopic guidance of the target more proximally as marked by the catheter from the IJ as shown in Fig. 3. The computed tomography scan was used in addition to the catheter position to confirm where to direct the needle from the IVC to the RA (Fig. 1). Once a tract was created, the guidewire was snared in the RA with a 10-mm Snare (Cook). Intravascular ultrasound was then performed to assess the diameter of the IVC prior to dilation and stenting. A Mustang 6.0 mm × 40 mm balloon (Boston Scientific, Natick, MA, USA) was inflated at 10.0 atmosphere followed by deployment of a 10 mm × 29 mm Palmaz Stent on a 20 mm × 4 cm balloon (BIB Balloon Cordis Corporation, Bridgewater, NJ, USA) in the IVC occlusion (Fig. 4). Post-intervention, the IVC was completely patent at the level of the lesion with brisk flow as shown in Fig. 5.