

Single-access liver floss technique with antegrade hepatic vein access and recanalization in Budd-Chiari syndrome

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ABSTRACT

A 14-year-old boy presented with several months of increasing abdominal girth and fatigue. Imaging confirmed massive ascites and hepatic congestion secondary to central hepatic venous obstruction. Several large intrahepatic collateral veins were seen draining via caudate and emissary veins. After an unsuccessful attempt at retrograde recanalization utilizing intravascular ultrasound, the right hepatic vein was recanalized in an antegrade fashion by way of a prominent caudate collateral vein, and subsequently stented. We herein discuss the established treatment options for Budd-Chiari syndrome and describe our experience employing a single-access liver floss technique.

Budd-Chiari syndrome encompasses a variety of conditions, all characterized by hepatic outflow obstruction but heterogeneous in etiology and morphology of venous occlusion as well as clinical manifestations of hepatic congestion. Prompt, appropriate treatment improves prognosis. A transjugular intrahepatic portosystemic shunt (TIPS) may be created to manage sequelae of portal hypertension and provide a bridge to transplant if significant hepatic injury has occurred. Liver transplant may be the sole treatment option if the disease is identified in late stages. When early in the disease process, however, parenchymal damage may be mitigated by reestablishing hepatic venous outflow which has classically been achieved by transjugular or percutaneous transhepatic access approaches when transjugular approaches prove infeasible.

TECHNIQUE

A 14-year-old male presented with a 6-month history of progressive exercise intolerance and abdominal distension. Evaluation at an outside hospital identified massive ascites and concluded with a provisional diagnosis of cirrhosis on the basis of ascites and the abnormal appearance of the liver on abdominal computed tomography (CT). The patient was subsequently transferred to our institution for further evaluation. On presentation, he was found to have a tense and distended abdomen without other signs of liver failure. Initial serologic workup including liver enzymes, ceruloplasmin, alpha-1 antitrypsin pi typing, autoimmune hepatitis serologies, and hepatitis B and C studies were negative, and he was referred to interventional radiology for paracentesis and transjugular liver biopsy.

Right internal jugular vein access was obtained. Attempts to cannulate the hepatic veins were unsuccessful despite several catheter configurations. A single plane digital subtraction cavogram was acquired, notable for the absence of reflux into normal hepatic veins at the level of the venous confluence (Fig. 1a). Additional evaluation of the inferior vena cava (IVC) and tributaries was performed with axial intravascular ultrasound (Volcano Visions PV .035, Philips), demonstrating the absence of right (RHV), middle (MHV) and left hepatic veins (LHV) at the hepatic vein confluence. An engorged caudate vein was identified, connecting to the IVC via a small orifice. A 5 French (F) angled hydrophilic catheter (Glidecath, Terumo) and 0.035-inch hydrophilic guidewire (Glidewire, Terumo) were introduced and used to cannulate the identified orifice and were subsequently advanced into the right intrahepatic venous system. Planar digital subtraction venogram (Fig. 1b) and three-dimensional cone beam CT venogram were acquired. These images confirmed seg-

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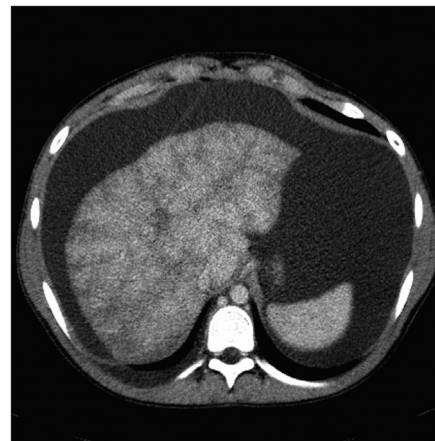
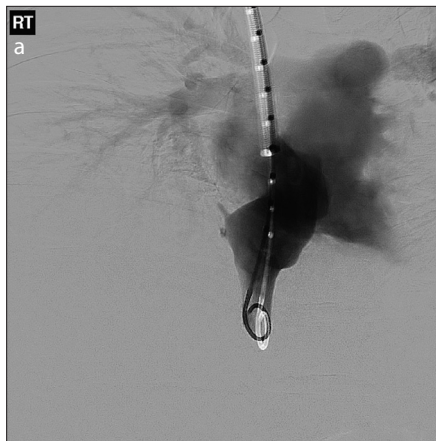


Figure 1. a, b. Imaging at time of attempted transjugular liver biopsy including (a) digital subtraction suprahepatic cavogram with no reflux into or inflow artifact from the hepatic veins and (b) digital subtraction venogram of the right hepatic vein demonstrating a long segment occlusion.

Figure 2. Axial image from a contrast-enhanced CT of the abdomen demonstrating large volume simple ascites and mottled parenchymal hepatic enhancement.

mental occlusion of the cephalad aspects of the RHV, MHV and LHV with hepatic venous drainage via intrahepatic venous arcades coalescing at the accessory caudate venous orifice. An intrahepatic confluence of veins was identified within the right liver, suspected to represent the inferior aspect of the RHV occlusion.

Exchange was made for a balloon occlusion catheter (Python, Applied Medical). Wedged hepatic, free hepatic and right atrial pressures were transduced, measuring 27 mmHg, 26 mmHg, and 9 mmHg, respectively (trans-sinusoidal gradient of 1 mmHg and post-sinusoidal gradient of 17 mmHg). The jugular access sheath and catheters were removed. Paracentesis was carried out with removal of 10 liters of serous ascites and intravenous albumin replacement.

The patient was presented at our interdisciplinary transplant hepatology meeting. The outside hospital abdominal CT images

were acquired in the interval, demonstrating nonvisualized central hepatic veins, mottled hepatic parenchymal enhancement and caudate hypertrophy typical of Budd-Chiari syndrome (Fig. 2). A consensus decision was made to attempt venous outflow augmentation and, if unsuccessful, endovascular portosystemic shunt prior to liver transplant listing.

The patient returned to interventional radiology two weeks after the initial venogram. Paracentesis was carried out with removal of 13 liters of serous ascites and intravenous albumin replacement. Right internal jugular vein access was obtained. Attempts to cannulate the cephalad aspects of the occluded hepatic veins via the IVC were unsuccessful despite several catheter configurations and placement of a stiffened cannula for improved caval wall apposition (Rösch-Uchida Transjugular Liver Access Set, Cook Medical). Appropriate positioning was confirmed with use of an 8 F right femoral vein approach side-firing intravascular ultrasound catheter (AcuNav, Acuson). The caudate vein orifice was then cannulated using a 5 F Glidecath and 0.035-inch Glide-wire which were then navigated to the right liver intrahepatic venous confluence. Using a 0.035-inch stiffened hydrophilic guidewire (GlideWire, Terumo) and the 5 F Glidecath, the occluded RHV was traversed under continuous intravascular ultrasound guidance. The wire was snared within the right atrium using a 6 F tri-lobed snare (EN Snare, Merit Medical Systems) from the existing right jugular sheath, achieving single access liver floss (Fig. 3a). The Glidecath was reintroduced in a retrograde direction (Fig. 3b) and the hydrophilic wire was exchanged for a stiff 0.035-inch guide wire (Amplatz Super-

Stiff, Boston Scientific). Angioplasty of the RHV was performed with a 6×40 mm angioplasty balloon (Mustang, Boston Scientific) (Fig. 3c). A 10×40×20 mm partially polytetrafluoroethylene-covered stent-graft (Viatorr, WL Gore and Associates) was deployed. The uncovered portion was placed within the right lobe intrahepatic venous confluence. The covered portions spanned the native, recanalized RHV. Completion venography confirmed position and demonstrated brisk flow to the right atrium. Completion post-sinusoidal pressure gradient was reduced to 2 mmHg. After sheath removal and appropriate hemostasis, the patient was placed on a heparin drip and subsequently transitioned to a therapeutic dose enoxaparin on the first postprocedure day.

The patient returned 6 weeks post-procedure for a transjugular liver biopsy, which revealed focal scarring and minimal perivenular fibrosis. An intra-procedural digital subtraction venogram confirmed a widely patent stent (Fig. 4). Intrahepatic venous arcades persisted but drained antegrade via the stent to the right atrium. Liver enzymes were stable and ascites had not recurred. He was transitioned from enoxaparin to aspirin 81 mg per oral once daily. An extensive hypercoagulability workup was unrevealing. Sequential ultrasounds, most recently 15 months post-procedure, have confirmed a widely patent stent (Fig. 5). The patient remains symptom free at the time of this report, now 2 years post-procedure, with self-reported excellent energy levels and no clinical evidence of recurrent ascites or liver dysfunction. Imaging surveillance of the stent will continue with yearly ultrasound.

Main points

- Budd-Chiari syndrome encompasses a heterogeneous group of disorders characterized by obstruction of hepatic venous outflow at any point from the small perisinusoidal hepatic veins to the right atrium.
- Prompt identification of Budd-Chiari syndrome and decompression of hepatic venous outflow minimizes damage to hepatocytes, and endovascular methods are preferred over surgery.
- Enlarged accessory hepatic and caudate veins are frequently present in Budd-Chiari syndrome and provide alternative routes into the intrahepatic venous system for recanalization of the central outflow obstruction.

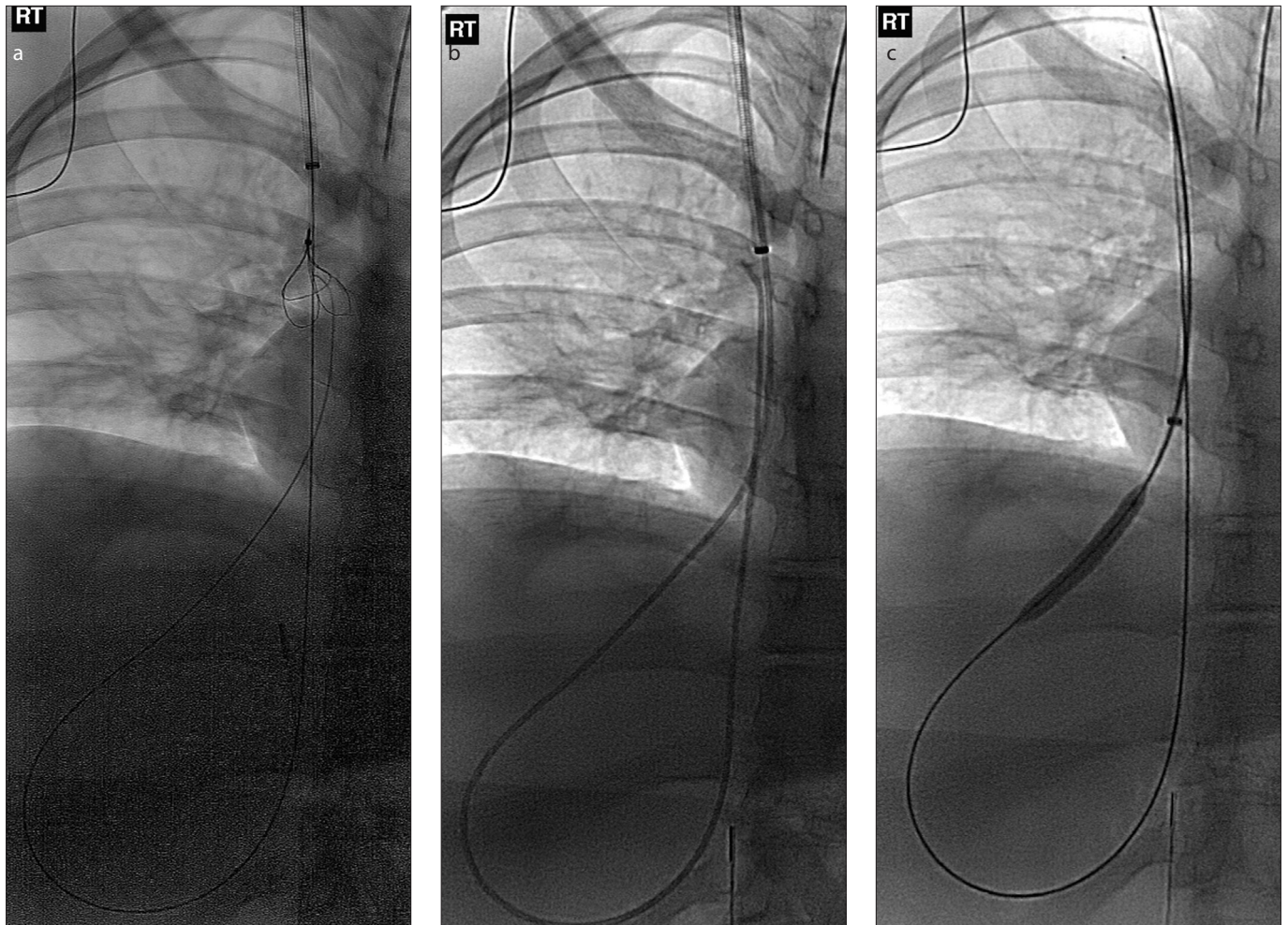


Figure 3. a–c. Intraprocedural fluoroscopic images including (a) antegrade wire recanalization of the occluded right hepatic vein segment and wire snaring within the right atrium, (b) directional reversal with a catheter inserted from the right jugular sheath, and (c) angioplasty of the occluded right hepatic vein segment.

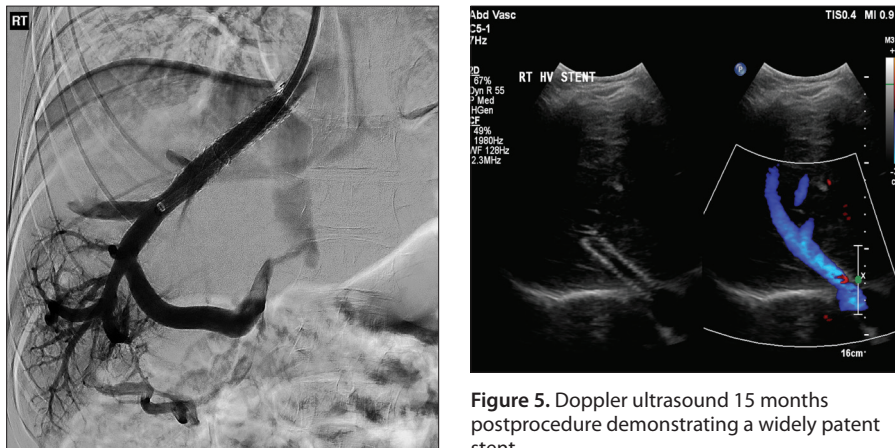


Figure 4. Digital subtraction venogram 6 weeks postprocedure demonstrating a widely patent stent.

Figure 5. Doppler ultrasound 15 months postprocedure demonstrating a widely patent stent.

Discussion

Budd-Chiari syndrome encompasses a heterogeneous group of disorders characterized by obstruction of hepatic ve-

nous outflow at any point from the small perisinusoidal hepatic veins to the right atrium (1). Persistent outflow obstruction results in increased sinusoidal pressure and hepatic congestion with subsequent hypoxic injury, hepatocyte necrosis, and progressive fibrosis. Patients with Budd-Chiari syndrome may

be asymptomatic and discovered incidentally or present with symptoms on a spectrum ranging from mild abdominal pain to fulminant liver failure. The severity of disease on presentation is dictated by the degree and rate of onset of obstruction, as well as the development of compensatory collateral outflow (2). Following diagnosis, the goal of treatment is to decompress hepatic venous outflow in a timely manner to minimize damage to hepatocytes. Without treatment, Budd-Chiari syndrome leads to liver failure and ultimately death.

The treatment options for Budd-Chiari syndrome are well established and include medical management, endovascular procedures, and surgical interventions. Medical management consists of anticoagulation for all patients, diuretics, low sodium diet, and paracentesis with albumin replacement. These therapies may alleviate symptoms temporarily but are not definitive treatments, and relying solely on medical

management is associated with poor long-term outcomes. The well accepted definitive treatments of Budd-Chiari syndrome are endovascular (hepatic vein recanalization, TIPS) and surgical (shunt creation, liver transplant) procedures (2). Endovascular methods of decompression are preferred over surgical shunt creation as they are associated with lower morbidity and mortality, and liver transplantation is limited by donor availability and is reserved for patients with irreversible liver damage and advanced hepatocellular dysfunction.

Both TIPS and hepatic vein recanalization provide good clinical outcomes with comparable patency and survival rates, but recanalization has lower procedural complication rates and is not associated with hepatic encephalopathy (3). TIPS can effectively reduce portal hypertension and serve as a bridge to transplant, but hepatic vein recanalization is the first-line endovascular treatment of choice. If recanalization fails, then the next step is to attempt a TIPS (4). However, TIPS may be considered the first-line treatment in the case of long segmental obstruction with diminutive intrahepatic veins due to high failure rates of recanalization in these settings (5). Hepatic vein recanalization and balloon angioplasty could traditionally be performed with or without subsequent stent placement, but stenting has been shown to decrease the restenosis rate and has become standard practice (6).

Hepatic vein recanalization has been classically achieved through internal jugular vein or percutaneous transhepatic approaches. Transjugular access with continuous ultrasound guidance and micropuncture access technique is now routinely performed and associated with few complications, even in patients with coagulopathy. Femoral vein access has also been used but may be technically challenging due to the acute angle between the hepatic veins and IVC (7). Percutaneous transhepatic access is more technically challenging and the possibility of potentially serious bleeding complications must be considered. One recent study of 93 patients with Budd-Chiari syndrome treated with transhepatic venous balloon angioplasty reported major complications in six patients (6.45%) (8). Transhepatic procedures of any kind are associated with elevated risks, particularly in patients with coagulopathy, liver dysfunction, and ascites. Ascites, frequently present in Budd-Chiari syndrome patients, is associated with a 30% risk of bleeding following percutaneous transhepatic portal vein access without

appropriate tract embolization. When gaining transhepatic access, an access point may be chosen in-line with the target hepatic vein outflow, augmenting ergonomics and the translation of the forces during recanalization. When the transhepatic access site is used exclusively for traversing the obstruction, with subsequent angioplasty and/or stent from the jugular approach, transhepatic access equipment size may be kept small caliber to minimize hepatic bleeding risk. Bleeding risk may be decreased further by tract embolization, of particular importance when larger transhepatic access is required or in the case of coagulopathic patients. However, avoiding transhepatic access completely essentially eliminates capsular bleeding risk as well as potential transpleural access related complications such as pleural effusion, pneumothorax, hemothorax, and bilothorax. When gaining transjugular access, the traditional route for recanalization involves retrograde catheterization of the desired hepatic vein through the obstructed segment. However, in some cases of extensive occlusion this may prove infeasible. Given the aforementioned risks associated with transhepatic access, devising another alternative approach may be desirable in these situations.

Case studies describing the successful utilization of alternative routes for accessing and recanalizing hepatic veins have been previously reported. Baptista et al. (6) described a similar single access liver floss technique, using a retrograde approach to reach the right hepatic vein via a patent caudate lobe accessory hepatic vein and an intrahepatic shunt. This approach required an engorged caudate lobe hepatic vein, which is frequent in patients with Budd-Chiari syndrome. Sun et al. (7) employed a femoral vein approach to cannulate enlarged accessory hepatic veins. They then used the accessory hepatic vein to reach occluded main hepatic veins through intrahepatic communicating branched veins and subsequently formed a loop to the IVC, allowing for successful recanalization in two separate cases of short segment hepatic vein occlusion. In addition to providing a route of access for recanalization of main hepatic veins, some larger accessory hepatic veins can be utilized/recanalized to independently provide adequate decompression in select cases of extensive occlusion of multiple hepatic veins (8). Although no complications were reported in these cases, we suspect caudate vein access risks venous avulsion and hemorrhage as well as damage to a critical route of hepatic venous drainage.

Endovascular procedures are the preferred definitive treatment of Budd-Chiari syndrome, and recanalization of obstructed outflow tracts is generally accepted as the ideal initial strategy. While traditional approaches involving internal jugular vein and transhepatic access are generally effective and relatively safe, certain situations may require alternative techniques to maximize procedural efficacy and patient safety. The anatomic configuration in the patient presented certainly enabled the techniques employed and will not be reproducible in all patients with Budd-Chiari syndrome, particularly those with difficult to cannulate caudate or emissary veins, tortuous intrahepatic venous collaterals or excessive distances from dominant intrahepatic veins to the native hepatic vein confluence.

In conclusion, the single access liver floss technique is an elegant technique for hepatic vein recanalization in Budd-Chiari syndrome and an attractive alternative to percutaneous transhepatic vein recanalization in patients with favorable venous anatomy.

Conflict of interest disclosure

The authors declared no conflicts of interest.

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