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 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.

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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 Glidcath and 0.035-inch Glidewire 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 Glidcath, 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 Glidcath 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 (Via- torr, 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 intraprocedural 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 ultrasonograms, most recently 15 months postprocedure, have confirmed a widely patent stent (Fig. 5). The patient remains symptom free at the time of this report, now 2 years postprocedure, 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.
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 (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 inciden- tally or present with symptoms on a spectrum ranging from mild abdominal pain to fulmi- nant 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 replace- ment. These therapies may alleviate symp- toms temporarily but are not definitive treatments, and relying solely on medical

Discussion

Budd-Chiari syndrome encompasses a heterogeneous group of disorders char-acterized by obstruction of hepatic ve-
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 micro-puncture 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 bilithorax. 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 lobe technique, using a retrograde approach to reach the right hepatic vein via a patent caudate lobe accessary 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 lobe 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.

References

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