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Pediatric Hepatobiliary Interventions in the Setting of Intrahepatic Vascular Malformations, Portal Hypertension, and Liver Transplant

Eric J. Monroe, MD,[*,†] and Giridhar M. Shivaram, MD[*,†]

Within the broad spectrum of pediatric hepatobiliary disorders, hepatic vascular malformations, portal hypertension, and hepatic transplant interventions pose numerous challenges. The role of interventional radiology within each of these conditions is discussed herein, beginning with endovascular management of high flow hepatic vascular malformations. Next, while becoming less common in adult populations, surgical portoportal and portosystemic shunts remain prevalent in many pediatric centers. Shunt anatomy is reviewed along with endovascular management techniques for shunt dysfunction. Next, the growing experience with pediatric transjugular intrahepatic portosystemic shunt placement is reviewed along with tips for success in pediatric patients. Finally, pediatric hepatic transplant interventions are discussed with technical notes pertinent to split liver anatomy.
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High Flow Hepatic Vascular Malformations

Congenital vascular malformations of the liver are a rare but important entity due to the potential for development of portal hypertension, liver dysfunction/failure, or heart failure. High flow malformations, or those malformations with an arterial component, present the highest risk for development of these complications. A multidisciplinary approach is required for accurate diagnosis and effective management. Patient presentation may occur in the prenatal period, with detection of liver or secondary cardiac abnormalities on obstetric US. While advanced prenatal imaging such as fetal MRI can potentially characterize a vascular liver abnormality before birth, Doppler US and CT/MRI imaging in the postnatal period are critical to establishing an accurate diagnosis. Neonates with hepatic vascular abnormalities can present with anemia, hepatomegaly, and signs of cardiac failure.[1] Infants in whom a prenatal diagnosis was not established may present in delayed fashion with these signs as well as failure to thrive. If heart failure is severe, management of patients in the neonatal or pediatric intensive care unit for respiratory and circulatory support is almost always required.

The first step in treatment planning is careful imaging diagnosis of the hepatic vascular abnormality. Broadly, lesions can be classified into high flow or low flow physiology. High-flow lesions by definition have an arterial component, and these include arterioportal or arteriovenous malformations or vascular hepatic tumors including but not limited to infantile or congenital hemangioma, hemangioendothelioma, and metastatic tumors.[1,2] Low flow lesions include portosystemic shunts, venous or capillary malformations, and hypovascular tumors without shunting.[3] At the authors' institution, Doppler US is performed for initial noninvasive evaluation of the hepatic lesion followed by CT or MR angiography.[4] Once a lesion has been established as high flow based on Doppler waveform analysis or arterial supply to the lesion is seen on cross-sectional imaging, lesion morphology is assessed to characterize it as mass-like or nonmass-like, with malformations usually appearing more diffuse and without an associated circumscribed soft tissue component. Malformations are usually nonmass-like because

*Department of Radiology, Seattle Children's Hospital, Seattle, WA.
†Department of Radiology, University of Washington, Seattle, WA.
Address reprint requests to Eric Monroe, MD, Department of Radiology, Seattle Children's Hospital, MA.7.220 - Radiology, 4800 Sand Point Way NE, Seattle, WA 98105. E-mail: eric.monroe@seattlechildrens.org

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of, often innumerable, ill-defined arteriovenous connections without an associated prominent interstitial abnormality. Additionally, for hepatic hemangiomas, the presence of extrahepatic hemangiomas or GLUT-1 staining on biopsy can help differentiate between these entities. Additional information gleaned on preprocedure imaging include assessment of portal hypertension, ascites, extent of liver parenchyma involved, and angioarchitecture of high-flow lesions. Arterialized portal venous flow can lead to portal hypertension, altered portal Doppler waveform, and sequelae such as splenomegaly with sequestration, ascites, or variceal bleeding.

Arterioportal malformations are congenital vascular abnormalities of the liver characterized by inappropriate connections between hepatic arteries and portal veins that bypass the sinusoidal architecture of the liver parenchyma.[5] Lesions can be focal or diffuse but often involve the left hepatic lobe. Associated arterial to hepatic venous shunting or shunting between portal and hepatic veins may also be present in complex malformations. Arterioportal fistulas, when congenital, can be considered as a subset of arterioportal malformations but with a solitary or single connection between artery and vein. Like other arteriovenous malformations, the angioarchitecture of arterioportal malformations is characterized by a central nidus which represents the extracapillary connections between artery and vein. Any intervention targeted toward the malformation must involve obliteration of the nidus, as proximal arterial embolization without embolization of the nidus often leads to aggressive collateral vessel recruitment.[6]

After completion of preprocedure imaging, multidisciplinary evaluation should occur to discuss the options of medical management of heart failure, surgical therapy, or interventional approaches. While surgical resection or transplantation is often appropriate for management of hepatic tumors, interventional treatment is usually a first-line approach for arterioportal malformations.[5] Often, medical management of heart failure can allow for stabilization and growth of the child, which decreases the risk of procedural complications.

For arterioportal malformations, transarterial and/or transhepatic portal venous embolization can be performed depending on the angioarchitecture of the lesion (Fig. 1). Often, there is a dominant portal venous varix or aneurysm just beyond the nidus of the malformation, which can be percutaneously accessed. Regardless of the approach, embolization is usually performed with a combination of coils and liquid embolic agents. Particulate agents are avoided because of the high risks of systemic nontarget embolization and ischemic damage. For lesions with fewer than 3 supplying arteries, a transarterial approach can be employed for complete obliteration of the nidus.[5] However, if there is complex arterial supply to the lesion, which is more common, a combination of transarterial and transportal embolization is usually required. Key principles of effective embolization are obliteration of the nidus and portal venous varix, or connection, between hepatic artery and portal vein, and avoiding embolization of normal arterial or portal supply uninvolved in the lesion. Excessively proximal embolization can also lead to treatment failure, as only temporary cessation of flow within the lesion is achieved, with recurrence occurring due to development arterial collateralization, which may be hard to access for future procedures.

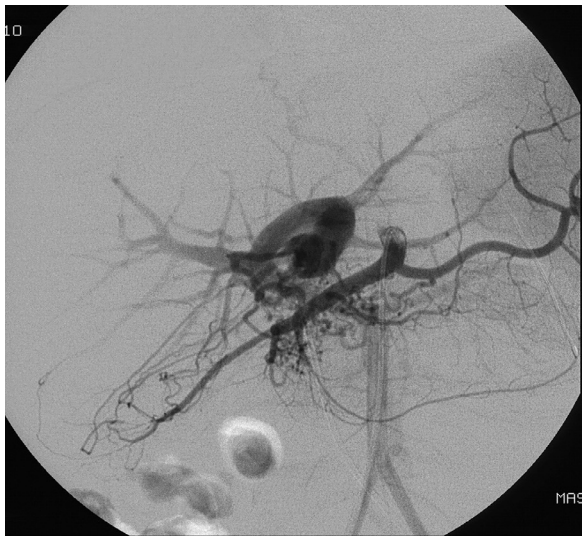

Figure 1 Arterioportal malformation. A 6-month-old male with Trisomy 21, portal hypertension, and variceal bleeding was found to have a hepatic vascular abnormality on Doppler US. Conventional celiac arteriogram showed a complex arterioportal malformation with a left portal vein varix. Embolization and surgical hepatic artery ligation were performed, followed by surgical splenorenal shunt creation to address persistent portal hypertension. The patient died at 4 years of age due to complications of short gut syndrome.

The authors' preference for embolization technique is to use a coaxial system where a guiding catheter is inserted into the celiac or superior mesenteric arteries, depending on supply to the lesion, with placement of a coaxial microcatheter for interrogation and embolization of more distal feeding arteries. For neonates weighing less than 10 kg, the authors often insert the microcatheter directly from femoral artery access, with or without a 3 French sheath, to decrease the risk of catheter-related occlusion of the femoral artery. For patients weighing less than 10 kg who are not being imaged for bleeding, the authors empirically bolus heparin 25-50 U/kg after access to reduce the risk of femoral artery thrombosis.

Care must be taken to not perform embolization that is too proximal as this can lead to only temporary decrease in translesional flow. Collateralized arterial flow can form following proximal embolization and access into these collateralized channels for future embolization can be difficult if not impossible. If there is a sizable nidus, liquid embolics such as n-BCA glue or Onyx (maximum dose 0.2 mL/kg) can be delivered to effectively penetrate into the nidus. Glue reflux into normal arteries should be avoided as well as inadvertent gluing of the delivery catheter. However, if there is not a sizeable nidus, coil embolization should be considered as distally as possible without extending coil material into normal portal veins. For complex arterioportal malformations, staged embolization should be considered. An additional important consideration is abrupt decrease in arterialized portal flow

following embolization. This can lead to portal thrombosis and significant morbidity.

For complex malformations, complete nidal obliteration is often difficult. Multiple staged procedures may be required as well as adjunctive surgical resection. For lesions other than arterioportal malformations, including venous malformations and hepatic tumors, transarterial embolization may be useful in decreasing intralesional shunting. For hepatic tumors, definitive therapy is usually surgical resection. Regardless of the type of lesion being addressed, if there is diffuse involvement of the liver parenchyma with resultant hepatic failure, liver transplantation may be eventually required.

For patients not undergoing liver transplantation, imaging and clinical follow-up is targeted towards detecting early recurrence of the malformation and assessing the need for repeat intervention. At the authors' institution, Doppler ultrasound is used as the primary modality for post-treatment surveillance. For arterioportal malformations, parameters assessed include persistent arterialization of portal flow, improvement or persistence of splenomegaly, and recurrence of the arterioportal nidus. Any of these findings should prompt repeat evaluation either with CT, MR, or conventional arteriography. The advantage of conventional arteriography is that more subtle areas of recurrence can be detected due to intra-arterial injection of contrast and treatment can be performed at the same setting.

In summary, high flow hepatic vascular malformations are very complex lesions that present early in life, often carry a high morbidity, require extensive diagnostic work-up, and necessitate a multidisciplinary team for treatment planning.

Portal Hypertension

Uncompensated obstruction of portal venous flow system results in portal hypertension with associated risks of variceal gastrointestinal hemorrhage, ascites, exudative enteropathy, hepatic encephalopathy, splenomegaly, and thrombocytopenia (sequestration).[7,8] Flow impedance typically occurs at the intrahepatic level but may result from obstruction within the hepatic venous outflow tract (Budd-Chiari syndrome) or proximally within the portal venous system. Unlike the adult population where viral hepatitis and alcoholic cirrhosis prevail, pediatric intrahepatic obstruction most commonly results from biliary atresia, congenital hepatic fibrosis, alpha-1 antitrypsin deficiency, autoimmune hepatitis, and cystic fibrosis-associated liver disease.[7] In adults, portosystemic pressure gradients >10-12 mm Hg are associated with the formation of varices, gastrointestinal bleeding and liver decompensation.[8,9] Normal portosystemic pressure gradients in children, however, are not well established. Baveno V Consensus Workshop on Methodology of Diagnosis and Therapy in Portal Hypertension recommends extrapolating from adult guidelines while acknowledging potential flaws due to insufficient literature specific to pediatric patients.[8]

Extrahepatic portal venous obstruction (EHPVO) presents acutely (thrombosis, thromboembolism) or chronically as a result of remote portal vein occlusion and subsequent cavernous transformation.[10] Acute portal thrombosis and thromboembolism in pediatric patients can be safely and effectively treated with catheter directed thrombolysis.[11] Chronic EHPVO represents the most common etiology of pediatric upper gastrointestinal bleeding in developing countries and presents unique treatment challenges.[12,13] Hepatic parenchymal function is often well preserved.

Flow diversion for the alleviation of portal hypertension carries risks of decompensated liver disease, hepatic encephalopathy and hepatopulmonary syndrome. The pediatric end−stage liver disease model was developed and implemented to facilitate graft allocation among pediatric orthotopic liver transplant candidates.[14] Beyond prediction of transplant waitlist mortality and post-transplant survival, the authors apply the pediatric end−stage liver disease to anticipate tolerance of various flow diverting treatments of portal hypertension, particularly transjugular intrahepatic portosystemic shunt (TIPS). Given the relative paucity of pediatric TIPS experience, the authors extrapolate from MELD score risk stratification during consultation. Pediatric patients with portal hypertension of any cause are well served by local expertise and interdisciplinary collaboration between pediatric interventional radiology, surgery, gastroenterology, and hepatology.

Surgical Shunts

Surgical shunts for the alleviation of portal hypertension include portal bypass from the coronary vein or remnant portal venous confluence to the left portal vein via the hepatic Rex recess (meso-Rex shunt) or a variety of portosystemic conduits including splenorenal, splenocaval, or mesocaval shunts. The meso-Rex shunt requires continuity of the intrahepatic left and right portal veins for adequate function. When feasible, the resultant bypass of a main portal vein occlusion preferred over portosystemic shunting as it reestablishes physiologic portal flow and may be more effective in reducing hypersplenism.[15] While shunt creation is ultimately surgical in these instances, the interventional radiologist may provide preoperative anatomic characterization via wedged hepatic vein carbon dioxide portography or percutaneous portal venous imaging and may later assist with postoperative endovascular rescue and maintenance of meso-Rex shunts.[16-20] Portal venous access in the later situation is typically percutaneous transhepatic or trans-splenic, with subsequent procedural considerations generally paralleling the discussion below.

Surgical portosystemic shunts may be created for cases of EHPVO not amenable to meso-Rex shunt or patients with intrahepatic portal hypertension in whom surgical shunt creation is felt to preferable to TIPS placement. In cases of a patent portal vein, the distal splenorenal shunt carries less risk of overshunting and preferred over mesocaval shunting as it offers a more balanced reduction in portosystemic pressure gradients. Surgical shunts are effective in the alleviation of portal hypertension and may delay the need for liver transplant in some pediatric patients.[21-27]

Scheduled clinical follow-up with surveillance imaging affords detection and management of shunt dysfunction

prior to recurrent complications of portal hypertension. Specifically, clinical evidence of splenic sequestration and/or elevated peak splenorenal anastomotic velocities on Doppler US should prompt catheter venography and endovascular intervention.[28] While splenorenal shunts may be approached via the percutaneous trans-splenic approach, systemic side access (typically femoral venous) is felt to be safer with respect to bleeding complications and provide better ergonomics for catheterization and intervention.

For typical distal splenorenal shunts, reverse curve catheter access from the left renal vein facilitates access, naturally extrapolated from phrenico-adrenal access techniques during adrenal vein sampling or spontaneous splenorenal shunt access during balloon occluded transvenous obliteration procedures given similar anatomic configuration and shunt outflow on the superior aspect of the renal vein. If angioplasty is required, the authors heparinize 50-100 U/kg (goal activated clotting time >250 s) all patients unless contraindicated. Stenting is generally reserved for stenoses following angioplasty (Fig. 2) and the authors transition patients to long-term antiplatelet therapy postprocedurally. Aspirin is used primarily, with alternative agents such as clopidogrel reserved for patient with enteric or other intolerances of aspirin. Stent selection is individualized; while self-expanding stents more readily assume any rounded configurations encountered, balloon expandable stents offer more precise placement, and later expansion to accommodate for patient growth.

Transjugular Intrahepatic Portosystemic Shunts

TIPS creation effectively reduces portosystemic pressure gradients and robust literature supports its use in adults for managing a wide variety of portal hypertensive sequela, most commonly treatment and prevention of acute gastroesophageal variceal bleeding.[29,30] Comparatively limited experience in the pediatric population has demonstrated similar technical success and clinical efficacy and expert consensus supports its use for the narrow indications of esophageal variceal bleeding refractory to pharmacologic and endoscopic management.[8,31-39] Reported technical success rates have been high (78%-100%) and significant experience in both adult TIPS placement and pediatric interventional radiology is recommended to replicate these results.[31-39] Technical challenges to TIPS creation in small children abound.[40,41] While a full discussion is beyond the scope of this review, several of the most pertinent nuances deserve attention.

EHPVO complicated by variceal bleeding may not always be amenable to meso-Rex shunt creation due to extensive intrahepatic portal venous occlusions. Although once thought infeasible, TIPS creation in EHPVO offers potentially life-saving portal decompression. Transhepatic or trans-splenic portal venous access facilitate transjugular portal venous access via snare or balloon targeting in cases of EHPVO. Less commonly, a dominant channel may be targetable via intravascular ultrasound (IVUS). When a dominant channel is not appreciable, authors favor trans-splenic snare targeting as it allows withdrawal of the snared access wire

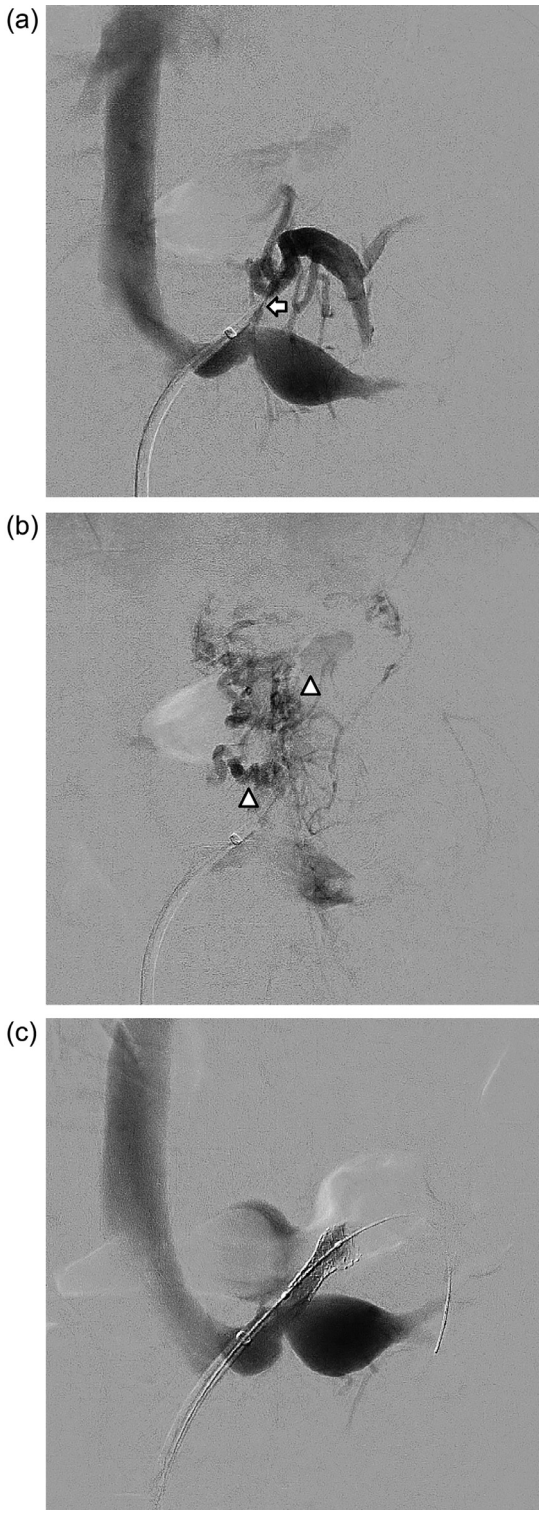

Figure 2 Splenorenal shunt angioplasty and stent. A 3-year-old male with cavernous transformation postsynthetic interposition graft distal splenorenal shunt presented with splenic sequestration and elevated shunt velocities on surveillance ultrasound. Early (a) and late (b) digital subtraction venographic images demonstrated severe shunt stenosis (arrow) with splenogastric collateral drainage (arrowheads). The stenosis was refractory to balloon angioplasty. An 8 × 20 mm balloon expandable stent (Formula 418, Cook, Bloomington, IN) was placed (c) with restoration of brisk splenic vein outflow, nonvisualization of previous collateral pathways and reduction in the portosystemic gradient from 15 to 2 mm Hg.

through the portal confluence and out the splenic access, rapidly surmounting the tortuosity of cavernous transformation and achieving a highly stable "body floss" access. In a series of 28 pediatric patients with EHPVO, Lv et al achieved a 60.1% technical success rate for TIPS creation.[37] Stent dysfunction at 1- and 3-years (24.3 and 31.9%, respectively) and rebleeding after successful TIPS creation (41.2%) were notably high in this series, although likely attributable in part due to the use of bare metals stents rather than endografts.

Regardless of main portal vein patency, side-firing IVUS (AcuNav, Acuson, Mountain View, CA) has dramatically augmented TIPS placement in the authors' practice and enabled the additional option of transcaudate direct intrahepatic portosystemic shunt creation. Patent portal veins and parenchymal needle passage are readily visible (Fig. 3), reducing portal access attempts and procedure time. Guidance through anatomic challenges such as small anatomy, cavernous transformation and Kasai portoenterostomies, decreasing procedural risks in complex cases. In addition to transjugular portal venous targeting, IVUS provides opportunities for subsequent procedural guidance and fluoroscopic and contrast dose reduction. The authors use IVUS for confirmation of wire passage through the portal confluence and into the superior mesenteric vein as well as fluoroscopic demarcation

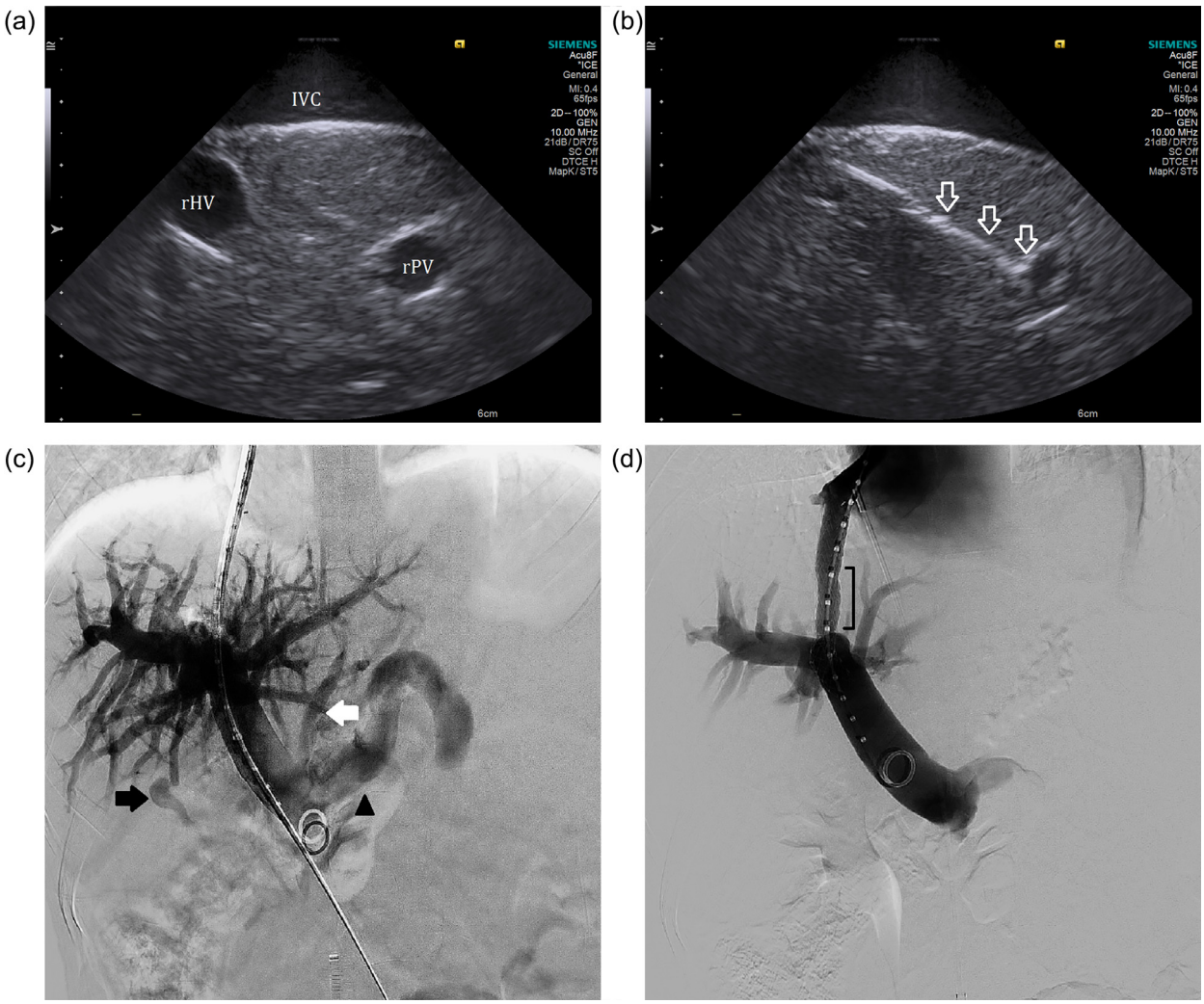

Figure 3 Transjugular intrahepatic portosystemic shunt (TIPS). A 17-year-old male with autoimmune hepatitis and portal hypertension presented with recurrent variceal bleeding postvariceal banding. Planning intravascular ultrasound (IVUS) image (a) following placement of an 8 French side-firing probe (AcuNav, Acuson, Mountain View, CA) within the inferior vena cava (IVC) demonstrated the anticipated parenchymal tract between the right hepatic vein (rHV) and posterior branch of the right portal vein (rPV). Following sheath placement into the right hepatic vein, the access needle and catheter (Rösch-Uchida Liver Access Set, Cook, Bloomington, IN) were introduced and directed into the target portal vein (b) (open arrows). Subsequent portal venogram (c) demonstrated a patent portal vein with stigmata of portal hypertension including an enlarged and nearly static splenic vein (black arrowhead), reversed coronary vein (white arrow), and recanalized paraumbilical vein (black arrow). Portal venogram (d) following placement of a 10 mm controlled expansion stent-graft (Viatorr CX; WL Gore and Associates, Flagstaff, AZ) demonstrated a patent TIPS with hepatopetal flow. The central aspects of the TIPS were left underdilated to 8 mm (bracket) following an acceptable gradient reduction from 12 to 4 mm Hg.

of the hepatic venous confluence, thus obviating the need for sheath withdrawal and dual injection venogram for stent graft selection.

Following portal access, the authors place partially Polytetrafluoroethylene (PTFE)-covered stent-graft used for TIPS creation (Viatorr; WL Gore and Associates, Flagstaff, AZ) with rare exceptions. Therefore, standard Rösch-Uchida and Haskal sets, rather than pediatric modifications, are used with their included 10 French sheath necessary for Viatorr delivery. Therapeutic pediatric portosystemic gradient reductions following TIPS are not well established and gradient reduction goals are generally extrapolated from the adult literature. The authors strive for a final gradient less than 10 or, if bleeding is encountered with an initial portosystemic gradient less than 10 mm Hg, a 50% reduction in the gradient. Legacy Viatorr stent-grafts are well known to passively expand to their nominal diameter within several weeks of placement, risking overshunting and related complications.[42,43] A newer version (Viatorr CX; WL Gore and Associates, Flagstaff, AZ) features an additional PTFE sleave to resist autodilation. If clinically successful as hoped, the modification may lessen the need for clever techniques to combat this issue at the time of TIPS creation or after overshunting has developed.[44-48]

Following successful TIPS creation, a baseline US is acquired in 4-7 days to allow for dissipation of stent-graft air artefact. Surveillance exams are then obtained at 1-, 6-, and every 12-month thereafter. Normal appearance following TIPS in pediatric patients is not well established. Instead, the authors partially extrapolate from the adult experience but pay close attention to interval changes from an individual patient's baseline. Clinical signs of shunt dysfunction trump indeterminate US findings and warrant venographic evaluation.

While endovascular technical advances and growing experience with pediatric TIPS may lessen the role of surgical portosystemic shunts, surgical shunts will likely remain an important treatment option for the foreseeable future. Henderson et al compared TIPS and distal splenorenal shunts in adult patients in a multi-institutional randomized trial, finding similar clinical outcomes but a higher rate of reintervention in patients undergoing TIPS.[49] However, uncovered nitinol stents rather than the modern standard of PTFE-covered TIPS endografts were used exclusively in this study and have been shown to have inferior durability as compared to contemporary partially-covered PTFE stents.[50] Length of stay in patients undergoing surgical shunting was notably longer. Comparative studies regarding clinical efficacy and durability in pediatric patients are notably lacking and the available relatively small retrospective series reporting on each individual modality are potentially limited by institutional biases and/or expertise.

Liver Transplant Interventions

Biliary

Due to small patient and graft size, pediatric liver transplantation is often complicated by anastomotic or nonanastomotic biliary strictures.[51] As the primary supply to the biliary system is via hepatic arterial perfusion, the usual etiology of these strictures is arterial insufficiency. Biliary complications can occur in up to 40% of pediatric transplant recipients of split grafts.[52]

Mechanical stricture at the biliary anastomosis usually present early in the postoperative transplant course and is detected by clinical signs of jaundice, acholic stool, and/or rising serum bilirubin levels or other biochemical markers of liver function. If detected in the first few weeks of transplantation, surgical correction is often warranted.[53] If detected later, percutaneous or endoscopic intervention can be performed. On the other hand, ischemic biliary strictures related to perioperative arterial insufficiency can have a delayed presentation. Ischemic biliary strictures are often multifocal and can lead to an insidious worsening of transplant liver function due to developing cholestasis and inflammatory damage to the liver parenchyma.[53] These strictures may only be partially occlusive, resulting in lack of appearance of overt clinical signs of jaundice or acholic stools. The diagnosis is usually made with a combination of altered biochemical markers of liver function and biopsy showing ductular proliferation or cholestasis. These patients also may have a history of perioperative arterial insufficiency. The main differential diagnosis in these patients is rejection.

Once a suspicion of biliary obstruction is present, initial evaluation can be performed with US or Magnetic resonance cholangiopancreatography (MRCP). However, transplant biliary obstruction does not always result in marked dilation of peripheral bile ducts and may be difficult to detect on US. Additionally, central bile ducts can be difficult to visualize on US. In contrast, MRCP is a more sensitive technique for evaluation of central biliary ducts and may be useful in cases where an invasive procedure is not felt to be desirable as a first step. If central biliary strictures are confirmed, endoscopic evaluation and possible treatment is generally preferred as this avoids percutaneous access into the liver, which can be complicated by bleeding or infection. However, many children have Roux-en-Y biliary-enteric anastomoses, or may be too small, precluding endoscopic access.

Preparation for percutaneous transhepatic cholangiography should include careful review of preprocedure imaging to identify liver transplant anatomy and suitable routes of access. Children often receive split or reduced liver allografts in which the lateral segments (2,3 and sometimes 4) are transplanted. Segment 3 access is usually more advantageous due to a more inferior location from the costal margin. General anesthesia with pharmacologic paralysis offers the ability for breath-holding to optimize access. All patients should receive prophylactic intravenous antibiotics for protection against biliary sepsis.

After administering local anesthesia, a 21 gauge needle is advanced toward a peripheral bile duct, which sometimes cannot be clearly visualized if there is not a complete central obstruction. Cannulation of the bile duct is confirmed by central passage of a 0.018-inch guidewire or by contrast injection opacifying the biliary tree. Due to often diminutive size of the bile ducts, multiple needle passes are often

required for successful cannulation. If a large number of passes are performed (greater than 30), the authors usually terminate the procedure under the assumption that there may be a lack of obstruction and that other causes of liver dysfunction should be explored.

Once successful access has been gained into the biliary system, contrast injection is performed to delineate the type and number of strictures (Fig. 4a). If there is felt to be insufficient drainage from liver segments not accessed, additional drainage may be required. The authors' preference is to perform cholangioplasty of focal lesions at the initial procedure and then place an 8 French internal-external biliary drainage catheter. Patients then return at 2-3 week intervals for further cholangioplasty and upsizing of drainage catheters until a 12 French drainage catheter is placed. Following this, if there is sufficient dilation and spontaneous antegrade drainage, attempt at drain externalization with subsequent removal is performed (Fig. 4b).

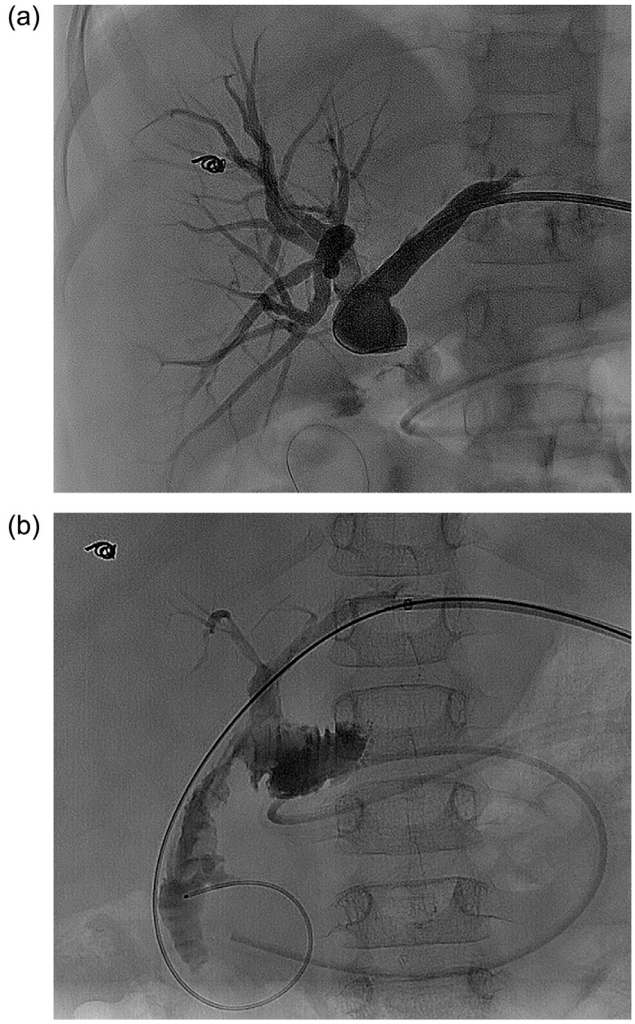

Figure 4 Transplant biliary stricture. A 4-year-old female status post whole liver transplant found to have rising bilirubin and US evidence for biliary stricture. Percutaneous cholangiogram demonstrated occlusion at the biliary-enteric anastomosis (a). Following serial cholangioplasty and drain upsizing over a 2-month period, resolution of the stricture was achieved (b).

Future avenues for investigation include assessing for the possibility of shorter treatment courses with more aggressive cholangioplasty at the initial procedure or use of bioresorbable biliary stents.[54]

Hepatic Vein

Hepatic venous outflow obstruction (HVOO) at any level promotes hepatic congestion and postsinusoidal portal hypertension. If not recognized and corrected, significant congestion will result in graft dysfunction and eventually graft failure. Vigilant surveillance and endovascular management affords graft-saving intervention. While piggyback IVC anastomoses are now nearly ubiquitous in adult liver transplantation, technical variant allograft outflow anastomoses including reconstruction of the recipient hepatic veins into a common channel ("cloaca") or an end-to-side anastomosis between the donor LHV and recipient IVC are frequently utilized in pediatric liver transplantation.[55-59]

Greater variability in reconstruction technique and patient size results in unique hemodynamics within the hepatic venous outflow, creating challenges during the interpretation of surveillance Doppler US at this location. The presence of Doppler US triphasicity within the donor hepatic venous outflow essentially excludes clinically relevant HVOO. Loss of triphasicity, however, is nonspecific as parenchymal processes such as graft rejection may alter the Doppler US waveform phasicity even in the absence of mechanical outflow obstruction.[60,61] While rising anastomotic velocities generally correlate with the likelihood of a hemodynamically significant stenosis, establishing a threshold value that results in both high sensitivity and specificity is challenging.[62] Evidence of hepatic congestion or portal hypertension such as ascites, right-sided pleural effusion, or splenomegaly should prompt outflow venography, and pressure measurements, when US results are inconclusive.

Endovascular management for acute, subacute and chronic pediatric transplant HVOO is safe and effective.[57,58,63-65] The hepatic outflow is catheterized from a retrograde jugular approach with rare exceptions; percutaneous transhepatic antegrade access may facilitate recanalization for difficult occlusions. Multiplanar digital subtraction venography with breath holding or ventilator pause assesses for both anastomotic narrowing and indirect signs of congestion such as delayed intrahepatic contrast retention. Pressure measurements and waveform assessment are critical components of the evaluation. Axial IVUS (Volcano Corporation, Rancho Cordova, CA) can be used for intravascular sonographic evaluation in difficult cases. For patients requiring intervention, patients are heparinized with 50-100 U/kg intravenous. Angioplasty balloon diameters of 100%-120% of the hepatic venous outflow channel diameter are used in the treatment of anastomoses created more than 1 month prior. Stenting is reserved for refractory/recurrent stenoses (Fig. 5) or in the management of iatrogenic injuries resulting from angioplasty. Occasionally, a twisting configuration is observed as a result of torsion with graft settling; no resistance during balloon inflation and immediate return to baseline venographic appearance are typical.

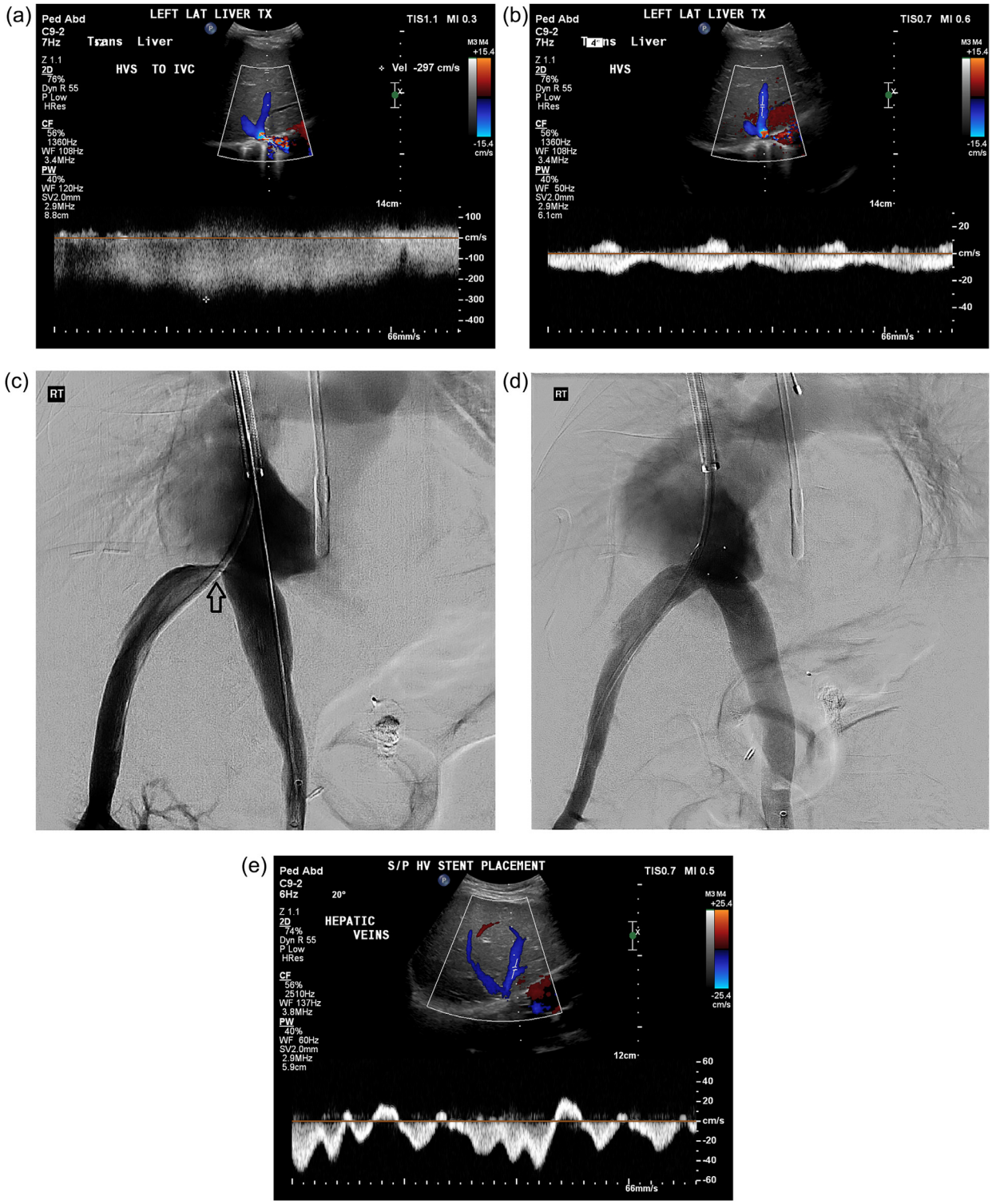

Figure 5 Transplant portal vein interposition graft angioplasty and stent. An 11-month-old female post whole liver transplant presents with recurrent portal vein interposition graft stenosis 2 months postangioplasty. Digital subtraction portal venogram following percutaneous transhepatic access (a) demonstrated recurrent stenoses at both mesenteric (white arrow) and hepatic (open arrow) aspects of the interposition graft. Planning venogram following stent positioning (b) was acquired to confirm coverage of both stenoses without unnecessary extension across the superior mesenteric vein (black arrow). A 6 × 30 mm balloon expandable stent (Formula 418, Cook, Bloomington, IN) was deployed (c) with restoration of brisk hepatopetal flow.

For patients undergoing stent placement, the authors will keep patients on a heparin drip (goal unfractionated heparin activity 0.3-0.6 IU/mL) until reassuring Doppler US findings are observed, subsequently transitioning to a six weeks course of enoxaparin and then indefinite antiplatelet therapy unless contraindicated, although universally accepted anticoagulation/

antiplatelet therapy guidelines following stent placement in pediatric patients have not been established.

Early HVOO may result from graft edema, anastomotic stenosis or thrombosis. While self-limited graft edema will be accompanied by improvements in Doppler US, the later conditions may occasionally necessitate surgical outflow revision in the early postoperative period.[63] When venography is pursued within four weeks of transplant and angioplasty is deemed necessary, the authors will proceed cautiously with undersized balloons (50%-75% of hepatic venous outflow diameter); the theoretically increased need for secondary intervention is preferred over risking potentially catastrophic anastomotic rupture. Catheter directed pharmacologic thrombolysis for acute outflow thrombosis carries a high risk of intra-abdominal hemorrhage and should not be entertained unless there are compelling contraindications to surgical thrombectomy and outflow reconstruction.

Portal Vein

For left liver allografts, a portal anastomosis is created between the recipient main portal vein and donor left portal vein. In cases of a diminutive recipient portal vein or cavernous transformation, a synthetic or allogeneic venous interposition graft may be placed to bridge the gap. Of all Doppler US parameters, rising anastomotic velocities correlate best with anastomotic narrowing; a maximum anastomotic velocity of 180 cm/s in pediatric patients led to a sensitivity of 83% and a specificity of 71% in predicting portal vein stenosis.[66] Despite these threshold parameters, portal vein stenosis can be challenging to diagnose with Doppler US alone. Additional clinical signs that may be present in the setting of portal vein stenosis and necessitate invasive splenoportography and intervention include splenic sequestration (splenomegaly and/or thrombocytopenia), ascites, or variceal bleeding.

The authors' typical transhepatic and trans-splenic portal venous access techniques are as follows. Four- and 6 French access will suffice for most diagnostic and interventional procedures, respectively. A peripheral portal venous or splenic branch is targeted with a 21 gauge needle. Traversing 1-2 cm of parenchyma prior to vein entry facilitates tract embolization. Subcostal approaches are desirable to minimize the risk of hemothorax. A 4 French micropuncture introducer set is placed, allowing over-the-wire venography to confirm position as well as baseline assessment to determine the need for additional imaging and/or intervention. A 0.014-inch pressure transducing wire may be helpful at this point to determine the hemodynamic significance of ambiguous stenoses. While gradients of 2-5 mm Hg suggest hemodynamic significance and may be used for increased justification of intervention, gradients of 5 mm Hg or higher are unequivocally abnormal. If intervention is required, the micro puncture set is exchanged for a 6 French triaxial catheter set which allows parallel placement of a 0.035-inch wire. A 4- or 6 French vascular sheath is placed over the 0.035-inch wire, through which additional imaging and intervention is performed. The 0.018-inch wire remains in place, secured to the side for access preservation. While a variety of 0.014- to 0.018-inch balloon catheters are available with profiles that would allow angioplasty procedures through a 4 French sheath, the authors typically opt for 6 French sheaths during interventions so as not to be prepared for urgent "bail-out" stent placement if complications are encountered during angioplasty, such as acute thrombosis or rupture.

Hand injection venography will suffice for most pediatric patients. For cases of portal venous occlusion, dual injection venography (simultaneously from catheter and sheath) is useful to determine the occlusion length. In addition to morphologic stenoses, venography may demonstrate additional signs of hemodynamic significance such as delayed mesenteric or splenic vein drainage and flow through collateral pathways. Pressure gradients are useful to confirm hemodynamic significance as well as tract progress of subsequent interventions. Patients undergoing intervention are heparinized with 50-100 units/kg intravenous and bolused to keep activated clotting time above 250 seconds. Angioplasty is performed to the diameter of the smaller, usually recipient, portal vein. Primary stent placement is reserved for refractory stenoses with exception of stenotic interposition grafts (Fig. 6). If the stented segment is $\leq$ 8 mm, the authors will often opt for balloon expanded stents to allow future over dilation to accommodate patient growth. Following angioplasty, patients are maintained on heparin overnight until a reassuring US is acquired and then transitioned to enoxaparin for 6 weeks. However, anticoagulation/antiplatelet regimens are often institution-specific in pediatric patients. Following stent placement, patients are transitioned to an oral antiplatelet after 6 weeks of enoxaparin unless contraindicated.

Access related bleeding risk is proportional to sheath size, which can be mitigated with tract embolization.[17,18] Heparinization is allowed to partially subside for an ACT of 200 or less. Tract embolization is performed for access sizes 4-6 French using gelatin. Gelatin in pledget form is placed through the sheath using a cut dilator as a stylet in a similar manner to that described for pediatric hepatic biopsy.[67] Pledget form is preferred over a slurry form for a lower risk of nontarget embolization. Vascular plugs are considered for access $\geq$ 6 French and deployed within the parenchymal tract with a coaxial catheter of 0.038-inch lumen or greater.

Endovascular management for transplant portal venous stenoses in children is safe and effective.[68-73] Recently, Shim et al described their experience in 50 pediatric transplant patients, finding no statistically significant difference in long-term primary patency rates between angioplasty, percutaneous stent placement and intraoperative transmesenteric stent placement.[74] In the authors' experience, angioplasty alone is typically successful, paralleling the experience of Shim et al. Stenoses of portal vein interposition grafts, however, frequently recur after angioplasty alone and primary stenting in this setting is preferred.

Hepatic Artery

One of the challenges of pediatric liver transplantation is the often small size of the recipient, requiring allograft reduction

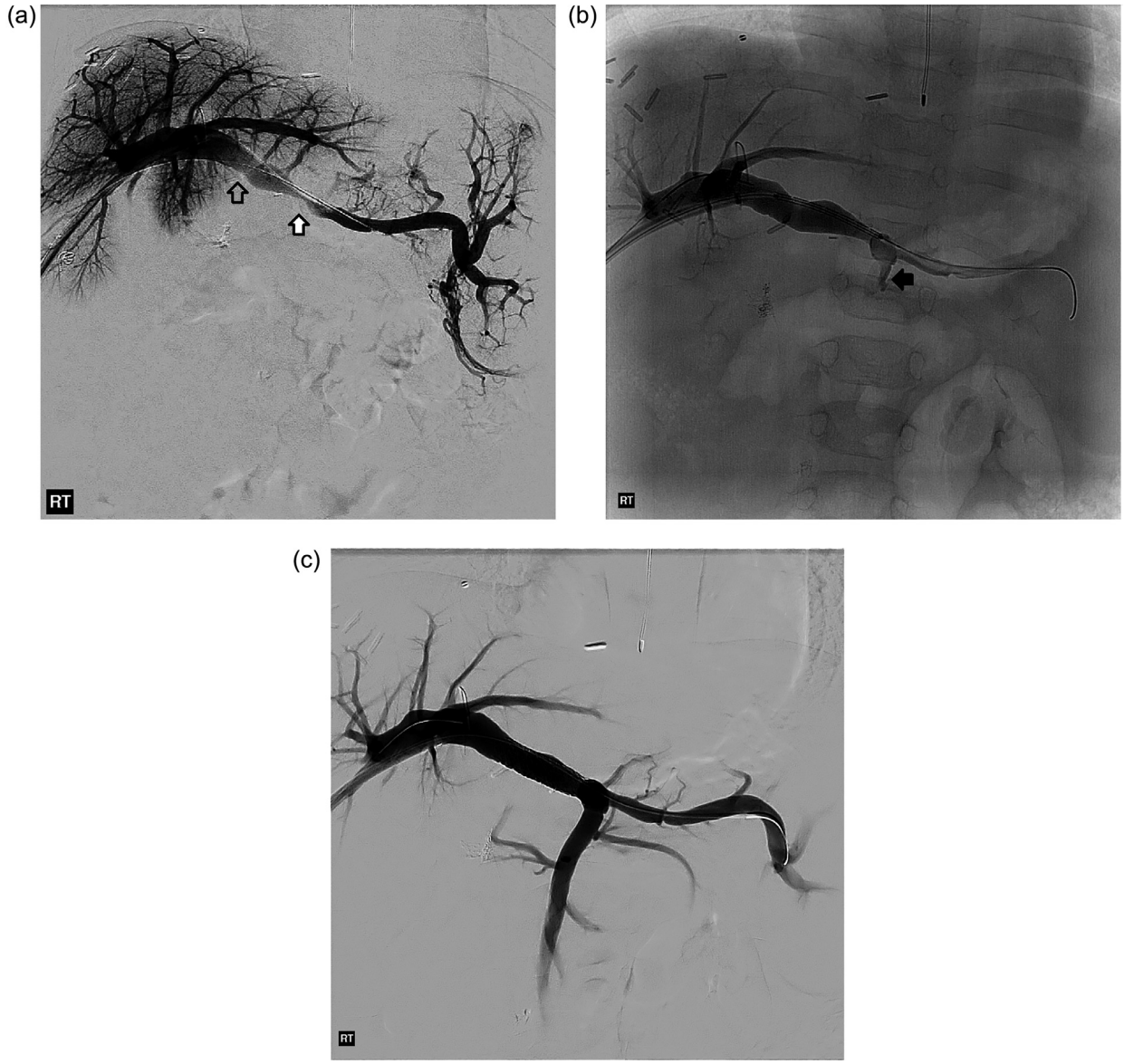

Figure 6 Transplant hepatic vein stent. A 2-year-old female with history of biliary atresia and split liver transplant presented with recurrent hepatic vein stenosis postangioplasty 2 weeks prior. Preprocedure ultrasound demonstrated elevated anastomotic velocities and turbulence (a) as well as blunted intrahepatic phasicity (b). Initial digital subtraction hepatic venogram (c) demonstrated a severe anastomotic stenosis (arrow). Following angioplasty and placement of a 12 × 40 mm self-expanding nitinol stent (Zilver, Cook, Bloomington, IN) (d) there was resolution of the stenosis and brisk hepatic outflow. Ultrasound on postprocedure day 1 (e) demonstrated restoration of a triphasic waveform.

and advanced techniques for creation of vascular anastomoses. The hepatic arterial anastomosis can be difficult to fashion for these reasons and jump grafts are sometimes required. Up to 14% of pediatric liver transplant may be complicated by arterial anastomotic stenosis.[75,76] Intraoperative and close postoperative Doppler US surveillance is performed to monitor intrahepatic arterial waveforms and resistive indices.[77] Arterial insufficiency can have devastating consequences for the health of the allograft, as biliary necrosis or more chronic biliary ischemia can develop. Biliary complications can in turn lead to need for prolonged percutaneous or endoscopic management or graft failure. While most anastomotic arterial strictures are clinically significant, specifically severe biliary injury, some patients will develop sufficient collateralization and maintain acceptable graft.[78]

When hepatic arterial anastomotic strictures become apparent in the early postoperative period, surgical correction is usually pursued. If there are equivocal findings on Doppler US regarding the presence of an arterial anastomotic stricture, CT angiography or catheter angiography is often performed. While there is no definite time interval following transplantation when the safety margin of transarterial correction of an arterial anastomotic stricture is safely pursued, at the authors' institution endovascular intervention is not attempted within the first 2 weeks of transplant due to concern of the integrity of the anastomosis if instrumented endovascularly.

Transplant arteriography is performed by obtaining femoral artery access, usually with a 3 or 4 French sheath depending on patient size. For patients weighing less than 10 kg, 25-50 U/kg of heparin are bloused intravenously to protect the femoral artery against thrombosis. If subsequent arterial intervention is performed, then additional heparin IV boluses are given as necessary to achieve an activated clotting time of greater than 250 seconds. A 3 or 4 French diagnostic catheter is then advanced into the ascending aorta and celiac arteriography is performed (Fig. 7a). If there is normal antegrade flow without angiographic evidence for stenosis, a decision may be made to terminate the procedure. Conversely, if there is evidence for complete occlusion of the hepatic artery with robust surrounding collateralization, a decision may also be made to not pursue further intervention. For angiographically apparent stenoses, the authors prefer crossing the lesion with a 0.014-inch pressure transducing guidewire, which allows for minimally disruptive translesional pressure gradients. Usually, a systolic pressure gradient of 10 mm Hg or more to the abdominal aorta is considered hemodynamically significant, but lower gradients may not preclude intervention.

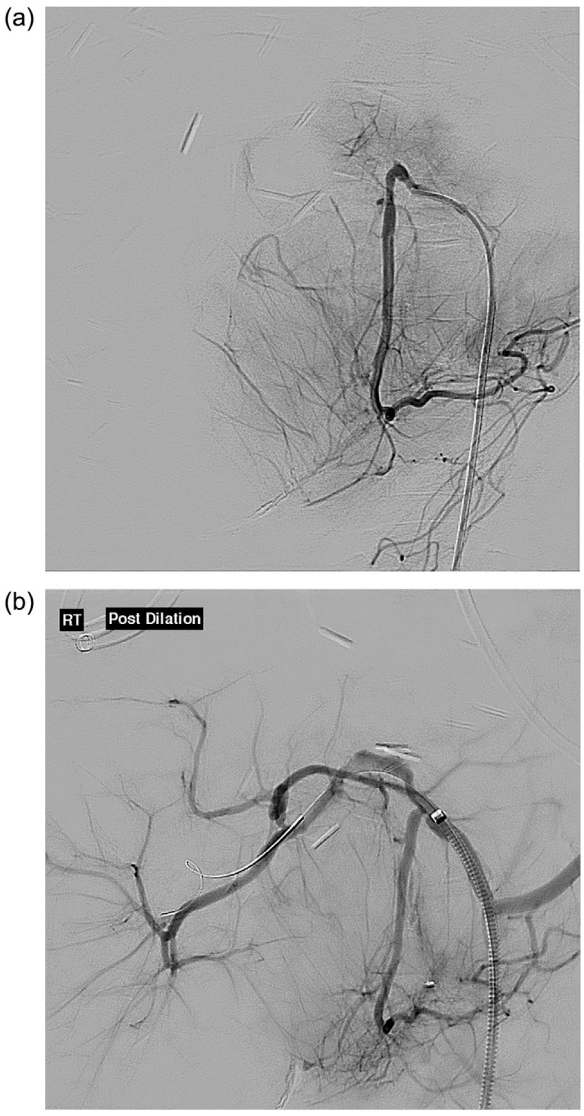

Figure 7 Transplant hepatic artery occlusion. A 13-month-old female with history of biliary atresia status postlateral segment liver transplant. The patient was found to have elevated liver enzymes and Doppler imaging of the liver suggesting arterial hypoperfusion. Conventional hepatic arteriogram (a) shows a complete occlusion at the arterial anastomosis. Following angioplasty to 2 mm, patency was restored (b) with antegrade flow into the transplant.

After access has been gained across the stenosis and a decision has been made to intervene, a 4 or 5 French sheath is advanced up to or just into the celiac artery. Care must be taken to select the appropriately sized sheath to allow for stent placement in case angioplasty is insufficient or there is a rupture or flow-limiting dissection of the artery. Prophylactic verapamil (0.5-1mg boluses) or nitroglycerine (1-2 microgram/kg boluses) is administered intra-arterially to reduce iatrogenic vasospasm, although substantial arterial vasospasm following wire manipulation in children is not uncommon. Initial angioplasty should be done with a low profile balloon, taking care not to overdilate or rupture the anastomosis or perianstomotic arteries. Following dilation, significant arteriospasm may occur, which can be relieved with administration of additional intra-arterial vasodilators. If there is a flow-limiting dissection or rupture, placement of a covered stent should be considered. However, primary stent placement is usually not necessary as balloon angioplasty alone can provide sufficient revascularization (Fig. 7b).

Following angioplasty, patients are maintained on heparin overnight until a reassuring US is acquired and then transitioned to enoxaparin for 6 weeks. An oral anti platelet is initiated immediately, in addition to heparin, for stents and continued for 6 months. Imaging follow-up post angioplasty and/or stent can be performed with Doppler US, typically at 1-, 3-, 6-, and 12-month intervals followed by yearly evaluation. Reocclusion of the treated artery may occur, but if there is interval development of sufficient collateralization, this may be an acceptable outcome for graft survival.

Summary

The treatment of hepatic vascular malformations, portal hypertension and hepatic transplant dysfunction in pediatrics patients poses numerous challenges and endovascular management plays a critical role. An experienced interventionalist, familiar with relevant pathophysiology, postsurgical anatomy and the technical nuances pediatric intervention, can provide potentially organ- and life-saving treatment for these patients.

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