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Techniques in Vascular and Interventional Radiology

Portal Venous Interventions: How to Recognize, Avoid, or Get Out of Trouble in Transjugular Intrahepatic Portosystemic Shunt (TIPS), Balloon Occlusion Sclerosis (ie, BRTO), and Portal Vein Embolization (PVE)

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Portal venous interventions comprise a large portion of many Interventional Radiology practices today, and remain some of the more technically challenging cases in one's repertoire of procedures. The patients upon whom these procedures are performed are often critically ill, have decompensated disease, or are burdened with comorbid conditions such that they are poor surgical candidates. This leaves them with few options outside the care of Interventional Radiology.

Some portal venous interventions, such as transjugular intrahepatic portosystemic shunt, have an established history of excellent clinical success with numerous technical advancements over the years helping to improve outcomes. Others, like balloon occlusion sclerotherapy or portal venous recanalization, are less well established but are nonetheless invaluable in the treatment of portal venous diseases. The goal of this article is to help dispel some of the anxiety experienced by individuals performing the three main procedures of the portal venous system, namely transjugular intrahepatic portosystemic shunt, balloon-occlusion retrograde transvenous obliteration, and portal vein embolization.

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Introduction

Transjugular intrahepatic portosystemic shunt (TIPS) placement is a well-established procedure to treat the sequelae of portal hypertension and is arguably one of the most extensively studied procedures performed by Interventional Radiologists. Indications for TIPS include uncontrollable variceal hemorrhage, recurrent variceal hemorrhage not amenable to initial or continued endoscopic therapy, portal hypertensive gastropathy or colopathy, refractory ascites, hepatic hydrothorax, and Budd Chiari syndrome.[1] Although TIPS is considered a safe procedure, complications can occur in up to 20% of cases.[2]

Several randomized controlled trials have demonstrated significant benefit of TIPS in recurrent variceal bleeding.[3-6] Large meta-analyses comparing TIPS to other forms of endoscopic therapy have shown a greater than threefold reduction in risk of recurrent variceal bleeding.[7,8] Rates of rebleeding are lower with TIPS ranging from 9% to 40.6%, compared to endoscopic therapy 20.5%-60.6%. There is also no difference in all-cause mortality rates between TIPS and other endoscopic therapy.

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Randomized controlled trials studying TIPS in refractory ascites demonstrated a 7.1-fold decrease in the risk of recurrence of tense ascites after TIPS.[9] Ascites improved in 38%-84% of patient after TIPS, compared to 0%-43% after large volume paracentesis. For the treatment of Budd Chiari syndrome, TIPS has been studied in 6 European Centers,[10] in which 124 patients with an elevated median MELD score of 17 received TIPS for varying indications. The 1-year liver transplant free survival was 88% and the 5-year liver transplant free survival was 78%. Finally, TIPS for the treatment of hepatic hydrothorax has been associated with a complete resolution of symptoms in 57%-71% of patients, and partial improvement in clinical symptoms in 68%-82% of patients.[11]

Procedural Steps

The procedural steps to TIPS placement have been described extensively in the literature. For an updated description of the conventional approach to TIPS, the authors refer the reader to Fidelman et al.[12]

For many Interventional Radiology (IR) physicians, the most anxiety-provoking and hazardous aspect of TIPS is gaining transparenchymal access to the portal vein. The standard approach remains a similar technique as when TIPS was first envisioned by pioneers in the field of IR. From a right jugular vein approach, a large bore needle is advanced from the right hepatic vein to the right portal vein in a calculated but "blind" puncture under fluoroscopic guidance. Wedged or balloon occlusion carbon dioxide hepatic venography has been performed since the inception of the procedure to minimize the "blind" aspect to this approach. Many have searched for safer methods to gain portal vein access including ultrasound or fluoroscopic-guided transhepatic placement of loops snares, marker wires, or sheaths to provide a target for which to aim.[13] An increasing number of operators are using intravascular ultrasound (IVUS) to guide their punctures from hepatic veins to the portal venous system and have shown fewer capsular perforations and less radiation.[14] Additionally, IVUS has shown improved inexperienced operator times to TIPS placement compared to conventional techniques with experienced operators deriving less benefit from IVUS. Some are strong advocates for a direct intrahepatic portosystemic shunt (DIPS) using IVUS to guide transcaval portal vein puncture.[15]

Regardless of the method of gaining access to portal venous system, a soft tipped guidewire (Benson) or hydrophilic wire is advanced into the SMV or splenic vein followed by placement of a diagnostic catheter. Portal manometry and a contrast portal venogram are performed. Portal venography should be meticulously scrutinized at this juncture, as the operator has the latitude to abort the procedure or reattempt access should the point of entry into the portal vein be deemed unsafe (ie, extrahepatic main portal vein access or transgression of other critical structures such as large biliary ducts or hepatic artery branches). Rotational computed tomography (CT) imaging (cone-beam CT) can be used to evaluate this in more detail. Once balloon angioplasty of the parenchymal track has been performed, the operator is committed to TIPS placement. On rare occasions, portal manometry will indicate an absence of significant portal hypertension in which case TIPS is not indicated. Ideally, one should either have evidence of portal hypertension or perform wedged portal manometry prior to embarking upon the portal vein puncture.

The tail end of the TIPS procedure involves placement of a Polytetrafluoroethylene (PTFE)-coated stent graft (Viatorr, W.L. Gore). The safest approach to TIPS stent deployment is to advance one's sheath into the main portal vein due to the self-deploying characteristic of the caudal 2 cm uncovered portion of the Viatorr stent graft. Balloon angioplasty of the parenchymal track with a 6 mm balloon will facilitate advancement of the sheath into the main portal vein. It is relatively widely accepted that the rostral aspect of the TIPS stent should terminate at the junction of the right hepatic vein (RHV) and Inferior vena cava (IVC) or within the IVC itself. This minimizes acute thrombosis of the TIPS stent due to outflow obstruction. Secondary stents can be overlapped to reduce angulation and to reach the desired termination target.

After TIPS stent deployment, manometry is again performed in the main portal vein and right atrium. The goal post TIPS portosystemic gradient in patients with variceal bleeding is <12 mmHg,[16] and for refractory ascites the goal may be as low as <8 mmHg.[17] The optimal diameter of the deployed TIPS stent (typically 8-10 mm) varies according to the preprocedure TIPS gradient, the patient's hemodynamic status, and aspects of the worsening disease state. Larger diameter TIPS are associated with increased rates of encephalopathy, and therefore the TIPS diameter should be determined on an individual patient basis, but smaller diameter TIPS are favored.

Intraprocedural Technical Challenges and Complications of TIPS

Acute Hemorrhage

Acute hemorrhage is the major concern during TIPS placement, which most often occurs during attempts to access the portal venous system. When intraprocedural hemorrhage occurs, patients present with hemodynamic instability, rapidly increasing abdominal distention or hematemesis. Attention should immediately be turned to identification of the source of hemorrhage and maneuvers implemented for expeditious control. Postprocedural hemorrhage can manifest as hemobilia, melena, or hematochezia in the setting of inadvertent arterial injury and arteriobiliary fistula. Intraperitoneal hemorrhage, which often occurs during the procedure, has various sources. These include arterial injury, portal vein laceration, or liver capsule disruption.

Accidental hepatic arterial puncture can lead to complications of pseudoaneurysm formation, arterioportal fistula creation, hemorrhage, dissection, or occlusion. The incidence of accidental puncture of the hepatic artery during a TIPS procedure is 6%,[2,18] and the rate of symptomatic arterial injury is less than 2%. Using a smaller 21 gauge system to access

the portal system would seem intuitively safer than the standard 16-gauge sheathed system (ie, Colapinto needle). Haskal et al demonstrated no significant difference in arterial injury when comparing the two systems, albeit the numbers of patients studied was low.[18] Hepatic arterial injury likely occurs more commonly than most practitioners realize and is self-limited.

Portal venous laceration with intraperitoneal hemorrhage can be catastrophic. In the setting of portal hypertension, hemorrhage into the lower pressure intraperitoneal compartment is brisk and technically very challenging to control during surgical rescue attempts. Additionally, a majority of patients undergoing TIPS have decompensated liver function with minimal reserve such that portal venous hemorrhage is often fatal. Avoidance of central portal vein puncture minimizes these injuries and, thankfully, portal venous laceration with life-threatening hemorrhage is rare.

Liver capsule injuries are another source of potentially significant hemorrhage during TIPS and can occur during wedged hepatic venography and/or manometry or transparenchymal needle passes. Breach of the liver capsule can occur with the needle and/or catheter combination during TIPS needle passes in up to 33% of cases, and can result in intraperitoneal hemorrhage in 1%-2% of cases.[12] Liver capsule disruption most commonly results in injury to the gallbladder, with consequences of hemobilia, cholangitis, and intrabiliary clot. Injury to the kidney, colon, and duodenum has been reported without severe clinical compromise.[19] Injury to the liver parenchyma can occur during the process of obtaining wedged hepatic vein pressure, and can be severe enough to require surgical repair.[20] Liver laceration during hepatic venography is more commonly seen using iodinated contrast, compared to carbon dioxide with incidence rates reported at 7.5% and 1.8%, respectively.[21]

Techniques for Avoidance and Management

TIPS begins with meticulous ultrasound-guided right internal jugular vein access to avoid carotid artery injury, and use of fluoroscopy to guide the TIPS sheath over a wire into the IVC to avoid right atrial injury. Modern techniques make these injuries highly unlikely.

To reduce the risk of hepatic arterial injury, preprocedural imaging and careful analysis of the arterial tree and portal veins are necessary. If hepatic arterial bleeding is suspected, angiography should be performed immediately with the use of covered stents or embolization to control bleeding.[22] Rotational 3-D CT angiography (ie, cone-beam CT) is an excellent adjunctive tool to identify the source of bleeding and to direct therapy.

Injury to the portal venous system carries a high risk of fatal venous hemorrhage, which cannot be treated with embolization but may be well controlled with successful TIPS insertion. Once catheter access to the portal venous system is obtained, the initial portal venogram should be meticulously scrutinized to identify any evidence of portal venous injury. If present, this can prompt resuscitative techniques while the procedure is completed. Portal vein hemorrhage is often only identified after balloon angioplasty of the transparenchymal track has occurred. Prior to balloon angioplasty, portal venography with cone-beam CT assistance can identify the precise location of portal venous puncture (Fig. 1). Comparing cone-beam CT imaging to preprocedural contrast CT imaging can prove invaluable to avoiding complications. The portal vein should ideally be accessed in an intrahepatic location to avoid hemorrhagic complications. Unfortunately, anatomical studies demonstrate that 50% of portal vein bifurcations are extrahepatic,[23] and placement of TIPS onto peripheral portal veins often results in unsatisfactory curvature of the stent, and can lead to stenosis on the portal venous aspect and subsequent TIPS failure. Extrahepatic portal vein punctures were likely more concerning in the formative years of TIPS when bare metal stents were used as the primary conduit. This assumption is based upon the fact that numerous operators have reported successful endovascular rescue of extrahepatic portal vein hemorrhage with the use of covered stents extending beyond the site of injury.[23] With the near ubiquitous use of the Viatorr stent-grafts as the primary TIPS conduit, extrahepatic punctures are better tolerated but should still be avoided if possible.

The most common etiology of acute intraprocedural hemorrhage is probably inadvertent puncture to the inferior hepatic capsule, with extravasation of high-pressure portal venous blood into the low-pressure peritoneal space. This puncture can occur quite easily but fortunately exsanguination is rare. Operators have embolized the puncture site with some success, but the best way of minimizing hemorrhage is reducing the portal venous pressure by completing the TIPS. The covered portion of the TIPS stent can be extended further into the portal venous system if necessary to exclude portal veins perfusing the area of active extravasation. Although this may result in portal venous thrombosis, the survival of the patient is usually paramount.

Advice to avoid liver trauma during wedged hepatic venography includes the use of balloon occlusion catheter technique and slow injection of carbon dioxide by hand.[20,21] This technique allows less viscous carbon dioxide to be distributed over a larger area, and avoids the direct injection of more viscous iodinated contrast into the liver parenchyma. Management of liver capsule rupture and/or laceration during wedged venography includes balloon tamponade and coil embolization of the bleeding vessel.[19]

In general, the presence of ascites reduces the possibility of local tamponade effect and has been associated with increased risk of significant bleeding from parenchymal injury.[20] Removing ascites at the onset of the TIPS procedure not only helps with tamponade effect but also helps to stabilize the liver during transparenchymal puncture. Leaving the paracentesis catheter in situ throughout the procedure will help to identify instances of occult intraperitoneal bleeding, and prompt immediate reactive maneuvers.

Prolonged Procedural Time

Historically, TIPS has been associated with long fluoroscopy times and high levels of radiation exposure to both the operator and patient. The rate-limiting step is portal venous

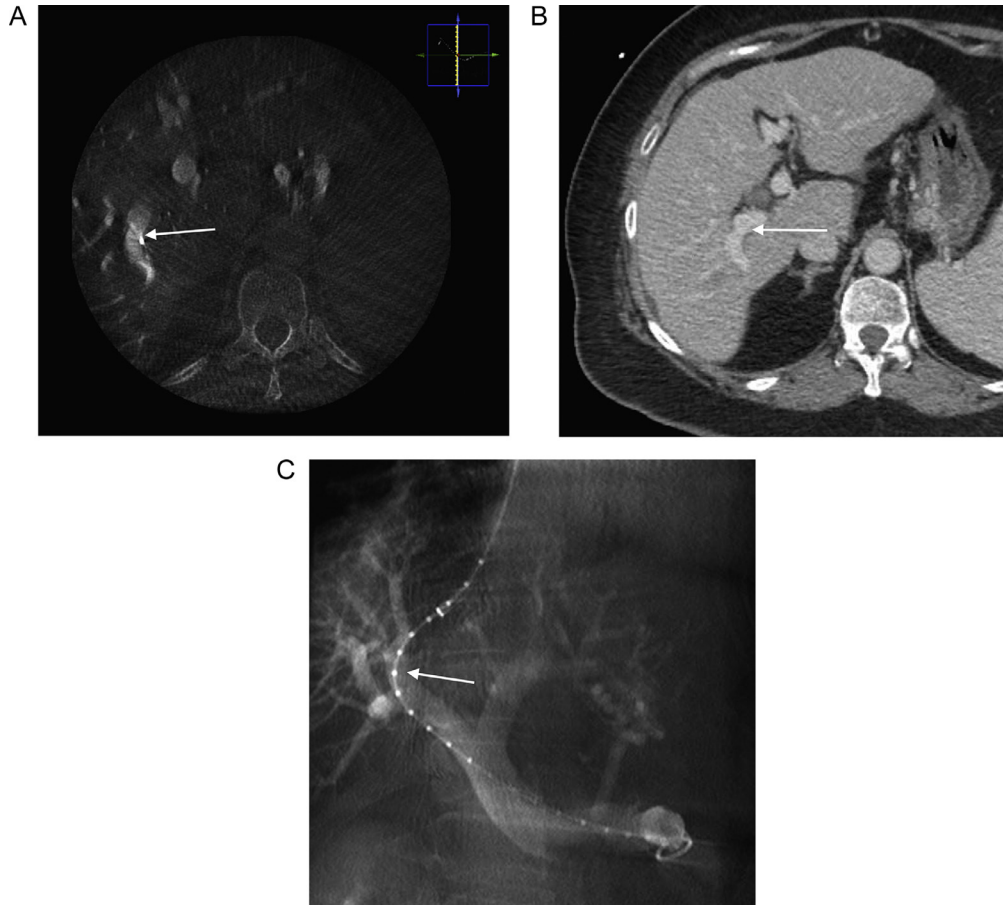

Figure 1 (A) Rotational CT during portal venogram demonstrates the location of catheter access into the portal venous system at the bifurcation of the anterior and posterior branches of the right portal vein while intrahepatic (arrow). (B) The majority of this patient's portal venous system is noted to be extrahepatic. Comparing rotational CT to preprocedural CT imaging (shown) demonstrates safe access while the portal venous system is intrahepatic. (C) Reformatted minimum intensity projection (MIP) images corroborate location of portal venous access (arrow). CT, computed tomography.

access, and any procedural techniques that reduce the time to portal vein access will thereby reduce procedural time.

Techniques for Avoidance and Management

Wedged hepatic venography is crucial to reducing procedural times during conventional TIPS. However, even in cases where wedged hepatic venography is used, the TIPS procedure can be prolonged. Alternatives to this include placing a temporary loop snare or radiopaque marker wire (Fig. 2) via the transhepatic approach into the portal vein as a target for the TIPS needle.[13] Alternatively, gunsight technique has been employed in cases of extreme angulation between hepatic and portal vein (Fig. 3). If the loop snare technique is used, ultrasound-guided access to the left portal vein is sometimes preferred. The loop snare is still placed within the right portal vein, but the left portal vein approach allows for the operator to pull the transparenchymal TIPS wire into the portal venous system rather than pushing the wire from the right side, which can be difficult.

Placing a genitourinary access sheath system such as the Accustick (Boston Scientific, Natick, MA) or Neff set (Cook Medical, Bloomington, IN) into the portal vein from transhepatic approach allows for contrast portal venograms which can provide an improved roadmap target than that of wedged carbon dioxide images. The transhepatic sheath may also provide high quality 3-D CT portal venograms to facilitate TIPS placement and use of needle-assist software in cases of

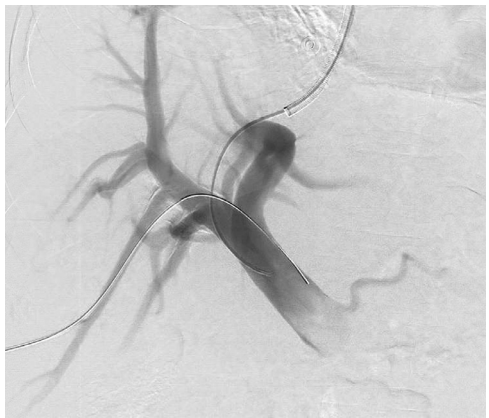

Figure 2 Transhepatic placement of a 0.018 wire can facilitate portal vein puncture. Puncture can be directed toward the radiopaque transition site on the wire, which is left in the exact location of intended puncture.

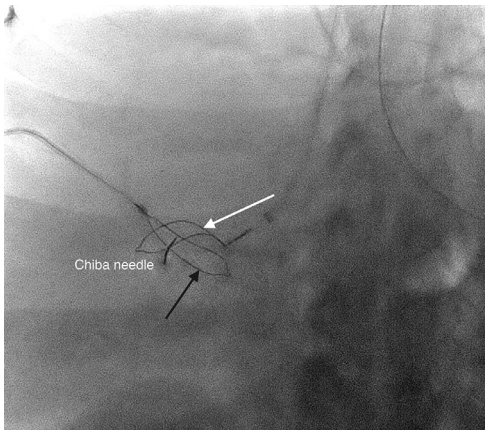

Figure 3 Gunsight technique demonstrated. Two loop snares are placed within the right hepatic vein (white arrow) and right portal vein (black arrow) from right IJ and transhepatic access respectively. The image projection is the rotated such that the 2 loop snares overlap one another. A 22-gauge Chiba needle (shown) is then used to puncture between the loop snares and 0.018 wire flossing access obtained. IJ, internal jugular.

difficult access. The 5 Fr or 6 Fr sheath components can generally be removed without concern for capsular hemorrhage post-TIPS. If the operator is concerned, the transhepatic entry site can be embolized with gelatin sponge pledgets, coils, and/or glue into the tract while retracting the sheath (Fig. 4).[24] Intravascular ultrasound avoids transhepatic access altogether and, as previously mentioned, can reduce unintended needle puncture and procedural time (Fig. 5).

TIPS Malfunction During Preparation

Premature or benchtop deployment of the Viatorr endoprosthesis relates to two potential errors during preparation.[25] Accidental removal of the clear plastic access sheath that restrains the Viatorr endoprosthesis causes exposure of the bare metal portion of the endoprosthesis, which at this stage renders the device nonfunctional. The second error occurs when the clear sleeve covering the bare portion of the stent graft is advanced through the hemostatic valve of the introducer sheath. If the clear plastic access sleeve is incompletely loaded into the sheath until the black line is aligned with the sheath valve, partial maldeployment of the bare portion of the endoprosthesis can occur in the hub of the sheath and prevent the ability to advance the stent graft beyond the hub into the sheath. If this occurs, the stent graft is also rendered nonfunctional.

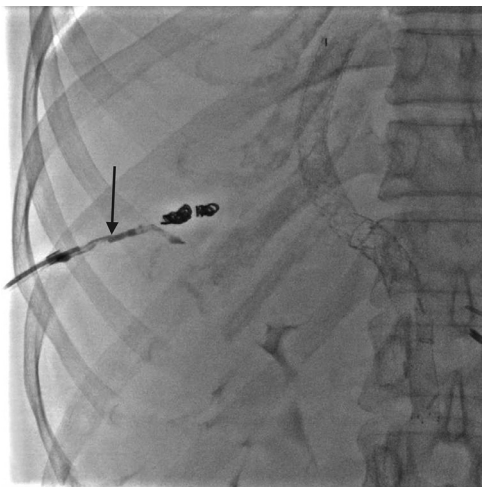

Figure 4 Embolization of transhepatic tract following TIPS placement. Two 4 mm coils are deployed just outside the portal vein access location and then a catheter is used to embolize the tract with NBCA glue (arrow) while the sheath is retracted. NBCA, N-butyl cyanoacrylate.

Techniques for Avoidance and Management

Premature deployment of the endoprosthesis can be avoided in the hands of an experienced operator, and with careful assessment of the components of the kit prior to starting the procedure. The Gore Corporation provides a warning flag pin in the Viatorr stent regarding the perils of removing the plastic sheath on the benchtop, however we suspect that this is occasionally ignored, especially by male operators with an aversion to reading directions.

Injury to the Biliary Tree

The formation of accidental biliary fistula during TIPS placement has an incidence of less than 5%.[2] Isolated biliary injury to the bile ducts or gallbladder is usually well tolerated,[2] however biliary and vascular fistulas may lead to hemobilia, cholangitis, sepsis, and stent infection.[26,27] Communication between the TIPS stent and the biliary system may lead to accelerated pseudointimal hyperplasia and occlusion.[2]

Techniques for Avoidance and Management

Advice to reduce the risk of accidental fistula formation between the biliary and arterial system includes the controlled needle advancement and fewer number of needle punctures.[19] The management of biliary-arterial fistula formation includes internal or internal-external biliary diversion. Fistulous communication between the biliary system and a TIPS stent is predominantly of concern when bare metal stents are used. This is managed with placement of a covered stent within the parenchymal tract.[28] The majority of practitioners in the United States likely use the Viatorr stent-graft for TIPS placement, and therefore biliary to TIPS stent fistulas are rare.

Shunt Malalignment

Ideally, a TIPS stent should reside with the proximal covered portion spanning the hepatic parenchymal tract and the uncovered portion within the portal vein. The gold marker band on the Viatorr stent should reside at the portal vein point of entry. The hepatic venous aspect of the stent should ideally extend to the level of the intrahepatic IVC. Stenosis at the hepatic venous end is the most common etiology of TIPS thrombosis and most often results when the stent is left terminating in the hepatic veins. On the other hand, if the stent

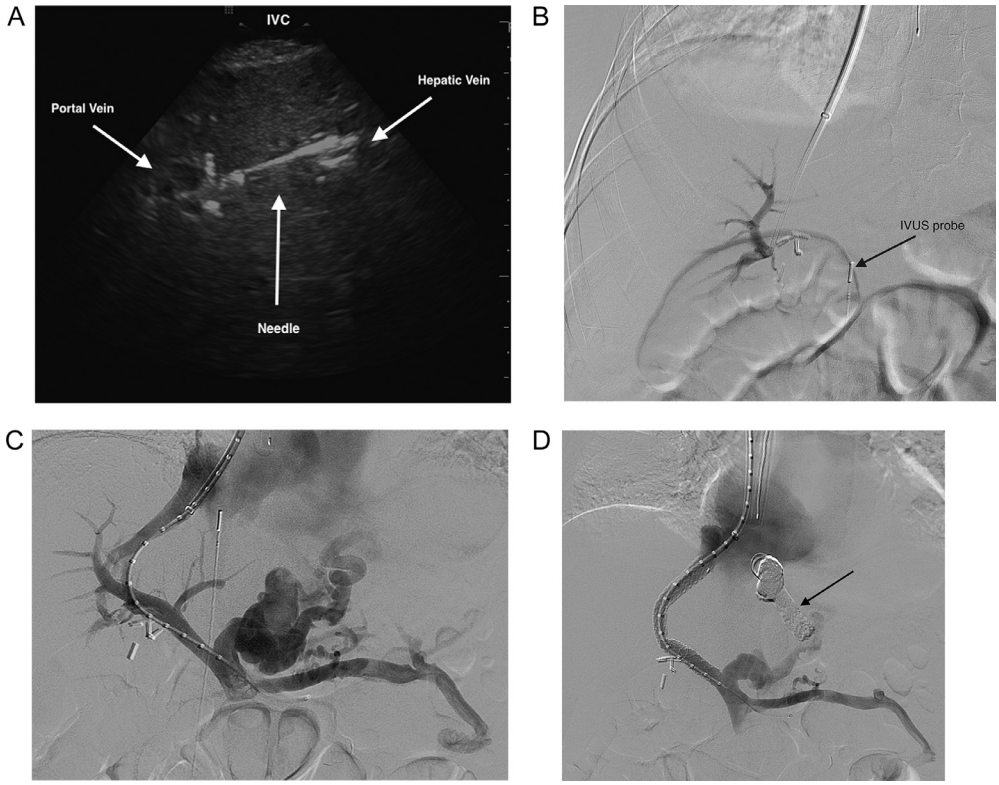

Figure 5 (A) Intravascular ultrasound image using a side probe (5 MHz transducer, depth 7.0) demonstrating a needle passage from the hepatic vein branch to the portal vein branch. (B). A portal vein branch is accessed, and direct portal venography is performed. The IVUS probe is noted within the IVC (arrow). (C) Direct portal venography performed demonstrates opacification of large paraesophageal varices. The desired length of the TIPS is measured using a marker pigtail catheter. (D) Postembolization portogram, demonstrating drainage through a widely patent TIPS and coils/NBCA glue (arrow) within embolized varices. IVUS, intravascular ultrasound; IVUS, transjugular intrahepatic portosystemic shunt; NBCA, N-butyl cyanoacrylate.

extends from the hepatic IVC into the right atrium, challenges may occur during subsequent liver transplantation.[19]

Techniques for Avoidance and Management

A gold radiopaque marker band is generally present to differentiate the covered and uncovered portions of the shunt. The uncovered 2 cm of the stent should be deployed within the main portal vein and then retracted to the point of entry into the portal venous system. Extending the covered portion of the stent into the portal vein could potentially exclude portions of the right portal vein, resulting in occlusion of these branches. The stent can be intentionally deployed in this fashion in the setting of portal vein injury or extravasation.

The proximal or hepatic venous end of the stent should extend to or within 1 cm of the hepatic vein-IVC confluence.[19] If the main stent terminates shortly, the TIPS can be extended with a coaxial uncovered stent such as self-deploying S.M.A.R.T. (Cordis Corp, Fremont, CA) or Wallstent (Boston Scientific, Natick, MA). Alternatively, a second Viatorr stent-graft can be used, although this proves costly. An ancillary benefit to placing a second coaxial stent to the TIPS is straightening of the shunt thereby improving outflow.

Shunt Migration

Covered stents have an increased propensity to migrate compared to bare metal stents.[19] Cephalad TIPS migration has been associated with cardiac rupture including hemopericardium, cardiovascular fistula formation, and valve damage.[29] It may also increase surgical complexity during future liver transplantation[30] due to difficulty with IVC cross clamping. Migration of the TIPS further into the main portal vein can result in portal or mesenteric vein thrombosis.

Techniques for Avoidance and Management

We recommend careful advancement and withdrawal of wires and catheters after shunt placement to avoid shunt migration.[19] Shunt migration is more inclined to occur with balloon dilation and manipulation. One should oversheath the balloon with each deflation, which minimizes traction on the TIPS stent and ensures complete deflation

prior to balloon removal. This technique also prevents the sheath from engaging the rostral aspect of the TIPS stent due to mismatched sheath-wire diameter in the absence of the sheath stylet. To that end, the inner stylet of the sheath should always be inserted if the operator wishes to advances the TIPS sheath through the shunt into the portal venous system.

Should the operator be in the unfortunate scenario of TIPS migration, management should focus upon attempted stent repositioning or removal. Oversnaring a TIPS stent can prove technically challenging. Loop snares can be loaded over an angioplasty balloon matched to the TIPS stent diameter. Inflating a balloon partially within the rostral aspect of the TIPS can facilitate oversnaring the stent and then attempting removal.[26] Alternatively, surgical removal may be required.[31]

Liver Ischemia and Infarction

Liver ischemia and infarction have been reported after TIPS due to the shunting of flow from the portal vein into the systemic circulation, thereby reducing sinusoidal blood flow.[19] Hepatic perfusion after TIPS is negatively correlated to the Child-Pugh score[18] and is dependent on the arterial buffer reserve.[32] Compression of the hepatic artery by the shunt is associated with hepatic ischemia and infarction.[33]

Techniques for Avoidance and Management

Liver failure after TIPS is related to the sudden change in portosystemic pressure gradients related to shunt placement, and avoiding critically low portosystemic pressure gradients after TIPS is essential in preventing liver failure.[34] Hepatic failure related to portosystemic shunting can be achieved by reducing shunt caliber and in intractable cases may require closure of the shunt altogether (described in more detail below).

Postprocedural Clinical Complications After TIPS

Hepatic Encephalopathy

Post-TIPS hepatic encephalopathy occurs in up to 35% of cases, with severe cases reported in up to 3%.[35] The onset of post-TIPS hepatic encephalopathy can occur up to 210 days after the TIPS procedure.[35]

Techniques for Avoidance and Management

A majority of cases of hepatic encephalopathy can be managed with a combination of protein restricted diet, lactulose, or branch-chain amino acids. If patients develop acute liver failure or medically refractory encephalopathy, TIPS downsizing or closure should be considered. TIPS closure can be accomplished by a variety of techniques including using an angioplasty balloon for at least 12 hours to prompt thrombosis[35] or by using a vascular occlusion plug.[36] The benefit of the former is that the thrombosis is potentially reversible since TIPS occlusion can be fatal[37] and will lead to reversal of improvement in portal hypertension. If TIPS occlusion is performed, it is recommended to embolize and/or sclerose varices prior to TIPS closure in an effort to minimize recurrent variceal bleeding. Recurrent ascites can be managed with aggressive medical

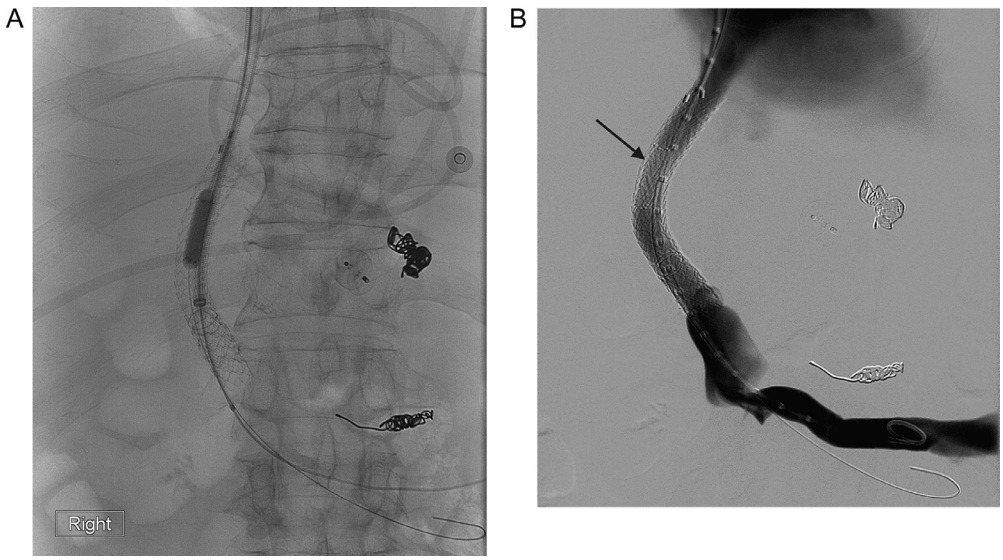

Figure 6 (A) TIPS revision performed for a patient with refractory ascites. Downsizing was required due to intractable hepatic encephalopathy. A 5 mm ParaMount balloon mounted biliary stent (Medtronic, Minneapolis, MN) is placed adjacent to a second Viatorr stent to create an hourglass configuration and narrow the midportion of the TIPS. (B) Direct portal venography through the TIPS stent after adjacent biliary stent (arrow) and second Viatorr stent placement reveals reduced flow through the stent. The gradient prior to TIPS revision was 7 mmHg, after TIPS revision was 11 mmHg. TIPS, Intrahepatic Portosystemic Shunt.

therapy or other interventional techniques (ie, Denver shunt or therapeutic catheter-assisted paracentesis).

TIPS downsizing (ie, reduction in shunt caliber) can be achieved with a variety of techniques. The general concept of TIPS downsizing is to insert a second coaxial stent-graft and create an hourglass shape to the stent-graft using a variety of techniques. These include balloon-dilating only the caudal and rostral aspects of the stent-graft in various ways, presuturing a self-expanding stent graft to deploy within the TIPS, using a "lasso" technique and using a "parallel" stent technique[38-43] (Fig. 6).

Recurrent Ascites or Variceal Bleeding

Recurrent ascites or variceal bleeding after TIPS can suggest stenosis of the TIPS stent, and warrants investigation of patency by Doppler ultrasound, TIPS venography, and portosystemic gradient assessment. The Viatorr endoprosthesis has a long-term patency of 76% at 2 years,[44] much improved from the conventionally used bare metal stents for TIPS which had primary patency rates of 8%-48% at 2 years.[45]

Techniques for Avoidance and Management

Hepatic vein stenosis at the TIPS margin, and intra-TIPS stenosis are typically managed by balloon angioplasty.[46] Lesions refractory to angioplasty are managed by relining the TIPS with a new internal endoprosthesis.[47] A caveat to placing secondary stents is a progressive narrowing of the TIPS stent lumen. A persistently elevated portosystemic gradient without appreciable anatomical stenosis by TIPS venography can be managed by empiric angioplasty of the entire TIPS to the IVC. Rarely, a parallel TIPS is required to reduce the gradient which is generally reserved for recurrent variceal bleeding.

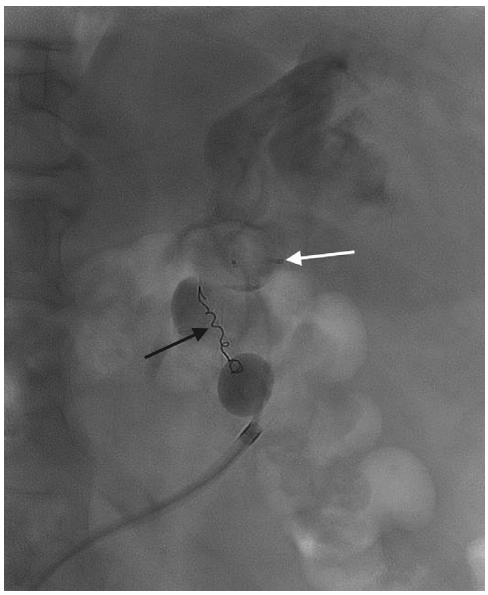

Figure 7 Image demonstrating a standard groin approach BRTO. A straight flexor sheath and inflated occlusion balloon reside within the caudal aspect of the splenorenal shunt. A microcatheter is advanced distally within the varices (white arrow). Embolization coils are noted within a collateral draining vein (black arrow). BRTO, balloon-occlusion retrograde transvenous obliteration

Use of IVUS to Facilitate TIPS

Intravascular Ultrasound (IVUS)

DIPS is a technique introduced in 2001, which involves direct puncture of the portal vein from the inferior vena cava through the caudate lobe, under real-time IVUS guidance.[15] During a DIPS procedure, an IVUS probe is advanced into the intrahepatic IVC, and a modified access set is used only to perform the needle puncture into the portal vein before resuming the remaining steps under fluoroscopic guidance. The advantage of using IVUS is to eliminate the repeated attempts at blind portal vein puncture, reducing complications of inadvertent arterial, biliary or extrahepatic puncture, and kinking of the sheath at the IVC[15] (Fig. 6). IVUS is also associated with reduced radiation exposure, contrast agent volume, and procedure duration compared with fluoroscopically guided TIPS creation.[13,14]

Balloon-Occlusion Retrograde Transvenous Obliteration of Varices (BRTO)

Introduction

Bleeding from varices is one of the major complications of cirrhosis-related portal hypertension. Relative to esophageal variceal rupture, gastric varices rupture infrequently but carry a much worse prognosis and higher mortality.[48] Gastric varices are less well managed by endoscopic injection of sclerosant, as the flow within the varices is often extremely high. Additionally, TIPS has been shown to be less effective in the treatment of gastric varices.[49]

BRTO is an adjunctive or alternative procedure to TIPS for the treatment of gastric varices. The procedure can also be used to treat medically refractory hepatic encephalopathy by closing de novo portosystemic shunts. It has advantages to TIPS in that it is less invasive and can be performed on patients with poor hepatic reserve.

The procedure involves inflating an endovascular balloon to slow or stagnate flow within gastric varices interposing a portosystemic shunt. Sclerosant is then injected with the goal of filling the entire gastric variceal complex and closing all portosystemic connections to avoid revascularization of varices. The embolization end point is minimal reflux of sclerosant into the afferent portal vasculature. Critical to the procedure is flow stagnation within the gastric variceal complex to allow prolonged sclerosant endothelium contact time and avoid reflux of sclerosant into the portal or systemic circulation, which may result in serious complications.

If performed in a retrograde fashion from the systemic circulation side of the shunt, the procedure is referred to as a BRTO (Fig. 7) and is used as an alternative to patients with contraindications to TIPS (ie, severe hepatic encephalopathy or MELD > 19). The procedure can also be used in

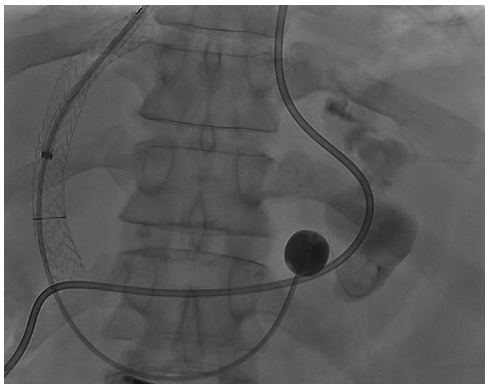

Figure 8 Balloon occlusion antegrade transvenous obliteration of varices (BATO) used as an adjunctive therapy to TIPS in this patient with persistent gastric variceal bleeding. An inflated balloon occlusion catheter and microcatheter are demonstrated residing within the left gastric vein. TIPS, transjugular intrahepatic portosystemic shunt.

combination with TIPS (Fig. 8), or from a transhepatic approach or percutaneous transhepatic obliteration (Fig. 9) whereby balloon occlusion is initiated from the portal venous system in an antegrade fashion, termed "BATO."[50] Although the basic procedural steps of BRTO are relatively straightforward, flow dynamics within the portosystemic shunt can be highly complex and inaccurate, or incomplete recognition can lead to complications if not appropriately understood. For this discussion and the sake of brevity, we will focus on the standard BRTO in the presence of a gastrorenal shunt.

Hazard Types and Recognition

Sclerosant Reflux Into the Portal or Systemic Circulation

Sclerosant reflux into the systemic circulation can occur as a result of incomplete balloon occlusion, balloon rupture, or failure to recognize efferent veins draining the gastric variceal complex aside from the main gastrorenal outflow. Identifying systemic reflux of sclerosant begins with meticulous mapping of the anatomical configuration of the gastric varices and detailing the flow of the gastorenal shunt using digital subtraction venography. Systemic reflux is best recognized

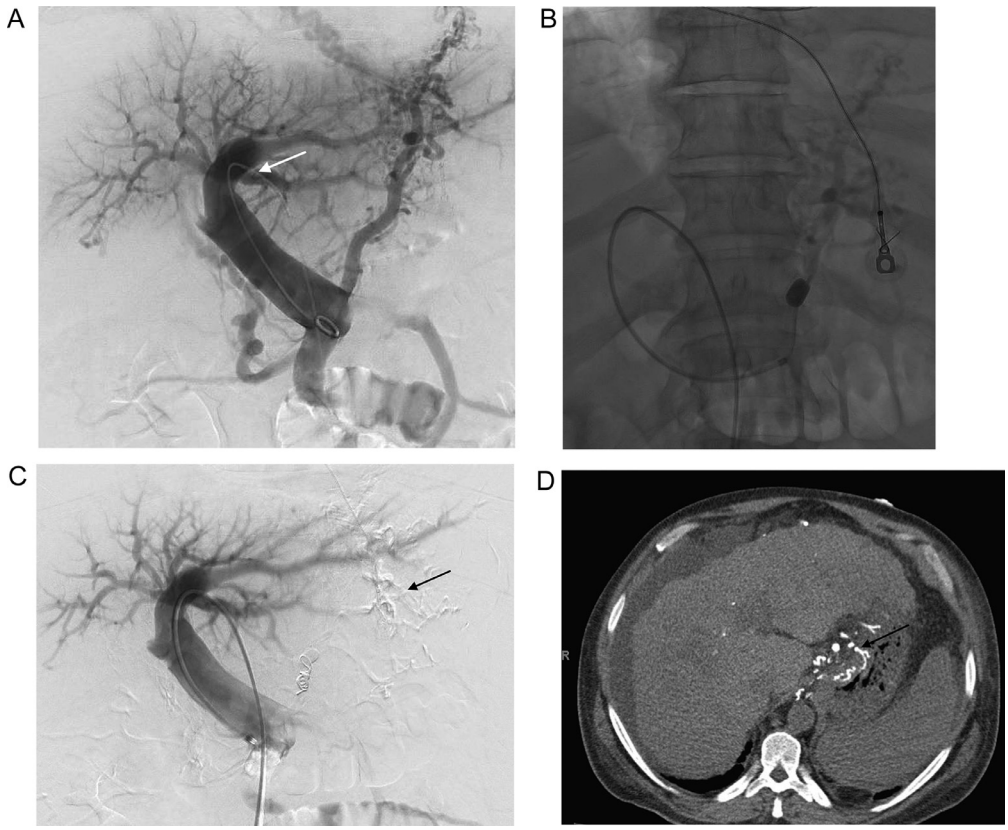

Figure 9 (A-D) A 60-year-old male with metastatic stage 4 lung adenocarcinoma and cirrhosis presenting with esophageal variceal hemorrhage. Patient with elevated MELD and hepatic encephalopathy precluding TIPS as well as no definable portosystemic shunt amenable to BRTO. (A) Percutaneous transhepatic obliteration (PTO) is shown with 7 Fr sheath in the left portal vein (arrow). Portal venogram through flush catheter shows large gastroesophageal varices arising from the left gastric vein. (B) Inflated balloon occlusion catheter within the left gastric vein and Sotradecol sclerotherapy performed. (C) Completion portal venogram shows sclerosant trapped within gastroesophageal varices (arrow) and embolization coils within the left gastric vein. (D) Follow-up noncontrast CT demonstrates Lipiodol and sclerosant trapped within gastric varices (arrow). TIPS, transjugular intrahepatic portosystemic shunt; BRTO, balloon-occlusion retrograde transvenous obliteration; CT, computed tomography.

as a "washing out" of the sclerosant from the gastric varices. Actual washout around the balloon is often difficult to visualize as the flow is slowed as a result of balloon occlusion. If the configuration of the sclerosant within the varix changes with dwell time, one should suspect systemic reflux. Balloon rupture is obvious to detect, and sclerosant washout will be rapid.

One can recognize portal reflux of sclerosant by using a reference venographic image of portosystemic shunt configuration and flow dynamics. Injecting foam Sotradecol sclerosant into a gastrorenal shunt is a slow process, with regular fluoroscopic spot images allowing for evaluation of flow dynamics. Sudden contrast opacification of portal structures overlying the liver indicates nontarget reflux. Additionally, injection of foam sclerosant has a characteristic pressurized sensation upon one's hands during injection. If there is a sudden depressurization from the syringe then one should suspect reflux into the portal circulation. The sensation is akin to injecting air through a nephrostomy catheter already filled with contrast solution.

Inability to Occlude the Gastrorenal Shunt

Occasionally, gastrorenal shunt outflow veins outsize the maximal diameter of commercially available balloons. This precludes one's ability to occlude the shunt. This can often be pre-emptively avoided by reviewing preprocedural contrast-enhanced CT or magnetic resonance imaging, which we believe is crucial to the planning of a BRTO.

Occlusion Balloon Rupture

Sclerosing agents have the ability to rupture occlusion balloons if administered via the balloon occlusion catheter directly and sclerosant comes in direct contact with the balloon. Recognition is straightforward with sudden rupture of the balloon and decompression of the gastrorenal shunt via the left renal vein.

Variceal Rupture

The appearance of variceal rupture is similar to that of rupture of any vascular structure during endovascular procedures. Contrast spills out of the confined vascular lumen into either the peritoneum, retroperitoneum, or gastric lumen. This most often occurs with wire manipulation and aggressive contrast injection via 0.035 base catheters within the varix.

Hemoglobinuria-Related Nephrotoxicity

Hemoglobinuria-related nephrotoxicity is recognized as "hematuria" and elevated creatinine immediately following the procedure. This complication is associated with the use of the sclerosing agent ethanolamine oleate (EO). This complication is less commonly seen in the United States due to the unavailability of the antidote haptoglobin secondary to absence of U.S. Food and Drug Administration approval.

Techniques to Avoid Hazards or to Get Out of Them

Inability to Occlude the Gastrorenal Shunt Outflow Vein

Imperative to a successful BRTO is preprocedural imaging to evaluate the characteristics of the portal venous shunts and size of outflow veins.[51] At our institution, we perform contrast-enhanced CT scans of the abdomen in portal venous phase due to the ease and rapidity of performing this study in oftentimes critically ill patients. Magnetic resonance imaging of the abdomen with contrast is an excellent alternative and can provide similar information. Using contrast-enhanced CT scans, we are able to accurately measure the outflow vein to decide whether occlusion of the shunt is possible. Several balloon occlusion catheters are available in the United States including the standard occlusion balloon catheter (Boston Scientific, Marlborough, MA), the Equalizer (Boston Scientific), Coda balloon (Cook Inc., Bloomington, IN), and Python occlusion balloon (Applied Medical, Rancho Santa Margarita, CA) to list a few. The shunt should be measured at the narrowest point which, in the scenario of a gastrorenal shunt, is at the confluence of the left inferior phrenic vein with the left renal vein. Inability to occlude the outflow vein is one of the primary means of failure to perform BRTO.

Cross-sectional imaging will also dictate whether BRTO would be best performed from an Internal Jugular or Femoral Venous approach. Occlusion of the gastrorenal shunt outflow not only requires a balloon adequately sized for the shunt, but also an angle of approach that is relatively parallel to the outflow vein. Significant torque on the outflow vein results in poor balloon-wall apposition and sclerosant leakage.

From an internal jugular vein approach, we elect to use a 7 Fr Flexor Ansel 2 sheath (Cook Inc., Bloomington, IN) and a Cobra 2 catheter (also Cook Inc.; Fig. 10). From a femoral approach, a straight sheath of operator preference is used and a 4 or 5 Fr Simmons reverse catheter facilitates access to the gastrorenal shunt outflow vein. If feasible, the sheath should be advanced a short way into the outflow vein to ensure stability.

The catheter is then exchanged over a Rosen wire for the occlusion balloon. With the balloon inflated, gastrorenal shunt venography is performed to map the configuration of the gastric varices and evaluate for appropriate stagnation. Draining veins >2 cm in diameter often proves difficult to occlude with standard occlusion balloons. A relatively reliable anatomical feature of gastrorenal shunts is the presence of a venous valve at the junction of the left adrenal vein and left renal vein (Fig. 11). Although this valve can make catheterizing the shunt troublesome, it often provides a bottleneck narrowing of the gastrorenal shunt where the outflow vein is narrowed. Advancing the balloon beyond the valve, inflating the balloon, and then retracting proximally will enable

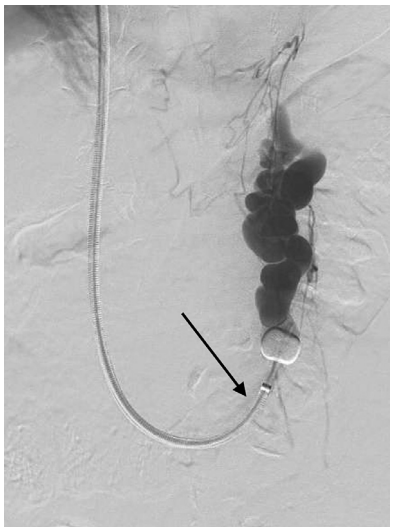

Figure 10 Internal jugular vein approach BRTO. IJ approach employed due to extreme caudal angulation of the left renal vein arising from the IVC. A double angle 7 Fr Flexor Ansel 2 sheath (arrow) provides excellent positioning within the outflow vein of the gastrorenal shunt. BRTO, balloon-occlusion retrograde transvenous obliteration.

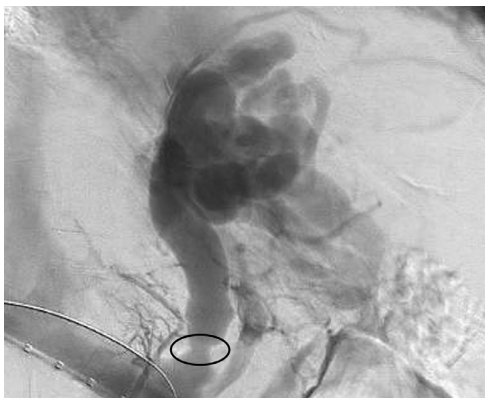

Figure 11 Magnified image of a large gastric variceal complex draining through a splenorenal shunt into the left renal vein and subsequently IVC. The image shown is a delayed venogram after portal vein injection. The black oval demonstrates the characteristic venous valve at the terminal aspect of the gastrorenal shunt outflow vein at the confluence with the left renal vein. This provides a bottleneck upon which to "cork" an occlusion balloon for BRTO (not shown). BRTO, balloon-occlusion retrograde transvenous obliteration.

"corking" of the shunt. This will often allow occlusion of shunts measuring larger than the nominal diameter of the occlusion balloon.

Aside from measuring the outflow vein size and characterizing the shunt, preprocedural cross-sectional imaging will provide additional information that may preclude performing a BRTO. The presence of ascites usually indicates decompensated cirrhosis and will worsen following BRTO. This may preclude performing an elective BRTO in the setting of encephalopathy for instance. Portal vein thrombosis is a relative contraindication to BRTO since without portal outflow, occlusion of portosystemic shunts can incite mesenteric venous hypertension and bowel ischemia. Portal venous thrombosis should be evaluated on a case-by-case basis as robust portal venous collaterals (also known as cavernous transformation) may allow for adequate portal venous outflow after BRTO.

Inability to Stagnate Flow Within the Shunt

The primary limiting factor to variceal sclerosis is inability to balloon-occlude the outflow vein and stagnate flow within the varices (described above). A secondary reason for the inability to stagnate flow is accessory outflow veins draining the varices into the systemic circulation. Gastric variceal portosystemic shunts can be categorized into four types based on efferent venous drainage.[52] Type A is characterized by a single draining vein from the variceal complex usually a gastrorenal shunt draining through the left renal vein via the left adrenal vein or a direct gastrocaval drainage through the left inferior phrenic vein. These are often easily occluded and treatable. Type B and C on the other hand prove more difficult to evaluate and treat. Type B venous drainage involves the single main shunt and one or numerous collateral draining veins. These collateral veins do not form a discrete shunt, but rather drain through a plexus of vessels into the systemic circulation (Fig. 12A). These draining veins include pericardial, inferior phrenic, intercostal, lumbar, and perivertebral veins. Their presence makes full opacification of the main variceal complex and subsequent stagnation of flow difficult. If the operator is fortunate, the collateral veins can be catheterized with a microcatheter and embolized with metallic coils (Fig. 12B). In less common instances where these collateral veins cannot be catheterized, we offer a few potential solutions. One option is to advance the balloon occlusion catheter beyond the origin of these collaterals further into the varices.

If flow stagnates with the occlusion, then sclerosis can be performed. There are caveats to this technique. In general, the farther you advance into the gastrorenal shunt, the larger the lumen becomes (at least initially). The variceal lumen often becomes oversized for most occlusion balloons and the aforementioned advantage of "corking" the occlusion balloon on the left adrenal vein valve is lost. Additionally, these varices are nearly always extremely tortuous, and performing this technique is technically very difficult due to the stiffness of balloon occlusion catheters. Gaining purchase into the varices with a microcatheter and then advancing the balloon over a stiff microwire and/or microcatheter complex can be helpful.[53] Caution must be taken as the risk of variceal rupture increases with luminal tension from stiff occlusion balloons. An alternative treatment strategy is to inject Gelfoam pledgets or foam sclerosant and rely on flow-directed embolization to embolize these veins. Finally, if one can advance the microcatheter far enough beyond these collateral draining veins into the varices and obtain stagnation in the presence of these collaterals then this can be done. Type C varices describe the presence of both a gastrorenal shunt and a gastrocaval shunt. If the secondary shunt can be catheterized, then metallic coil embolization can be performed and

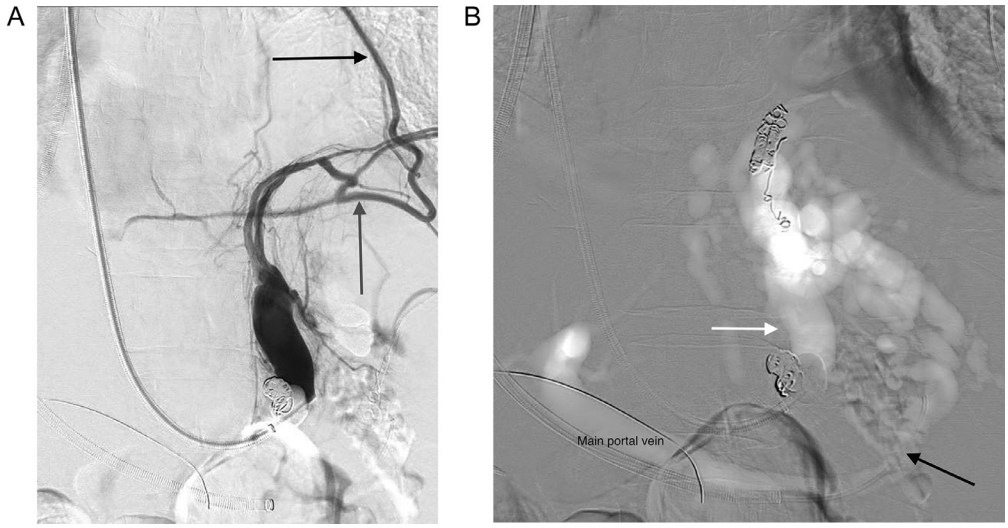

Figure 12 (A) Internal jugular vein approach BRTO shown. An occlusion balloon is inflated within the terminal aspect of the gastrorenal shunt. Large pericardial collaterals (black arrow) and intercostal collaterals (gray arrow) compete for shunt outflow such that visualization of the gastric variceal component of the shunt is not possible with retrograde balloon occlusion venography. (B) The collateral veins were embolized with coils and carbon dioxide balloon occlusion retrograde venography performed. Notice the significant improvement in the visualization of the shunt. This is attributed to embolization of competing outflow and the antidependent flow nature of CO_2. The white arrow denotes the systemic side of the shunt whereas the black arrow indicates reflux of CO_2 into the portal vein where a catheter and sheath are seen residing. BRTO, balloon-occlusion retrograde transvenous obliteration.

standard BRTO technique employed. Alternatively, a second venous access site can be obtained and balloon occlusion of both shunts performed to stagnate flow.[52]

Gastrorenal shunts can also be characterized by the afferent (inflow) venous anatomy.[52] The afferent vein to gastric varices is often either the left or posterior gastric vein, less commonly short gastric veins. A type 1 gastrorenal shunt describes a single afferent vein, and the BRTO procedure needs no alterations to the standard technique. A type 2 system describes multiple afferent supplying veins, that is, the left gastric and posterior gastric or short gastric contribution (Fig. 13). If the two afferent veins have similar pressures, then BRTO can be performed in standard

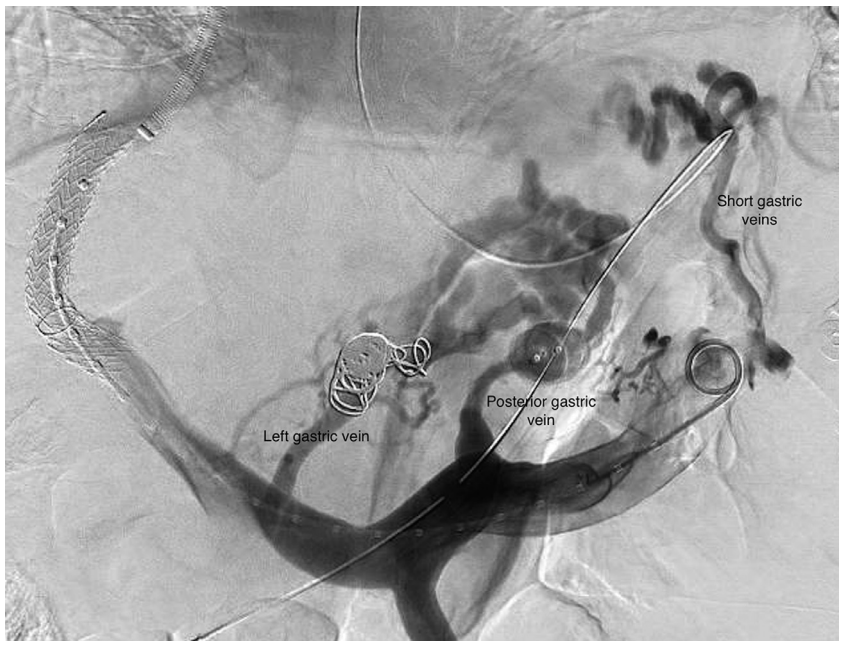

Figure 13 Portal venogram performed during TIPS revision due to recurrent variceal bleeding after TIPS placement and variceal embolization ~1 year prior. The venogram demonstrates the most common afferent veins supplying gastric varices (left gastric, posterior gastric, and short gastric veins). Two of these afferent veins have recanalized despite previous coil embolization, which gives credence to those practitioners who advocate for sclerosis of gastric varices at the time of TIPS placement, rather than coil embolization. TIPS, transjugular intrahepatic portosystemic shunt.

technique. Oftentimes however, the afferent veins have different pressures and therefore sclerosant injection will fill the lowest pressure vein first. Continued injection will result in sclerosant reflux into the portal circulation. Injection of sclerosant until the lower pressure vein reflux is seen will result in partial variceal treatment, as the higher-pressure afferent vein will continue to feed a portion of the gastric variceal complex. Our approach in this setting is to stage the procedure with treatment of the lower pressure afferent vein first and then repeat several weeks later to allow the lower pressure afferent vein to thrombose.

Finally a type 3 afferent venous system describes a scenario whereby a separate afferent vein has input directly into the primary shunt without contribution to the gastric varices. In this scenario, balloon-occluded injection of sclerosant could bypass the gastric varices and reflux immediately into the portal circulation. The approach to treatment in this scenario would be to advance a microcatheter as deeply into the gastric varices as feasible and then perform a venogram to document stagnation and reflux into the primary afferent vein. If this is not possible, then transhepatic or transjugular embolization of the separate afferent draining vein must be performed.

Occlusion Balloon Rupture

Rupture of the occlusion balloon during sclerosant dwell is one of the primary feared complications of BRTO. The underlying fear is migration of a large volume of sclerosing agent into the pulmonary vasculature with associated sequelae of pulmonary embolism. A secondary, delayed complication is one of incomplete gastric variceal thrombosis and/or sclerosis, with subsequent risks for variceal hemorrhage. Our approach to avoiding balloon rupture is to minimize contact of the occlusion balloon with sclerosing agent and/or Lipiodol by advancing a microcatheter as far distally into the gastric varices as feasible. We know from our experience with chemoembolization that Lipiodol has the ability to dissolve the plastic components of equipment with prolonged contact time.

A study by Park et al provides some insight into the risk factors of balloon rupture. They observed an incidence of balloon rupture of 8.7%.[54] Bench testing of occlusions balloon by immersion in sclerosant and Lipiodol showed no evidence of rupture. Using contrast medium in exchange for Lipiodol did not alter the rate of balloon rupture. Additionally, they found no association between the use of a microcatheter to administer sclerosant vs administration through the occlusion balloon catheter. The authors proposed that mechanical forces might play a role in balloon rupture. Some key differences in their study to our approach is the use of EO as a sclerosing agent that is not commonly used in the United States. Additionally, a majority of their patients studied had prolonged sclerosant dwell time of 3-24 hours.[54]

We have adopted the accelerated coil and/or plug-assisted BRTO whereby we begin to nearly immediately embolize the gastrorenal shunt outflow vein with coils and/or a vascular plug to allow for sooner deflation of the occlusion balloon. In doing so, our sclerosant contact time with the occlusion balloon is minimized to <1 hour and our experience with balloon rupture is likely much lower than 8.7%. This approach is beneficial to not only minimize balloon rupture and patient complications, but minimizes patient discomfort, need for higher level of monitoring postoperatively (ie, intensive care), and hospital logistical challenges. With that in mind, Park et al did note that rapid sclerosant washout was more commonly seen when the balloon ruptured <1 hour into the dwell time, and associated complications were significant (ie, two deaths). Balloon ruptures >3 hours, on the other hand, demonstrated much less sclerosant migration likely due to time allowed for variceal thrombosis. One option is to avoid balloon occlusion altogether with coil-assisted retrograde balloon occlusion[55] and plug-assisted retrograde balloon occlusion[56] techniques recently described in the literature.

We propose the following techniques to minimize balloon rupture:

1. Select the best endovascular approach into the gastrorenal shunt to minimize mechanical torqueing forces on the balloon (sheath and catheter techniques described above).

2. Use the lowest profile, most flexible balloon occlusion catheter available.

3. Do not overinflate the balloon beyond manufacturer recommendations. The balloon should be inflated until minimal deformation is noted along the walls and an ovoid appearance noted. If this appearance cannot be achieved, then a larger balloon should be selected.

4. Minimize the volume of sclerosing agent used to avoid systemic complications in the event of balloon rupture. If using EO, <40 mL total is recommended as a maximum dose.[57] The use of EO is highly limited in the United States due to an absence of U.S. Food and Drug Administration approval for haptoglobin, an antidote for hemoglobinemia resulting from hemolysis caused by EO. Foam EO can be made with the following formula: 10 mL of 10% EO: 10 mL contrast: 20 mL of air. One or three percent sodium tetradecyl sulfate (STS) is the most common sclerosing agent used in the United States. The amount of Sotradecol varies depending upon the shunt. Three percent STS comes in standard 2 mL vials of 30 mg/mL (60 mg per vial). As an unmixed liquid, ~30 mL (mean 10-65 mL) of Sotradecol is commonly used. However, we highly recommend against using unmixed liquid STS. Instead we recommend converting the Sotradecol to a foam solution. This has been shown to reduce the volume of STS required to ~10 mL (mean 1-20 mL). Additionally, foaming the STS sclerosant results in improved endothelial contact and efficacy, as well as allows antidependent flow of the sclerosant further into the varices. Our standard formula for STS foam is 1 mL Lipiodol: 2 mL STS: 3 mL air. In the majority of cases, we use <10 mL of STS (a total of 30 mL foam with above formula). These volumes are in accordance with a study of gastric variceal obliteration with STS by Sabri et al.[58] Their mean overall dosage of STS was 300 mg (10 mL, or five 2 cc vials of 30 mg/mL) and mean total foam sclerosant mixture used was ~34 mL. Even in very large gastrorenal shunts, it is unusual to administer >20 mL of STS (60 mL of foam). One should question whether appropriate stagnation of flow has been obtained if >20 mL of STS is required to fully opacify the shunt. At these doses, pulmonary or systemic complications are rare even in the event of

balloon rupture. Foaming sclerosant works best by the time of contact with the endothelium and concentration. Systemic dilution and high blood flow through the lungs is likely to minimize the effects of sclerosis in the pulmonary vasculature. A third foam sclerosant option is polidocanol in a ratio of 2 mL of 3% polidocanol: 8 mL of air.

5. Administer sclerosant via a microcatheter positioned as deeply into the varices as feasible.

6. If performing coil and/or plug-assisted BRTO then begin to deploy coils through the balloon occlusion catheter as soon after sclerosant administration as possible. The coils and/or plug will act as a potential backstop in the event of balloon rupture.

7. In the event of balloon rupture, administer oxygen as needed for dyspnea and medically manage pulmonary complications.

Outcomes

The technical success of BRTO can be divided into single session, multi-session, and BRTO + rescue endoscopic therapy or BATO approach. A comprehensive review of the literature by Saad[59] describes single-session BRTO success for gastrorenal shunts without rescue therapy as ranging from 79% to 100%. Multi-session BRTO had success of 71%, 88%, and 91% during first, second, and third treatments, respectively.[59] The most likely etiology of technical failure is due to inability to opacify, "trap" and sclerose a multicollateral gastrorenal shunt complex,[59] but data are admittedly limited due to variable reporting standards, low numbers of reported failures, and overall high success of the procedure.

Clinical success of BRTO can be divided into the indications for which the procedure was performed. For active gastric variceal hemorrhage, reported success is 91%-100%.[60,61] BRTO similarly shows a very high success rate when performed for intractable hepatic encephalopathy (~100%).[61,62] Rebleeding following BRTO may arise from multiple sources, that is, gastric varices, esophageal varices, duodenal varices, or portal gastropathy. Sources of rebleeding are often not cited in the literature but a few studies cite specific etiologies of rebleeding. Rebleeding related to gastric varices, intent-to-treat gastric varices (including technical failures), and global variceal rebleeding are 3.2%-8.7%, 10%-20%, and 19%-31%, respectively.[60,63,64] Bleeding related to aggravation of esophageal varices depends upon the thoroughness of follow-up, but is estimated at around 27%-35%, 45%-66%, and 45%-91% at 1, 2, and 3 years, respectively.[65,66]

Portal Vein Embolization (PVE)

Introduction

Primary and secondary liver tumors are increasing in incidence and portend a poor prognosis.[67,68] Surgical resection of the tumor-bearing liver improves survival for patients with both primary and secondary liver cancers who are not candidates for liver transplantation. Only 20%-30% of the patients with primary and 10%-20% of the secondary liver tumors are candidates for surgical resection.[69] A major limiting factor when considering surgical resection is the capacity of the future liver remnant (FLR) to sustain the liver functions and avoid liver failure. FLR of less than 20% in normal livers, 30% in patients subjected to heavy chemotherapy, and 40% in chronic liver disease, respectively, are associated with increased incidence of liver failure.[70] PVE has become a standard procedure to increase the FLR by diverting the portal blood flow away from the tumor-bearing liver.

Preventing Complications

Preprocedure Evaluation

A multidisciplinary team, which includes a surgical oncologist, medical oncologist, hepatologist, and interventional radiologist, should meet and discuss an individualized patient plan.[71] A clear communication between the multidisciplinary team is vital for the technical and clinical success of the procedure and avoiding complications.

The interventional radiologist should clinically evaluate the patient. During the evaluation, a detailed history and physical should be performed. Conditions that potentially hinder liver regeneration including diabetes, portal hypertension, biliary obstruction, age, nutritional status, baseline liver function, continuing use of alcohol, and status of hepatitis, should be considered in making a decision to proceed with the procedure.[72,73] Laboratory tests including complete blood cell count, prothrombin time, liver function tests, blood urea nitrogen and/or creatinine levels and viral screening are essential before PVE. Elevation of total bilirubin >3 mg/dL or platelet count <10,000/dL are predictors of poor response to PVE.[74-76] A triple phase contrast-enhanced liver protocol CT scan to evaluate for extrahepatic and intrahepatic disease, anatomical variations of the portal circulation, and calculating the FLR should be performed.[77, 78] Preprocedure antibiotics to cover gram negatives and anaerobes should be administered to prevent biliary sepsis.[74]

Indications for PVE

The indication for PVE is to increase the FLR so that postoperative liver failure does not occur. Liver resection is the procedure of choice in patients with good liver function, favorable tumor distribution, and who are not transplant candidates with primary (hepatocellular cancer or cholangiocarcinoma) or metastatic liver cancers. A minimum FLR of at least 20% for normal livers and 30% for patient who had prolonged (>12 weeks) chemotherapy and 40% for Child A cirrhosis is recommended.[79-82]

Contraindications for PVE

The contraindications for PVE include extrahepatic disease, pathologic periportal lymphadenopathy, portal vein thrombosis, and poor liver reserve as indicated by the indocyanin green retention (ICG 15) rate at 15 minutes of less than

10%. Other contraindications include uncorrectable coagulopathy, extensive liver tumors precluding a safe percutaneous access, and extensive multilobar disease. If biliary obstruction is present, an internal-external drain should be placed to reduce the serum bilirubin to less than 3 mg/dL before the PVE is performed.[71]

Procedural Steps

Portal Venous Access[74,78-84]

Three access techniques are described for PVE: contralateral, ipsilateral, and ileocolic access (described individually below). Access technique depends on anatomy, extent of PVE, and operator preference. A preprocedure CT is used to evaluate for access feasibility and anatomical variations. The portal venous access is obtained under ultrasound guidance and the procedure is usually performed with moderate sedation. During right-sided portal vein access, one should be cognizant of the intercostal artery travelling just beneath the ribs to avoid arterial injury. Injury to the central hepatic artery should be avoided by targeting the peripheral branch of the portal vein. Once the portal vein is accessed, a flush catheter is placed in the main portal vein, and a venogram with baseline portal venous pressures should be performed. If there is sluggish flow, flow reversal in the portal vein, or portal hypertension, then proceeding with the procedure should be reconsidered.

Contralateral Access

A peripheral portal vein branch in the FLR is utilized (most commonly left hemiliver access). This technique generally allows for easy catheterization of the right portal vein branches due to a more linear approach to embolization. Extended embolization of segment 4 portal vein branches is also generally technically easier from the contralateral approach relative to ipsilateral. A potential risk of the contralateral approach is damage to the FLR.

Ipsilateral Access

A peripheral portal vein branch in the tumor-bearing liver is utilized (most commonly right hemiliver access). Ipsilateral approach has the advantage of not going through the FLR. Utilization of reverse curve catheters to access the portal vein branches may be more technically challenging and increase procedure time and radiation exposure. Care should be directed to avoid traversing the tumor while accessing the portal vein, and avoid tumor tract seeding or potential peritoneal seeding.

Transileocolic Access

Transileocolic access is a surgical technique utilizing the ileocolic vein for PVE. This is an open procedure that is rarely performed and not described further in this review.

Embolic Material Selection

Several embolic materials may be used to perform PVE. These include particles of varying types and sizes, N-butyl cyanoacrylate (NBCA) glue, gelfoam, alcohol, polidocanol foam, coils and nitinol plugs.[82,85-87] Particles and coils are likely the most commonly used agent in North America. Tris-acryl gelatin microsphere 300-500 μm particles are commonly used, with an initial goal to achieve distal embolization. If large portal-to-hepatic venous shunts are identified, appropriate particle size adjustments need to be made in an effort to close the shunts prior to proceeding with embolization using intended smaller particle size.

Suboptimal and/or poor distal embolization has been reported to increase the likelihood of inadequate hypertrophy.[88] As the distal circulation occludes, the particle size is increased to 700-900 μm. After stasis is achieved, the proximal segmental branches of the portal veins are occluded with coils or amplatzer plugs. Care should be used to preserve a 1 cm portion of the right or left portal veins so that surgical clamping can be performed.

Another popular embolic material is NBCA glue. Depending on the operator experience and approach to embolization, the ratio of NBCA to Lipiodol will vary. In general, an appropriate consistency of glue is such that distal portal venous embolization occurs. If the consistency is such that rapid polymerization occurs, then proximal segmental PVE will result in a less robust hypertrophy of the FLR. If glue is used alone, some authors suggest a ratio of 1:1 or 1:2 NBCA:Lipiodol.[89] Alternatively, some practitioners will embolize the proximal right portal vein with a metallic plug and subsequently use a 1:8 or 1:10 NBCA:Lipiodol ratio.[90] With the latter approach, a "thinner" NBCA consistency is favored due to the absence of portal venous flow assisted distal embolization.

Glue is best utilized from a contralateral approach relative to ipsilateral. The procedural time is reduced, and some authors have suggested improved hypertrophy with glue.[90] It is however difficult to compare the data due to variation in selection criteria, technique, and follow-up periods to name a few.[82,91] Nevertheless, the current consensus generally accepts that outcomes are similar. Using glue requires experience, and the procedure can cause pain due to the exothermic nature of the polymerization of the NBCA glue.

Other embolic materials stated earlier will not be addressed in detail, but the authors recommend establishing familiarity with one embolic agent to reduce nontarget embolization, improve one's experience, and likely translate to better PVE results.

Postprocedure Considerations

PVE is generally significantly less painful compared to other embolization procedures and patients generally tolerate same-day discharge very well. Mild to moderate pain has been reported in 20%-30% of patients which can be managed by oral analgesics on an outpatient basis.[71]

Recognizing and Managing Complications

Minor complications occur in 20%-25% of the procedures and include abdominal pain, fever, and nausea.[71] These symptoms are generally well managed by conservative treatment. Major complications occur in <5% of procedures and include liver abscess, cholangitis, portal vein thrombosis, nontarget embolization, liver failure, bile leak, and hematomas. In general, major complications of PVE can be categorized into access-related complications and nontarget embolization.

Access-Related Complications

Vascular injuries occur in 2%-4% of patients who undergo PVE.[77,92] Subcapsular and intraperitoneal hemorrhage, arteriovenous fistula, pseudoaneurysm, and transient hemobilia have been reported after PVE (Fig. 14). Subcapsular hemorrhage can present immediately or be delayed by a few days to weeks after the procedure. The most common symptoms are pain and hypotension. Cross-sectional imaging with CT is required to evaluate the extent of bleeding. Bleeding can occur from several sources including the intercostal artery, hepatic artery, and portal vein. Most of the cases can be managed conservatively with blood transfusion and pain management. If bleeding is massive, a transarterial embolization may be warranted.

Techniques for Avoidance and Management

Ultrasound guidance is recommended when obtaining portal venous access. Use of ultrasound minimizes the number of liver passes and allows for direct visualization of intervening hepatic arteries, biliary structures or tumors en route to portal vein access. Central portal venous access should be avoided.

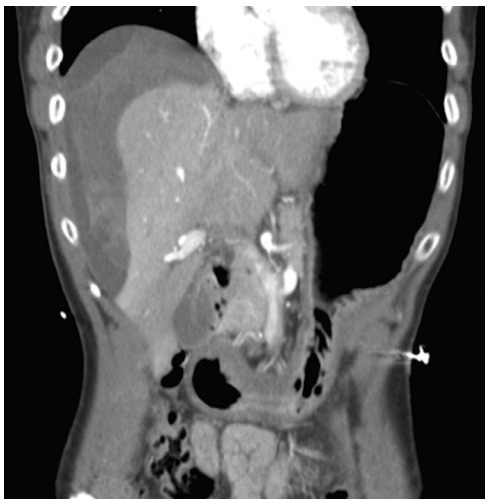

Figure 14 A 56-year-old female underwent attempted access of the right portal vein for ipsilateral PVE. After initial needle passage she developed severe pain and the procedure was stopped. In the holding area she became hypotensive and a CT with contrast for arterial injury was performed. Contrast enhanced CT scan shows large hematoma around the right lobe of the liver with fluid layering. Figures 15-20: Patient with Colorectal Cancer with right lobe metastatic disease. Local tumor was resected. Right + Segment 4 hepatectomy recommended by the multidisciplinary team but concern for undersized FLR therefore PVE (Right + 4) recommended. PVE, portal vein embolization; CT, computed tomography; FLR, future liver remnant.

Postprocedural Bilomas or Infection

Significant bile duct injury during transhepatic access can be avoided by choosing a peripheral portal vein target. Bilomas manifest as persistent abdominal pain following the procedure. When infected, the patient may have an elevated white count with symptoms of sepsis. Bile leak can also present with symptoms of biliary peritonitis. Cross-sectional imaging with CT shows a low attenuating collection with Hounsfield (HU) < 20. Percutaneous drainage is the initial treatment of choice. Laboratory analysis of this fluid should demonstrate a bile content 3 times greater than the serum level. Loculated collections may not be amenable to percutaneous drainage and may require surgical intervention. Cholangitis and hepatic abscesses are rare complications of PVE.

Techniques for Avoidance and Management

One should target a portal vein branch with an appropriate distance from the capsule. A branch within the middle third, not central or exceedingly peripheral, would be ideal. The Interventional Radiology practitioner should have a low threshold to drain intra-abdominal collections after PVE.

Nontarget Embolization

Thrombosis of the main portal vein occurs in 0.5%-4% of cases and reduces the possibility of resection.[77] Complete occlusion of the portal vein can result in hepatic infarction and acute liver failure. FLR portal vein nontarget embolization may also occur with ipsilateral access during segment 4 embolization or from contralateral access. Recognition and timely intervention by mechanical or pharmacomechanical thrombectomy to reestablish flow should be performed. Do not hesitate to perform catheter-directed thrombolysis or conservative management in select cases (Figs. 15-20).

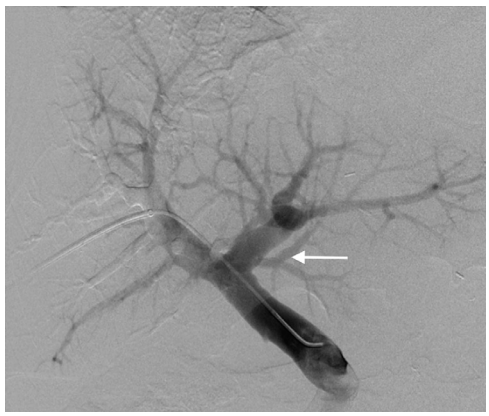

Figure 15 Initial portal venogram shows patent Segment 4 and caudate lobe vein (arrow).

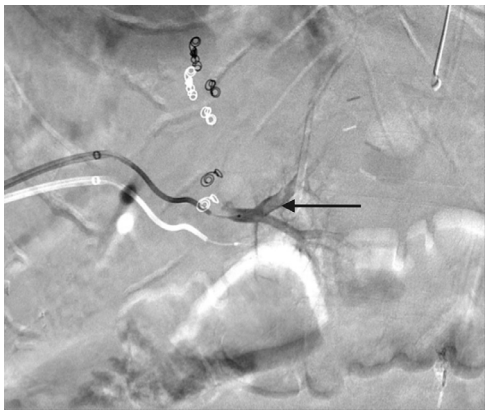

Figure 16 Venogram during Segment 4 embolization shows thrombosis of caudate lobe vein (arrow).

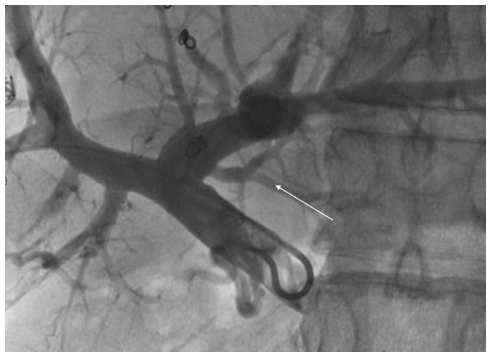

Figure 19 Reattempt at PVE 2 months later, initial portal venogram shows clearance of the previous clot in caudate lobe vein (arrow). Successful Right + 4 PVE was performed. PVE, portal vein embolization.

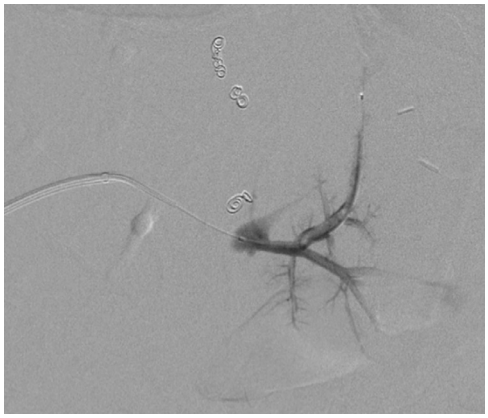

Figure 17 Decision taken to abandon procedure and leave a tPA infusion catheter in the caudate lobe vein.

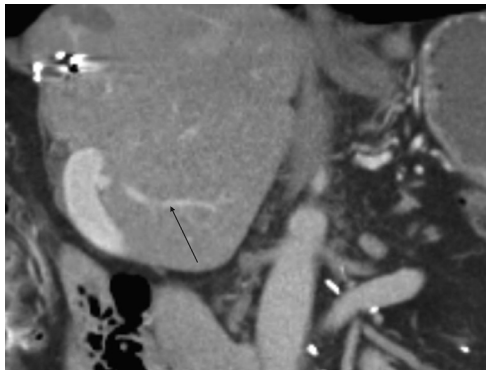

Figure 20 Patient went on to receive successful hepatectomy and the sequence of CT images demonstrate 7 year follow-up CT with patent caudate lobe vein (arrow). Courtesy Armeen Mawash. CT, computed tomography.

Migration of the embolic material into the portal vein supplying the FLR occurs in 1% of PVE procedures.[77] This may affect the capacity of the FLR to hypertrophy. Familiarity with the embolic material is key to avoiding this complication (Figs. 21-24). If partially occlusive embolic material results in portal vein stenosis, angioplasty of the narrowed segment can be attempted as a last resort, however this often initiates a potentially vicious cycle of thrombosis. Surgical intervention with a bypass graft may be required to avoid loss of liver parenchyma in the FLR.

Techniques for Avoidance and Management

Avoid excessive manipulation of reverse curve catheters in the portal vein or when performing segment 4 embolization to minimize intimal injury and resultant thrombosis. Consider aborting the procedure in patients with very sluggish

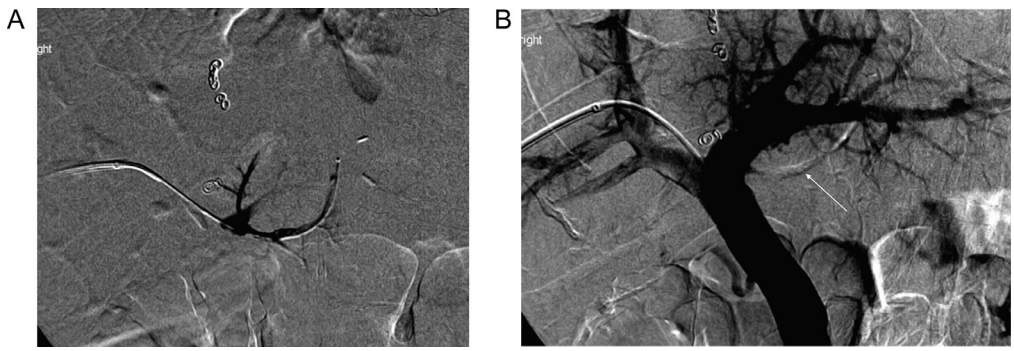

Figure 18 (A, B) Twenty hours post-thrombolysis, minimal clot reduction. Poor response to lysis thought to be due to a combination of nontarget embolization and in situ clot. A decision was made to abandon procedure and reattempt after 2 months.

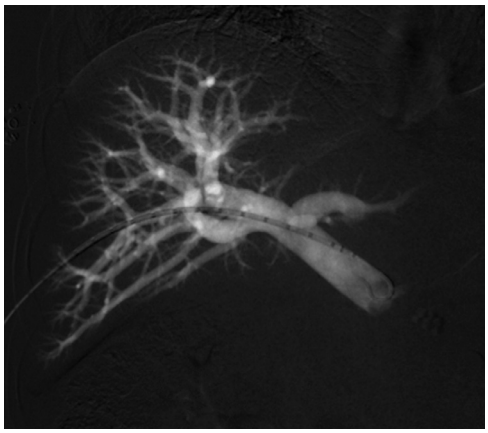

Figure 21 A 50-year-old man with metastatic sigmoid colon cancer to the liver. Initial access portal venogram.

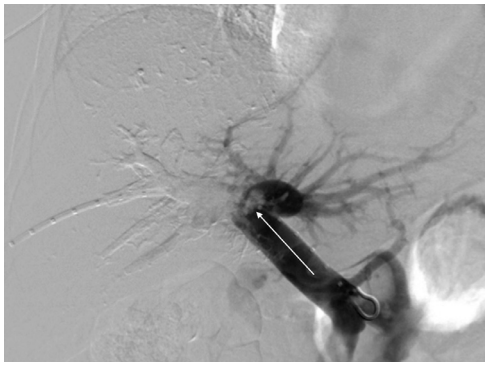

Figure 22 Portal vein glue with embolization via an ipsilateral access. Final images shows significant encroachment of the FLR left main portal vein (arrow). FLR, future liver remnant.

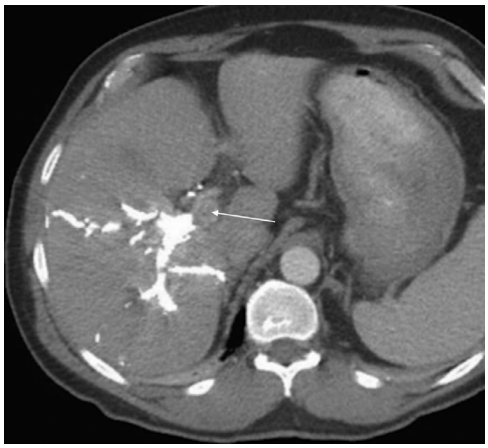

Figure 23 CT scan obtained 24 hours later confirmed glue extension and significant flow compromise of the left portal vein (arrow). CT, computed tomography.

flow in the main portal vein. Suction thrombectomy (eg, Penumbra CAT8, Inc., Alameda, CA) or hydrodynamic thrombectomy (eg, Angiojet, Boston Scientific, Marlborough, MA) can be used to remove bland thrombus. Consider loop snare retrieval of nontarget glue embolization.[93] TPA lysis can be used, but the patient should be monitored closely for signs of hepatic bleeding.

Pearls of Wisdom to Recognize or Avoid Trouble

- **For all portal venous interventions, meticulous review of preprocedural contrast-enhanced CT imaging can minimize complications.**
- **Minimizing the number of transparenchymal passes en route to obtaining portal venous access during TIPS will result in low rates of complications and reduce procedural times. Consider placement of temporary transhepatic targets (loops, snares, and wires) or using adjunctive imaging (intraprocedural rotational CT or IVUS) to improve procedural safety.**
- **Acute hemorrhage during TIPS can be catastrophic and most likely results from inferior hepatic capsule puncture. This is best managed by completing the TIPS to lower portal venous pressure.**
- **Recommended post-TIPS portosystemic gradient is <12 mmHg for variceal bleeding, and may be as low as <8 mmHg for refractory ascites.**
- **Avoid overdilation of the TIPS stent to minimize hepatic encephalopathy, which is best managed medically. Downsizing the TIPS can be accomplished by inserting a second coaxial stent-graft and creating an hourglass shape to the stent-graft using a variety of techniques.**
- **BRTO is a valuable adjunctive or alternative procedure to TIPS for the treatment of gastric varices. The procedure is less invasive, can be used to treat patients with poor hepatic reserve, and improves hepatic encephalopathy.**
- **Paramount to a successful BRTO is flow stagnation within the gastric variceal complex to prolong sclerosant endothelium contact time.**
- **Prevent balloon rupture and associated reflux of sclerosant into the systemic circulation by minimizing sclerosant contact time with the occlusion balloon. This can be achieved by sclerosing through a microcatheter deep in the varices and embolizing the shunt through the balloon occlusion catheter as soon as feasible.**
- **For PVE, if "Right + 4" is done, then one should consider general anesthesia as this may take especially long to complete.**
- **To quantify post-PVE hypertrophy, a contrast-enhanced CT scan should be performed at 3-6 weeks and FLR calculated. Waiting too long risks potential tumor growth secondary to trophic factor release via the splanchnic circulation.**
- **To avoid PVE access complications, use ultrasound guidance to minimize liver passes, avoid central access, and avoid passage through tumor (ipsilateral approach).**
- **Nontarget thrombosis during PVE can be managed with suction thrombectomy (ie, Penumbra), hydrodynamic thrombectomy (ie, Angiojet), or overnight tPA lysis.**

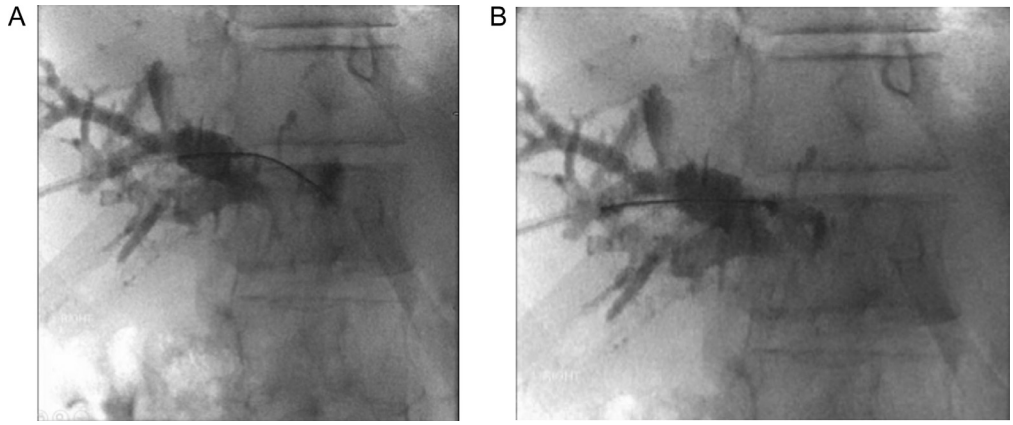

Figure 24 (A, B) The nontarget glue embolus was snared from the left and dragged into the right portal vein. Courtesy Ravi Srinivasa.

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