Image-guided Intervention in Management of Complications of Portal Hypertension: More than TIPS for Success

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Mehran Midia, MD, FRCPC

Management of clinically important sequelae of portal hypertension, such as variceal bleeding and ascites, may involve a combination of medical, endoscopic, surgical, and interventional approaches and procedures. Although clinically significant esophageal and rectal varices are typically visible endoscopically, ectopic varices may require multiplanar portal venous phase computed tomography or magnetic resonance imaging for diagnosis. A detailed understanding of individual vascular anatomy, flow dynamics, and patient-related factors such as cardiac and hepatic status is necessary for appropriate treatment selection in patients with complicated portal hypertension. The hepatic venous pressure gradient is the key indirect measurement of portal venous pressure. Transjugular intrahepatic portosystemic shunt (TIPS) placement is regarded as the archetypal intervention for treating complicated portal hypertension by reducing portal pressure. Various modifications, such as direct portocaval shunt, may be used in patients with challenging vascular anatomy. A subset of patients with obstructed hepatic venous outflow or portal venous inflow should be considered for recanalization. Splenic artery embolization may be considered for reduction of portal pressure in selected patients, particularly when hypersplenism or splenic vein occlusion is a prominent feature. Gastric and ectopic varices may bleed even when the portal pressure is low, and balloon-occluded retrograde transvenous obliteration (BRTO) in such patients may lead to equal or improved outcome compared with TIPS placement. BRTO is not limited by poor hepatic reserve or encephalopathy; however, it does not reduce portal pressure and may aggravate esophageal varices. Interventional radiology plays an important role in maintaining the patency of surgically created portosystemic shunts, and it remains at the forefront of new approaches in shunt design and placement. Supplental material available at http://radiographics.rsna.org/lookup/suppl/doi:10.1148/rg.335125166/-/DC1.

Abbreviations: APF = arterioporal fistula, BRTO = balloon-occluded retrograde transvenous obliteration, DIPS = direct intrahepatic portocaval shunt, HVPG = hepatic venous pressure gradient, IVC = inferior vena cava, PSE = partial splenic embolization, PTE = percutaneous transhepatic variceal embolization, PVT = portal vein thrombosis, TIPS = transjugular intrahepatic portosystemic shunt

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Introduction

An in-depth knowledge of the indications, contraindications, and limitations of several image-guided therapies, in addition to a detailed understanding of individual vascular anatomy and flow dynamics, is necessary in choosing an appropriate treatment option in patients with complicated portal hypertension.

Transjugular intrahepatic portosystemic shunt (TIPS) placement has a recognized role in the management of variceal hemorrhage and intractable ascites. Several alternate image-guided modifications may be considered for patients in whom TIPS placement has failed or is contraindicated (absolute or relative). Data for some of these alternate procedures suggest equal or improved outcome compared with TIPS placement in selected patients—for example, balloon-occluded retrograde transvenous obliteration (BRTO) for gastric varices (1–4). Partial splenic embolization (PSE), either alone or in combination with other techniques, has proved beneficial in case series and nonrandomized studies on the management of variceal hemorrhage (5). Image-guided embolization is a useful alternative to TIPS placement in patients with ectopic varices (6), which may bleed even when portal venous pressure is low (7–9).

In this article, we discuss image-guided alternatives to TIPS placement in terms of pathoanatomy, indications and contraindications, and technical considerations.

Portal Hypertension

Portal hypertension is defined as a portal pressure gradient (the difference in pressure between the portal veins and the hepatic veins) of 5 mm Hg or greater. It is generally classified as prehepatic, hepatic, or posthepatic, depending on the location of the primary block to blood flow (Table 1). In hepatic (sinusoidal) cirrhosis, portal hypertension results from both increased resistance to portal flow and increased portal venous inflow. Increased resistance is both structural (due to fibrosis and regenerative nodules) and dynamic (increased hepatic vascular tone due to endothelial dysfunction mediated by increased endothelin-1 [ET-1] production and decreased nitric oxide bioavailability) (10).

When the portal pressure gradient exceeds a certain threshold, collateral vessels develop at sites of communication between the portal and systemic circulations and decompress or shunt the portal flow into the systemic circulation. This process is modulated by angiogenic factors. In addition, portal venous inflow increases as a result of splanchnic vasodilatation and increased cardiac output. Increased portal flow maintains and exacerbates portal hypertension (11). In severe portal hypertension, blood flow may change direction in the portal vein from hepatopedal to hepatofugal and may reverse in the superior mesenteric vein, leading to the formation of mesenteric varices.

Wedged hepatic venous pressure (a transducer measurement obtained through either a wedged end-hole catheter or an occlusion balloon in a distal hepatic vein branch) is the pressure transmitted through the sinusoids from the portal vein. In patients with intra- and posthepatic obstruction, the HVPG (wedged pressure-free [unoccluded] hepatic venous pressure) is an accurate indirect measurement of portal pressure. It has been validated in multiple clinical settings as an independent predictor for adverse outcome, including the risk of developing varices, ascites, and clinical decompensation in cirrhotic patients.
Changes in HVPG can be used as a surrogate for a patient’s portal pressure response to pharmacologic therapy, since bleeding risk in chronic HVPG responders is extremely low. For patients with acute variceal bleeding, an HVPG of 20 mm Hg or greater independently predicts a poor outcome and may help identify high-risk patients in whom early use of TIPS placement is preferable to combined pharmacologic and endoscopic therapy.

In prehepatic obstruction, portal blood flow is reduced before it reaches the hepatic sinusoids, such that the wedged hepatic venous pressure does not reflect the portal pressure (Table 1). Portal vein thrombosis (PVT) is the classic example, but other causes of presinusoidal portal hypertension, including “downhill” varices (from obstruction of the superior vena cava with or without obstruction of the azygos vein) and the presence of a competing portosystemic shunt, should also be considered in patients who present with acute variceal hemorrhage and a normal hepatic pressure gradient.

### Table 1
**Anatomic Causes of Portal Hypertension with Expected Hepatic Pressures and Interventional Treatment Options**

<table>
<thead>
<tr>
<th>Cause</th>
<th>Examples</th>
<th>Wedged Hepatic Venous Pressure</th>
<th>Free Hepatic Venous Pressure</th>
<th>HVPG</th>
<th>Treatment Options*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prehepatic</td>
<td>Portal or splenic vein thrombosis, congenital portal vein stenosis, arteriovenous fistula, SVC occlusion (downhill varices)</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
<td>Recanalize portal vein, occlude portosystemic collateral vessels, decrease demand (arterial or systemic venous inflow) on portal system</td>
</tr>
<tr>
<td>Hepatic</td>
<td>Presinusoidal: primary biliary cirrhosis</td>
<td>Increased†</td>
<td>Normal</td>
<td>Increased†</td>
<td>Create portosystemic shunt, occlude portosystemic collateral vessels, decrease demand (arterial inflow) on portal system</td>
</tr>
<tr>
<td></td>
<td>Sinusoidal: cirrhosis, infiltrative liver disease, idiopathic portal hypertension, congenital hepatic fibrosis, nodular regenerative hyperplasia, polycystic liver disease</td>
<td>Increased†</td>
<td>Increased†</td>
<td>Increased†</td>
<td>Recanalize hepatic vein, create portosystemic shunt, occlude portosystemic collateral vessels, decrease demand (arterial inflow) on portal system</td>
</tr>
<tr>
<td></td>
<td>Postsinusoidal: veno-occlusive disease</td>
<td>Increased†</td>
<td>Increased†</td>
<td>Increased†</td>
<td>Recanalize hepatic vein, occlude portosystemic collateral vessels, decrease demand (arterial inflow) on portal system</td>
</tr>
</tbody>
</table>

Note.—HVPG = hepatic venous pressure gradient, IVC = inferior vena cava, SVC = superior vena cava.

*Symptomatic thoracentesis or paracentesis may be appropriate in all patients.
†Patients with large portosystemic collateral vessels may have normal pressures.

Portosystemic Collateral Vessels
Clinically significant esophageal and rectal varices are usually visible endoscopically, but paraoesophageal and gastric varices may be less clearly, or incompletely, identified (Fig 1, Table 2). Ectopic varices outside the cardioesophageal region may be identified at endoscopic ultrasonography (US) but are often identified only at cross-sectional imaging. Bleeding from ectopic varices outside the gastroesophageal region is uncommon, accounting
Duodenal varices account for one-third of these cases; other sites of involvement include the small bowel, colon, bile duct, stomas, retroperitoneum, ovaries, vagina, and bladder. Multiplanar portal venous phase computed tomography (CT) and magnetic resonance (MR) imaging are now readily available in most radiology departments and facilitate identification of these complex collateral pathways (13,14).

**Interventions in Portal Hypertension**

The major sequelae of portal hypertension include variceal hemorrhage, hypersplenism, hepatogenic ascites, and hydrothorax. The primary goal in the treatment of portal hypertension is reduction in portal pressure (Table 3). The hemodynamic effects of portal hypertension may be modified through the use of certain systemic drugs. Parenteral splanchnic vasoconstrictors such as vasopres-
sin and somatostatin decrease mesenteric arterial flow, thereby decreasing portal venous inflow. Oral nonselective beta-adrenergic blockers, such as pro-
pranolol and nadolol, affect portal flow by means of both $\beta_1$ blockade (reduction of cardiac output) and $\beta_2$ blockade (splanchnic vasoconstriction). Nitrates, adrenergic inhibitors, and angiotensin blockers act by inducing intrahepatic vasodilatation and may have a synergistic effect. The disadvantages of these agents include relatively common contraindications and side effects (fatigue and shortness of breath), which may preclude treatment or require discontinuation in 15%–20% of patients. In patients with medium-sized or large esophageal varices, either nonselective beta-blockers or endoscopic variceal ligation can be used; a

### Table 2

<table>
<thead>
<tr>
<th>Location</th>
<th>Portal Veins</th>
<th>Systemic Veins</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lower esophagus</td>
<td>Left gastric (coronary)</td>
<td>Azygos</td>
</tr>
<tr>
<td>Stomach</td>
<td>Short gastric or gastroepiploic</td>
<td>Left renal</td>
</tr>
<tr>
<td>Spleen</td>
<td>Splenic</td>
<td>Left renal or adrenal</td>
</tr>
<tr>
<td>Umbilicus</td>
<td>Veins of ligamentum teres</td>
<td>Superior or inferior epigastric</td>
</tr>
<tr>
<td>Rectum</td>
<td>Superior rectal</td>
<td>Middle or inferior rectal</td>
</tr>
<tr>
<td>Retroperitoneum (veins of Retzius)</td>
<td>Colonic or mesenteric</td>
<td>Body wall veins or IVC</td>
</tr>
<tr>
<td>Bare area of liver</td>
<td>Hepatic or portal</td>
<td>Inferior phrenic</td>
</tr>
<tr>
<td>Ductus venosus</td>
<td>Left portal</td>
<td>IVC</td>
</tr>
</tbody>
</table>
A meta-analysis of high-quality, randomized, controlled trials has shown equivalent efficacy and no differences in survival (15). Patients who do not respond to these measures should be considered for image-guided therapy.

Depending on the underlying cause, it may be possible to reduce portal pressure by establishing or reestablishing patency in an occluded vein, creating a shunt to allow bypass flow, or decreasing inflow from the arterial side. When it is not possible to achieve this primary goal, alternate procedures may be used to palliate or control symptoms related to portal hypertension, such as the occlusion of portosystemic collateral vessels or the removal of transudate-exudate fluid from serosal spaces (Table 3).

### Selection of Procedure and Access

Choosing the appropriate procedure for an individual patient should take into account the following considerations: (a) etiology; (b) patient presentation; (c) the patient’s medical condition and comorbidities (liver status, presence of cardiac or renal disease); (d) vascular anatomy and pathoanatomy (including access route, prior surgery, and location of varices); and (e) local expertise and available resources.

Access to the portal venous system for intervention can typically be gained through a percutaneous transjugular or transhepatic approach. Other approaches may be required in unusual circumstances, such as neck and hepatic vein occlusion, extensive hepatic malignancy and polycystic disease, and portal vein occlusion. Access routes through the IVC, spleen, ectopic varices, and native portosystemic collateral vessels (including the umbilical vein) are well recognized (Fig 2). Direct jejunal vein puncture by means of laparotomy has been successfully used to access bleeding jejunal varices (16). Rozenblit et al (17) reported good success in a series of 61 patients who underwent intrahepatic portosystemic shunt placement with a combination of transfemoral access to the hepatic vein and transmesenteric access to the portal system by means of a mini-laparotomy. The authors found the method to be safer and more efficient than the transjugular approach (17).

### TIPS: Indications, Limitations, and Challenges

TIPS is a transjugular portosystemic shunt created within the liver parenchyma between the hepatic vein and the portal vein to achieve a portosystemic gradient of less than 12 mm Hg. Several excellent reviews of the technical aspects of covered TIPS placement and management of shunt dysfunction have been published (18–20). In pediatric patients,
the technique is similar, although carbon dioxide (CO₂) venography is not recommended for very young patients due to the risk of hepatic laceration; and balloon-expandable bare or uncovered stents are preferred, allowing further dilation during the growth of the child (21).

Indications for TIPS placement include uncontrollable variceal bleeding, recurrent variceal bleeding in patients who have failed endoscopic and medical therapy, refractory ascites or hydrothorax, acute gastropathy, hepatorenal syndrome, Budd-Chiari syndrome, and hepatopulmonary syndrome.

Absolute contraindications to TIPS placement include right heart failure and pulmonary arterial hypertension, severe polycystic liver disease, uncontrolled systemic infection or sepsis, liver abscess, unrelied biliary obstruction, and portal hypertension from arterioporal fistula (APF).

Relative contraindications include severe liver failure (Child-Pugh class C cirrhosis, Model for End-Stage Liver Disease (MELD) score > 22, serum bilirubin level > 3 mg/dL), for which TIPS placement has an unclear survival benefit, and preexisting encephalopathy. Other relative contraindications include hepatoma (especially if central), obstruction of hepatic veins, PVT, hepatic vein thrombosis, severe coagulopathy (international normalized ratio [INR] > 5), thrombocytopenia (< 20,000/cm³), moderate pulmonary hypertension, and severe stenosis or occlusion of the celiac or hepatic artery (22).

It is now well recognized that, compared with intermittent large-volume paracentesis, TIPS placement significantly improves transplant-free survival in cirrhotic patients with refractory ascites (23). For esophageal variceal bleeding, TIPS placement also compares favorably with combined medical and endoscopic therapy (24, 25). For patients with Budd-Chiari syndrome, TIPS placement is associated with less morbidity than is surgery. In published case series, ascites control following TIPS placement for Budd-Chiari syndrome approaches 100%, and improvement in liver function can eliminate the need for transplantation (26, 27). The benefits of TIPS are less clear for gastric varices (the portosystemic pressure gradient may be low or even normal due to shunting) and for ectopic varices, which are not specifically targeted and have high recurrent bleeding rates (7).

Safe, accurate, and timely puncture of the portal vein is desirable during a TIPS procedure but can be challenging. Cross-sectional imaging with postprocessing, such as thick-slab venous phase maximum intensity projection of CT data, can help identify anatomic variations and optimal angles for puncture. The success rate for intraprocedural portal vein visualization with CO₂ wedged or balloon-occluded hepatic venography is approximately 90%. Opacification of the portal vein can also be achieved directly by means of percutaneous transhepatic portal vein or direct variceal puncture, or by means of indirect portal venography with either superior mesenteric arteriography or CO₂ splenoportography.

Several modifications have been described that can facilitate TIPS placement in patients with challenging vascular anatomy. A left jugular vein access site has potential technical advantages, including a possible straighter course to the preferred target portal vein site. A recent series reported no difference in complication rates for TIPS placement performed with a right- or left-sided jugular approach, whereas technical success improved with the left jugular approach and greater physician experience with TIPS procedures (28).

In some patients, direct portocaval puncture, essentially creating a side-to-side portocaval shunt, may be required when the hepatic veins are occluded, as in Budd-Chiari syndrome or in the presence of unfavorable hepatic venous anatomy due to a shrunken right hepatic lobe with a transverse or cephalic course of the hepatic vein.

Portocaval puncture can be performed with fluoroscopy alone using a fine-needle system (Angiodynamics, Queensbury, NY) with a combined transhepatic and transjugular “gunsight” technique (29, 30). US guidance can be used for percutaneous transhepatic puncture through the portal vein to the IVC with snaring of a guidewire via a jugular vein puncture. This technique has been shown to be useful in patients with Budd-Chiari syndrome (31, 32). These patients typically have an enlarged caudate lobe, complicating access to the portal vein and causing intrahepatic caval compression. The accuracy of US-guided puncture of the intrahepatic IVC may be enhanced with use of a caval balloon (Fig 3). CT- and endoscopic US–guided puncture have also been described (33).

Direct intrahepatic portocaval shunt (DIPS) placement is a further modification of the TIPS procedure in which intravascular US guidance is used for puncture from the intrahepatic IVC.
Figure 3. Portocaval shunt placement in a 13-year-old girl with a 5-day history of abdominal pain and swelling. Contrast-enhanced CT demonstrated features consistent with acute Budd-Chiari syndrome, including early enhancement of an enlarged caudate lobe, delayed enhancement of the periphery of the liver, and nonenhancement of the hepatic veins. (a, b) Transhepatic US images illustrate how a needle was advanced across the proximal left portal vein (arrow in a) into the IVC (arrowhead in a, arrow in b) for “through and through” puncture. (c) Fluoroscopic image shows the use of a percutaneous needle to access the left portal vein. (d) Digital subtraction venogram obtained following caval puncture shows contrast material in the IVC and right atrium. The IVC balloon is inflated. (e) Digital subtraction venogram shows a guidewire that was snared through the caval sheath, allowing transjugular access into the portal system, and demonstrates the portal vein anatomy. (f) Digital subtraction venogram shows a patent portocaval stent.
after TIPS or whether it should be reserved for certain anatomic subsets, such as patients with previous or recent bleeding, a persistently elevated portosystemic pressure gradient, or gastric varices; or used to reduce the risk of chronic liver failure in patients with large shunts and a low portosystemic gradient by improving hepatic portal perfusion. An Amplatzer vascular plug (AGA Medical, Golden Valley, Minn) may be useful for the occlusion of large gastric varices and spontaneous gastrorenal or splenorenal shunts (35–37).

Recanalization of the Hepatic Vein

Budd-Chiari syndrome is characterized by venous outflow obstruction at the level of the hepatic veins, IVC, or right atrium. Recanalizing the hepatic vein or IVC by means of balloon angioplasty or stent placement can relieve hepatic congestion and prevent progression to irreversible...
ible liver damage. Li et al (38) reported satisfactory midterm outcomes (primary and secondary patency rates of 76% and 84%, respectively, at 2 years) for balloon angioplasty for hepatic vein occlusion in patients with Budd-Chiari syndrome. The technical success rate was 91% (92 of 101 patients), with a higher rate of technical failure in patients with long-segment vein occlusion, although stents were used in only two patients (38).

Transabdominal US may be used to guide a catheter and wire through a transjugular liver biopsy cannula (positioned in the IVC) across ostial or short segmental occlusions. This technique obviates percutaneous transhepatic puncture, drainage of ascites, and normalization of bleeding parameters (39).

**Figure 5.** Percutaneous transhepatic recanalization of the portal vein in a 59-year-old woman who presented with esophageal varices, ascites, and hemorrhoids. The patient had undergone right trisegmentectomy 2 years earlier for a presumed hilar cholangiocarcinoma, which proved to be a benign bile duct papillary adenoma at pathologic analysis. (a, b) Transhepatic portal venograms demonstrate irregular narrowing along the main portal vein. (c) Digital subtraction image shows placement of a 10-mm × 6-cm Zilver stent (Cook Medical, Bloomington, Ind), which resulted in initial resolution of the clinical symptoms. The patient presented again 2 years later with variceal bleeding and stent occlusion, which necessitated coil embolization of the coronary vein. Results of a nonfocal liver biopsy performed at that time confirmed previously undiagnosed α1 antitrypsin deficiency, steatohepatitis, and fibrosis.

**Recanalization of the Portal Vein and Its Tributaries**

Extrahepatic obstruction of the portal vein or its branches accounts for 5%–10% of all cases of portal hypertension (higher in children). The cause of obstruction can be benign or malignant, and patients usually present with variceal bleeding, ascites, or abdominal pain. Recanalization of the blocked vein by means of variceal bleeding and stent placement will reduce these symptoms and is typically performed using either a transjugular or percutaneous transhepatic (Figs 4, 5) approach (40,41). Placement of an additional TIPS may be necessary to prevent thrombosis if blood flow through the recanalized channel is slow or the portosystemic gradient after recanalization
is greater than 12 mm Hg (42). Tuite et al (43) reported the use of the transsplenic route in three patients with chronic PVT who underwent successful portal vein–spleenic vein recanalization. Two patients underwent concomitant TIPS placement and variceal embolization with conventional catheter and wire techniques (43).

**Embolization of APF**

APFs are a rare cause of portal hypertension (44). They may be congenital or secondary to trauma, surgery, percutaneous procedures such as biopsy, or liver tumors. Most APFs are asymptomatic, although clinical symptoms such as gastrointestinal bleeding, ascites, and congestive heart failure can occur. Decades may elapse between fistula formation and detection. Guzman et al (45) classified APFs into small peripheral type 1 (asymptomatic, typically resolving spontaneously), large central type 2, and congenital type 3. Small APFs are rarely of concern (except perhaps in patients being considered for hepatic artery chemoembolization or radioembolization). Types 2 and 3 may require surgical or interventional treatment. The preferred treatment is transarterial embolization of the feeding artery using coils, glue, or the Amplatzer vascular plug (AGA Medical) (46). In general, coils can be used successfully in simple arterioportal shunts (with a single feeding artery), whereas more complex shunts, with multiple feeding arteries due to cirrhosis, tumor, or previous surgery, typically require a liquid or particle agent to abolish the shunt.

**Partial Splenic Embolization**

Hypersplenism and platelet sequestration may contribute to thrombocytopenia in patients with cirrhosis and portal hypertension. Endoscopic variceal ligation may be effective but does not address the underlying portal hypertension. Although a portosystemic shunt reduces the risk of hemorrhage by reducing the underlying portal pressure, it may not be suitable in all patients (eg, patients with advanced liver dysfunction and encephalopathy or PVT). Open or laparoscopic splenectomy has been proposed but has not gained widespread acceptance because of the high risk of complications (up to 10%), especially of PVT. Furthermore, there is a subset of patients in whom esophagogastric varices (short gastric and gastroepiploic veins) may develop as a consequence of isolated splenic vein thrombosis (eg, secondary to pancreatitis), a condition known as sinistral portal hypertension. In such patients, TIPS placement should not be performed because splenic artery embolization or splenectomy can successfully eliminate the source of gastric varices.

PSE is performed to diminish inflow of blood into the portal vein, with secondary reduction in portal pressure (Figs 4, 6). This procedure achieves reduction in splenic size, improvement in hypersplenism-induced thrombocytopenia due to decreased splenic sequestration, increased white cell count due to decreased splenic pooling, and increased red blood cell count attributed to increased erythrocyte survival time (5).

PSE can result in a variable decrease in portal pressure gradient in patients with cirrhosis and portal hypertension; however, changes in variceal flow rather than changes in portal pressure pressure are thought to be more predictive of outcome. A spleen-to-liver volume ratio greater than 0.5 based on CT findings is reported to be a positive predictor of a decrease of over 20% in portal pressure gradient, and this cutoff value can be used to identify patients who may benefit from obliteration of splenic artery inflow as a method of decreasing portal hypertension (47). Embolization may be performed alone or in combination with other interventions, such as endoscopic ligation or retrograde transvenous variceal obliteration (48–50). Hepatofugal flow is regarded as a contraindication to PSE due to the increased risk of PVT. A systematic review from 2007 points to a lack of sufficient high-quality evidence to allow an evidence-based evaluation of PSE in achieving control of variceal hemorrhage (5).

Xu et al (48) reported their experience with endoscopic variceal ligation and PSE in 41 patients. Esophageal varices and hypersplenism were well controlled, without recurrent hemorrhage (mean follow-up interval, 9.9 months), and there was a significant reduction in flow rate and maximum flow velocity in the main portal vein. Postembolization splenic abscess occurred in one patient, and another patient died of a pulmonary embolus (48). In a nonrandomized study, Chikamori et al (51) performed PSE 7–14 days before BRTO in 14 patients. The 3-year cumulative occurrence rate of esophageal varices was 9%, compared with 45% in a similar group who underwent BRTO alone (51). Pålsson et al (52) reviewed 26 patients who underwent a total of 52 PSE procedures, mainly for thrombocytopenia due to bleeding from esophageal varices. The mean hemoglobin value, leukocyte count, and platelet count increased significantly after partial
embolization, and the frequency of bleeding from esophageal varices decreased significantly. One patient died as a result of severe hepatic insufficiency, and another patient died of complete splenic infarction with abscess, total PVT, and cardiac insufficiency (52).

Figure 6. PSE in a 60-year-old woman with hepatitis B cirrhosis and large gastric fundal varices. (a, b) Axial arterial phase (a) and coronal venous phase (b) CT images demonstrate several splenic artery aneurysms and extensive varices with splenorenal shunting. (c) Digital subtraction angiogram shows coil embolization of the splenic artery. (d, e) Follow-up contrast material–enhanced MR images through two levels of the splenic hilum obtained with gadoxetate disodium (Primovist; Bayer Healthcare Pharmaceuticals, Montville, NJ) show thrombosis of the aneurysms, approximately 50% splenic parenchymal infarction, and reduction in the size of the varices.

Embolic agents for PSE include polyvinyl alcohol particles, embospheres, and absorbable gelatin sponge (Gelfoam; Pharmacia Upjohn, Kalamazoo, Mich). Concomitant antibiotics or
embolic agents soaked in antibiotic solutions may reduce the risk of splenic abscess. The efficacy of PSE for thrombocytopenia is dependent on shrinking the parenchymal volume by at least 50% (53). Embolization of over 70% of splenic volume may increase the risk of complications, including pleural effusion and abscess (53). In particular, avoiding the cranial aspect of the spleen may reduce the likelihood of an inflammatory reaction that might induce diaphragmatic irritation and pleural effusion. Postembolization symptoms may require hospitalization (42).

**Figure 7.** Percutaneous transhepatic embolization in a 72-year-old woman with acute rectal bleeding from extensive rectal varices. The patient had Child-Pugh class B cirrhosis and a small right hepatic vein that was unsuitable for TIPS placement. (a) Wedged hepatic venogram through the middle hepatic vein shows asymptomatic contrast material extravasation (arrowheads) through a capsular tear (arrow) (see Movie 2). The following day, percutaneous transhepatic access was obtained through the left portal vein into the inferior mesenteric vein. (b, c) Venograms through an 8.5-mm occlusion balloon (Boston Scientific, Natick, Mass) placed through a 7-F sheath demonstrate extensive rectal varices (see Movie 3). Foam sclerotherapy with 4 mL of 3% tetradecyl sulfate (Tromboject; Omega Laboratories, Montreal, Canada) and 4 mL of air was performed with the occlusion balloon left inflated overnight. Two coils were placed before balloon removal. (d) Follow-up venogram shows satisfactory thrombosis, and the patient had no further bleeding.

**Percutaneous Transhepatic Variceal Embolization**

Percutaneous transhepatic variceal embolization (PTE) was described in 1974 by Lunderquist and Vang (54) for the treatment of intractable variceal bleeding. Although this initially appeared to be a highly effective procedure, successfully controlling acute bleeding in 70%–90% of patients, recurrent bleeding was seen in 10%–60% of cases, since the underlying portal hypertension was unaffected. A failure rate of up to 9% was reported, particularly in patients with PVT or small livers with marked ascites (55), and PTE itself was implicated in inducing transient PVT in
up to 36% of patients (56). Some authors recommend the prophylactic administration of subcutaneous low-molecular-weight heparin for 5–7 days after PTE for this reason (57). Despite these reservations, PTE remains safe and effective for the treatment of special types of varices with portal hypertension and may be considered in cases in which TIPS placement fails or is contraindicated; and in patients with (a) bleeding ectopic varices, including rectal (Fig 7), stomal (Fig 8), and duodenal (Fig 9) varices, among others; (b) splenic vein thrombosis with isolated gastric varices (58); or (c) gastric varices without a catheterizable draining vein. A variety of embolic agents have
been used, including coils, cyanoacrylate glue, absorbable gelatin sponge, and sclerosants such as 50% dextrose, ethanol, sodium tetradecyl sulfate (Sotradecol; Angiodynamics, Queensbury, NY), and ethanolamine.

In 2007, Chikamori et al (59) reported successful PTE in 13 patients with use of a variety of complex shunts and varices. To our knowledge, there have been no randomized controlled trials comparing TIPS placement and PTE in the management of bleeding ectopic varices, but available data suggest that recurrent bleeding can occur in

Figure 9. Percutaneous transhepatic embolization in an 82-year-old woman with advanced-stage hepatitis C cirrhosis who presented with unstable gastrointestinal bleeding from duodenal varices. (a) Venous phase CT image shows extensive duodenal varices (arrows). (b, c) Percutaneous transhepatic venograms show large varices draining into the IVC (arrow in c). An ascitic drain (*) is noted. (d, e) Digital subtraction venograms show coil-sclerosant embolization of the varices, which resulted in cessation of bleeding. Unfortunately, the patient was lost to follow-up beyond 1 month.
Figure 10. BRTO of gastric varices in a 57-year-old man with alcoholic cirrhosis, hepatocellular carcinoma, and upper gastrointestinal bleeding from a large gastric fundal varix (seen at endoscopy). (a) Axial venous phase CT image shows the presence of gastric fundal varices (arrow) and hepatocellular carcinoma (*). (b) Retrograde venogram shows an 8.5-mm occlusion balloon (Boston Scientific) (arrowhead) that was placed into a gastrorenal shunt using a retrograde transfemoral venous approach. Coil occlusion of the inferior phrenic vein (arrow) could not be achieved due to vessel tortuosity. A mixture of 3% tetradecyl sulfate and lipiodol was injected under fluoroscopic guidance into the large gastric fundal varix, and the balloon was left inflated for 5 hours. (c, d) Follow-up CT images demonstrate both lipiodol-sclerosant–containing thrombus and bland thrombus (arrow in c) in the varices. The patient had no further bleeding but died 4 months later following an episode of bacterial peritonitis.

25%–40% of cases despite hemodynamic targets being achieved (60). The use of adjuvant embolization following TIPS placement appears to result in greater efficacy (60).

BRTO of Varices

The behavior of gastric varices varies depending on their location. Most isolated fundal or fundal-cardiac varices drain through a developed gastrorenal shunt, such that the portal pressure in such patients is quite low. Tripathi et al. (61) showed that a significant proportion of gastric varices bleed at a portal pressure gradient of 12 mm Hg or lower, and that TIPS placement improved mortality only in patients with gastric variceal bleeding at a portal pressure gradient greater than 12 mm Hg.

BRTO is a technique that was popularized in Japan for control of gastric varices (62,63). Technical feasibility requires a natural gastrorenal or gastrophrenic shunt (up to 95% of gastric varices), which is typically well demonstrated at contrast-enhanced CT. The technique involves advancing a balloon catheter from the femoral or jugular vein into the outlet of the gastrorenal shunt–left adrenal vein. Following balloon occlusion of the shunt, a sclerosant (typically 5% ethanolamine olate with lipiodol or iopamidol) is injected retrogradely to fill the gastric varices, either directly or through a coaxial microcatheter (Fig 10) (1,58). If ethanolamine is unavailable, the use of other sclerosants such as alcohol, 50% dextrose, sodium tetradecyl sulfate, or an ethylene vinyl alcohol copolymer (Onyx, eV3-Endovascular) can be considered. An infusion of haptoglobin is commonly used to protect against renal dysfunction caused by ethanolamine-induced hemolysis. Balloon rupture is uncommon but may cause rapid migration of sclerosant, pulmonary embolism, or recurrent gastric variceal bleeding.
A detailed understanding of the venous anatomy and flow patterns is the most important factor in ensuring successful treatment. Hirota et al (63) classified gastric varices and collateral veins into five grades according to the results of adrenal venography during balloon occlusion in a series of 20 patients. In grade 1, gastric varices are well opacified with no evidence of collateral veins (such as inferior phrenic, hemiazygos, or pericardial veins). In grade 2, contrast material remains in the gastric varices for 3 minutes or more, with collateral veins small and few in number. In grade 3, contrast material fills the gastric varices only partially and disappears within 3 minutes, and collateral veins are medium sized to large and few in number. In grade 4, gastric varices are not opacified and there are many large collateral veins. In grade 5, the left adrenal vein cannot be occluded with the balloon catheter because of a very large gastrorenal shunt with rapid blood flow (63). This classification system may help in selecting the optimum technique for BRTO (eg, additional or staged embolization, or combined with PSE or transhepatic embolization). The authors suggest that occlusion of collateral veins is essential for the obliteration of gastric varices higher than grade 2 (63).

Kiyosue et al (64) introduced a modified classification system for gastric varices based on the hemodynamic pattern of varices before and during embolization in a series of 60 patients. This included the pattern of both afferent and efferent veins. Type 1 gastric varices are supplied by a single afferent gastric vein; type 2, by multiple afferent gastric veins; and type 3, by single or multiple gastric veins with coexistent gastric veins that are directly contiguous with the gastrorenal shunt but do not contribute to the varices. Type A draining veins are contiguous with a single shunt; type B are contiguous with a single shunt and collateral draining veins (there are three subtypes: B-1 [small, low flow], B-2 [medium sized, low flow], and B-3 [high flow]); type C are contiguous with both gastrorenal and gastrocaval shunts; and type D are not contiguous with a catheterizable shunt (64).

Additional techniques that can be considered in BRTO of complex shunts (Kiyosue type 2 or 3, B or C) include stepwise injection of the sclerosing agent, selective injection of the agent via a microcatheter (to decrease the volume of sclerosant required), coil embolization of the afferent gastric veins or collateral draining veins, double-catheter technique (for collateral vessels that cannot be catheterized), double-balloon technique (for gastrocaval or inferior phrenic shunt) (65), and BRTO performed with percutaneous transhepatic portal venous access or transileocolic venous access (Fig 11) (66). Concomitant balloon occlusion of the splenic artery may improve distribution of sclerosant in the gastric varix and has the potential to improve outcome through more extensive thrombosis.

BRTO is considered by many investigators to be as effective as TIPS placement in controlling gastric variceal bleeding (1–3). Potential advantages of BRTO over TIPS placement include augmentation of portal blood flow, preserving or improving liver function in patients with a poor hepatic functional reserve, and prevention of encephalopathy (1,2). It has been suggested that patients with large gastric fundal varices bleed at a lower portal pressure than those with esophageal varices, due to a combination of shunt formation and large variceal size. In these patients, TIPS placement is less successful than in patients with esophageal varices (67), and BRTO may be a particularly suitable treatment option (4).

Oclusion of a gastrorenal shunt may aggravate existing esophageal varices or lead to the development of new varices and may result in PVT (3). Cho et al (68) reported that ascites developed or was aggravated in 82% of patients, and that the spleen showed enlargement in 56% of patients within 1 week of BRTO.

Cho et al (69) reported thrombus in major systemic or portal veins in 15% of 60 patients following BRTO. Interestingly, high-attenuation thrombus (n = 7), which was thought to represent iodized oil deposition, showed complete resolution at follow-up imaging, whereas low-attenuation thrombus (n = 2) was associated with late venous occlusion (69).

Figure 11. BRTO and transhepatic variceal obliteration in a 72-year-old woman with autoimmune hepatitis, Child-Pugh class C cirrhosis, and bleeding gastric varices. (a) Coronal venous phase CT image shows gastric varices and a gastrorenal shunt (arrow). (b) Retrograde venogram shows an 8.5-F Berenstein occlusion balloon (Boston Scientific) that was placed into the left adrenal vein; however, the varices are poorly opacified. A coil used for embolization of a small pericardiophrenic vein is faintly visible (arrow). A combined transhepatic approach was chosen to identify and sclerose the inflow vessels. (c–e) Digital subtraction venograms obtained with the occlusion balloon inflated (c) and deflated (d, e) show drainage through the gastrorenal shunt. (f) Digital subtraction venogram from the portal inflow. Cessation of bleeding was achieved with the administration of a foam mixture consisting of 8 mL of lipiodol, 8 mL of 3% tetradecyl sulfate, and 8 mL of air, in addition to the placement of several inflow coils. (g) Follow-up CT image demonstrates satisfactory occlusion.
Figure 12. US-guided access for sclerotherapy in a 77-year-old man with both primary hepatoma and colorectal cancer who presented with bleeding ileostomy varices in the right lower quadrant. Abdominal CT demonstrated extraluminal portal vein compression with intraluminal portal vein thrombus and established collateral vessels. (a, b) Digital subtraction venograms obtained following direct US-guided micropuncture set access show peristomal varices draining into the external iliac vein (arrow in b). (c) Fluorostore image obtained during injection of a sclerosant mixture of 2 mL of 3% tetradecyl sulfate, 2 mL of iodinated contrast material, and 2 mL of air, with compression over the outflow. (d) Postsclerotherapy venogram shows a satisfactory result. The patient had no further bleeding but died of hepatorenal syndrome 2 months later.

Miyamoto et al (70) suggest that the elevated portal pressure seen immediately after BRTO returns to baseline after 4 weeks as alternate collateral vessels develop. Ninoi et al (2) followed up 78 patients (mean, 700 days) who had undergone BRTO. The 5-year gastric varices recurrence rate was 2.7%, and the 5-year bleeding rate was 1.5% (3.5% in patients with a history of bleeding before BRTO) (2). Worsening of esophageal varices was observed in 37% of patients. Multivariate analysis demonstrated that the presence of esophageal varices before BRTO was a prognostic factor for worsening (2).

Tanihata et al (71) reported aggravation of esophageal varices in 11 of 19 patients (57%) 18 months after BRTO. The authors found that a portosystemic pressure gradient greater than 5 mm Hg was more strongly related to aggravation of esophageal varices than the presence of esophageal varices before BRTO.

Long-term results from BRTO are encouraging. In a study of 33 patients with initial bleeding from gastric varices, Hiraga et al (72) reported a 7-year cumulative worsening rate of esophageal varices of 52%. All patients were successfully managed endoscopically, with a recurrent bleeding rate of just 10% (72).
Figure 13. US-guided access for sclerotherapy in a 59-year-old man with a history of gallstone pancreatitis complicated by abscess and pseudocyst formation. The patient presented with significant upper gastrointestinal bleeding. Endoscopy demonstrated a large clot in the first part of the duodenum and several small ulcers in the second part. (a) CT angiogram is negative for pseudoaneurysm and active bleeding but demonstrates cavernous transformation of the portal vein with extensive periduodenal varices (arrow). A gas-containing peripancreatic collection (*) is also noted. Empirical gastroduodenal artery embolization was performed; however, the patient continued to require transfusion for ongoing melena and underwent repeat mesenteric arteriography (with negative results) 10 days after embolization. Following large-volume paracentesis, US was used to access the right gastroepiploic vein. (b) Venogram demonstrates extensive periportal varices (arrows). A total of 6 mL of a mixture of 2 mL of air, 2 mL of 3% tetradecyl sulfate, and 2 mL of lipiodol was injected through a 4-F Cobra glide catheter (Terumo, Somerset, NJ). This mixture partly filled the varices. More distal access was not possible due to vessel tortuosity. (c) Postsclerotherapy digital subtraction venogram shows no variceal filling and a patent gastroepiploic vein. The access tract was occluded using a Gelfoam (Pharmacia Upjohn) slurry. The bleeding ceased, and, although the patient had another episode of melena 4 months later, further intervention was not attempted.

Ectopic Varices: Percutaneous Access and Sclerotherapy

Stomal variceal bleeding can develop in patients with underlying cirrhosis and portal hypertension. Most patients are best treated with TIPS placement because this procedure addresses the underlying portal hypertension; however, stomal varices may bleed even when the transsinusoidal pressure is less than 12 mm Hg (7–9). For acute bleeding, transhepatic coil embolization of stomal varices has shown good results, although vessel recanalization or new varices formation may eventually mandate further intervention (Fig 8) (9). Freehand (non–image-guided) sclerotherapy is not recommended due to high rates of stomal damage (9); however, more proximal access with image-guided embolization can be a useful alternative in patients with contraindications to TIPS placement (6).

A thorough review of cross-sectional imaging findings is required to identify suitable veins. The use of US guidance to facilitate coil embolization or sclerotherapy of stomal varices has recently been reported (Fig 12) (6,73). Other veins may occasionally provide access to the portal system. Even with a micropuncture set, however, percutaneous access may be challenging (Fig 13). Minami et al (74) have reported a cutdown
technique for access to the left superficial epigastric vein to perform BRTO for stomal varices, and Nakata et al (75) have recently used a similar access route for sclerotherapy of jejunal varices. If a sclerosant is used, fluoroscopy-guided compression over drainage veins can decrease early outflow into the systemic system.

Surgical Shunts
In addition to TIPS and DIPS placement, there are several surgical portosystemic shunt placement procedures such as portocaval, mesocaval, mesoportal, or distal splenorenal shunt placement that can be performed in patients with portal hypertension. Unlike the use of endoscopic therapy (sclerotherapy or banding) for prevention of recurrent variceal bleeding in patients with cirrhosis, there is no consensus as to which approach is preferable. A Cochrane review (76) of 22 randomized clinical trials evaluating 1409 patients found that all shunts resulted in a significantly lower recurrent bleeding rate, but at the expense of a higher incidence of encephalopathy. No survival advantage was demonstrated with any shunt. Most studies comparing surgical shunts with TIPS have found the rates of thrombosis, stent stenosis, and reintervention to be significantly higher in the “TIPS” group. However, there are comparatively little data from the covered stent era. In specialized centers, patients with Budd-Chiari syndrome and cirrhosis can nearly always be treated with a combination of endoscopy, interventional radiology, and liver transplantation (77). In those rare instances in which these therapies fail in patients with cirrhosis, side-to-side splenorenal shunt placement remains a good option. Percutaneous intervention is typically successful in managing shunt dysfunction, including thrombosis and stenosis (Fig 14). In children, in whom extrahepatic portal vein occlusion is a relatively common cause of portal hypertension and bare stents may still be required, mesocaval or mesoportal shunts have demonstrated good long-term patency (78).

Peritoneovenous Shunt
Drainage of peritoneal fluid into the systemic venous circulation through a tunneled catheter with a nonreturn valve and a subcutaneous control pump can be safely performed by interventional radiologists (79,80). Guidelines from the American Association for the Study of Liver Diseases recommend peritoneovenous shunt placement only in patients who are diuretic resistant and are not candidates for transplantation or serial therapeutic paracentesis. In addition, patients require sufficient cardiorespiratory and renal function to minimize fluid overload from shunting of ascites into the intravascular compartment. Complications have been reported in up to 30% of patients and include shunt occlusion (e.g., fibrin sheath), infection, vena caval thrombosis, pulmonary edema, bleeding from varices, and disseminated intravascular coagulopathy.

In a randomized trial, bare stent TIPS showed significantly better assisted patency and ascites control than peritoneovenous shunts in the treatment of medically intractable ascites (81). Arai et al (82) have recently described the use of a novel transjugular transhepatic peritoneovenous shunt in patients with malignant ascites, with access into the abdominal cavity via a TIPS needle. This technique avoids the long subcutaneous tunneling required for Denver shunt placement and may be less invasive and more advantageous if catheter exchange is needed (82).

Conclusion
Interventional radiology can provide a number of image-guided procedures for the management of complications of portal hypertension in patients in whom archetypal TIPS placement has failed or is contraindicated. A careful review of individual pathophysiology, vascular anatomy, and hemodynamic flow patterns is crucial in selecting an appropriate intervention.

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References
Figure 14. Mesocaval shunt angioplasty in a 45-year-old man with Budd-Chiari syndrome who had undergone placement of a Dacron mesocaval shunt (DuPont, Wilmington, Del) 23 years earlier. The patient presented with increasing ascites. (a) Axial CT image demonstrates shunt stenosis with calcification (arrow). (b) Doppler US image suggests stenosis or occlusion of the shunt. SMV = superior mesenteric vein. (c) Digital subtraction venogram demonstrates moderate stenosis (arrow). (d) Fluorostore image shows an inflated angioplasty balloon across the stenosis. (e) Doppler US image obtained following dilation with 6-mm and 10-mm angioplasty balloons shows restoration of flow, which resulted in resolution of the ascites.


Image-guided Intervention in Management of Complications of Portal Hypertension: More than TIPS for Success

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Pages 1474
In patients with intra- and posthepatic obstruction, the HVPG (wedged pressure–free [unoccluded] hepatic venous pressure) is an accurate indirect measurement of portal pressure. It has been validated in multiple clinical settings as an independent predictor for adverse outcome, including the risk of developing varices, ascites, and clinical decompensation in cirrhotic patients.

Pages 1475
Ectopic varices outside the cardioesophageal region may be identified at endoscopic ultrasonography (US) but are often identified only at cross-sectional imaging.

Page 1485
Despite these reservations, PTE remains safe and effective for the treatment of special types of varices with portal hypertension and may be considered in cases in which TIPS placement fails or is contraindicated; and in patients with (a) bleeding ectopic varices, including rectal, stomal, and duodenal varices, among others; (b) splenic vein thrombosis with isolated gastric varices; or (c) gastric varices without a catheterizable draining vein.

Page 1488
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Page 1491
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