Endovascular Treatment for Variceal Hemorrhage: TIPS, BRTO, and Combined Approaches

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Semin Intervent Radiol 2018;35:169–184

Abstract

Keywords

- variceal hemorrhage
- gastric varices
- esophageal varices
- transjugular intrahepatic portosystemic shunt
- balloon-occluded retrograde transvenous obliteration

Variceal hemorrhage is a feared complication of portal hypertension, with high rates of morbidity and mortality. Optimal management requires a thoughtful, multidisciplinary approach. In cases of refractory or recurrent esophageal hemorrhage, endovascular approaches such as transjugular intrahepatic portosystemic shunt (TIPS) have a well-defined role. For hemorrhage related to gastric varices, the optimal treatment remains to be established; however, there is increasing adoption of balloon-occluded retrograde transvenous obliteration (BRTO). This article will review the concept, history, patient selection, basic technique, and outcomes for TIPS, BRTO, and combined TIPS + BRTO procedures for variceal hemorrhage.

Objectives: Upon completion of this article, the reader will be able to explain the indications for the use of TIPS, BRTO, and combined approaches in the treatment of esophageal and gastric variceal hemorrhage.

Accreditation: This activity has been planned and implemented in accordance with the accreditation requirements and policies of the Accreditation Council for Continuing Medical Education (ACCME) through the joint providership of Tufts University School of Medicine (TUSM) and Thieme Medical Publishers, New York. TUSM is accredited by the ACCME to provide continuing medical education for physicians.

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Variceal hemorrhage (VH) is a major complication of gastroesophageal varices (GEV) and is a leading cause of mortality in patients with cirrhosis.^{1,2} This review discusses esophageal varices (EV) and gastric varices (GV) as distinct anatomic and

pathologic entities, highlighting both historical and emerging trends in the utilization of transjugular intrahepatic portosystemic shunt (TIPS) and/or balloon-occluded retrograde transvenous obliteration (BRTO) to address VH.

TIPS and BRTO have evolved in relative isolation as different philosophical strategies to address VH.³ TIPS is more commonly utilized in North America and Europe where portal decompression with or without adjunctive embolization of varices⁴ has been a mainstay of endovascular strategy. Conversely, BRTO evolved in Asia as a direct treatment of VH by obliterating GEV (particularly GV) via sclerosis.³ These approaches were previously viewed as in conflict with one another as BRTO eliminates the gastrorenal shunt (GRS), aggravating portal hypertension, whereas TIPS is designed to reduce portal hypertension.³ Increasingly, TIPS and BRTO are viewed as complementary procedures that can be combined in specific clinical scenarios to reduce bleeding risk, while ameliorating sequelae of portal hypertension. Consideration of TIPS with/without adjunctive embolization, BRTO, or a combination of the procedures in VH requires an understanding of GEV progression and VH.

Pathophysiology and Epidemiology of Esophageal and Gastric Varices

Portal venous congestion in longstanding liver cirrhosis triggers a multifactorial process establishing collateral flow through GEV.⁵ A hepatic venous pressure gradient (HVP) ≥ 10 mm Hg is a critical threshold above which varices are known to form.⁶ EV are present in 52% of screened patients and are more prevalent with disease progression, seen in up to 43% of patients classified as Child–Pugh class A versus 72% in Child–Pugh class B/C.⁷ Comparatively, GV are less common and are classically thought to be present in 5 to 33% of patients with portal hypertension.^{8,9}

GEV progression is dynamic, with both vessel diameter and growth correlating with disease severity.¹⁰ Varices are typically classified as “small” or “large” at a threshold of 5 mm,^{11,12} with further subdivision incorporating a “medium” size based on morphology.¹² Conservatively, the incidence of new EV has been reported at 7 to 9% per year^{13–15} and progression from small to medium or large varices is reported to be 10 to 15% per year.^{14–16} GV development is thought to occur in a similar process, although studies have demonstrated that GV exist at lower pressures than EV (11–18 vs. 16–24 mm Hg).^{17,18}

The law of Laplace and Bernoulli's principle describe the pathophysiology of VH.^{15,19,20} GEV wall thickness decreases as vessel diameters expand due to increased transmural pressures from an elevated HVP. Ultimately, hemorrhage occurs when wall tension limits are exceeded.^{15,21} Although they share similar pathophysiologic principles, esophageal variceal hemorrhage (EVH) and gastric variceal hemorrhage (GVH) can be stratified as unique clinical events requiring varying treatment options.

EVH is highly prevalent given the high rates of EV in the cirrhotic population.^{8,9,22} EVH occurs at a rate varying between 5% yearly when small varices are present and 15% yearly in the setting of medium or large varices.²³ Spontaneous resolution of EVH has been reported in up to 40% of patients with EVH, but 6-week mortality approaches 20% for acute VH.^{12,24–26} A recent model for end-stage liver disease (MELD)-based analysis of VH reported an overall 16% mortality rate at 6 weeks, with mortality rates worsening with disease severity, varying between 5% (MELD < 11) and 20% (MELD ≥ 19).²⁷ Furthermore, VH can recur in 60% of patients within 1 to 2 years if untreated, with mortality rates as high as 33%.^{28,29} A unique population is decompensated patients with HPVG > 20 mm Hg within the first 24 hours of an acute VH. These patients exhibit higher rates of recurrent bleeding within 1 week, failure to control bleeding (up to 83%), and higher mortality (up to 64%).^{12,30,31}

Comparatively, GVH is much less common and accounts for only 10 to 30% of VH. Although GV bleed 50% less often than EV, GVH exhibit severe bleeding with higher transfusion requirements.⁸ Furthermore, bleeding rates are reported as high as 16% at 1 year, 36% at 3 years, and 44% at 5 years.⁹ Critically, GVH can occur at a lower portosystemic gradient (PSG) than EVH. In a recent retrospective analysis, 95 patients with acute VH demonstrated EVH at significantly higher pressures than GVH (23 vs. 17–20 mm Hg).³² These

findings mirror a previous study where bleeding occurred at a higher PSG in EV than GV (21.4 vs. 15.8 mm Hg).³³ Moreover, 10% of GV bled at a PSG < 12 mm Hg (compared with 2% for EV),³² echoing a cohort where 16% of patients with bleeding GV had a baseline HPVG < 12 mm Hg.³⁴

Anatomical Considerations

EV and GV exhibit anatomical characteristics that may drive pathological behavior and response to current treatment regimens. The Sarin classification system (→Fig. 1) is often used to delineate GV patterns⁸: gastroesophageal varices type 1 (GOV1) at the lesser curvature of the stomach, gastroesophageal varices type 2 (GOV2) at the greater curvature of the stomach, isolated gastric varices type 1 (IGV1) at the fundal region, and isolated gastric varices type 2 (IGV2) which are ectopic and rarely encountered. It should be noted that BRTO typically target GOV2 and IGV1 type varices.

EV are generally supplied by the left gastric vein (94–100%) and demonstrate “uphill” venous outflow (77–100%) through the azygos/hemiazygos venous system. Less commonly, EV may be supplied by the posterior gastric vein (16–25%) or short gastric vein (3–11%).^{22,35} GV demonstrate a more complex framework, with “downhill” drainage via a GRS predominating (~85%)^{18,35,36} or mix “uphill–downhill” venous outflow given their proximity to the EV.²² Overall, GOV are commonly supplied by the left gastric vein (86%) and posterior gastric vein (67%), but less so by the short gastric vein (4%). IGV demonstrate more complex patterning and are supplied with more frequency by the posterior gastric vein (64–70%) and the short gastric vein (25–64%) when compared with EV.^{22,35} These anatomic considerations make the management of GVH a more complicated endeavor than that of EVH. →Fig. 2 represents an idealized schematic of portal venous supply and systemic venous drainage for EV and GV.

Management of Variceal Hemorrhage

Primary prophylaxis for VH is recommended in high-risk patients with medium or large-sized GEV, utilizing either nonselective β blockade or endoscopic variceal band ligation (EVL).^{10,37} Upon failure of primary prophylaxis, management of VH involves a multidisciplinary approach that is beginning to more aptly address EVH and GVH as differentiated.^{10,37}

Presenting with gastrointestinal bleeding, cirrhotic patients undergo hemodynamic resuscitation with a hemoglobin transfusion threshold of 7 g/dL and antibiotic prophylaxis, given their susceptibility to bacterial translocation.^{2,10} Endoscopy is performed to determine if the bleeding source is due to VH or an arterial etiology such as peptic ulcer bleeding. Furthermore, endoscopy provides visualization of the bleeding site, an opportunity for EVL or sclerotherapy, and exploration of variceal anatomy.^{10,37} This approach is usually effective in all but 10 to 20% of patients.³⁸ Balloon tamponade and metallic stents may also be considered as bridge therapies until more definitive measures may be attempted.^{10,37} VH that is refractory to the aforementioned medical and endoscopic treatments may be referred for endovascular intervention.

Fig. 1 Sarin classification scheme for gastroesophageal varices.⁸ The percentages in the stomach (black text) represent the incidence of the variceal subtype, while the percentages to the left of the stomach (red text) represent the bleeding incidence for that subtype. The circled subtypes (GOV-2 and IGV-1) are typically treatable with BROTO. GOV, gastroesophageal varices; IGV, isolated gastric varices.

Fig. 2 Diagram of the primary portal venous supply and systemic venous drainage pathways for gastric and esophageal varices. Note that gastric varices are typically supplied via posterior gastric vein and short gastric veins, and drain via a gastrosplenic shunt, whereas esophageal varices are typically supplied via left gastric vein and drain via the hemiazygos vein; however, contributions and drainage pathways can be variable. A direct collateral from the splenic vein to the left renal vein without a submucosal (variceal) component is depicted here as a splenoportal shunt. White arrowheads denote afferent portal venous inflow, and black arrowheads denote efferent systemic venous drainage. AZY, azygos vein; EV, esophageal varices; GRS, gastrosplenic shunt; GV, gastric varices; H. AZY, hemiazygos vein; IVC, inferior vena cava; LGV, left gastric vein; LPV, left portal vein; LRV, left renal vein; PGV, posterior gastric vein; PV, portal vein; RPV, right portal vein; SGV, short gastric vein; SMV, superior mesenteric vein; SPV, splenic vein; SRS, splenoportal shunt.

TIPS

History

Conceptualized in the late 1960s by Rösch et al,^{39,40} TIPS has developed over nearly five decades.⁴¹ Initial experimentation involved Teflon coaxial tubing or silicone copolymer-coated spring coils in dogs. The silicone-coated shunt remained open for up to 2 weeks until thrombosis whereupon the tract remained patent for 24 to 48 hours after removal of the occluded device.⁴⁰ Further advancements were made utilizing a human cadaveric model⁴² and experimentation with a balloon-dilated shunt tract in an animal model.⁴³ By 1982, a balloon-angioplasty shunt tract was utilized alongside variceal embolization⁴⁴ in a patient with acute GEV hemorrhage. This pivotal report led to exploration of balloon-angioplasty tract formation in a series of 20 patients with acute GEV hemorrhage.⁴⁵

TIPS, as it is understood today, came to fruition in the late 1980s and early 1990s⁴¹ with the development of expandable metallic stents in an animal model.⁴⁶ The first metallic stents were deployed in patients in the late 1980s and early 1990s^{41,47–51} but faced patency issues.^{52,53} Biliary tract fistulas were contributing to shunt occlusion.^{3,54,55} The advent of polytetrafluoroethylene (PTFE)-covered stent grafts has greatly improved patency.^{56–58}

Indications/Contraindications

Indications for TIPS in the setting of VH (EV and GV) are well demarcated by American Association for the Study of Liver Diseases (AASLD) Practice Guidelines: (1) TIPS is indicated in rescue therapy for acute VH that is refractory to medical and endoscopic intervention; (2) it is also indicated in recurrent VH despite medical and endoscopic treatment; (3) an emerging indication is the early TIPS creation within 24 to 72 hours of acute VH.¹⁰

Contraindications to TIPS include prevention of VH in varices that have not previously bled, prevention of bleeding from gastric antral vascular ectasia, or primary therapy for portal hypertensive gastropathy.^{22,59} The Society of Interventional Radiology recognizes multiple relative contraindications including elevated heart pressures (left or right), heart failure or severe valvular insufficiency, rapidly progressive liver failure, severe or uncontrolled hepatic encephalopathy, uncontrolled systemic infection or sepsis, unrelieved biliary obstruction, polycystic liver disease, extensive primary or metastatic hepatic malignancy, and a severe, uncontrolled coagulopathy.⁶⁰

Preprocedure Evaluation

Patient selection includes a preprocedure work-up involving a combination of a complete blood count, metabolic panel, liver function test, bilirubin levels, and coagulation profile. Objective risk stratification can be performed utilizing the MELD score, which has demonstrated efficacy in early prognosis.⁶¹ Individual patients should be evaluated on a case-by-case basis.

Cross-sectional imaging with contrast-enhanced computed tomography (CT) or magnetic resonance imaging (MRI) can elucidate variceal pathways, demonstrate patency of the portal, splenic, and hepatic venous systems, and reveal

unrelieved biliary obstruction, and is critical for treatment planning. Ultrasound with Doppler can also be utilized to demonstrate venous patency when time constraints or renal insufficiency is present.

Technique

TIPS creation has been detailed many times over the years.⁶² Briefly, the procedure involves bridging a hepatic vein and a portal vein with a bile-impermeable Viatorr (W.L. Gore, Flagstaff, AZ) covered stent graft—which are purposely designed for TIPS. Shunt creation with an expandable covered stent graft is preferred over bare metal stents to maintain durable patency.⁶³ Most commonly the right hepatic vein is selected and wedged hepatic venography is performed to identify the right portal vein. A needle is then directed to the target portal vein, and once successful access to a portal vein branch is achieved, direct portal pressures are obtained and used to calculate a PSG. Splenoportal venogram is obtained to evaluate suitability of accessed portal vein branch, identify varices, and determine the correct length of stent, which is then deployed through the hepatic parenchymal tract. In patients with VH, incremental dilatation can be made with an 8- or 10-mm balloon to reduce the PSG to a target of 12 mm Hg or less.⁶⁴ Following TIPS placement, coil embolization of varices—typically left gastric vein (coronary vein)—is performed per the operator or institutional preference. When and how to embolize varices remains a point of debate, with many choosing to embolize only varices that persistently opacify on portal venography performed following TIPS placement (→Fig. 3).

Postprocedure Follow-up

Follow-up includes postprocedural intensive care to monitor for resolution of VH, complications of TIPS (including procedural-related hemorrhage), and renal function, and neurological monitoring for signs of hepatic encephalopathy. Liver function and coagulation profiles are also routinely obtained. Clinical follow-up includes ultrasound surveillance with Doppler at 1, 3, and 6 months to ensure shunt patency and adequate flow.

Major complications from TIPS creation are uncommon and are reported at a rate of 3%. Selected major complications include hemoperitoneum, biliary peritonitis, stent malposition, hemobilia, hepatic infarction, renal failure, accelerated liver failure, and severe or uncontrolled hepatic encephalopathy.⁶⁰ Rarely, the decision will be made to reduce or occlude a TIPS in the setting of uncontrollable hepatic encephalopathy or hepatic decompensation.⁶⁵ Minor complications are reported at a rate of 4% and include hepatic encephalopathy (controlled with medical management), transient renal failure due to intraprocedural contrast administration, transient pulmonary edema, fever, and access site hematoma.⁶⁰

Outcomes for TIPS in Variceal Hemorrhage

Decompression of an elevated HPVG through TIPS creation has a longstanding tradition in North America and Europe, and a wide breadth of data are available to support its use for the indications listed in the previous section. However, data

Fig. 3 (a) Digital subtraction splenoportal venogram prior to TIPS creation depicts a left gastric vein (arrow) supplying esophageal varices (arrowheads). (b) Following TIPS placement (arrowheads) and coil embolization of the left gastric vein (arrow), the esophageal varices are no longer visualized.

on the efficacy of TIPS in the setting of EV/EVH when compared with GV/GVH are sparse because datasets often combine these entities into GEV. Moreover, historical studies have relied heavily on bare metal stents although PTFE-covered stents are favored today. Contemporary debate has bloomed around the efficacy of TIPS and the role of adjunctive embolization, especially in the setting of GV.

Embolotherapy and TIPS

Variceal embolotherapy is routinely practiced during TIPS creation^{41,43} and has been historically reported in 24 to 48% of patients,^{66–68} though studies have reported success in controlling VH without adjunctive embolotherapy.^{52,69,70} Timing of embolization (pre-TIPS vs. post-TIPS embolization), selection of varices for embolization, and benefits of embolotherapy remain contentious. In the setting of EVH, the left gastric vein (coronary vein) is the most frequently embolized (► Fig. 3).

Conceptually, embolotherapy of collateral varices is a protective measure. In the era of bare metal TIPS, embolization of collateral varices would exclude a competing shunt

that may decrease TIPS patency, limit variceal filling, and thus limit recurrent VH due to shunt dysfunction.^{66,71} Pre-TIPS embolization has the technical benefit of increased GEV visualization due to variceal filling and decreased risk of nontarget systemic coil embolization, as no large-caliber shunt is present to allow systemic coil migration. Comparatively, post-TIPS embolotherapy provides the technical benefit of having a patent shunt to access and assessment of TIPS efficacy for variceal nonfilling.

Tesdal et al compared TIPS with embolotherapy to TIPS alone in a prospective study of 95 patients. Patients receiving TIPS and embolotherapy experienced a lower rebleeding rate (19 vs. 47%) at 4 years and also determined that embolotherapy had an independent effect on rebleeding.⁷² A recent meta-analysis of six studies by Qi et al concluded that TIPS combined with embolization conferred a significant reduction in rebleeding and improved TIPS patency at 6 months, although no significant differences in the incidence of death were observed.⁷³ Overall, studies generally demonstrate reduced rates of rebleeding with combined TIPS and embolotherapy when compared with TIPS alone (► Table 1).

Table 1 Sample studies evaluating rebleeding rates after TIPS with embolization versus TIPS alone

Study	Year	Patients	Study type	Rebleeding rate TIPS + Embo	Rebleeding rate TIPS alone	Significant difference
Tesdal et al	2005	95	Prospective	19	47	Yes
Wu et al	2009	263	Retrospective	17	26	Yes
Gaba et al	2011	52	Retrospective	7	22	No
Xue et al	2011	80	Retrospective	25	57	No
Xiao et al	2011	79	Retrospective	21	22	No
Wei et al	2011	122	Prospective	20	42	Yes
Chen et al	2013	106	Prospective	6	20	Yes
Shi et al	2014	101	Retrospective	6	19	Yes

This significant reduction in rebleeding with TIPS and embolotherapy was also observed in several other series including 349 patients (retrospective),⁷⁴ 122 patients (prospective),⁷⁵ 106 patients (prospective),⁷⁶ and 101 patients (retrospective).⁷⁷ However, a series of 79 patients compared TIPS with embolotherapy to TIPS alone, observing no significant differences in rebleeding or survival.⁷⁸ Similarly, no benefit in the risk of rebleeding was seen in another retrospective series of 137 patients.⁷⁹ Although suggestive that adjunct embolization may reduce rebleeding, this interpretation requires caution given the frequent association of rebleeding with reduced TIPS patency, which may have limited value with the emergence of PTFE-covered stents and raises questions of relevance in current practice.

Current rebleeding rates range up to 10%^{56,80–82} and may be related to decreased shunt dysfunction with use of PTFE-covered stents. The use of bare metal stents was identified as an independent risk factor for shunt dysfunction in a recent prospective series of 154 patients with VH undergoing TIPS and embolotherapy.⁸³ Moreover, shunt dysfunction has been reported in up to 70% of patients. Critically, though shunt dysfunction played a critical role in the era of bare metal stents, the utilization of PTFE-covered stents has demonstrated high patency with low rates of dysfunction.⁵⁷ A recent meta-analysis comparing covered stents and metallic stents analyzed 14 studies (4 randomized clinical trials, 2 prospective nonrandomized studies, and 8 retrospective studies; 2,519 patients total) and demonstrated that odds of patency (odds ratio, OR = 4.75) and survival (OR = 1.85) were significantly higher for PTFE-covered stent grafts, while odds of rebleeding were significantly lower (OR = 0.37).⁸⁴ Thus, the role of embolotherapy remains uncertain in our current era and questions about its use have been raised given involved cost, radiation exposure, procedure duration, and risk of postembolization syndrome.^{72,85}

TIPS in VH, Rescue, and Emerging Early TIPS

TIPS is highly effective for rescue therapy for both EVH and GVH. TIPS creation is almost always technically feasible and achieves bleeding cessation in over 90% of cases of EVH^{57,61} and GVH.^{86–91} Historical rebleeding rates in GV may be exaggerated in the era of PTFE-covered stents. Rebleeding rates post-TIPS are recently reported to be nearly 10% in EVH^{80,81} and as low as 10 to 20% in GVH.^{86,87,89–92}

Moreover, TIPS has also long been shown to be an effective modality for the secondary prevention of EVH. A recent meta-analysis of 10 randomized controlled trials involving 1,076 patients compared EVL, combination EVL and medical management, and TIPS for secondary prophylaxis of esophageal variceal bleeding⁹³: TIPS most effectively reduced bleeding-related mortality (relative risk, RR = 5.66) and rebleeding (RR = 2.20), though overall mortality was not significantly variable.

An emerging indication is early (nonsalvage) TIPS in the setting of acute VH. In a landmark study, 63 patients with acute Child–Pugh B/C VH were randomly assigned to early TIPS creation within 72 hours with a PTFE stent versus medical and endoscopic therapy. Early PTFE TIPS creation

yielded higher rates of bleeding control (97 vs. 50%), 6-week survival (97 vs. 67%), and 1-year actuarial survival (86 vs. 61%).⁸⁰ These results were validated in 2013 with similar bleeding control (93 vs. 53%) and 1-year actuarial survival reported (86 vs. 70%) in a retrospective analysis.^{81,94} However, in a prospective study matching 31 patients receiving early TIPS and medical/endoscopic treatment with historical cohort whose patients did not receive early TIPS, no significant difference was noted in actuarial 1-year survival, though significantly more patients who received early TIPS remained free of rebleeding events (97 vs. 51%).⁹⁵

Furthermore, a meta-analysis analyzing early TIPS within the first 5 days of VH in 608 cirrhotic patients revealed a significant risk reduction in 1-year mortality (RR = 0.68) and variceal rebleeding (RR = 0.28).⁹⁶ Similarly, a retrospective analysis of 142,539 patients with EVH revealed that when compared with no TIPS, early TIPS demonstrated significantly decreased inpatient mortality (RR = 0.87) and rebleeding (RR = 0.56) without significant increases in hepatic encephalopathy (HE).⁹⁷ A prospective observational studying of 964 patients with gastrointestinal bleeding in France revealed that 1-year actuarial survival was significantly increased in the 22 patients receiving early TIPS (86 vs. 59%), though rebleeding events were not recorded.⁹⁸

Effectiveness in EV versus GV

Although TIPS can establish control of acute GVH, it may not be as efficacious in this setting as GV persist and bleed at lower PSGs than EV.^{3,17,32,33} Though historically studies have often discussed the two as a single entity and direct comparisons between rebleeding rates in EV and GV have demonstrated no difference in rates of rebleeding,^{33,86,99} emerging data demonstrate that EV and GV respond differently to current treatment strategies. In a recent retrospective study of 78 patients with GVH who underwent TIPS creation with or without adjunctive embolization, GV remained patent (65%) with high rates of rebleeding (27%) and mortality at 90 days reported to be 15%. These findings echo a similar result where 50% of patients undergoing TIPS had persistent GV and 27% rebleed rate was reported.¹⁰⁰

There are several postulations as to why TIPS may not be as effective for establishing control of GV: (1) “proximity,” (2) “throughput,” and (3) “recruitment.” The “proximity” theory³ suggests that EV are well decompressed by TIPS because the primary vessel supplying these varices—the left gastric vein—is small enough and close enough to benefit from shunt creation. Conversely, as GV are larger caliber and anatomically remote from the TIPS shunt, they are likely not as susceptible to decompression as EV and have been observed to be persistent in this scenario.^{72,101} The “throughput” theory³ is based on the concept of low-pressure shunts from large-caliber inflow and outflow vessels associated with GV. These vessels may ultimately compete with TIPS and, in effect, decompress the shunt itself. The “recruitment” theory¹⁰¹ describes the development of new inflow feeding vessels after embolization of a GV complex, analogous to treating an arteriovenous malformation. Thus, the challenging nature of these varices prompts consideration of obliterative approaches.

BRTO

History

The first case example of retrograde gastric variceal obliteration in the literature was by Olson et al in 1984 at the University of Indiana. A patient who had undergone two previous transhepatic coil embolizations of bleeding EV was found to have numerous short gastric veins feeding large GV, which drained via a spontaneous GRS. With too many feeding vessels to coil, the authors had the insight to place an occlusion balloon in the spontaneous GRS above the inflow into the left renal vein, and reflux absolute ethanol into the varices, thereby sclerosing them.¹⁰² In context, the first TIPS was performed in a human 4 years later by Richter et al in Germany.¹⁰³

The modern concept and technical advancement of the procedure as it is now known was developed by Kanagawa et al in Japan in the 1990s, and he coined the term *balloon-occluded retrograde transvenous obliteration* (BRTO).¹⁰⁴

Indications/Contraindications

After long experience in Asia, BRTO has been increasingly adopted in the United States and Europe for the treatment of GV over the past decade. It is used primarily for prevention of recurrent GV bleeding, and has recently been included in the American Association for the Study of Liver Diseases (AASLD) practice guidelines for this indication alongside TIPS.¹⁰ Some experienced centers have also used BRTO as primary prophylaxis, but this indication has not been widely adopted—similar to TIPS, BRTO is not recommended in the new AASLD guidelines for primary prophylaxis of GV bleeding.^{10,105} Despite decades of use in Asia, and increasing adoption in the United States and Europe, defined patient selection for BRTO are lacking. Typically, it is utilized in patients who are at high risk for TIPS including elevated MELD (>18), right-sided heart failure, or hepatic encephalopathy.

Major contraindications include portal or splenic vein thrombosis without other portosystemic collaterals to provide adequate mesenteric or splenic venous outflow following BRTO, which can result in bowel or splenic ischemia secondary to venous congestion. Relative contraindications include a lack of a GRS, although experienced centers have success performing BRTO via nontraditional shunts such as the inferior phrenic vein.¹⁰⁶

Preprocedure Evaluation

As with TIPS, patients should previously have undergone endoscopic evaluation to confirm presence of GV as source of hemorrhage, or be at high risk for bleeding. Endoscopy is also necessary to evaluate for presence of preexisting EV, which may need to be banded. Cross-sectional (CT or MRI) imaging is critical to establishing candidacy for BRTO and for planning purposes. Evaluation of imaging includes: (1) confirming presence of catheterizable GRS (►Fig. 4c); (2) confirmation and location of GV (►Fig. 4a); (3) patency of portal and splenic veins; (4) presence of additional systemic collaterals that may need to be embolized (e.g., inferior phrenic vein); (5) identification of inflow portal branches (e.g., short and posterior gastric veins; ►Fig. 4b); and (6) identify additional

sequelae of portal hypertension such as ascites that may suggest benefit of adjunctive TIPS placement. Good preprocedure imaging evaluation can ensure that appropriate equipment is available for the procedure (e.g., occlusion balloon of appropriate size for the GRS), and save time by suggesting the best approach to catheterizing the shunt (e.g., transfemoral vs. transjugular) based on the coronal relationship of the inferior vena cava (IVC), left renal vein, and GRS. Laboratory evaluation including renal and liver panels, complete blood count, and coagulation panel should be obtained, and coagulopathy addressed as necessary. MELD calculation is useful to stratify mortality related to TIPS placement, and can be helpful to suggest which patients may benefit from BRTO based on elevated risk of TIPS placement.

Technique

Multiple detailed descriptions of the basic technique and modifications are available.^{104,107–111} Briefly, the goal of BRTO is to trap sclerosant within the GV by controlling both inflow and the outflow of the GV. Inflow is controlled by relying on the inflow portal pressure to ensure sclerosant is not refluxed into the portal venous system. The outflow is controlled by temporarily occluding the outflow GRS with an occlusion balloon. Thus, the first step is to catheterize the GRS. This can be performed via transfemoral or transjugular approaches, with the optimal approach dependent on operator preference and anatomic relationship of the GRS, left renal vein, and IVC. Once the GRS is catheterized, an appropriately size occlusion balloon is positioned above the left adrenal vein confluence and inflated to arrest flow. In large shunts, a balloon smaller than the maximum diameter of the shunt can often be placed above a web or stenosis that frequently exists in the GRS, and can gently be pulled down after inflating, a technique referred to as “corking” the balloon. With the occlusion balloon inflated, contrast is injected by hand, and a balloon-occluded retrograde venogram (BORV) is obtained. If systemic collateral outflow vessels are identified and result in significant “leakage” without visualization of the GV, they are embolized using coils, vascular plugs, gelatin sponge (Gelfoam; Pfizer, New York, NY), or sclerosant. After embolization of the systemic collaterals, repeat BORV is performed to confirm visualization of the GV (►Fig. 4d). A microcatheter is placed coaxially through the occlusion balloon, ideally all the way into the GV, and the sclerosant is instilled via the microcatheter (►Fig. 4f). Multiple sclerosant agents have been used. In the United States, most published experience is with 3% sodium tetradecyl sulfate (STS), which is mixed with air to create a foam, and ethiodized oil for fluoroscopic visibility. A typical mixture consists of a 3:2:1 mixture of air to sclerosant to ethiodized oil¹¹² (►Fig. 4e). Foamed sclerosant is advantageous as it maximized contact with the vessel wall, minimizing the overall amount required compared with pure liquid sclerosis and theoretically minimizing systemic toxicity. Instillation of the sclerosant is stopped after the GV are filled, and portal inflow vessel is identified. Intraprocedural C-arm cone-beam CT can be helpful to confirm adequate sclerosant in the GV, identify the portal inflow vessel,

confirming the end point for sclerosant administration (►Fig. 4g,h). The microcatheter is removed, and the sclerosant is allowed to dwell for a minimum of 4 hours while the occlusion balloon remains inflated. When the occlusion balloon is deflated, fluoroscopy is used to ensure stability of the sclerosant in the GV prior to balloon removal. Modifications exist which replace occlusion balloon with coils or vascular plugs to expedite this step and improve logistics.¹¹³ Recent adaptations of the technique use coils (coil-assisted retrograde transvenous obliteration) or plugs (plug-assisted retrograde transvenous obliteration) to occlude the GRS rather than an occlusion balloon, and gelatin sponge slurry rather than foamed sclerosant to obliterate the GV.^{114,115} These techniques can enhance logistics by eliminating the 4-hour balloon inflation time, and can result in obliteration of large-diameter shunts with smaller-diameter devices.

Postprocedure Follow-up

Following BRTO, the patient is observed overnight in the hospital. A postembolization syndrome including upper abdominal or flank pain, fevers, and occasionally leukocy-

tosis can be encountered, and may be related to the large clot burden intentionally generated during the procedure. Symptoms typically respond to conservative management with anti-inflammatory and analgesic medications as necessary. Hydration is encouraged to minimize systemic toxicity of sclerosants. Cross-sectional imaging is obtained within 1 week to ensure complete thrombosis of the varices, and early repeat treatment with BRTO, BATO, or endoscopic obliteration should be performed if there is persistent flow in the varices to minimize bleeding risk. Coordination with the referring hepatologist is critical to maintain routine scheduled upper endoscopy to evaluate and treat aggravation of EV, although formal guidelines are lacking.

The complication profile of BRTO can be subdivided into early procedural-related complications and later physiologic “complications” related to expected increase in portal hypertension. A large meta-analysis including 1,016 patients demonstrated a low overall major complication rate of 2.6%, and included small numbers of pulmonary emboli (4 patients), portal or splenic vein thrombosis (11 patients), renal vein thrombosis (3 patients), extravasation of sclerosant (5 patients),

Fig. 4 (a) Axial postcontrast CT of the abdomen demonstrates fundal gastric varices (white circle). (b) More caudal axial image from same CT demonstrates inflow via posterior gastric vein (white arrow). (c) Coronal CT image demonstrates gastroduodenal shunt (white circle) joining the left renal vein (black asterisk). Also seen is the inflow posterior gastric vein (white arrow). (d) Balloon-occluded retrograde venogram after coil embolization of an inferior phrenic vein (black arrow) demonstrates filling of the gastric varices (black circle). (e) Mixing sclerosant foam with the Tessari method. (f) Spot image following instillation of foamed sclerosant via coaxial microcatheter (black arrowhead) placed coaxially through occlusion balloon (black asterisk) shows filling of gastric varices (black circle) and early filling of posterior gastric vein (black arrow). (g) Intraprocedure cone-beam CT shows filling of fundal gastric varices (white circle) and (h) posterior gastric vein (white arrow) with good correspondence to preprocedure CT of the same structures (a, b).

Fig. 4 (Continued)

and death (2 patients).¹¹⁶ Major vessel thrombosis rates have been noted to be higher in other studies; however, major vessel thrombosis appears to be self-limited and well tolerated when it results from spilling of the foamed sclerosant.¹¹⁷ Balloon rupture has been reported in up to 9%.¹¹⁸ Respiratory effects related to sclerosant usage is relatively uncommon, and appears even less frequent with use of STS compared with ethanolamine oleate, the sclerosing agent most typically used in Japan.^{119,120}

Technically successful BRTO by definition closes a naturally occurring decompressive portosystemic shunt, and

would be expected to increase the PSG, thereby potentially exacerbating sequelae of portal hypertension. This has been validated by studies demonstrating an increased incidence or worsening of preexisting EV. The reported rates are variable, and it appears to be a time-dependent phenomena, with one study reporting worsening rates of 27, 58, and 66% at 1, 3, and 5 years post-BRTO, respectively.¹²¹ Similarly, worsening of ascites is variably reported in up to 35%, and likely depends on whether detected incidentally on imaging or defined as clinically evident or requiring therapy.¹²²

Outcomes

A recent meta-analysis by Park et al compiled postprocedure outcomes in 1,016 patients across 24 studies for those who underwent BRTO for the management of GV,¹¹⁶ and reflects results representative of the modern experience with gastric variceal obliteration. Technical success (also called immediate clinical success)—defined as complete thrombosis of GV on immediate or short-term follow-up imaging, successful injection of sclerosing agent into GV, and control of actively bleeding GV—was observed in 96.4% (95% confidence interval [CI]: 93.7–98.3) of patients. Clinical success, defined as no recurrence or rebleeding of bleeding GV (or no future bleed in high-risk GV) or complete obliteration of varices on subsequent imaging, was evident in 97.3% (95% CI: 95.2–98.8) of patients. Overall, these results suggest that BRTO is an effective and safe intervention for the management of GV. Of note, however, 23 of the included studies were retrospective, while only 1 was prospective, relegating a majority of the evidence to level 2 data. Additionally, only one of the studies included patients from the United States, while the other 23 spanned patients from either Japan or Korea. This regional disparity narrows the overall generalizability of the analysis due to population demographics. Moreover, the slant toward studies from the far east may artificially raise the cumulative clinical success rate, as BRTO is more often performed as prophylaxis in nonbleeding high-risk varices (for which it is unclear that bleeding would have ever occurred) in Asia, versus for bleeding GV in the United States.

The clinical outcomes of TIPS versus BRTO were described in a meta-analysis performed by Wang et al in 2016.¹²³ In collating 353 total patients (210 BRTO, 143 TIPS patients), the authors found no significant difference in procedure technical success (BRTO: 88% vs. TIPS: 98%, $p = 0.06$), hemostasis rate (BRTO: 100% vs. TIPS: 94%, $p = 0.30$), or complication rate (BRTO: 10% vs. TIPS: 5%, $p = 0.79$). However, rebleeding incidence (BRTO: 7% vs. TIPS: 23%, $p = 0.02$) and HE rate (BRTO: 1% vs. TIPS: 30%, $p < 0.00001$) favored obliteration.

Combination TIPS + Obliteration

The decision to pursue decompressive strategies via TIPS, or oblitative strategies via BRTO, does not need to be binary, but rather can be combined to utilize the comparative advantages of each procedure. This is particularly helpful in the treatment of GV. As discussed, GV are often related to large, high-flow GRSs. Thus, obliteration of the GV results in the elimination of this shunt, reducing the bleeding risk inherent to GV. The resultant expected increase in portal pressures and attendant increased risk of new/worsening ascites, aggravation of EV, and formation of difficult-to-treat ectopic varices can be ameliorated by the simultaneous or staged placement of TIPS. The combined result is the elimination of a high-flow spontaneous portosystemic shunt that is at risk for life-threatening hemorrhage (the GRS and gastric variceal complex), which is replaced by a man-made endovascularly created portosystemic shunt (TIPS) that is not at risk for rupture. Because flow rates are often higher through the GRS than through the TIPS, portosystemic shunting is expected to be reduced following the combined procedure, theoretically resulting in decreased risk

of encephalopathy compared with TIPS placement alone. Furthermore, TIPS patency may also be improved, as closure of competitive portosystemic shunts has been shown to improve short-term TIPS patency, also decreasing short-term variceal rebleeding rates.⁷⁶

As mentioned previously, there is increasing evidence that EV and GV are distinct both in terms of anatomy and response to therapy. This is supported by studies demonstrating GV bleeding at lower PSG compared with EV, including evidence that GV can bleed at $\text{PSG} < 12 \text{ mm Hg}$.³² Furthermore, GV were found to have similar patency rates after TIPS placement, whether or not they were previously embolized.¹⁰¹ This suggests that definitive treatment of GV may require obliteration rather than simple embolization. Thus, in the case of GV, TIPS, even with embolization, may be inadequate therapy, although a reduction in bleeding rates from the addition of BRTO to TIPS has yet to be described.¹²⁴

In addition to the physiologic advantages proposed, combining the procedures results in some technical advantages as well. When performing traditional BRTO, particularly early in one's experience, it can be difficult to elucidate the relevant anatomy. This is predominantly a result of the retrograde venography in which all of the afferent feeding veins may not be apparent during BORV. Additionally, complex systemic outflow may require time-consuming embolization prior to the instillation of sclerosant. When performing an obliteration after TIPS, the entirety of the afferent supply and efferent drainage of the GV can be seen during the trans-TIPS portal venogram. This improved anatomic delineation of the GV supply and drainage allows for easier planning of the ultimate GV obliteration. Finally, having control of the inflow (via a combination of embolization and occlusion balloons) in addition to standard control of the outflow (via plug embolization) may result in improved trapping of the sclerosant in the target GV and reduced likelihood of sclerosant migration into non-target vascular territories.

Conversely, TIPS can occasionally be made technically less challenging when staged after BRTO. This is because it is often noted that patients with large GV have relatively diminutive portal veins as the spontaneous shunt siphons large amounts of blood away from the liver. These small portal veins can be challenging to target during a standard TIPS and may require adjunctive techniques to gain portal vein access, which can increase procedure time, risk, and expense.^{103,125–128} In the authors' experience, following BRTO the portal vein is often larger in caliber as the previously shunted blood is redirected to the liver, resulting in easier targeting during subsequent TIPS.

Finally, there can be a logistical advantage of combining or staging TIPS + obliteration by eliminating the need for prolonged balloon inflation times used in traditional BRTO techniques. As described in the technique section in the following, the GV are securely "trapped" by embolizing the inflow and outflow vessels with a combination of vascular plugs and coils, and an occlusion balloon is only briefly necessary during the instillation of sclerosant, after which it is quickly removed following embolization of that final remaining inflow vessel with coils. This removes the need for

observation in the intensive care unit (ICU) during which the balloon is inflated, and decreases the overall time of the procedure as well as the associated resource utilization.

Technique

At the authors' institution, we typically prefer to perform TIPS + obliteration in a planned staged fashion, beginning with TIPS. Following TIPS placement via previously described techniques, a thorough splenoportal venogram is obtained (► Fig. 5c,d). It is imperative to image from the splenic hilum all the way to the newly created TIPS, to identify all potential inflow vessels to the GV. The predominate inflow vessel to the GV is identified and initially preserved, while all other inflows are embolized via coils or vascular plugs. Next, the GRS is catheterized in a retrograde fashion from the left renal vein via a transfemoral or transjugular approach in a similar fashion as a BRTO. Suitably sized vascular plugs or coils are used to embolize the outflow. An occlusion balloon is placed

into the remaining inflow vessel via a trans-TIPS approach, and a microcatheter is placed coaxially through the balloon deep into the GV. The sclerosant is then instilled via the microcatheter until the GV are filled (► Fig. 5e). The sclerosant foam is the same 3:2:1 mixture of air: sclerosant: lipiodol as described for BRTO. The microcatheter is then used to coil embolize the remaining inflow vessel prior to deflation and removal of the occlusion balloon. Final splenoportal venogram is performed to confirm no further flow through the GV (► Fig. 5f).

Postprocedure Follow-up

Follow-up combines elements of both TIPS and BRTO follow-up algorithms. The patient is observed for complications overnight in the hospital—this can be in an ICU setting if the TIPS was difficult or complicated. As with BRTO, intravenous hydration is encouraged to minimize systemic toxicity of sclerosants. Cross-sectional imaging is obtained within

Fig. 5 (a) Axial postcontrast CT of the abdomen demonstrates paraesophageal varices (white circle). (b) More caudal axial image from same CT demonstrates large gastric varices (black circle). Early- (c) and late-phase (d) splenoportal venogram after TIPS (white arrowheads) placement shows persistent filling of large gastric varices (white oval) with primary inflow via short gastric vein (black arrow) and contribution from left gastric vein (black arrowheads), which also fills esophageal varices (black oval). Outflow is via gastroduodenal shunt (white asterisk). (e) Following embolization of the left gastric vein with coils and vascular plug (black arrowheads) and the gastroduodenal shunt outflow with vascular plugs (white asterisks), foamed sclerosant is instilled into the gastric varices (white circle) via a coaxial microcatheter placed through an occlusion balloon (black arrow) positioned in the short gastric vein. (Adjacent coils are in an additional small short gastric vein). (f) Final splenoportal venogram demonstrates widely patent TIPS (white arrowheads) with no filling of the gastric or esophageal varices. Additional coils (black asterisk) were placed in the short gastric vein prior to removal of the occlusion balloon. (g) Follow-up axial postcontrast CT of the abdomen demonstrates retained lipiodol in the gastric varices (black circle) and patent TIPS (black arrow). Metallic streak artifact is from coils in the left gastric vein. LRV, left renal vein; PV, portal vein; SV, splenic vein.

Fig. 5 (Continued)

1 week to ensure complete thrombosis of the varices, and repeat treatment with BATO should be performed if there is persistent flow in the varices to minimize bleeding risk. Routine scheduled ultrasound surveillance of the TIPS should be obtained to optimize patency. Routine endoscopic follow-up is typically not necessary assuming technically successful obliteration and final PSG < 12 mm Hg, and the TIPS remains patent on follow-up ultrasound.

Outcomes

There is a paucity of data with internal controls evaluating the effectiveness of TIPS with obliteration. A 2013 study by Saad et al evaluated 36 patients undergoing BRTO for GV, of which 9 had TIPS (either previously placed or concurrently placed). The ascites/hydrothorax free rate for BRTO only versus BRTO + TIPS at 6, 12, and 24 months was 58, 43, and 29% versus 100, 100, and 100%, respectively ($p = 0.01$),¹²⁴ suggesting a protective effect of portal decompression complementing GV obliteration from BRTO. There was also a significant reduction in recurrent hemorrhage for the BRTO versus BRTO + TIPS groups at the same time points: 9, 9, and 21% versus 0, 0, and 0%, respectively ($p = 0.03$),¹²⁴

also suggestive of a protective effect of TIPS against other sources of portal hypertension-related bleeding besides GV, which could be aggravated by BRTO.

Conclusion

Over the past few decades, TIPS and BRTO have cemented themselves as foundational procedures in the interventional radiology armamentarium for the treatment of variceal bleeding. While varying in their underlying mechanism—namely portal pressure decompression versus direct variceal eradication—both are associated with high technical and clinical success rates, allowing effective control of hemorrhage from GEV. Future experience will dictate whether different variceal subtypes warrant specific therapies, and whether combination approaches confer benefit over either therapy alone.

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