Balloon-occluded Retrograde Transvenous Obliteration (BRTO): Technical Results and Outcomes

Wael E. A. Saad, M.D., F.S.I.R.,[1] and Saher S. Sabri, M.D.[1]

ABSTRACT

Variceal bleeding is one of the major complications of portal hypertension. Gastric variceal (GV) bleeding is less common than esophageal variceal (EV) bleeding, however, is associated with a high morbidity and mortality. Balloon-occluded retrograde transvenous obliteration (BRTO) is an established procedure for the management of gastric varices in Japan and has shown promising results in the past decade. The technical success rate, intent-to-treat (including technically failed BRTO-procedures) obliteration rate, and the obliteration rate of gastric varices of technically successful BRTO procedures was 91% (79–100%), 86% (73–100%), and 94% (75–100), respectively. BRTO is successful in controlling active gastric variceal bleeding in 95% of cases (91–100%) and in significantly reducing or resolving encephalopathy in 100% of cases. However, BRTO diverts blood into the portal circulation and increases the portal hypertension, thus aggravating esophageal varices with their potential for bleeding. The 1-, 2-, and 3-year esophageal variceal aggravation rates are 27–35%, 45–66%, and 45–91%, respectively. The gastric variceal rebleed rate of successful BRTO procedures, the intent-to-treat gastric variceal rebleed rate, and the global (all types of varices) variceal rebleed rate are 3.2–8.7%, 10–20%, and 19–31%, respectively. However, the advantage of diverting blood into the portal circulation and potentially toward the liver is improved hepatic function and possible patient survival. Unfortunately, the improved hepatic function is transient (for 6–12 months); however, it is preserved in the long-term (1–3 years). Patient 1-, 2-, 3-, and 5-year survival rates are 83–98%, 76–79%, 66–85%, and 39–69%, respectively. Patient survival is determined by baseline hepatic reserve and the presence of hepatocellular carcinoma.

KEYWORDS: BRTO, transvenous obliteration, varices, rebleeding, hepatic function, survival

Objectives: Upon completion of this article, the reader should be able to explain the expected results and outcomes of balloon-occluded retrograde transvenous obliteration (BRTO) and identify the limitations of the BRTO procedure, as well as the limitations of the literature discussing the BRTO procedure.

[1]Division of Vascular Interventional Radiology, Department of Radiology, University of Virginia Health System, Charlottesville, Virginia.

Address for correspondence and reprint requests: Wael E. A. Saad, M.D., F.S.I.R., Department of Radiology and Medical Imaging, University of Virginia Health System, Box 800170, 1215 Lee Street, Charlottesville, VA 22908 (e-mail: wspikes@yahoo.com).

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Variceal bleeding is one of the major complications of portal hypertension. Esophageal variceal bleeding is more common due to portal hypertensive varices (70–80%)[1] and the role of minimally invasive procedures is well established.[2] Gastric variceal bleeding is less common (20–30%)[1,3,4]; however, it is associated with a high morbidity and mortality (45–55% mortality) and its management is largely uncharted due to a relatively less-established literature.[2,5–7] Furthermore, endoscopic management of gastric varices is less effective when compared with its role in the management of esophageal varices.[7–11]

From an interventional radiology standpoint, gastric varices can be managed with the transjugular intrahepatic portosystemic shunt (TIPS) and/or balloon-occluded transvenous obliteration (BRTO).[3,6,12–14] In Japan the primary school of management in the presence of a gastrorenal shunt is to address the gastric varices specifically by sclerosing them utilizing the BRTO procedure. The gastrorenal shunt acts as the tranvenous (transfemororenal or transjugulorenal) shunt to the gastric varices (please see "Balloon-occluded Retrograde Transvenous Obliteration (BRTO): Technique and Intraprocedural Imaging" in this issue). This spontaneous gastrorenal (a natural portosytemic decompressive) shunt is occluded in the BRTO procedure resulting in diversion of blood flow toward the portal circulation and in turn the liver.[15–18] This blood diversion has advantages and disadvantages.

In this article we discuss the technical, anatomic, and clinical outcome of the traditional balloon-occluded retrograde transvenous obliteration (BRTO) procedure. Modifications and alternative route transvenous obliteration is discussed in "Variations of Balloon-occluded Retrograde Transvenous Obliteration (BRTO): Balloon-occluded Antegrade Transvenous Obliteration (BATO) and Alternative/Adjunctive Routes for BRTO" in this issue.

TECHNCAL AND ANATOMIC RESULTS OF BRTO

Candidacy of the BRTO Procedure

Candidacy for the traditional "transrenal" balloon-occluded retrograde transvenous obliteration[19,20] varies depending on definition in its simplest and most obvious form, the definition refers to patients with gastric varices without gastrorenal or gastrosplenorenal shunts. Gastrorenal shunts occur in 39–85% of patients with gastric varices.[1,3,21] Patients without a gastrorenal or gastrosplenorenal shunt are not candidates for the "traditional/classic" BRTO-procedure. Unfortunately, it is usually difficult to audit these patients in retrospective studies in which the vast majority of BRTO studies are classified[1,3,4,6,14–18,22–45] and incidence is probably underestimated in the studies that do disclose their frequency (8.6% of patients).[39] There is a wide disparity in the rate of gastrorenal shunts in patients with gastric varices (39–85%).[1,3,21] Patients with gastrorenal shunts who have a shunt that is too wide to be occluded/trapped by the largest available occlusion catheter/sheath are not candidates for BRTO.[6] However, this is supposed to be a form of technical failure and is categorized a type-V shunt according to Fukuda et al.[6] A third set of non-candidacy (the first and second being having no shunt or having too large a shunt) are patients that have a contraindication to the BRTO procedure despite having no gastrorenal shunts.[18] These contraindications include a hepatocellular carcinoma greater than 5 cm in diameter and large volume intractable ascites.[18] These occur in 3.7% of patients.[18]

Technical Success of the BRTO Procedure

Overall the technical success of patients with gastrorenal shunts (noncandidates not included) for BRTO only without adjunctive endoscopic sclerotherapy and/or BATO rescue ranges from 79–100%.[1,3,4,6,14–18,27–31,37–39,42,43] Two studies clearly identified primary treatment of gastric varices with BRTO, reserving BATO via a percutaneous transhepatic route as a rescue.[6,27] These two studies had a BRTO and/or BATO-rescue technical success of 84–98% (BRTO only) and 100% (BATO only or both BRTO and BATO), respectively.[6,27]

Another approach is to sclerose the gastric varices in multiple sessions to limit the sclerosant ethanolamine oleate to 20–30 mL per session in an attempt to reduce the dose-related complication of hemolysis and hemoglobinuria-induced renal dysfunction. Patients who do not have gastric varices obliterated from a prior session are returned for a subsequent session.[6,12,18,39,41] This approach/technique was first described by Sonumura et al in 1999.[28] In five studies adopting this approach in 210 BRTO procedures, the first, second, and third BRTO sessions were technically successful with complete obliteration of the gastric varix in 56–87%, 79–100%, and 81–100%, respectively.[6,12,18,39,41] One study had complete (100%) technical success in all cases by the second session.[39] Cumulatively, the first, second, and third session technical success rates of the 210 BRTO

cases in these five studies were 71% ($N = 150/210$), 88% ($N = 185/210$), and 91% ($N = 191/210$), respectively.[6,12,18,39,41]

In 10 studies with intent-to-treat gastric varices from one session, evaluating 457 BRTO procedures with and without BATO rescue, 419 cases (91.7%) were technically successful.[4,14–17,27,29–31,42] The range of technical success in each individual study of these 10 studies was 84–100%.[4,14–17,27,29–31,42]

We classify the causes of the technical failures as: type I—inability to cannulate the gastrorenal shunt with or without contrast or sclerosant extravasation; type II—unable to occlude the shunt due to undersized balloon (gastrorenal shunt is too wide; Type V shunt by Fukuda et al);[6] type III–unable to opacify the shunt despite a well-inflated and appropriately sized balloon due to a complex multicollateral gastrorenal/gastric variceal system (IIIa), or extravasation of contrast/sclerosant into the retroperitoneum (IIIb); and type IV–early (usually within 1–2 hours) occlusive-balloon rupture requiring a repeat BRTO. Balloon ruptures occur in 2.3–8.7% of BRTO cases;[37,45] however, not all occur early thus leading to technical failure (up to 50% of balloon ruptures are early and cause technical failure).[45] Unfortunately, not all technical failures are disclosed in detail and research reporting standards are required. Having said that, the most commonly but unquantified cause of technical failure is a complex multicollateral gastrorenal system and as a result the inability to fully opacify, "trap," and sclerose the gastric varix (Type III failure).[1,3,4,6,14,17,18,22–26,28–45] In three studies evaluating 160 BRTO procedures with 14 technical failures (91.3% technical success), type I failures (cannulation/extravasation) represented 14% ($N = 2/14$) of failures and 1.3% ($N = 2/160$) of BRTO cases; type II failures (small size for balloons) represented 36% ($N = 5/14$) of failures and 3.1% ($N = 5/160$) of BRTO cases; type III failures (unable to opacify and "trap") represented 43% ($N = 6/14$) of failures and 3.8% ($N = 6/160$) of BRTO cases; and type IV failures (early balloon rupture) represented 7.1% ($N = 1/14$) of failures and 0.6% ($N = 1/160$) of BRTO cases.[15,16,27]

The procedural and postprocedural (including long-term) complications are tabulated in Table 1.[1,3,4,6,14–18,26,28,29,31,34,37,42–45]

Table 1 Procedural Complications of Balloon-occluded Transvenous Obliteration (BRTO) Utilizing Ethanolamine Oleate

Complication Type	Incidence (%)
Procedural Complications	
Gross hematuria	15–100*
All pulmonary embolism	1.5–4.1
Symptomatic pulmonary embolism	1.4–2.5
Cardiac arrhythmia	1.5
Anaphylaxis	2.2–5.0
Rapid / fulminant hepatic failure	4.8–7.0
Death within 30 days from fulminant hepatic failure	0.0–4.1
Renal failure	4.8
Long-term complications	
Encephalopathy	17.6†
Portal hypertensive gastropathy	5.3–13.2
Post-BRTO gastropathy (not to extent of portal hypertensive gastropathy)	56.5
Aggravation of esophageal varices	14–68‡
Bleeding from esophageal varices	17–24‡
Duodenal varices	Up to 3.2
Bleeding duodenal varices	Up to 2.3
Ascites	0–43.5
Spontaneous bacterial peritonitis	Up to 8.2
Pleural effusion (hydrothorax)	5.3–7.9
Portal vein thrombosis	Up to 4.7
Renal vein thrombosis (no clinical consequences)	Up to 5.0

Based on data from references[1,3,4,6,14–18,26,28,29,31,34,37,42–45].
*This is a common complication and is usually without clinical consequences. The wide range is probably due to how well it is documented for the retrospective audit which most of studies are categorized as.
†The rate of encephalopathy is subject to definition and how closely there was clinical follow-up and may vary widely. This is a feature of the entire portal hypertension interventional radiology literature and is not specific to the BRTO literature.
‡The rate of variceal aggravation is subject to definition and how closely there was endoscopic follow-up and may vary widely. In addition (anecdotally), aggressive pre-BRTO banding may reduce the incidence esophageal varices and/or bleeding from these varices even with close post-BRTO endoscopic follow-up.

Anatomic Outcome of the BRTO Procedure

This refers to the complete obliteration of the gastric varices by follow-up imaging such as CT Venography, MR Venography, and/or Endoscopic Ultrasound (EUS) (please see "Balloon-occluded Retrograde Transvenous Obliteration (BRTO): Follow-up and Postprocedural Imaging" in this issue). In 14 studies evaluating post-BRTO gastric variceal obliteration in 580 BRTO procedures, the intent-to-treat (including technically failed BRTO procedures) obliteration rate and the obliteration rate of gastric varices of technically successful BRTO procedures was 73–100% and 75–100%, respectively.[1,3,4,6,14–16,18,28–30,37–39] Cumulatively for the same 14 studies the A = technical success rate, B = the intent-to-treat (including technically failed BRTO procedures) obliteration rate, and C = the the obliteration rate of gastric varices of technically successful BRTO procedures was A = 91% ($N = 529/580$), B = 86% ($N = 496/580$), and C = 94% ($N = 496/529$), respectively.[1,3,4,6,14–16,18,28–30,37–39]

Outcome of BRTO for Patients with Gastric Varices

The primary indications for BRTO is gastric variceal bleeding (or potential bleeding) and refractory

encephalopathy in the presence of a gastrorenal shunt.[1,3,4,6,14–18,22–45] The effectiveness of BRTO in controlling bleeding gastric varices is 91–100% in two studies evaluating 20 patients ($N = 19/20$, controlled: 95%).[4,6] The gastric variceal and global variceal rebleed rate are discussed below. In five studies evaluating 35 patients with encephalopathy, there was resolution or significant reduction in encephalopathy in all (100% success) patients.[3,6,18,32,42] Long-term postprocedural complications, when mentioned, are displayed in Table 1.[1,3,4,6,14–18,26,28,29,31,34,37,42–45]

The aggravation of nongastric (esophageal or duodenal) varices appears to be a major problem on the long-run and is reflective of increasing portal hypertension following BRTO.[1,3,4,6,14–16,26,31,34,43–45] It varies widely probably depending on the degree of vigilance, documentation, and thoroughness of follow-up endoscopy. However, in four main studies evaluating 160 patients who had undergone BRTO who had continuous endoscopic follow-up post-BRTO, the esophageal variceal aggravation rates (expressed as a Kaplan-Meier analysis) at 1, 2, and 3 years was 27–35%, 45–66%, and 45–91%, respectively.[14,18,31,40] In another two studies evaluating 117 patients with BRTO, the percentage of patients with aggravated esophageal varices was 30–68% and the patients that had bleeding esophageal varices was 17–24% of patients (36–57% of patients with aggravated esophageal varices went on to bleeding).[1,15] Again, one can argue that the percentage of esophageal variceal bleeding may be significantly reduced by a higher vigilance of endoscopic follow-up and more aggressive endoscopic therapy (esophageal banding and/or sclerotherapy). Other complications reflective of increased portal hypertension following BRTO is the development of portal hypertensive gastropathy (occurs in 5–13%) and possibly ascites (occurs in 0–44%) and hydrothorax/pleural effusion (occurs in 0–8%).[17,18,28,31,37,42]

The rebleeding rate following BRTO depends on how it is presented. We believe that there is a pressing need to standardize reporting of BRTO research. Most studies display a gastric variceal rebleed rate of patients who had undergone a successful BRTO procedure that ranges between zero and 20% (all studies except one with a gastric rebleed rate under 10%).[1,3,4,6,14–18,27–31,39,42,44] However, when factoring in an intent-to-treat basis (including technical failures) for the results, the gastric variceal rebleed rate is zero to 31.6%.[1,3,4,6,14–18,27–31,39,42,44] Many studies do not clearly state what, if any, is the global rebleed rate from gastric, esophageal, duodenal varices as well as portal hypertensive gastropathy.[1,3,4,6,14–18,27–31,39,42,44] In three clearly reported studies evaluating 141 patients who had undergone a BRTO procedure, the gastric variceal rebleed rate of successful BRTO procedures, the intent-to-treat gastric variceal rebleed rate, and the global (all types of varices) variceal rebleed rate was 3.2–8.7%, 10–20%, and 19–31%, respectively.[4,15,16]

One of the greatest advantages of BRTO is probably its preservation of hepatic function and its reduction in the risk of hepatic encephalopathy. In fact, one of the indications for BRTO is encephalopathy with the presence of a gastrorenal or gastro-splenorenal shunt.[3,6,18,32,33,38,42] In five studies evaluating 35 patients with encephalopathy, there was resolution or significant reduction in encephalopathy in all (100% success) patients.[3,6,18,32,42] The Kaplan-Meier survival rate after BRTO is impressive. The 1-, 2-, 3-, and 5-year survival rates range from 83–98%, 76–79%, 66–85%, and 39–69%, respectively.[3,27,31,35,40–42] Obviously, the greatest determinate of survival is the patient's hepatic reserve (determined by Child-Pugh score and/or MELD score).[6,15,38,39] However, hepatocellular carcinoma is also a significant determinate of survival[18,38,39] to the extent that prior authors have considered an intrahepatic hepatocellular carcinoma of >5 cm as a contraindication to BRTO.[18]

There are two recent and very interesting studies of note.[17,31] One discusses the reservation of hepatic function after BRTO and states that BRTO has a hepatic function protective value for patients with gastrorenal shunts.[17] The second study evaluates splenic artery embolization in an attempt to modulate the splenic vein contribution to the portal circulation and thus reduce the potentially adverse effects of increasing portal pressure post-BRTO.[31]

The study by Kumamoto et al consisted of three groups of patients.[17] One group consisted of patients with no gastrorenal shunt (the control group); the second had a gastrorenal shunt that was not treated (with BRTO or by any other means); and the third included patients with gastrorenal shunts that were treated with BRTO.[17] Those with untreated gastrorenal shunts had progressively deteriorating hepatic function (by Child-Pugh score), whereas those that had BRTO had transient improvement in hepatic function for 6–12 months and then a return (preserved) to baseline hepatic function up to 3 years. The patients without gastrorenal shunts (control group) had stable hepatic function similar to those patients with BRTO. This suggests that BRTO had a protective long-term role in preserving hepatic function and protecting the liver from portosystemic shunt syndrome.[17] This brings the discussion to another level for the indications of BRTO. Are the indications for BRTO gastric variceal management (history of bleeding, current bleeding, or impending bleeding) and/or hepatic encephalopathy? Alternatively, should we block all "significant" portosystemic collaterals for the sake of hepatic function preservation and longevity of cirrhotic livers and portal hypertensive patients?

The study by Chikamori et al consisted of two groups of patients.[31] One group had patients who underwent BRTO and the other group had patients who underwent BRTO and concomitant splenic artery embolization.[31] The intent was to reduce the adverse effects of increased portal hypertension that occurs subsequent to BRTO particularly the aggravation of esophageal varices (which are reflective of increased portal hypertension). The aggravation/development of esophageal varices at 6, 12, 24, and 36 months for the BRTO versus the BRTO + splenic embolization groups was 16, 27, 45, 45% versus 0, 0, 9, and 9%, respectively ($p < .05$).[31] This may be the solution to many of the untoward effects of increased portal hypertension by concomitantly modulating the splenic vein contribution to the portal circulation.[31] In fact, one can think of this as the endovascular alternative to the Hassab operation for gastric varices which is a splenectomy and a proximal (upper) gastric devascularization.[31,46,47] Unfortunately, the Hassab operation is not well tolerated by patients with comorbidities and poor hepatic reserve (advanced cirrhotics) in an attempt to modulate the splenic vein contribution to the portal circulation and thus reduce the potentially adverse effects of increasing portal pressure post-BRTO.[31,47]

CONCLUSION

The BRTO procedure is an effective moderately invasive procedure. It should be considered as a valuable procedure in the armamentarium of interventional radiologists for the management of gastric varices and/or encephalopathy in the presence of a gastrorenal shunt. BRTO most likely is a procedure that preserves the hepatic function and thus patients with poor hepatic reserve along with other non-TIPS candidates should be triaged to undergo a BRTO procedure if it is feasible (if a gastrorenal shunt exists).

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