

# **Case Report**

# Balloon-occluded retrograde transvenous obliteration for treatment of bleeding gastric varices: case report and review of literature

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#### ABSTRACT

Gastric variceal bleeding is a major complication of portal hypertension and is associated with high morbidity and mortality. While esophageal varices are more common, gastric varices are often more challenging to treat. Balloon-Occluded Retrograde Transvenous Obliteration is an interventional procedure whereby the portosystemic gastrorenal shunt is accessed via the left renal vein and the gastric varix outflow tract obliterated using direct sclerotherapy. Herein, we present a case of a 68-year-old female patient with cirrhosis who presented with bleeding gastric varices and successfully treated. This case highlights the procedural steps and the importance of detailed knowledge of the patient's portosystemic anatomy for determining suitability for balloon-occluded retrograde transvenous obliteration of gastric varices.

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## Introduction

Gastric varices (GV) develop in approximately 20% of patients with portal hypertension [1]. Most GV are associated with a left-sided spontaneous portosystemic shunt of varying complexity. Although GV bleed less frequently as compared to esophageal varices, GV bleeding is difficult to manage endoscopically due to their size, location, and high-volume blood flow [2]. Furthermore, GV are associated with a higher risk of rebleeding and increased mortality rate [3]. Balloon-occluded retrograde transvenous obliteration (BRTO) is a safe and effective procedure for treating GV and reducing the risk of rebleeding [4]. BRTO involves temporary occlusion of outflow veins of the portosystemic shunt followed by endovascular injection of a sclerosant into the varix. Over the last 2 decades, BRTO has been a common modality used for the prevention and treatment of bleeding GV in Japan and various parts of Asia. However, it has only recently gained wider attention in North America and is still underused for treatment of GV. This case describes the key clinical and anatomic features of GV in a patient who was a suitable candidate and was successfully treated with BRTO.

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## **Case report**

A 68-year-old woman with a history of liver disease was transferred from a regional hospital to a tertiary care hospital for urgent assessment and treatment of large GV along the greater curvature of the stomach with previous bleeding. Before arrival, the bleeding GV had been temporized with endoscopic clip placement. On admission, the patient was alert, oriented, and hemodynamically stable.

Laboratory investigations yielded the following results: Hgb 90 (normal, 120-160) g/L, red blood cell count 3.8 (normal, 3.8-5.8)  $\times 10^{12}$ /L, white blood cell count 7.8 (normal, 4.5-11)  $\times 10^{9}$ /L, and platelets 163 (normal, 150-350)  $\times 10^{9}$ /L. Her cardiovascular and respiratory examinations were unremarkable. Her abdomen was soft and nontender. There was no peripheral edema or any other stigmata of chronic liver disease (Child-Pugh class A). Her medical history was significant for anemia, diabetes, hypertension, dyslipidemia, cholecystectomy, and cirrhosis. Hepatology and interventional radiology services assessed her for possible consideration of BRTO for treatment of the GV.

Triphasic computed tomography (CT) of the abdomen and pelvis was performed with noncontrast, arterial, and portovenous phase multiplanar imaging. Very large GV were found in association with a splenorenal shunt draining into the left renal vein (LRV; Figs. 1 and 2A). There was no evidence of esophageal varices. The portal venous system, hepatic veins, and inferior vena cava were all patent. The liver demonstrated nodular contour and morphologic changes in keeping with cirrhosis. Based on the clinical and imaging findings of large GV with prior bleeding in the setting of a gastrorenal shunt, she was deemed a suitable candidate for BRTO treatment.

The BRTO procedural steps are illustrated in Figures 3A-F. Briefly, needle access to the right common femoral vein was achieved under ultrasound guidance. The vascular sheath and



Fig. 1 – Volume-rendered image from a preprocedure CT showing a large gastric varix with a gastrorenal shunt (arrow 1) draining into the left renal vein (LRV). The portal vein (arrow 2), LRV (arrow 3), splenic vein (arrow 4), and inferior vena cava (IVC) are also indicated.

C2-shaped catheter were advanced over the wire, and the LRV was selected. Venography was performed to identify the inferior phrenic vein (Figs 3A and B). A Berenstein catheter was used to select the gastrorenal shunt over a Glide wire (Figs 3C and D). Balloon-occluded venogram was performed through a 10-French sheath with an 11.5-mm occlusion balloon, outlining the entire extent of the GV with reflux into the splenic vein via the posterior gastric vein (Fig. 3E). A C-arm (cone-beam) CT was performed with the occlusion balloon inflated to outline the varices with trapped contrast. A total of 240 mg of 3% sodium tetradecyl sulphate foam was injected (air, sodium tetradecyl sulphate, and lipiodol ratio of 3:2:1; Fig. 3F). A C-arm CT was subsequently performed, which confirmed good filling of the GV with the sclerosing agent. With the occlusion balloon left in place, the patient was transferred to the step down unit and monitored during the sclerosant dwell time. Repeat fluoroscopy and C-arm CT were preformed 6 hours later. The patient had an uncomplicated recovery and was discharged home 4 days later. Follow-up CT performed 3 months later showed complete obliteration of her GV (Fig. 2B). Upper gastroscopy was performed 10 months post-BRTO which revealed the presence of small esophageal varices, but no GV were seen.

#### Discussion

GV are submucosal venous saccules in the wall of the stomach, which develop in about 20% of patients with portal hypertension [1]. They are classified according to Sarin et al [1] as either gastroesophageal varices (GOV) or isolated GV (IGV). GOV are further subdivided into two types: GOV1 (varices continuous with esophageal varices, extending down to the cardia, or lesser curve), and GOV2 (varices extending from the esophagus toward the fundus). IGV may be found in the fundus (IGV1) and are often tortuous and complex, or may be located elsewhere in the stomach (IGV2) such as the antrum, corpus, or around the pylorus [1,3]. GOV1 account for most GV (75%), however, according to a prospective study, the incidence of bleeding is significantly higher for IGV1 (78% for IGV1 vs 55% for GOV2, and 10% for GOV1 and IGV2) [1]. In comparison with esophageal varices, GV bleeding occurs less frequently but is associated with a poorer prognosis. GV bleeding results in greater hemorrhage and transfusion requirements, as well as increased risk of rebleeding and higher mortality rate [3]. Endoscopy is required to distinguish between an esophageal and gastric source of bleeding and is the first-line assessment modality for management of GV bleeding. However, a prospective study of patients with cirrhosis and GV hemorrhage and/or high-risk GV found that conventional endoscopic measures such as sclerotherapy may be associated with a higher rebleed rate as compared to BRTO [5].

The vast majority of GV are associated with a spontaneous left-sided portosystemic shunt, which can include gastrorenal, direct gastrocaval, and gastrocaval shunts via the inferior phrenic vein [2]. These shunts form to relieve portal hypertension or to bypass portal venous obstruction. Gastrorenal shunts are the most common, making up 80%-85% of left-sided portosystemic shunts [2]. They create an outflow



Fig. 2 – Computed tomography pre-BRTO and post-BRTO. (A) Axial CT images acquired at portal venous phase demonstrating large fundal GV (arrow). (B) Follow-up CT 3 months later showing resolution of varices after BRTO.

from the GV to the LRV and form a component of the portosystemic system. The gastric variceal system, which includes the varices and gastrorenal shunt, can vary in complexity, tortuosity, size, and blood flow. Hence, understanding the anatomy and hemodynamics of the gastric variceal system through preprocedural imaging studies using CT is critical for clinical management decisions [2].

BRTO involves occlusion of the portosystemic outflow veins with a balloon catheter, followed by injection of a sclerosing agent into the varix. The venous access site is the



Fig. 3 – BRTO procedural steps. (A) Left renal venogram via sheath with catheter tip at the renal hilum. Gonadal veins are incidentally filling inferiorly. (B) Inferior phrenic and/or adrenal vein confluence is catheterized. (C) A catheter is carefully advanced into the gastrorenal shunt for support. (D) Variceal outflow is delineated with digital subtraction contrast injection. (E) With occlusion balloon catheter inflated, the entirety of the gastrorenal shunt and GV are delineated back to the splenic vein origin. (F) Mixed density from the sclerosant foam injection throughout the shunt and/or variceal complex.

common femoral vein or internal jugular vein. The occlusion balloon is kept in place for hours to ensure that there is sufficient dwelling of the sclerosing material within the varix and to minimize complications due to reflux into systemic or portal vessels. The sclerosant results in thrombosis of the GV and draining portosystemic shunt, which marks the end point of the procedure. According to a retrospective study, balloon rupture may occur in about 15% of BRTO procedures, with no significant clinical or technical consequences [6]. However, technical failure may result if balloon rupture occurs early, before thrombosis and complete sclerosis is achieved [7].

There are 2 main clinical indications for BRTO: (1) impending, prior, or active gastric variceal bleeding and (2) GV with hepatic encephalopathy refractory to medical management [7]. Relative contraindications include: severe coagulopathy (often associated with liver failure), splenic vein thrombosis, portal vein thrombosis, and uncontrolled esophageal variceal bleeding unless BRTO is combined with transjugular intrahepatic portosystemic shunts (TIPS). Of these, the presence of chronic portal vein thrombosis may be the most serious contraindication, as the gastrorenal shunt could be the only splanchnic outflow tract. Thus, its obliteration with BRTO poses the greatest risk for adverse consequences due to splenic engorgement, thrombosis, and venous mesenteric ischemia [7]. It is therefore imperative to conduct preprocedural CT imaging to document the presence of a portosystemic shunt and assess the patency of the portal vein. Furthermore, due to variability in the portosystemic as well as afferent and efferent venous collaterals feeding the GV, knowledge of the patient's portosystemic anatomy is critical before performing the BRTO procedure.

Endovascular radiologic management of bleeding GV includes TIPS, which results in decompression of the portal circulation, and BRTO, which leads to obliteration of varices and their feeding shunts. Balloon-occluded antegrade transvenous obliteration is an alternate approach that occludes the inflow to the varices from the portal system and can be performed by direct transhepatic puncture of the portal veins or via access through a TIPS shunt. These strategies can be used alone or in combination depending on the patient's clinical characteristics and imaging findings. Guidelines for management of GV are less well established as compared with esophageal varices. According to a recent meta-analysis, TIPS and BRTO appear to be similar in efficacy for controlling bleeding GV [8]; however, TIPS has shown more consistent results in treatment of esophageal varices [2]. The inconsistency in TIPS outcomes for GV treatment is thought to be due to the large gastrorenal shunt and variability in blood flow. Furthermore, the effectiveness of TIPS in decompressing the GV may also be dependent on the pattern of gastric vein dominance [2]. TIPS is associated with a low but not insignificant complication rate, which includes aggravation or development of hepatic encephalopathy, bleeding, and even death.

One of the greatest advantages of BRTO over TIPS is that it improves hepatic blood flow and liver function, thereby improving hepatic encephalopathy. This has been demonstrated by several studies including a recent meta-analysis comparing BRTO to TIPS for treatment of GV [8]. However, BRTO also increases the risk of new-onset or worsening esophageal variceal bleeding by closing the portal outflow shunt and thus altering local hemodynamics and collateral flow [9]. Therefore, patients should be closely monitored with upper endoscopy post-BRTO for detection and management of esophageal varices [4]. Increased portal pressure may also increase the risk of ascites and pleural effusion in some cases [10]. Complications post-BRTO include fever, epigastric, chest and/or back pain, transient systemic hypertension, pleural effusion, and hemoglobinuria [10].

BRTO has excellent clinical and technical success rates (79%-100%) with a recent systematic review of 24 studies identifying BRTO as an efficacious and relatively safe procedure for treatment of GV [4]. Furthermore, in comparison to endoscopic-guided sclerotherapy or cyanoacrylate injection as primary treatment for GV, BRTO had a lower rebleed rate [5]. A meta-analysis comparing BRTO to TIPS in patients with GV and portal hypertension found them to be similar in terms of technical success rate, hemostasis rate, and incidence of postoperative complications, but BRTO was associated with a lower incidence of rebleeding and encephalopathy [8]. GV rebleed rates after a successful BRTO procedure range from 0% to 10% [9].

This case of isolated gastric variceal bleeding illustrates the portosystemic anatomy that allows for successful treatment with BRTO. In North America, expertise and utilization of BRTO for managing gastric variceal hemorrhage is still not widespread. Furthermore, large randomized controlled trials are lacking, and hence, more robust data are needed. None-theless, according to the best available evidence, if the technical expertise is available, BRTO should be considered for the management of significant GV [4,8].

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