

Balloon occluded retrograde transvenous obliteration for bleeding gastric varices: Eyes see what the mind knows

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Abstract

Approximately one in six patients with portal hypertension who develop varices at sites of portosystemic venous collaterals has gastric varices due to hepatofugal flow into the gastric veins. Bleeding from gastric varices, though less common, has a higher mortality and morbidity compared to bleeding esophageal varices, which are easier to manage endoscopically. The efferent channel for gastric varices is mostly the gastrosplenic shunt (GRS) which opens into the left renal vein. Balloon-occluded transvenous obliteration (BRTO) involves accessing the GRS with an aim to temporarily occlude its outflow using a balloon catheter and at the same time injecting sclerosant mixture within the varix so as to cause its thrombosis and thereby obliteration. BRTO is one of the mainstays of minimally invasive treatment for bleeding gastric varices. In the minority of cases where the GRS is absent, conventional BRTO is technically not possible. However, accessing the small alternate shunt from the inferior phrenic vein may be possible if one is aware of its existence.

Key words: Bleeding gastric varices; BRTO; gastrosplenic shunt; inferior phrenic vein

Introduction

Bleeding gastric varices (GVs) carry a higher morbidity and mortality than bleeding esophageal varices (EVs),^[1] which are easier to access and treat endoscopically using banding. Balloon occluded retrograde transvenous obliteration (BRTO) of GV is a minimally invasive treatment for such cases which relies on accessing the main efferent venous channel of these varices, i.e. the gastrosplenic shunt (GRS) via the left renal vein (LRV). In cases lacking a GRS, alternate efferent channels such as the left inferior phrenic vein (IPV) can be accessed and BRTO of varices is possible. To our knowledge, only one such previous case has been reported in which the

communication between the left IPV and left hepatic vein (LHV) was utilized to perform a BRTO.

Case History

A 70-year-old lady, a known case of hepatitis B virus (HBV) related cirrhosis presented to the emergency in Gastroenterology department with complaint of melena for 1 day. She had been referred to our hospital from another centre where endoscopy had been performed on her earlier in the day and had revealed gastric varices. Colonoscopy had also been performed which was normal. She reported 5 episodes of melena in the preceding 24 hours, with

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mild-to-moderate red discoloration of the stools. She also complained of generalized fatigue and weakness but her vital parameters were stable.

She was under medical management for chronic liver disease and cirrhosis for last many years. At presentation, her Child Pugh score was A6 with serum bilirubin of 1.4 mg%, serum albumin of 3.2 g%, international normalized ratio (INR) of 1.2 and absence of clinical ascites and encephalopathy. She had diabetes mellitus type 2 which was under medical control with oral hypoglycemic agents for many years.

Her serum hemoglobin was 8.3 g% with a hematocrit of 30.3% (normal range: 36–48%).

The liver enzymes (SGOT, SGPT, and alkaline phosphatase) and kidney function tests (blood urea nitrogen and serum creatinine) were within normal limits.

Gastric lavage performed with normal saline in the emergency room revealed presence of active bleeding in the aspirate. A preliminary diagnosis of liver cirrhosis with bleeding varices was made and esophago-gastric-duodenal endoscopy was performed for her 2 hours after admission. The same showed bleeding mid and distal esophageal varices (grade II) along with bleeding varices in the gastric cardia [Figure 1]. Banding was performed for the esophageal varices.

The patient underwent a triphasic computed tomography (CT) scan of the abdomen at the outside hospital on a 32 row scanner (Somatom Perspective, Siemens Healthcare, Erlangen, Germany). It showed cirrhotic liver morphology without any focal hepatic lesion. Minimal ascites was present. Varices were present along the lower esophagus and proximal lower curvature of stomach (type 1, Sarin Classification).^[2]

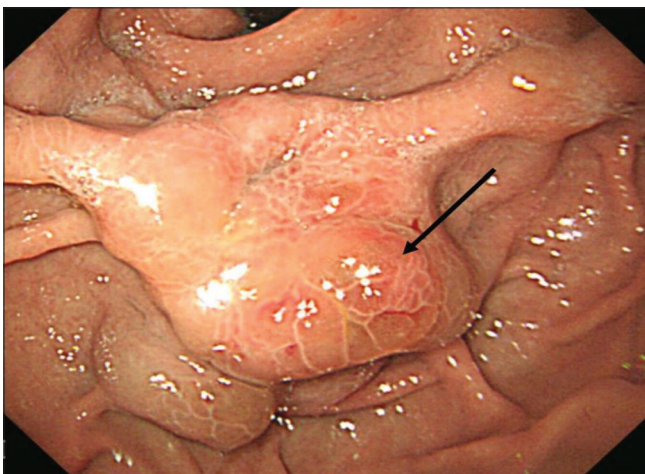


Figure 1: Upper GI endoscopy showing large varices in gastric cardia with raw red surface denoting recent bleeding (arrow)

Because there was no large connection between the left renal vein and GV, the patient was referred to the department of Radiology for transhepatic or transsplenic GV embolization. However, careful review of the images revealed the efferent channel of gastric varices to be the left IPV with one tributary leading into the left hepatic vein [Figure 2]. Because this channel was of moderate size (3–4 mm) and the patient was at high risk of re-bleeding from the GVs, it was decided to attempt a BRTO through this tributary.

The procedure was performed under local anesthesia. Under ultrasound guidance, the right femoral vein was accessed using a micropuncture introducer set (Flexor, Ansel 1 modification, Cook Medical, Bloomington, USA) and a 7 Fr, 70 cm sheath (Cook Medical, Bloomington, USA) was placed with its tip in the IVC over a 0.035" diameter, 260 cm length hydrophilic glide wire (Radifocus, Terumo Medical corporation, Tokyo, Japan).

A 90 cm, 5Fr Davis catheter (ANA MD, Sungnam, Kyunggi Province, Korea) was advanced over the wire. The catheter-wire combination was used to cannulate the LHV and was further advanced into the expected location of the IPV, as determined by CT. Once the location of the catheter within the IPV was confirmed by a venogram, the sheath negotiation into the IPV was attempted, however, it was not possible. Hence, the sheath was changed to a more flexible 7Fr, 65 cm sheath (Super Arrow-Flex, Arrow International Inc, Reading, USA) and advanced further into the IPV [Figure 3A].

The Davis catheter was exchanged for a 80 cm length, 6 Fr diameter occlusion balloon catheter (Terumo Medical Corporation, Tokyo, Japan) and the balloon was inflated using 2 ml of contrast. The venogram of the shunt was done, which revealed run off into the left phrenic vein, pericardiophrenic vein, and lower left intercostal veins with poor filling of the gastric varices [Figure 3B]. The phrenic vein branch which was not connected with GV was cannulated using a 110 cm, 2 Fr diameter microcatheter (Progreat AlphaTerumo, Tokyo, Japan) and 135 cm, 0.014" guidewire (Transcend, Boston Scientific Corporation, Marlborough, USA) advanced co-axially through the occlusion balloon catheter. It was embolized using three 3 mm × 4 cm Nester pushable micro coils (Cook Medical, Bloomington, USA). Following this, gelfoam slurry made using small gelfoam pieces (Cutanaplast, Mascia Brunelli Spa, Milan, Italy) and contrast (320 mg I/ml, Visipaque, GE Healthcare, Ireland) was injected to cause stasis within the intercostal vein and other small vein branches. The venogram was repeated which showed obliteration of the systemic run off and good filling of the gastric varices, as correlated with CT images [Figure 4A]. The schematic diagram of the procedure is shown in Figure 4B.

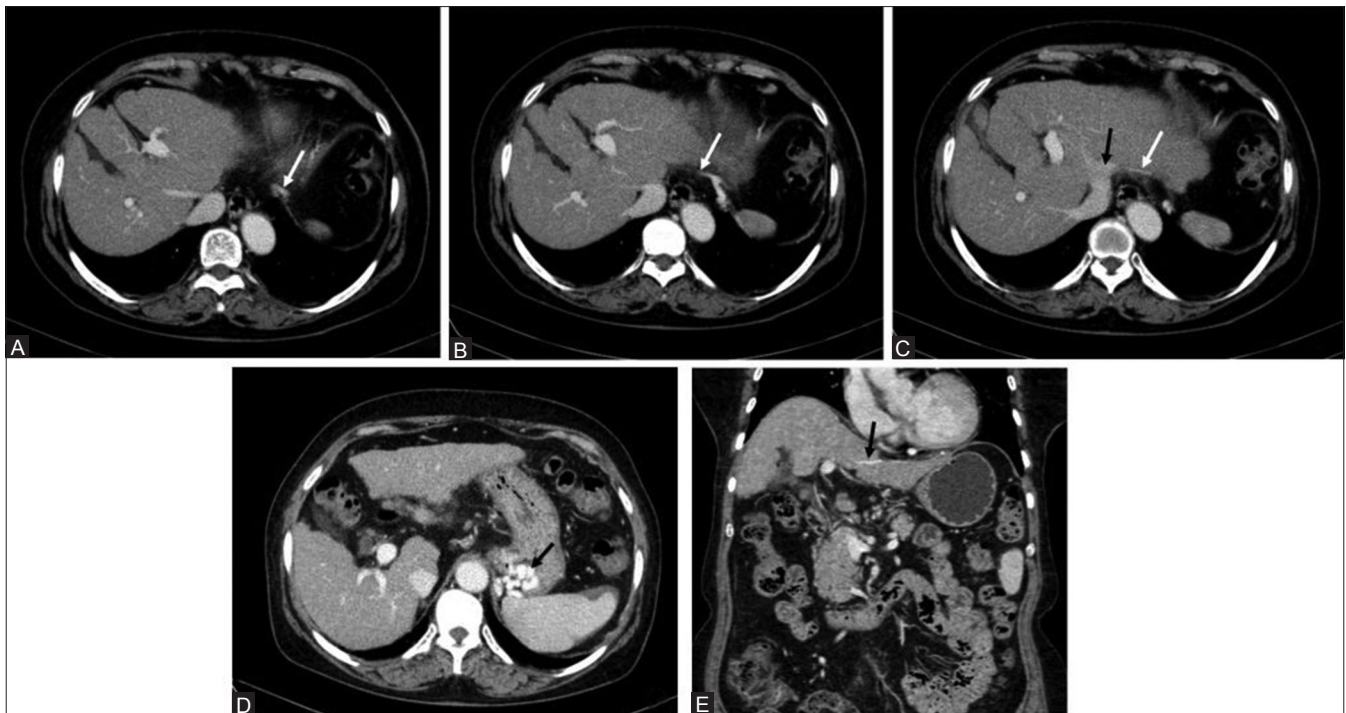


Figure 2 (A-E): Serial craniocaudal portal venous phase axial images (A-C) show the left IPV (black arrows) joining the LHV (white arrow in C). Axial image at a more caudal level (D) show the GV's in the region of cardia (arrow). Coronal reformatted image (E) shows the coursing left IPV (arrow). The prominent fissures in segment 8 (seen in A and B) indicate cirrhotic morphology of the liver

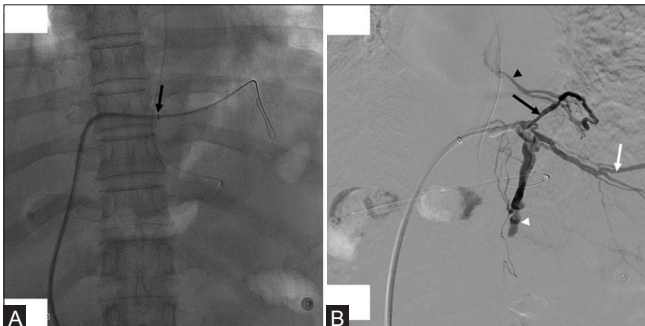


Figure 3 (A and B): Fluoroscopic image (A) shows the tip of sheath within the left IPV (arrow) with the 5Fr catheter and guidewire advanced further into the vein. (B) Venogram after inflation of the balloon within IPV shows contrast run off into the distal IPV (black arrow), pericardiophrenic vein (black arrowhead) and left lower intercostal veins (white arrow). Some filling of the GV's is seen (white arrowhead)

Thirty ml sclerosant mixture comprising 20 ml of 3% sodium tetradecyl sulphate (Tromboject, Omega, Montreal, Canada), 10 ml of contrast (320 mg I/ml, Visipaque, GE Healthcare, Ireland) and small gelfoam pieces were injected under fluoroscopic visualization till complete filling of the varices was achieved. The balloon was kept inflated and the patient was kept on the angiography table for an hour. Fluoroscopy was used every 15 minutes to confirm that the balloon remained inflated and in place with stasis within the varices. The patient was then shifted to the ward with instructions to keep the right leg straight for 12 hours. A triphasic CT of the abdomen was done after twelve hours on a 320 row scanner (Aquilion One, Toshiba Medical Systems,

Tokyo, Japan), which showed non-enhancement within the region of gastric varices consistent with thrombosis [Figure 5]. Mild ascites was seen which had increased since the previous CT. The balloon was deflated, the sheath removed, and hemostasis ensured by manual compression. At the time of submitting this article the patient has been on follow-up for 4 weeks and there has been no recurrence of bleeding. Her ascites has decreased on serial ultrasounds and the LFTs have shown no deterioration from pre-procedure levels.

Discussion

GVs can be caused by portal hypertension resulting from liver cirrhosis (90% of cases) or due to splenic vein thrombosis (10% of cases).^[3]

Patients with liver cirrhosis having portal hypertension have a 30% risk of portosystemic collaterals or varices, with GV's comprising only 10–20% of these. In other words, 3–6% of all cirrhotics have GV's.^[1]

Though risk of bleeding is lower with GV's compared to EV's, the morbidity and mortality with former is higher.^[1]

Upper GI endoscopy is usually the first line diagnostic and therapeutic tool for bleeding varices (using injection sclerotherapy or banding for EV's and injection sclerotherapy for GV's). However, it is not very effective for the latter.^[1,4] In addition, there is a risk of complications resulting from embolization of sclerosant (n-butyl-2-cyanoacrylate

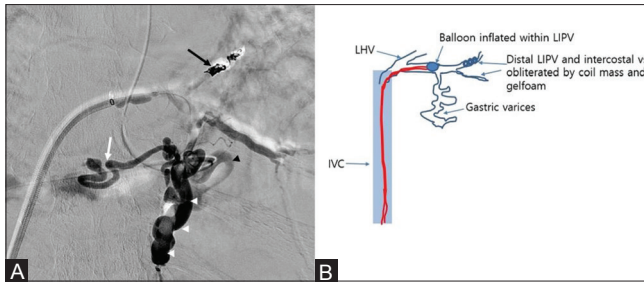


Figure 4 (A and B): Repeat venogram (A) after coiling of IPV (black arrow) and gelfoam embolization showed absence of flow within IPV, stasis within intercostal veins and good visualization of GV's (white arrowheads). The tortuous medially coursing channel (white arrow) is the left coronary vein and the faint vein (black arrowhead) is the posterior gastric vein (afferent to the varices). Sclerosant injection was done from this position. (B) Schematic diagram showing the anatomy relevant to the procedure

or isobutyl-2-cyanoacrylate) to other organs.^[5,6] When endoscopy fails to control variceal bleeding, a transjugular intrahepatic portosystemic shunt (TIPS) is commonly performed to decompress the portal venous system. However, it is not very effective in controlling bleeding GV's as it is for bleeding EV's.^[7,8] It can also lead to hepatic encephalopathy in 20–30% of patients.^[9,10]

The concept of BRTO involves accessing the GRS via the LRV through the femoral or jugular route and injecting a sclerosant agent such as ethanolamine oleate, absolute alcohol, gelfoam, or sodium tetradecyl sulphate into the varices after inflating a balloon in the GRS to obstruct the shunt outflow, thereby obliterating the varices^[11–14] (see the schematic diagram).

Olson *et al.* described the first attempt of balloon occluded sclerotherapy of GRS for GV's.^[14] Kanagawa *et al.* were the second authors of this technique.^[11] Over the years, its technique has evolved and it has become a well-established minimally invasive treatment for gastric varices both to control emergent bleeding or electively, especially in Japan and South Korea. The balloon is kept inflated for a period of 8–12 hours, during which time the varix undergoes thrombosis.

At present, the two main clinical indications for this procedure are bleeding gastric varices in both emergent and elective situation and in some cases refractory hepatic encephalopathy.^[15] In the latter indication, the closure of GRS by sclerosis leads to reduction of portosystemic bypass.

Discussion of detailed technique, patient selection and complications of this procedure is beyond the scope of this article and the reader is suggested to read the same from Saad, *et al.* and Watanabe, *et al.*^[16,17] The afferent channel for gastric varix is mostly from left gastric or posterior gastric veins.^[18–20]

The efferent channel for most GV's (80–85%) is the GRS, which opens into the LRV.^[19,21] However, gastric varices

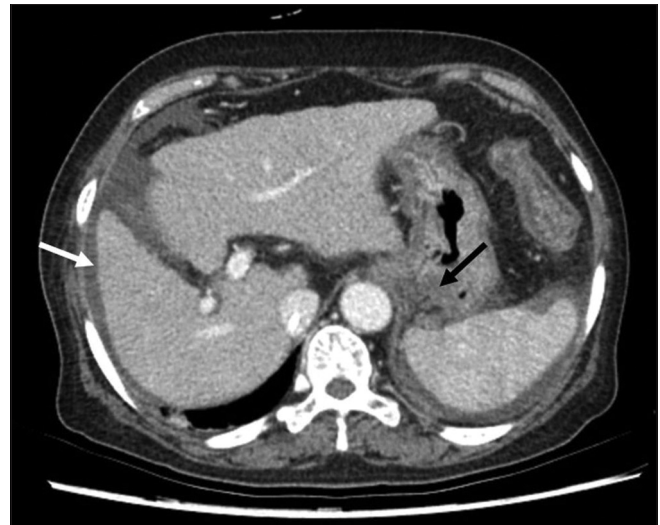


Figure 5: Twelve-hour axial portal phase CT image shows complete thrombosis within the varices (arrow). Mild ascites is also seen (arrow) which was not present earlier

lacking a GR shunt have also been observed in 10–15% of cases.^[12,19,21,22]

The major outflow in such cases includes the IPV, cardiophrenic vein, pericardial vein, retroperitoneal veins, and intercostal veins.^[22,23]

Because these outflow veins open into the IVC or other systemic veins with an anatomy that is unfavorable for entry into them through femoral or jugular routes, the options for treatment in these cases include TIPS followed by obliteration of varices through the TIPS tract or antegrade obliteration of varices through the transhepatic or transsplenic puncture of hepatic vein/splenic vein respectively (also known as BATO: Balloon occluded antegrade transvenous obliteration). Kameda *et al.* described BRTO in 6 of their patients who lacked a GRS but instead had a gastrocaval shunt through left IPV joining the IVC.^[22]

In our case, there was no GR shunt and initially a BRTO was not considered. However, careful review of axial and coronal reformatted CT images showed the relative large communication between the LHV and the left IPV and a BRTO could be done successfully. Extensive literature search revealed only one such previous case where Ibukuro *et al.* in their article have described a similar approach for BRTO in which the authors have approached the varices through the hepatic vein.^[24]

However, in their case the drainage of IPV into LHV could not be seen on CT but only on selective angiography of the splenic artery.

Other cases of BRTO that we came across during literature search for this article is by accessing the gastric varices

through the intercostal vein and pericardiophrenic vein.^[25-28]

Hemodynamically, our case is similar to the cases where the left IPV joins the IVC directly as described by Kameda *et al.*^[22]

Increase in the amount of ascites indicates increase in portal venous pressure as a result of variceal obliteration, which is a known side effect of BRTO in 20–30% of cases.^[17]

Our case highlights the importance of careful evaluation of CT images for alternate efferent pathways of a gastric varix lacking a GRS. Hence, the cross-sectional studies must be carefully analyzed for an alternate access to the varices in a case being evaluated for BRTO and lacking in a GRS. Also more importantly it underlines the fact that the varix can be treated using the cannulation of this small communication from the systemic vein.

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Conflicts of interest

There are no conflicts of interest.

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