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LETTER TO THE EDITOR

Splenic Venous Congestion after Balloon-Occluded Retrograde Transvenous Obliteration of Gastric Varices

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Introduction

Although esophageal veins are the main source of variceal bleeding due to portal hypertension, gastric varices are causative in 10–36% [1]. In the latter, variceal rupture is the most feared complication, with prevalence around 30% [2–4] and a mortality of 45–55% [2–6]. Transjugular intrahepatic portosystemic shunt (TIPS) for this indication has less favourable results than for esophageal variceal hemorrhage: success rates for controlling gastric varices range from 50–63% [6, 7] versus 81% in esophageal varices [8]. Variceal coiling requires either a percutaneous transhepatic approach (with its inherent risks), or TIPS creation to allow access. Balloon-occluded retrograde transvenous obliteration (BRTO) has been used to treat and prevent gastric variceal rupture, with bleeding control rates of 87–100%, and recurrence rates of 0–10% [6, 9, 10]. Balloon-occluded retrograde transvenous obliteration may potentially improve hepatopetal portal blood flow and hepatic function [6] and, compared to TIPS, provide lower cumulative rebleeding rates from gastric varices (2% at 1 year vs. 20% with TIPS) and higher cumulative survival rates (76% at 5 years vs. 40%) [11].

Splenic venous congestion may occur in association with sepsis, congestive heart failure, portal hypertension or splenic vein obstruction. However, to our knowledge, imaging findings of splenic venous congestion have not yet been reported after BRTO. We report a case of BRTO of extremely large gastric varices in the presence of partial splenoportal thrombosis, in which post procedural computed tomography (CT) appearance and clinical evolution suggested increased regional venous pressure and parenchymal splenic congestion.

Case Report

A 58-year-old man with known hepatic cirrhosis and remote alcoholism presented with increasing abdominal pain. Model for End-Stage Liver Disease score was 12. Contrast-enhanced CT (CECT) showed partially obstructing thrombus in the splenic and main portal veins, a large gastro renal shunt and gastric varices (Fig. 1). Anticoagulation for the thrombosis was contraindicated as a result of thrombocytopenia (range 20–50,000 mm^{-3}) and surgery was discounted, given the hemorrhagic risk. Gastric variceal BRTO was attempted with goals of variceal occlusion and improvement of antegrade portal blood flow, which might thereby reduce stasis and promote spontaneous, endogenous fibrinolysis of the splenoportal thrombus [6].

Balloon-occluded retrograde transvenous obliteration was performed via a transjugular venous approach (Fig. 2).

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Fig. 1 CT scan before BRTO shows the large gastric varices connected to the left renal vein through the gastrorenal shunt and the presence of a thrombus partially obstructing the splenic vein and main portal vein. Grade II esophageal varices were present as well (not shown)

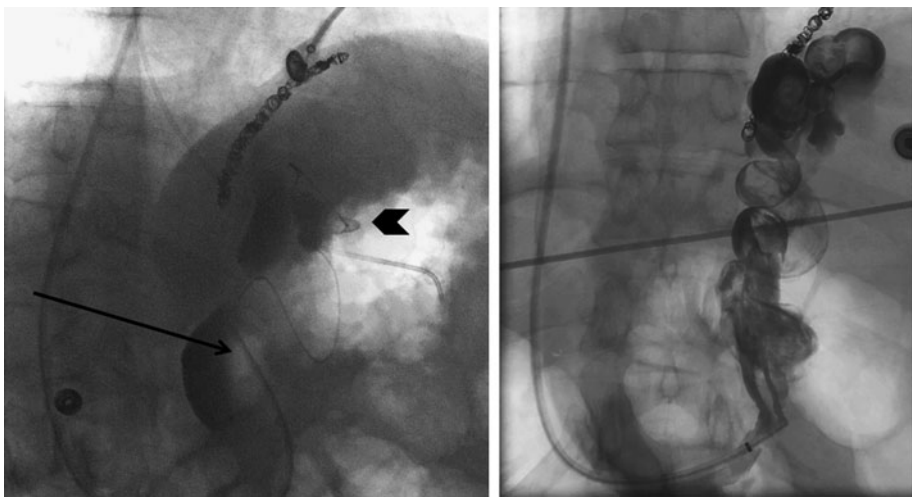

Fig. 2 BRTO procedure from a right internal jugular vein approach. (*Left*) The left inferior phrenic vein has been embolized with micro coils. The occlusion balloon (*arrow*) is in the caudal segment of the gastrorenal shunt. The coaxial microcatheter describes three loops before reaching the target gastric varices with its tip (*arrowhead*), as confirmed by dyna-CT (not shown). (*Right*) Follow-up venogram obtained 12 h after BRTO shows contrast stasis and large filling defects (thrombi) in the gastric varices and gastrorenal shunt, but no thrombus in the left renal vein

A wedge venogram obtained after placement of an occlusion balloon catheter in the gastrorenal shunt, showed satisfactory shunt occlusion and collateral opacification of a left inferior phrenic vein, supplying a pericardiophrenic vein (grade 3 varices in Hirota's classification) [6]. The left inferior phrenic vein was coil embolized. The microcatheter was redirected into the main gastric variceal outflow vein and, after retrograde catheterization of three large, tortuous varicose loops using dyna-CT imaging guidance (Siemens Axiom Artis, Siemens Medical Systems, Erlangen, Germany) [10], satisfactory microcatheter positioning in the target gastric varices was confirmed. Ten ml of liquid sclerosant (8 ml of sodium tetradecyl sulfate 3% mixed with 2 ml of iodinated contrast) was slowly injected through the microcatheter (Fig. 2). An additional 10 ml was provided 30 min later. The occlusion balloon remained

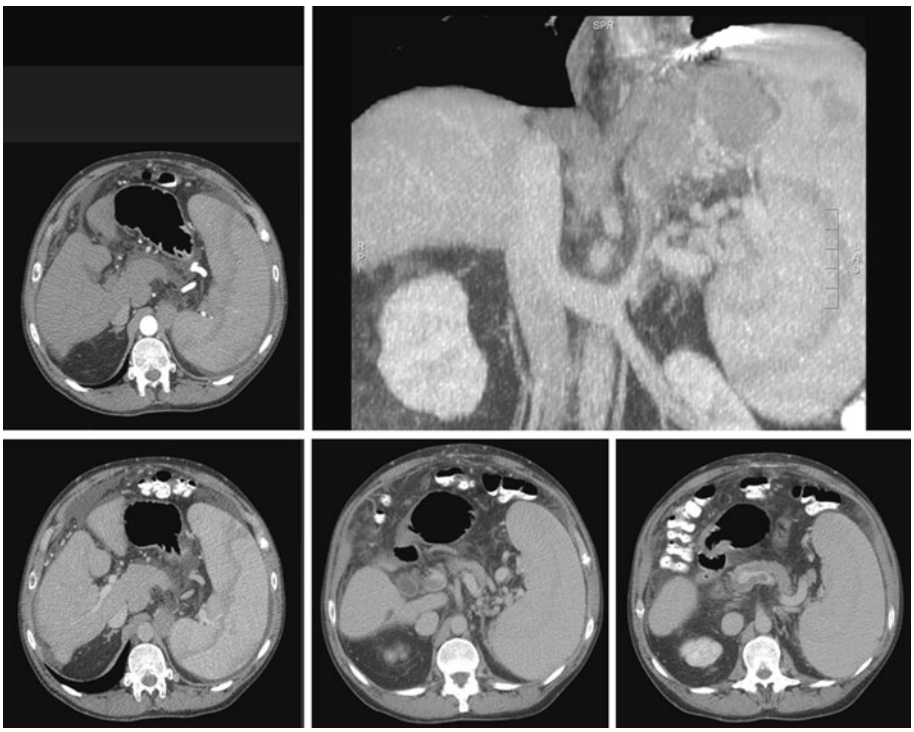

Fig. 3 CT scan acquired 9 days after BRTO shows occlusion of the sclerosed gastric varices, stable appearance of the preexisting splenoportal thrombus, mild ascites, and heterogeneous enhancement of the splenic parenchyma suggestive of splenic venous congestion

inflated for the entire procedure and until the posttreatment venogram the next morning. That study showed flow stasis and endoluminal defects filling most of the gastric varices and main draining vein, and no left renal vein or IVC thrombus extension. After discharge the next day, the patient had focal right upper quadrant pain, which gradually resolved while receiving oral analgesics. Contrast-enhanced CT obtained 1 week after BRTO (Fig. 3) showed complete thrombosis of the gastric varices and draining vein, no left renal vein thrombus, and mildly increased ascites. Ribbon-like bands of decreased splenic parenchymal enhancement were seen centered on the hilum. These were more prominent in the portal venous phase, with partial but incomplete improvement in the delayed phase. The appearance was thought to be consistent with splenic venous congestion [12] rather than arterial splenic infarcts.

Two months later, the patient was readmitted with hematemesis from esophageal varices, which were successfully treated by endoscopic banding. CECT (Fig. 4) showed interval shrinkage of the splenoportal thrombus, homogeneous splenic parenchymal enhancement, and persisting gastric variceal occlusion. Thereafter, the patient continued to develop recurrent ascites, treated by diuretics and paracentesis. He eventually underwent TIPS creation 16 months after BRTO because of refractory ascites. No splenoportal thrombus was found angiographically or on subsequent gadolinium-enhanced magnetic resonance imaging. Post-TIPS hepatic encephalopathy was medically controlled. There have been no further issues with ascites or gastrointestinal hemorrhage.

Fig. 4 CT scan 2 months after BRTO, obtained during readmission for esophageal variceal bleeding treated by endoscopic banding, shows interval worsening of ascites but return to more homogeneous parenchymal enhancement of the spleen. There has been interval shrinkage of the splenoportal thrombus and persisting occlusion of the gastric varices. Several small veins consistent with portal hypertension collaterals were seen outside of the splenic venous territory, notably in the caput medusa region

Discussion

This case was remarkable for the extremely large gastric varices, the existence—and eventually endogenous

fibrinolysis—of a partially obstructive splenoportal thrombus, and the development of heterogeneous splenic enhancement suggesting venous congestion after BRTO. Balloon occlusion of gastrorenal shunts has been shown to convert portal blood flow from hepatofugal to hepatopetal [13] and improve liver function tests [6]. Successful gastric variceal occlusion in this case may have improved hepatopetal flow around the splenoportal thrombus, thereby promoting spontaneous lysis. Patients with contraindications to TIPS (e.g., high hemorrhagic risk) might also benefit from BRTO [14–16]. Alternative management in this case could have been percutaneous transhepatic portal venous access followed by mechanical thrombectomy of the splenoportal thrombus. However, in situ thrombolysis would have had a higher hemorrhagic risk and the gastric varices and gastrorenal shunt would have persisted even after successful thrombolysis. We chose an option that would occlude the gastric varices and possibly promote both antegrade portal venous blood flow and endogenous fibrinolysis of the thrombus. Ascites and worsening of esophageal varices are common after BRTO [14, 16–18], as it increases preexisting portosystemic pressure gradients [19]. The latter occurs in 39% of cases at 3 years after BRTO and in 52% at 5 years, with variceal bleeding observed in 10–32% of patients [14, 20]. In contrast, splenic venous congestion has not been reported after BRTO. In our case, it may have been facilitated by the large varices, splenomegaly and the preexisting splenoportal thrombus. Although this thrombus might have caused some degree of sinistral portal hypertension, it is unlikely to be the only aetiology for the gastric varices: the underlying cirrhosis, ascites, and worsening esophageal varices after BRTO suggest that global portal hypertension was the dominant factor.

No single endovascular management technique is ideal for managing complex cases of portal hypertension sequelae. In patients who are not candidates for liver transplantation, combinations of these interventions should be considered. Balloon-occluded retrograde transvenous obliteration can be performed to eliminate the risk of gastric variceal bleeding; partial splenic embolization can reduce pancytopenia, splenomegaly, gastric variceal inflow, and portal hypertension; and TIPS provides safe shunt decompression and allows antegrade embolization of the veins feeding the varices [21]. In conclusion, splenic venous congestion is a rare but possible complication after BRTO, especially in the context of major splenomegaly. Partial splenic or portal thrombosis should not be considered to be an absolute contraindication for BRTO, as endogenous fibrinolysis may eventually occur after the procedure.

Conflict of Interest The authors declare that they have no conflict of interest.

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