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SPLENIC ARTERY EMBOLIZATION, A THERAPY THAT CAN BE LIFE-SAVING FOR SMALL BOWEL VARICEAL BLEEDING

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ABSTRACT

Introduction: Small bowel varices are a rare manifestation of portal hypertension, accounting for only 1–5% of variceal gastrointestinal bleeding. Their diagnosis and management remain challenging because of their uncommon occurrence, difficult anatomical accessibility, and lack of standardized treatment strategies. We report a case of refractory small bowel variceal bleeding successfully treated with splenic artery embolization after failure of conventional therapeutic options.

Case presentation: A 45-year-old man with liver cirrhosis, cholangiocarcinoma with bone metastases, ulcerative colitis, and previous partial hepatectomy presented with haemorrhagic shock secondary to severe lower gastrointestinal bleeding. Endoscopic evaluation suggested a small bowel source, while computed tomography angiography demonstrated portal vein stenosis, superior mesenteric vein thrombosis, portal hypertension, and actively bleeding small bowel varices. Portal vein stenting failed to control the haemorrhage, and transjugular intrahepatic portosystemic shunt (TIPS) placement was unsuccessful because of extensive hepatic vein thrombosis related to tumour burden. Salvage splenic artery embolization was subsequently performed using two vascular plugs, resulting in immediate haemostasis without recurrent bleeding. Follow-up imaging confirmed the absence of active bleeding. The only post-procedural complication was partial splenic infarction, which was managed conservatively.

Discussion: Splenic artery embolization reduces portal venous inflow by decreasing splenic blood flow, thereby lowering portal pressure and decompressing ectopic varices. Although primarily described for esophagogastric varices and other complications of portal hypertension, its use in small bowel variceal bleeding remains rarely reported. This case highlights splenic artery embolization as an effective rescue therapy when TIPS is technically impossible or unsuccessful.

Conclusion: Splenic artery embolization may represent a safe, minimally invasive, and life-saving therapeutic option for refractory small bowel variceal bleeding in carefully selected patients who are not candidates for conventional interventions. Multidisciplinary collaboration is essential to optimize outcomes, and further studies are needed to better define the role of this technique in the management of ectopic variceal bleeding.

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KEYWORDS

Splenic artery embolization, gastrointestinal bleeding, portal hypertension, small bowel varices

LEARNING POINTS

- Small bowel ectopic varices account for only 1–5% of cases of gastrointestinal bleeding.
- Due to its rarity in clinical practice, therapy is challenging.

INTRODUCTION

Bleeding from the gastrointestinal (GI) tract is a common problem encountered by gastroenterologists. Such bleeding is classified into three categories: upper GI bleeding, lower GI bleeding and small bowel bleeding (distal to the ligament of Treitz)[1,2]. Although small bowel bleeding is the least common, it is the most challenging to diagnose and manage due to the inaccessibility of the small intestine to endoscopy[3]. Among patients with cirrhosis, a proportion with small bowel varices experience life-threatening bleeding. Despite advancements in diagnostic and therapeutic modalities, a standardised treatment for small bowel varices has not been established due to its rarity (1–5% of cases). Management options vary depending on expertise and availability, and may include pharmacological, radiological, endoscopic or surgical approaches.

We report a rare case of small bowel variceal bleeding successfully managed with trans-arterial splenic embolization, underscoring its potential as a life-saving intervention.

CASE DESCRIPTION

A 45-year-old male smoker and alcoholic, known to have liver cirrhosis, cholangiocarcinoma with bone metastases, ulcerative colitis and a surgical history of prior partial hepatectomy, presented at the emergency department with hypotension, anaemia and active lower GI bleeding. Laboratory results included a white blood cell count of 9,000/mm^3, haemoglobin 5.3 g/dl, mean corpuscular volume 88 fl, platelet count 106,000/mm^3, INR 1.1 and partial thromboplastin time 32 seconds.

A computed tomography scan of the abdomen and pelvis revealed neoplastic infiltration of hepatic segments and spleen, bone metastases, ascites and portal hypertension. A gastroscopy was performed and revealed grade 3 oesophageal varices and a pyloric compressive mass without active bleeding. A colonoscopy showed evidence of active bleeding originating from the small bowel, with multiple clots in the cecum. Based on previous findings, abdominal computed tomography angiography was undertaken and demonstrated complete thrombosis of the superior mesenteric vein, stenosis of the portal vein causing severe hypertension in the mesenteric network and large collateral pathways. Small bowel varices with active bleeding were also noted *(Fig. 1A)*. A portal vein stent was inserted to alleviate the stenosis *(Fig. 1B and 1C)*; however, small bowel variceal bleeding persisted. A transjugular intrahepatic portosystemic shunt (TIPS) placement was attempted but failed due to thrombosis of the middle and anterior branches of the right hepatic vein, likely secondary to tumour burden. Finally, splenic artery embolization was performed *(Fig. 1D)*. The right common femoral artery was entered with a 5F introducer positioned in the external iliac artery. An immediate selective catheterisation of the splenic artery was performed using a 5 French catheter, with an angiogram showing a normal vasculature. The 5 French catheter and sheath were then exchanged for a 7 French guiding catheter positioned in the middle third of the splenic artery, which was successfully embolised using two vascular plugs of 12 mm and 14 mm in diameter. The final angiogram performed shows a satisfactory partial amputation of the splenic artery with no complication observed except for splenic infarct (43% of the splenic parenchyma was embolised, *Figure 1E*). The patient tolerated the procedure well and was transferred to the recovery room in a stable condition; it was a salvage therapy which led to a successful arrest of bleeding. The patient did not experience a new bleeding later on and did not have any new medical complaint except for splenic infarcts post embolization (follow-up laboratory results were WBC 11,000, Hb 7.5, platelets, 87,000). A follow-up computed tomography angiograph confirmed the absence of active bleeding.

DISCUSSION

Splenic artery embolization works like partially closing one of the main ‘taps’ feeding the portal system. By lowering the splenic contribution to portal flow, it decreases portal hypertension, which in turn reduces pressure in mesenteric varices and lowers bleeding risk. The portal vein receives blood from two main sources: the superior mesenteric vein and the splenic vein. These join to form the portal vein, which carries blood into the liver. In cirrhosis or other causes of increased intrahepatic resistance, blood cannot pass through the liver easily, causing portal hypertension. The elevated pressure pushes blood to find alternative pathways – portosystemic collaterals – such as gastroesophageal varices, rectal varices and ectopic varices (including mesenteric varices in the small intestine). The splenic vein contributes a large proportion of total portal blood flow (it can be 20–40%). In portal hypertension,



Figure 1. *This composite image illustrates the interventional radiology findings and procedures performed in this case. A) Congested small bowel varices: Angiographic evidence of variceal congestion due to portal hypertension; B) Portal venous stenosis: Radiological demonstration of significant narrowing in the portal vein; C) Portal vein stent placement: Stent insertion to address the stenosis and reduce portal venous pressure; D) Splenic artery embolization: Fluoroscopic image showing the catheterisation and embolization of the splenic artery; E) Splenic infarct post-embolization: CT scan depicting localised infarction in the spleen following successful embolization.*

the spleen often enlarges (congestive splenomegaly). This extra flow worsens portal hypertension and the pressure in varices. Splenic artery embolization (SAE) involves blocking the splenic artery (partially or totally) using coils, particles or plugs. Post embolization, the reduced arterial splenic input led to decreased splenic venous outflow and as a consequence, lower portal venous inflow. The reduced portal pressure will lead to less backpressure in mesenteric varices.

SAE has been utilised in various clinical scenarios, including recurrent gastrointestinal haemorrhage from oesophageal varices[4], portal venous pressure reduction during liver transplantation or major hepatectomy to enhance liver regeneration[5] and the management of iatrogenic spleen injury during esophagectomy operations[6]. Additionally, SAE is effective for haemorrhage control after blunt splenic injury[7]. A systematic review and meta-analysis of eighteen studies support the efficacy and safety of SAE in managing variceal bleeding due to portal hypertension, demonstrating significant reductions in bleeding, improvements in variceal grade, and increases in platelet counts[8]

In a study of nine patients with refractory portal hypertension-induced upper gastrointestinal bleeding who were ineligible for TIPS, partial splenic embolization (PSE) successfully prevented re-bleeding and eliminated the need for blood transfusions[9]. Another study involving 31 cirrhotic patients highlighted the effectiveness of PSE in managing portal hypertensive gastropathy, particularly in Child–Pugh class A patients[10]. Additionally, in a cohort of 25 patients with persistent oesophageal and gastric variceal haemorrhage, PSE proved to be a valuable rescue option in both emergency and non-emergency settings[11].

The utility of SAE has also been described in isolated left-sided portal hypertension secondary to splenic vein thrombosis. Kim et al. reported successful control of gastric variceal bleeding using SAE in a patient with splenic vein thrombosis complicating necrotising pancreatitis, highlighting the role of this technique as an effective minimally invasive alternative to surgery in selected cases. Their report further supports the concept that reducing splenic inflow can effectively decompress variceal circulation and achieve haemostasis, even in anatomically complex portal hypertensive states. Similar to our case, SAE served as a rescue therapy when conventional therapeutic options were either ineffective or unsuitable[12].

Although generally safe, SAE is sometimes associated with some complications such as splenic infarction, splenic abscess, sepsis, post-embolization syndrome and, in rare cases, coil migration or portal vein thrombosis[13].

CONCLUSION

Gastrointestinal bleeding in patients with portal hypertension is a life-threatening complication. TIPS remains the first-line treatment for uncontrolled bleeding when endoscopic and pharmacological therapies fail. However, in cases where TIPS is not feasible, SAE provides a minimally invasive and effective alternative. The interdisciplinary collaboration among hepatologists, endoscopists and interventional radiologists is essential to evaluate and execute the most suitable therapeutic option. Splenic embolization should be considered as a rescue treatment for variceal haemorrhage in patients for whom standard therapies fail.

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