Massive Gastric Bleed 20 Years After Splenopneumopexy

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ABSTRACT
Splenopneumopexy is an anastomosis performed for patients with portal hypertension secondary to veno-occlusive disease of the portal, splenic, or mesenteric veins. We present a case of an adult gentleman who presented with melena and was found to have pseudoaneurysm almost 20 years after his procedure. We also describe the clinical, laboratory, endoscopic, and radiological workup conducted to diagnose and manage gastric bleeding in this rare complication.

INTRODUCTION
Splenopneumopexy is a surgical procedure to create a parenchymatous venous anastomosis between the portal and pulmonary circulation systems, usually between the superior pole of the spleen and the left lung lower lobe vasculature. It was used mainly to treat patients with evidence of porto-splenic-mesenteric venous occlusion or patients with portal hypertension who are not candidates for

Figure 1. Pseudoaneurysm of splenopneumopexy as appears in this fundus lesion.

Figure 2. Active brisk bleeding from the fundus lesion after clip deployment.
traditional portosystemic shunts. The creation of this porto-pulmonary shunt was to prevent mortality from variceal bleeding and noncirrhotic portal hypertension such as Budd-Chiari syndrome. This intervention is believed to be a safe and effective approach to prevent gastrointestinal bleeding in selected patient groups. Our case report describes a massive gastric bleed from pseudoaneurysm 20 years after a splenopneumopexy.

**CASE DESCRIPTION**

A 74-year-old White man presented with 1 week of melena and general weakness. His medical history was significant for atrial fibrillation, antiphospholipid syndrome on coumadin, splenic vein thrombosis, and esophageal varices. He underwent splenopneumopexy approximately 20 years earlier.

Blood pressure on admission was 87/49 mm Hg measured on both arms; the heart rate was 96/min; the respiratory rate was 18/min; and he was afebrile. His physical examination revealed mild epigastric abdominal tenderness, otherwise the examination was unremarkable. Laboratory testing showed hemoglobin 9.4 g/dL (13.6–16.7 g/dL), hematocrit 29.3% (40%–49%), platelet count 85,000/mm³ (130,000–350,000/mm³), and an INR of 3.5 (0.83–1.14).

He was seen by the gastroenterology consulting service the next morning, and the decision was to further assess him with esophagogastroduodenoscopy to identify any potential source of bleeding. Overnight the INR was reversed to 1.6 with 2 units of fresh frozen plasma. His upper endoscopy revealed no active bleeding or old blood in the stomach. However, a distinct lesion was found in the fundus, which seemed high risk for rebleeding; it appeared similar to a visible vessel; however, it was not associated with an ulcer base (Figure 1). Given his significant medical history, a gastric varix was also suspected, although it did not have the typical appearance of an isolated varix. A decision was made to inject epinephrine and clip the lesion, and on deployment of the clip, the lesion began to bleed briskly (Figure 2).

He was emergently intubated and taken to the interventional radiology department for possible embolization of the bleeding vessel. He was found to have multifocal pseudoaneurysms arising from distal collateral branches beyond the previously embolized splenic artery, which were localized to the gastric fundus (Figure 3). Although his altered anatomy and vasculature made the case challenging, embolization of the splenic artery, proximal branch of the superior mesenteric artery, and distal right gastroepiploic artery were successfully performed with coils and onyx (Figure 4). Bleeding source control was achieved; he was extubated and ultimately discharged. On a follow-up visit, he continued to do well with no further evidence of gastrointestinal bleeding.

**DISCUSSION**

Splenopneumopexy is a surgically created portopulmonary shunt used to alleviate portal hypertension causing variceal bleeding and ascites in those with Budd-Chiari syndrome and porto-splenic-mesenteric venous occlusion. It was predominantly performed in the 1980s. Nowadays, it has been replaced by various modalities for portal hypertension management. Today, there is limited literature on the indications, safety, and management of potential complications.

Generally, splenopneumopexy is a well-tolerated, safe procedure. Few case series exist regarding its efficiency. Reese et al reported cessation of gastrointestinal bleeding up to 48 months after this intervention. Akita et al described mean splenic pulp pressure reduction after splenopneumopexy; it was also effective in minimizing esophageal variceal bleeding and controlling ascites in patients with Budd-Chiari syndrome.
Splenopneumopexy complications, although rare, include acute variceal bleeding. Ono et al described a 29-month follow-up period in 16 patients; only 1 patient died from liver failure, with no reported cases of gastrointestinal bleeding. Splenoportography is the radiological modality of choice to assess proper function of the shunt. Our case highlights a massive bleed from a pseudoaneurysm almost 20 years after splenopneumopexy.

Although the endoscopist should have high suspicion for variceal bleeding in the appropriate clinical setting. Atypical-appearing lesions should be approached with extreme caution and perhaps referred to interventional radiology. In our case, it emphasizes the importance of obtaining imaging and involving interventional radiology earlier in patients with altered anatomy and atypical-appearing lesions.

DISCLOSURES

Author contributions: A. Abulawi and AH Al-Tarbsheh: wrote – original draft, reviewed the literature, revised the manuscript for intellectual content, and approved the final manuscript; R. Bui and S. Richter: manuscript review and editing, revised the manuscript for intellectual content, and approved the final manuscript;. Seth Richter, MD is the article guarantor.

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REFERENCES

1. Reese JC, Fairchild RB, Brems JJ, Kaminski DL. Splenopneumopexy to treat portal hypertension produced by venous occlusive disease. Arch Surg. 1992;127(9):1129–32.
2. Zuckerman DA, Levitt RG. Splenopneumopexy: Evaluation with splenoportography. J Vasc Interv Radiol. 1992;3(1):91–4.
3. Akita H, Sakoda K. Portopulmonary shunt by splenopneumopexy as a surgical treatment of Budd-Chiari syndrome. Surgery. 1980;87(1):85–94.
4. Ono J, Katsuki T, Kodama Y. Combined therapy for esophageal varices: Sclerotherapy, embolization, and splenopneumopexy. Surgery. 1987;101(5):535–43.
5. Sutton JM, Nussbaum MS, Vu D, Diwan TS, Starnes SL, Shah SA. Splenopneumopexy: Decompression of portal hypertension in the setting of portal venous occlusive disease. Dig Dis Sci. 2015;60(4):1101–5.