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Gastric Variceal Bleeding Secondary to Splenic Vein Thrombosis: A Case of Left-Sided Portal Hypertension

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INTRODUCTION

Gastric varices are found most frequently in patients with portal hypertension.¹ While gastric varices bleed less than esophageal varices, bleeding from gastric varices is often more severe and life threatening. A less common but significant cause of gastric varices is due to isolated left-sided portal hypertension (LSPH).² We present a case of bleeding gastric varices secondary to LSPH caused by a splenic vein thrombosis (SVT).

CASE REPORT

A 71-year-old male presented to his local emergency department after several black stools, dizziness, and an episode of syncope. His medical and surgical history included coronary artery disease with multiple stent placements, a pacemaker placement, and warm autoimmune hemolytic anemia diagnosed a month prior to presentation. An esophagogastroduodenoscopy (EGD) at this outside facility revealed what was thought to be a gastric ulcer along with two gastric polyps. The polyps were biopsied and the patient was sent home to follow-up with a colonoscopy a few days later.

The patient presented to our hospital, a regional referral center, the following evening and was admitted due to persistent symptoms of dizziness and dark stools. An abdominal exam was unremarkable for any tenderness to palpation or organomegaly. His blood pressure was 97/75 mmHg, pulse rate was 114 bpm, hemoglobin level was 5.9 g/dL, platelet count was 65,000/ μ L, lactic acid was 3.2 mmol/L, lactate dehydrogenase was 326 U/L, and liver function tests were unremarkable. On day two of hospitalization, a Technetium-99m red blood cell scan failed to localize any active bleeding, however, a repeat EGD revealed a large amount of hematin material and non-bleeding gastric varices in the gastric fundus consistent with isolated gastric varices type 1 (IGV1) by Sarin classification (Figure 1).¹ No endoscopic therapy was done. An abdominal ultrasound showed a patent portal vein but body habitus precluded the visualization of the splenic vasculature. An abdominal computed tomography angiography (CTA) scan on hospital day three showed a splenic vein thrombosis and a small superior splenic infarction. Surrounding structures, including the pancreas, appeared normal. The patient underwent an uncomplicated splenectomy on hospital day five and was sent home on hospital day nine. He was to follow-up with his primary care provider 14 days after his splenectomy to receive pneumococcal, Haemophilus, and meningococcal vaccines.

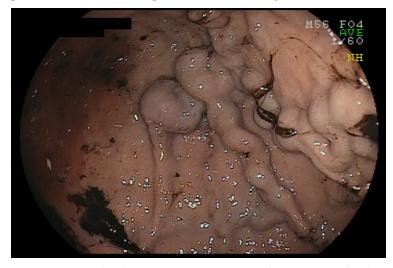


Figure 1. Isolated gastric varices in the gastric fundus.

DISCUSSION

LSPH is a less common, but significant cause of gastric varices that arises from any process that blocks blood flow through the splenic vein.^{3,4} Blockage results in left-sided venous hypertension forcing splenic blood to back up through collaterals, commonly the short gastric veins, forming varices in submucosal gastric vessels. Obstruction is usually intravascular due to a SVT, however, less common causes of splenic vein compression by nearby organs, tumors, and other processes have been documented. SVT most commonly is due to disease processes in the pancreas because it lies directly anterior to the splenic vein. Acute and chronic pancreatitis account for 60% of SVTs and pancreatic malignancies account for 9%.⁵

Most patients with LSPH secondary to SVT are asymptomatic and an SVT is found incidentally on imaging for other reasons.⁶⁻⁹ Symptomatic patients, such as ours, usually present with GI variceal bleeding (45 - 72%)^{5,10} and abdominal pain (25 - 38%).^{5,10,11} Splenomegaly is a variable finding and was not found in our patient. In a meta-analysis of patients with pancreatitis induced SVT, splenomegaly was only present in 51.9% of patients,¹² however, some studies suggest it is closer to 71%.¹⁰ Ascites is rarely found as a presenting sign in LSPH.¹³ Laboratory values can vary depending on the underlying etiology of the SVT, however, low hemoglobin suggests variceal bleeding. Nonetheless, a constellation of signs and symptoms should be

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considered. This was especially important with our patient who already had low hemoglobin secondary to hemolytic anemia.

Doppler ultrasonography (US) is a common preliminary test to evaluate for SVT in patients with symptomatic LSPH. While fairly sensitive and specific (93% and 83%)^{14,15} for finding portal vein thrombosis, the anatomic location of the splenic vein often makes it difficult to evaluate its patency, as was the case with our patient.¹⁵ In patients whose treatment likely will require splenectomy, more comprehensive imaging allowing visualization of the entire portal system, such as computed tomography (CT) or magnetic resonance imaging (MRI), is more favorable.¹⁶ In patients where pancreatic malignancy is suspected to be the cause of SVT, such as patients without a history of pancreatitis, endoscopic ultrasound (EUS) is used frequently because of its superiority to US and CT in diagnosing small pancreatic lesions and visualizing vascular invasion.^{17,18}

Once the diagnosis of gastric varices caused by SVT-induced LSPH has been made, treatment is based on whether the varices ever have bled. In patients with active bleeding, intravariceal cyanoacrylate injection is the first line therapy to achieve hemostasis when available.¹⁹ Other methods include banding or injection of a sclerosant. Once hemostasis is achieved or in patients with refractory bleeding, splenectomy is the treatment of choice,^{3,8,12,20-23} because rebleed rates without it range from 4 - 17%.^{7,8,24} Two studies have shown a 0% rebleed rate after splenectomy for previously bleeding gastric varices caused by LSPH.5,6 Cyanoacrylate injection can be used as definitive therapy in other types of gastric varices, but due to the amount of collateral connections between the stomach and splenic vein, splenectomy is favored in cases of varices due to SVT. Splenic artery embolization can be used as an alternative to splenectomy in high-risk surgical patients, however, splenic abscesses can occur in up to 7% of patients following the procedure.²⁵ Further studies are needed to confirm the efficacy of embolization for first line therapy.^{3,26} In the absence of any previous bleed, current literature suggests against prophylactic splenectomy.8 Heider et al.⁸ showed a bleeding rate of only 3.8% in patients with pancreatitis-induced SVT followed conservatively over 34 months.

Patients who undergo functional or surgical splenectomy require pneumococcal, Haemophilus, and meningococcal vaccines 14 days prior to the procedure. If urgent splenectomy is required, as was in our patient, vaccines should be administered at least 14 days following the procedure.²⁷ Studies with the pneumococcal vaccine suggest this timing allows for subsequently higher antibody concentrations.²⁸⁻³⁰ Revaccinations are required and should be administered in accordance with the Infectious Diseases Society of America (IDSA) recommendations for vaccination of patients with asplenia.²⁷

Prognosis is largely dependent on the underlying etiol-

ogy of the SVT. Treatment effectiveness often is evaluated by assessing for recurrence of bleeding, however, this is difficult because a large proportion of the patients have an underlying malignancy and their life expectancy is short.

Our case highlights the importance of accurate diagnosis and work-up of gastric varices caused by LSPH. It reminds physicians of the different causes of LSPH and what diagnostic and therapeutic approaches are available for addressing gastric varices caused by LSPH secondary to SVT.

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