

Introduction

Splenic vein thrombosis (SVT) typically occurs in the context of pancreatic diseases and leads to sinistral portal hypertension. Splenectomy is the treatment of choice and curative for bleeding from resultant isolated gastric varices (IGV). We report a unique approach to the management of gastric variceal bleeding secondary to SVT in the absence of malignancy or chronic pancreatitis. Hemostasis was achieved by recanalization of the splenic vein (SV) and subsequent embolization of gastric varices.

Case Presentation

A 43-year-old man with a prior history of alcoholic pancreatitis presented in hemorrhagic shock after copious hematemesis and multiple dark stools. Initial vitals were significant for sinus tachycardia. On examination, the abdomen was soft and nontender. His hemoglobin was 5.9 g/dL and platelet count 269,000/UL. Coagulation profile and liver function tests were normal. He was resuscitated with 3 units of packed RBCs. Esophagogastroduodenoscopy revealed active bleeding at the fundus, tempered intermittently with epinephrine injections. Computed tomography angiography abdomen showed a non-cirrhotic liver, mild splenomegaly with segmental SVT and perigastric varices; no paraesophageal varices noted. Repeat EGD found varices in the gastric fundus. Interventional radiology was consulted. A percutaneous transsplenic approach was employed. An SV branch was cannulated. Angiography revealed mid-vein near-occlusion and massive gastric varices (GV). Patient underwent splenic venography, venoplasty, stenting, and gastric variceal glue embolization via the short gastric veins. Heparin drip was given but soon afterwards but switched to apixaban for 6 months. Interval CT scan at 3 months showed occluded gastric varices, stable small splenic infarcts, and a patent stent in the splenic vein.

Images

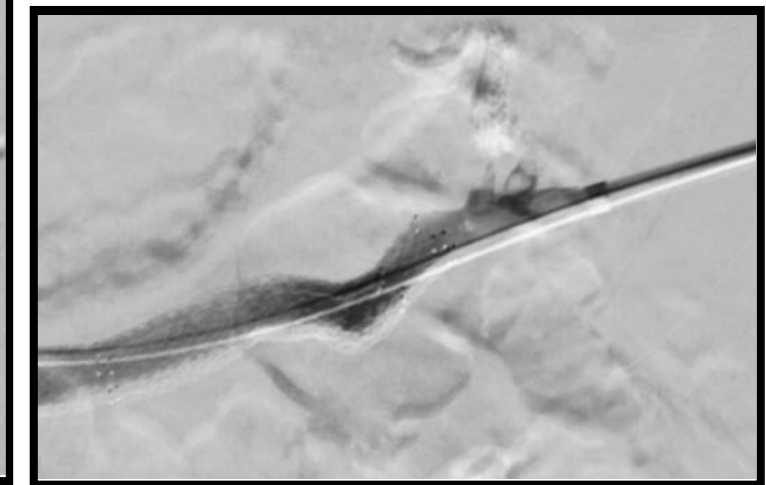
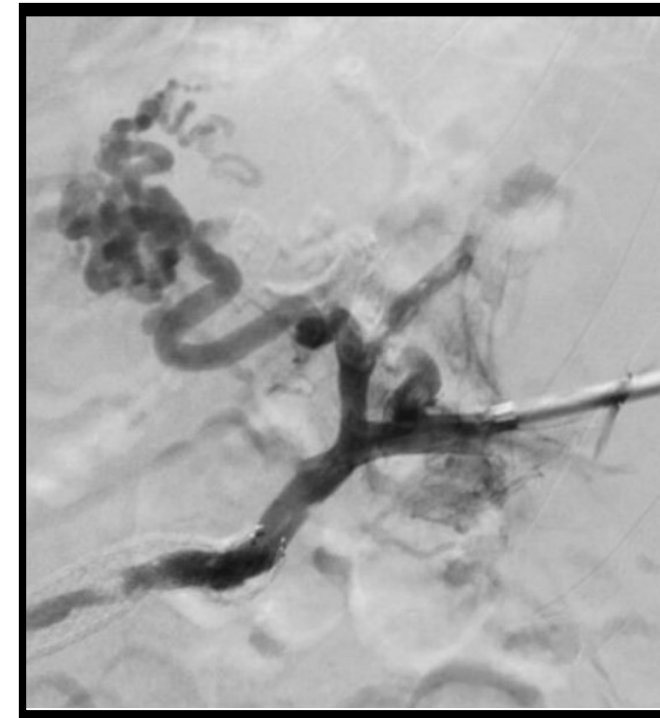
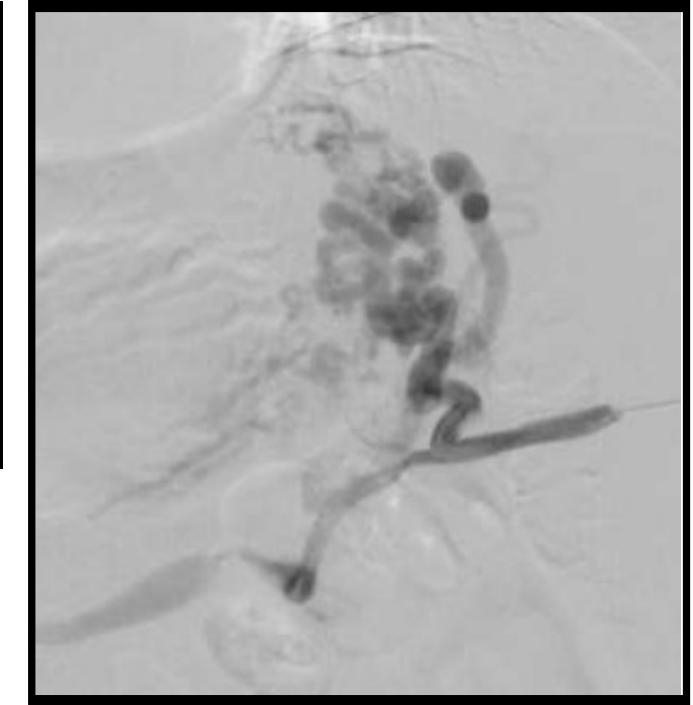
Top Left: Endoscopic view of fundic varices.

Splenic venography demonstrates near-complete splenic vein occlusion (**top right**). Splenic vein angioplasty, stenting (**bottom left**), and variceal embolization (**bottom right**) with restoration of rapid splenic in-line flow and cessation of variceal flow.

Discussion

Bleeding IGV secondary to SVT without underlying pancreatic lesions is rare and challenging to treat. Endovascular approaches, when available, provide spleen-sparing interventions for SVT with splenic vein stenting (SVS) favored over splenic artery embolization due to improved rebleeding risk. SVS preserves splenic parenchyma and immunologic function.

Our patient did develop punctate splenic infarcts, but have remained stable and asymptomatic, perhaps, given their small size, occupying a minute percentage of the total spleen volume. A transsplenic approach provides the most direct access to the affected vessel when compared with transjugular or transhepatic pathways. Our patient fared well with no abscess, refractory bleeding, or eventual splenectomy. We posit that, in SVT, splenic vein stenting should be considered in cases of sinistral hypertension, as a safe alternative to splenectomy.



Citations:

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