

Splenic Vein Stenting for Recurrent Chylous Ascites in Sinistral Portal Hypertension: A Case Report

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Research Article

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Abstract

Background:

Sinistral portal hypertension results from obstruction or stenosis of the splenic vein and is characterized by normal portal vein pressures and liver function tests. Gastrointestinal bleeding is the most common presentation and indication for treatment. Although sinistral portal hypertension-related chylous ascites is rare, several cases have described successful treatment with portal venous, rather than splenic venous, recanalization. Splenectomy is effective in the treatment of sinistral portal hypertension-related bleeding, although recent studies have evaluated splenic vein stenting and splenic arterial embolization as minimally-invasive treatment alternatives. Splenic vein stenting may be a viable option for other presentations of sinistral portal hypertension.

Case Presentation:

A 59-year-old gentleman with a history of necrotizing gallstone pancreatitis was referred to interventional radiology for management of recurrent chylous ascites. Analysis of ascites demonstrated a triglyceride level of 1,294 mg/dL. Computed tomography revealed splenic and superior mesenteric venous stricture. The patient elected to undergo minimally invasive transhepatic portal venography, which confirmed the presence of splenic vein and superior mesenteric vein stenosis. Venography of the splenic vein showed reversal of portal venous flow, multiple collaterals, and a pressure gradient of 14 mmHg. Two 10 mm x 40 mm Cordis stents were placed, which decreased the pressure gradient to 7 mmHg, and resolved the portosystemic collaterals. At 6 months follow-up, the patient had no recurrent episodes of ascites.

Conclusion:

The current case highlights successful treatment of sinistral portal hypertension-related intractable chylous ascites treated with transhepatic splenic vein stenting. Splenic venous stent patency rates of 92.9% at twelve months have been reported. Rebleeding rates of 7.1% for splenic vein stenting, 16% for splenectomy, and 47.8% for splenic arterial embolization have been reported in the treatment of sinistral portal hypertension-related gastrointestinal bleeding. The literature regarding splenic vein stenting for sinistral portal hypertension-related ascites is less robust. Technical and clinical success in the current case suggests that splenic vein recanalization may be a safe and viable option in other sinistral portal hypertension-related symptomatology.

Level of Evidence: Level 4, Case Report

Introduction:

Sinistral portal hypertension (SPH) results from obstruction or stenosis of the splenic vein and is characterized by normal portal vein pressures and liver function tests [1]. Gastrointestinal bleeding (GIB) is the most common presentation and indication for treatment [1]. Although SPH-related chylous ascites

is rare, several cases describe successful treatment with portal venous recanalization [2–4]. Splenectomy is effective in the treatment of SPH-related GIB, although recent studies have evaluated splenic vein stenting (SVS) and splenic arterial embolization (SAE) as minimally-invasive treatment alternatives [5–8]. Herein, a case of SPH-related recurrent chylous ascites successfully treated with SVS is described.

Case Report:

IRB approval was not required by our institution for this case report. A 59-year-old gentleman with a history of necrotizing gallstone pancreatitis was referred to interventional radiology for management of recurrent chylous ascites. The patient had gallstone pancreatitis two years prior to referral, which was complicated by the formation of recurrent and refractory infected pancreatic pseudocysts requiring repeat percutaneous and endoscopic drainage as well as a cyst-gastrostomy.

One month prior to referral, the patient developed chylous ascites requiring repeat paracentesis. Analysis of ascites demonstrated milky white fluid, with 89% neutrophils, 11% mononuclear cells, and a triglyceride level of 1,294 mg/dL. CT imaging demonstrated focal splenic and superior mesenteric venous stenoses, gastric varices, splenoportal collaterals, and large abdominal ascites. After discussion with the patient, the decision was made to proceed with percutaneous transhepatic venography with potential venoplasty and stenting.

Under moderate sedation, ultrasound-guided transhepatic right portal access was obtained with a 21 gauge chiba needle. Positioning was confirmed with injection of contrast under fluoroscopy. A 0.018" nitinol (Nitrex) guidewire with a 5 Fr KMP catheter (Cook) was advanced into the portal venous system. Portal venogram was normal, with a pressure of 3 mmHg. Superior mesenteric venogram demonstrated stricture adjacent to the portal confluence and an elevated pressure of 9 mmHg. Progressive superior mesenteric venoplasty was performed up to 7 mm with a Armada 35 balloon (Abbott Vascular). No change in the pressure gradient of 6 mmHg was demonstrated.

Splenic venogram demonstrated reversal of normal portal venous flow, multiple splenoportal venous collaterals, and a splenic venous pressure of 17 mmHg (Fig. 1). Progressive splenic venoplasty was performed with a Armada 35 balloon (Abbott Vascular) up to 1 cm with no angiographic or hemodynamic improvement. Two 10 mm x 40 mm Cordis stents were placed, which resulted in resolution of splenoportal collaterals and a decrease in the pressure gradient to 7 mmHg (Fig. 2). Track embolization was performed using two 8 mm x 14 cm coils and gelfoam slurry. The patient developed a post-procedure perihepatic hematoma but was discharged shortly thereafter, having received no additional interventions. He remained free of ascites six months after the procedure.

Discussion:

Splenic vein stenosis most commonly occurs as a sequela of pancreatic disease [6; 7]. The current patient's history of pancreatitis with infected pseudocyst was the likely etiology of splenic venous

stenosis. The most common clinical presentation of SPH is GIB, and most cases of splenic vein recanalization have treated this presentation [9]. Several studies have shown successful treatment of chylous ascites with portal venous recanalization [2–4].

Splenectomy is the historical treatment of SPH-related GIB, although SVS and SAE have been advocated as possible life-saving alternatives [8; 9]. Post-splenectomy sepsis from asplenia carries a reported mortality rate as high as 6% [10]. Rebleeding rates as high as 16% have been reported [10]. SAE carries the risk of splenic abscess, post-infarction syndrome, and incomplete therapeutic response requiring splenectomy [9].

Luo et al published a report on 11 patients diagnosed with SPH-related GIB who underwent transjugular splenic vein recanalization [6]. Six of these patients were identified as having splenic vein stenosis, and technical success was achieved in each case [6]. A recent retrospective comparative study between SAE and SVS for SPH-related GIB showed patients treated with SVS were less likely to develop rebleeding, with rebleeding rates of 7.1% and 47.8% in SVS versus SAE groups respectively [8]. The literature regarding SVS for SPH-related ascites is less robust, with few studies describing treatment of portal venous recanalization rather than SVS [2–4; 7]. Given that the described patient presented with a non-emergent presentation of SPH, SVS was felt to be the safest option in lieu of his multiple comorbidities and preference for minimally-invasive therapy.

Wei et al (2020) reported a cumulative stent patency rate of 92.9% twelve months after SVS [8]. A single splenic stent dysfunction was their only SVS-related complication [8]. At six months follow up, the current patient had no recurrence of ascites, but he did suffer a post-procedure perihepatic hematoma that resolved without further intervention. There has been some debate in the literature regarding a transjugular versus transhepatic approach, with some interventionalists avoiding a transhepatic approach due to perceived risk of increased bleeding [6]. Studies have shown success via both approaches, and a transhepatic approach was taken in the current case due to the relative ease of the technique and the patient's stable bloodwork [1; 5–7].

Conclusions:

The current case highlights successful treatment of SPH-related recurrent chylous ascites with transhepatic SVS. While evidence for SVS for SPH-related chylous ascites remains sparse, technical success and resolution of ascites suggest that splenic vein recanalization may be a safe and viable option. Although the current literature is promising, additional scientific studies are needed to assess the role of SVS for other SPH-related symptomatology.

Declarations

1. Ethics approval and consent to participate: All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national

research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. IRB approval was not required by our institution for this case report per HCA Healthcare ethics committee. .

2. Consent for publication: Written informed consent was obtained from the patient for publication of this case report and any accompanying images.
3. Availability of data and materials: All data generated or analysed during this study are included in this published article.
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6. Authors' contributions: All authors made substantial contributions to the conception of the work. BC drafted the manuscript. JM and RF substantially revised the manuscript. All authors have approved the submitted version and agree to be personally accountable for the author's own contributions. All authors ensure the integrity of this work.
7. Conflicts of Interest: The authors declare that they have no conflicts of interest.
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9. Authors' information: Not applicable

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Figures

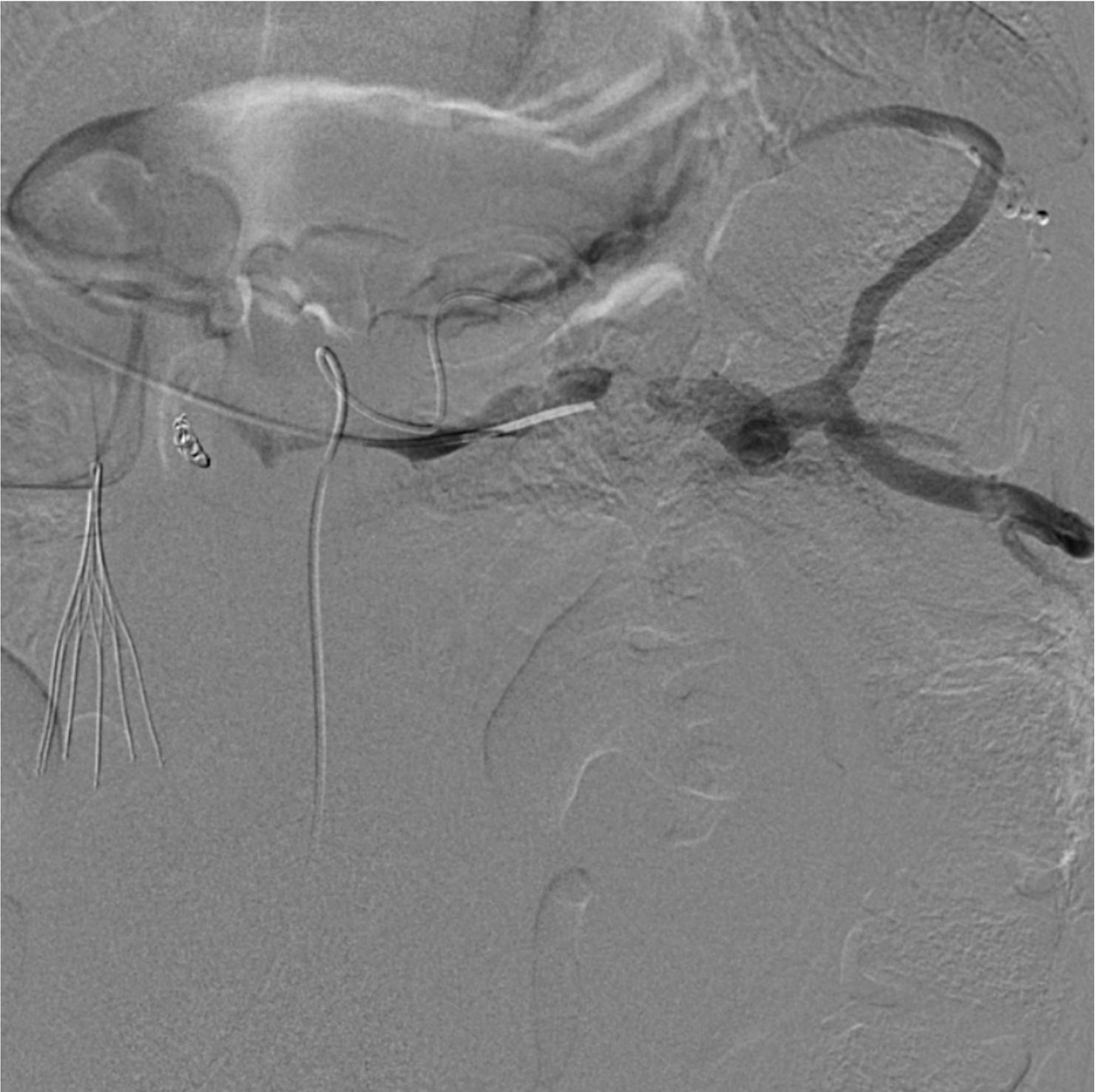


Figure 1

Digital subtraction venography of the splenic vein prior to venoplasty reveals multiple areas of stenosis and robust venous collaterals from portal hypertension.



Figure 2

Digital subtraction venography of the splenic vein after deployment of splenic stents shows increased splenic venous patency with resolution of splenoportal collaterals.