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Case Report

Gastroduodenal artery pseudoaneurysm and chronic superior mesenteric vein thrombosis treated with transcatheter embolization and stent dilatation, respectively: 7 year clinical and imaging follow-up [☆]

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ABSTRACT

Pancreatic pseudocyst formation, arterial pseudoaneurysm, and splanchnic vein thrombosis are complications of chronic pancreatitis that account for significant morbidity and mortality in this patient population. While the short-term utility of timely endovascular intervention for treating the vascular complications of chronic pancreatitis have been relatively well documented, there is a paucity of longitudinal follow-up in this patient population, therefore making it difficult to predict the long-term efficacy of these interventions. This report details a case of a gastroduodenal artery pseudoaneurysm embolization followed by symptomatic superior mesenteric vein chronic thrombosis treated by stent dilatation in a patient with chronic pancreatitis, with 7 years clinical and imaging follow-up.

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Introduction

Acute pancreatitis (AP) is a painful, inflammatory disease that targets the exocrine pancreas, arising as the result of premature intra-acinar enzymatic activation of pancreatic zymogens which cause local damage to pancreatic parenchyma and

surrounding abdominal organs [1]. It is overwhelmingly common, estimated to occur in approximately 40–110 in 100,000 adults globally [2]. While many cases may be managed in the outpatient setting, acute pancreatitis remains the third most common gastrointestinal cause for hospitalization in the United States, and in cases of persistent inflammation or frequent recurrence, may progress to chronic pancreatitis

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(CP) [3]. Chronic pancreatitis is a broad term describing a wide range of progressive fibro-inflammatory diseases of the exocrine pancreas that eventually lead to destruction and permanent damage to both endocrine and exocrine pancreatic parenchyma, and characteristically results in widespread calcium deposition [4]. Complications of both acute and chronic pancreatitis include necrosis, pancreatic pseudocyst formation, adjacent arterial pseudoaneurysm, and splanchnic vein thrombosis, which represent a source of significant morbidity and mortality in this patient population [5,6].

While a pseudoaneurysm is a relatively uncommon complication, with incident rates of 0.05% and 0.03% in acute and chronic pancreatitis respectively, it can lead to life-threatening hemorrhage. Angiographic embolization remains the preferred treatment, with clinical success rates at 12 months of 82%, 86%, and 88% respectively [5,7].

Meanwhile, splanchnic vein thrombosis (including the portal, splenic, and superior mesenteric veins), is much more common, with an estimated prevalence in patients with acute and chronic pancreatitis of 16.6% and 11.6% respectively [6]. Splanchnic vein thrombosis often manifests clinically as portal hypertension and its sequelae, including ascites, varices, hemorrhage, and bowel ischemia, further complicating management. Anticoagulation is the recommended first-line treatment in hemodynamically stable patients with symptomatic superior splanchnic vein thrombosis [8,9]. While less commonly performed, percutaneous stent dilatation has demonstrated efficacy in relieving portal hypertension while also providing symptom relief in patients with stenosis or occlusion of splanchnic veins and can serve as a definitive treatment in patients unresponsive to anticoagulation, or where anticoagulation is contraindicated [10–12].

While the short-term utility of endovascular intervention for treating the vascular complications of chronic pancreatitis have been relatively well documented, there is a paucity of longitudinal follow-up in this patient population, therefore making it difficult to predict the long-term efficacy of these interventions. This report details a case of a gastroduodenal artery pseudoaneurysm embolization followed by symptomatic superior mesenteric vein chronic thrombosis treated by stent dilatation in a patient with chronic pancreatitis, with 7 years clinical and imaging follow-up.

Case summary

A 64-year-old male with a history of chronic pancreatitis with pseudocysts and ascites presented to an outside hospital with increasing abdominal pain, hypoxic respiratory failure, and hypotension. He was intubated and transferred to our institution's medical intensive care unit (MICU) where he was treated for septic shock and found to have an infected pancreatic pseudocyst in the setting of severe necrotizing pancreatitis. CT imaging demonstrated a 10.2 × 7.8 cm pseudocyst within the head of the pancreas (Fig. 1), and thrombosis in the portal vein extending into the splenic and superior mesenteric veins (Fig. 2), which was diagnosed during a previous admission and treated with anticoagulation. Following aggressive resuscitation and pseudocyst drainage by interventional radiology (IR)



Fig. 1 – Axial contrast-enhanced CT image which demonstrates a large pancreatic pseudocyst measuring approximately 7.8 × 10.2 cm adjacent to the inferior right lobe of the liver.

(Fig. 3–4), his course was further complicated when he developed hematemesis and CT angiography subsequently demonstrated a focus of arterial phase contrast media extravasation within the large pancreatic head pseudocyst consistent with active hemorrhage from the gastroduodenal artery (GDA).

The patient was then emergently brought to IR for therapeutic transcatheter embolization. A conventional catheter angiogram of the celiac artery showed occlusion of the GDA likely related to vasospasm. A 4-French catheter was advanced into the GDA and an angiogram of the proximal GDA demonstrated a large bleeding pseudoaneurysm with extravasation into the pseudocyst (Fig. 5). A microcatheter and guidewire were then coaxially advanced into the pseudoaneurysm and 2 mechanical detachable micro-coils were deployed into the pseudoaneurysm, across the pseudoaneurysm neck, and into the distal and proximal GDA. A post-coil embolization angiogram of the celiac artery showed complete occlusion of the GDA and pseudoaneurysm and no evidence of extravasation (Fig. 6). A post embolization superior mesenteric artery angiogram showed no active hemorrhage. Over the course of 6 weeks, the patient's clinical state improved, and he was ultimately discharged on 6 months of anticoagulation to treat the superior mesenteric vein (SMV) thrombosis.

Three months later, the patient presented to an outside hospital following a fall with a hemothorax, pneumothorax, and worsening ascites and anasarca (Fig. 7). Prior to paracentesis he was found to have a supratherapeutic International Normalized Ratio (INR) of 4.9. The patient was then transfused with 5 units of Fresh Frozen Plasma and subsequently developed sudden cardiac arrest requiring cardiopulmonary resuscitation and 2 rounds of epinephrine administration for a suspected transfusion reaction. The patient was intubated and again transferred to our institution where two paracenteses were performed yielding 8.5 L and 4.3 L of transudative fluid, suggestive of portal venous hypertension. Subsequent CT angiography demonstrated persistent portal venous outflow ob-

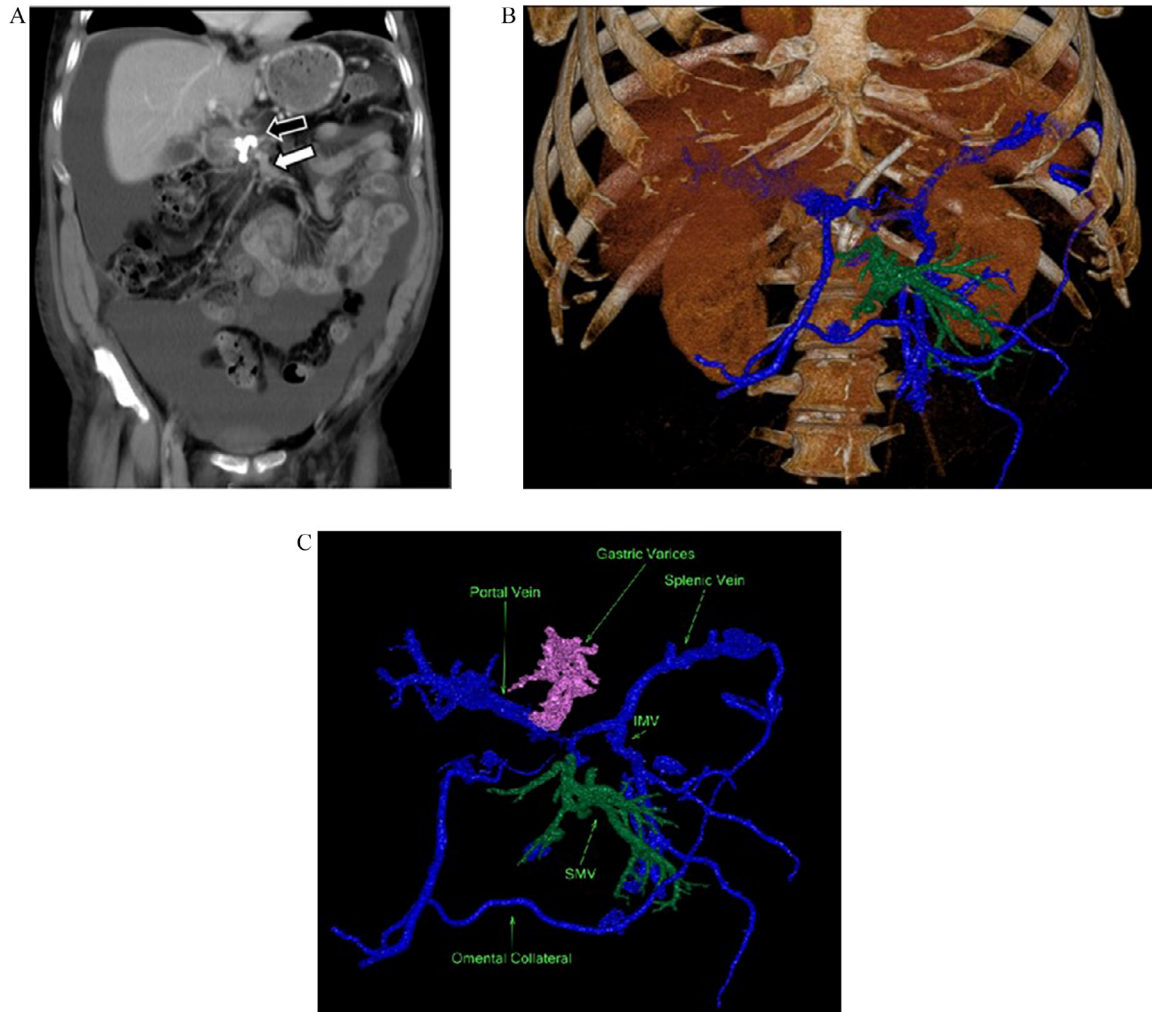


Fig. 2 – (A) Coronal contrast-enhanced CT showing region which corresponds to chronically occluded superior mesenteric vein (SMV) and portal vein. The white arrow with a black border corresponds to the patent portion of the SMV adjacent to the chronic occlusion. The portal vein is not well visualized in this image. The hyperdense focus in the center of the image denoted by the black arrow corresponds to both a coil from prior gastroduodenal artery aneurysm and a portion of the patient’s transgastric pseudocyst drainage tube, which appears C-shaped in the above image. **(B)** 3D volume rendering of CT scan of chronic SMV occlusion. The SMV is colored green, and the reconstituted portal vein adjacent to this is colored in blue. Of note, there is discontinuity between the SMV and portal vein due to chronic occlusion. **(C)** 3D volume rendering of abdominal vessels with bone and adjacent tissues subtracted. SMV = Superior mesenteric vein, IMV = Inferior mesenteric vein. Of note, direct communication between the SMV and Portal vein is absent due to chronic occlusion.

struction due to the chronic portal vein thrombosis extending into the SMV.

He was referred to IR for hepatic and portal vein venography, core needle liver biopsy, porto-systemic venous pressure measurement, and stent dilatation of the portal vein and SMV was then performed. Via a right trans-jugular vein approach, a 4-French catheter was advanced into the right hepatic vein and two 19-gauge transvenous core needle liver biopsies of the right lobe of the liver were obtained. Next, a sheath needle was used to puncture from the right hepatic vein and into a right portal vein branch. A 4 French catheter was advanced into the portal vein and a portal vein venogram was performed. This demonstrated chronic occlusion of the portal vein and SMV, with reconstitution of the portal vein in the porta hepatis from

collateral venous channels (Fig. 8A). The portal vein pressure was measured to be 20 Torr, and right hepatic vein pressure was found to be 17 Torr, for a portosystemic gradient of 3 Torr.

The 4 French catheter was exchanged over a guidewire for a long 4 French sheath. A 4 French catheter was advanced from the right hepatic vein, through the hepatic parenchymal track, into the portal vein, and up against the portal vein occlusion. Utilizing standard guidewire technique, the 4 French catheter was advanced through the chronically occluded segment of the portal vein and SMV and into the patent peripheral segment of the superior mesenteric vein. An SMV venogram was performed. This demonstrated a 4 cm long occlusion of the central aspect of the superior mesenteric vein, extending into the portal vein (Fig. 8B). A superior mesenteric vein pressure



Fig. 3 – Percutaneous drainage of pancreatic pseudocyst under CT guidance. Axial CT image shows a pigtail catheter coursing through the stomach, terminating in the pseudocyst. Oral contrast was used in this study.

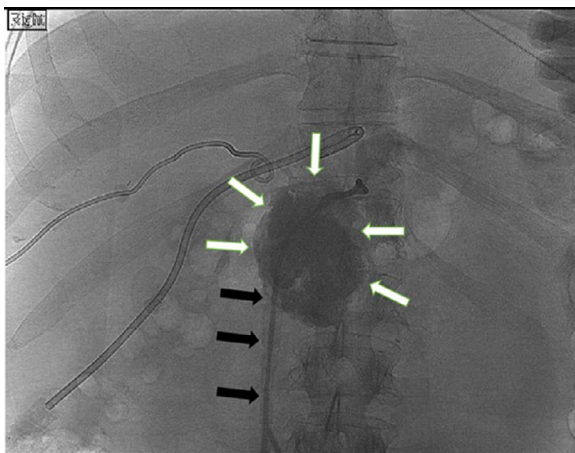


Fig. 4 – Transgastric pseudocyst drainage tube placement. Several tubes are present in this image related to other abscesses present in this patient, however the relevant catheter is denoted by black arrows, showing a transgastric pseudocyst drainage tube terminating within the pancreatic pseudocyst. The pseudocyst has been injected with iodinated contrast media and is denoted by white arrows.

of 26 Torr was measured. This represented a superior mesenteric vein to systemic pressure gradient of 9 Torr with a pressure gradient across the superior mesenteric and portal vein occlusion of 6 Torr.

Next, a 10 mm in diameter self-expanding Nitinol stent was deployed across the chronic occlusion of the superior mesenteric and central portal veins, and subsequently dilated with an 8-mm diameter angioplasty balloon. A post-stent dilatation SMV venogram demonstrated widely patent superior mesenteric and portal veins, without evidence of residual stenosis, extravasation, thrombosis, or dissection (Fig. 8C). Pressure measurements were then measured in the superior

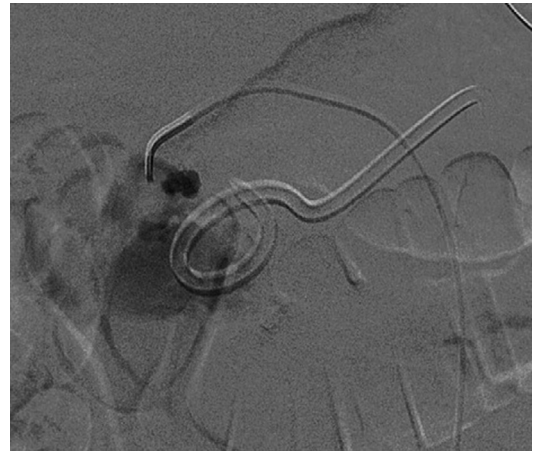


Fig. 5 – Proximal gastroduodenal artery (GDA) digital subtraction angiogram demonstrated a large bleeding pseudoaneurysm with extravasation into the duodenum.



Fig. 6 – Post-coil embolization angiogram showing occlusion of the gastroduodenal artery (GDA) and pseudoaneurysm.



Fig. 7 – Abdominal CT scan showing severe ascites in the setting of chronic SMV thrombosis. SMV = Superior mesenteric vein.

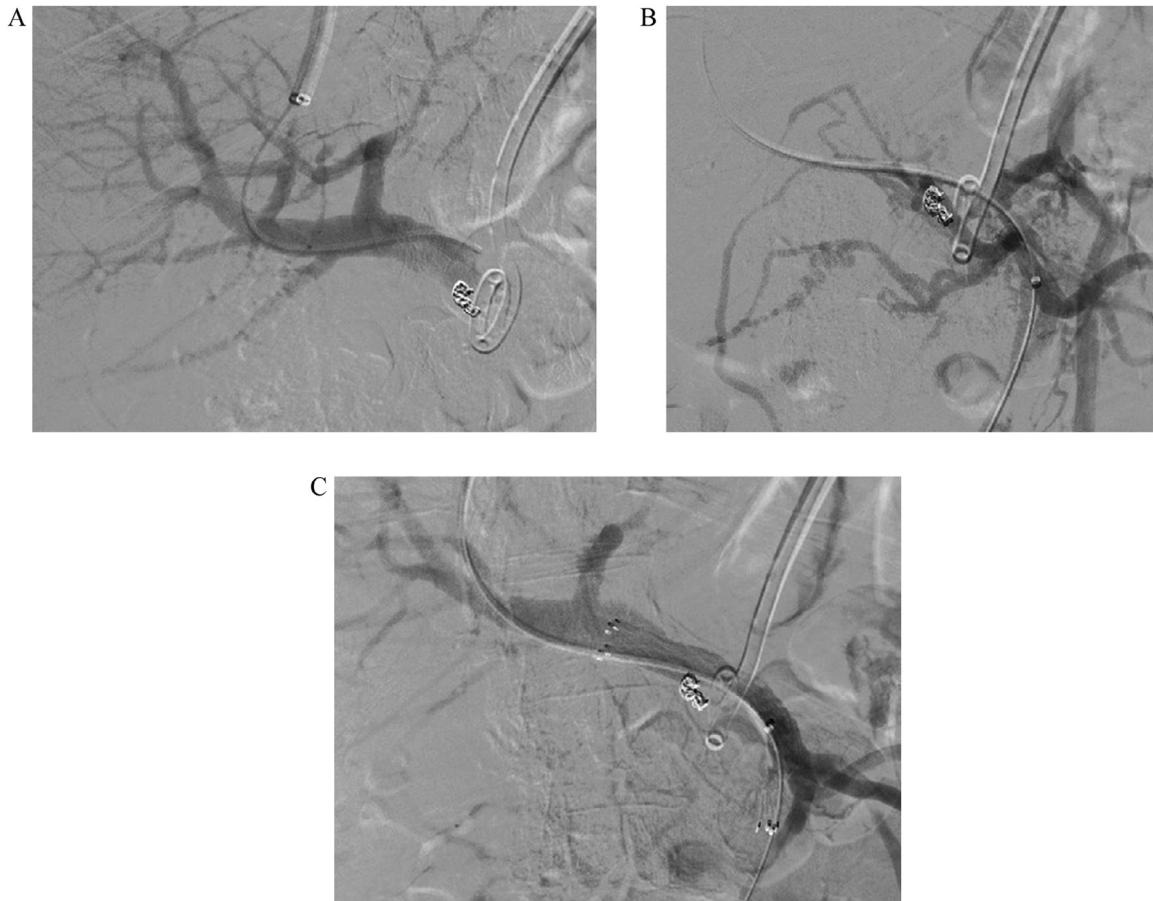


Fig. 8 – (A–C): A. Pre-stent transjugular venogram of chronic portal vein and SMV occlusion prior to traversing the chronic occlusion. (B) Venogram after traversing the chronic occlusion with the placement of the catheter into the patent portion of the SMV. This image demonstrates chronic occlusion of the portal vein and central SMV, with adjacent filling of a rich collateral venous circulation. (C) Post-stent dilatation SMV venogram demonstrating widely patent superior mesenteric and portal veins, without evidence of residual stenosis, extravasation, thrombosis, or dissection. SMV = Superior mesenteric vein.

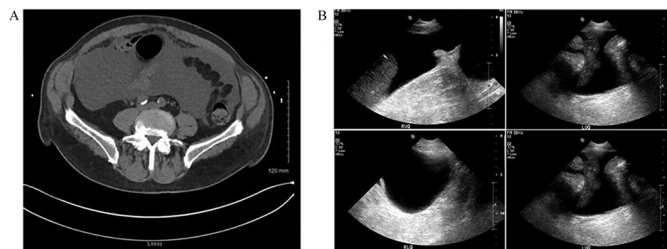


Fig. 9 – (A–B) A. Abdominal CT and ultrasound imaging demonstrating improvement in ascites 1-week post-stent placement. (B) Transabdominal ultrasound demonstrating improving ascites.

mesenteric and right hepatic veins, as well as the right atrium of the heart. The measurements obtained were, 25, 23, and 19 Torr, respectively, representing a reduction of the superior mesenteric vein to systemic pressure gradient from 9 to 2 Torr. The SMV venogram obtained prior to the stent dilatation procedure demonstrated innumerable venous collateral channels throughout the abdomen. These venous collateral channels were no longer identified on the post-stent dilatation SMV

venogram, which also demonstrated hepatopetal blood flow through the SMV, whereas it was previously hepatofugal. Clinically, the patient experienced significant improvement in his symptoms, and was discharged from the hospital the following day.

A week later the patient was readmitted for lethargy, hypotension, and recurrent, yet improved, ascites (Fig. 9A–B), and the SMV stent was found to be partially occluded. Anticoag-

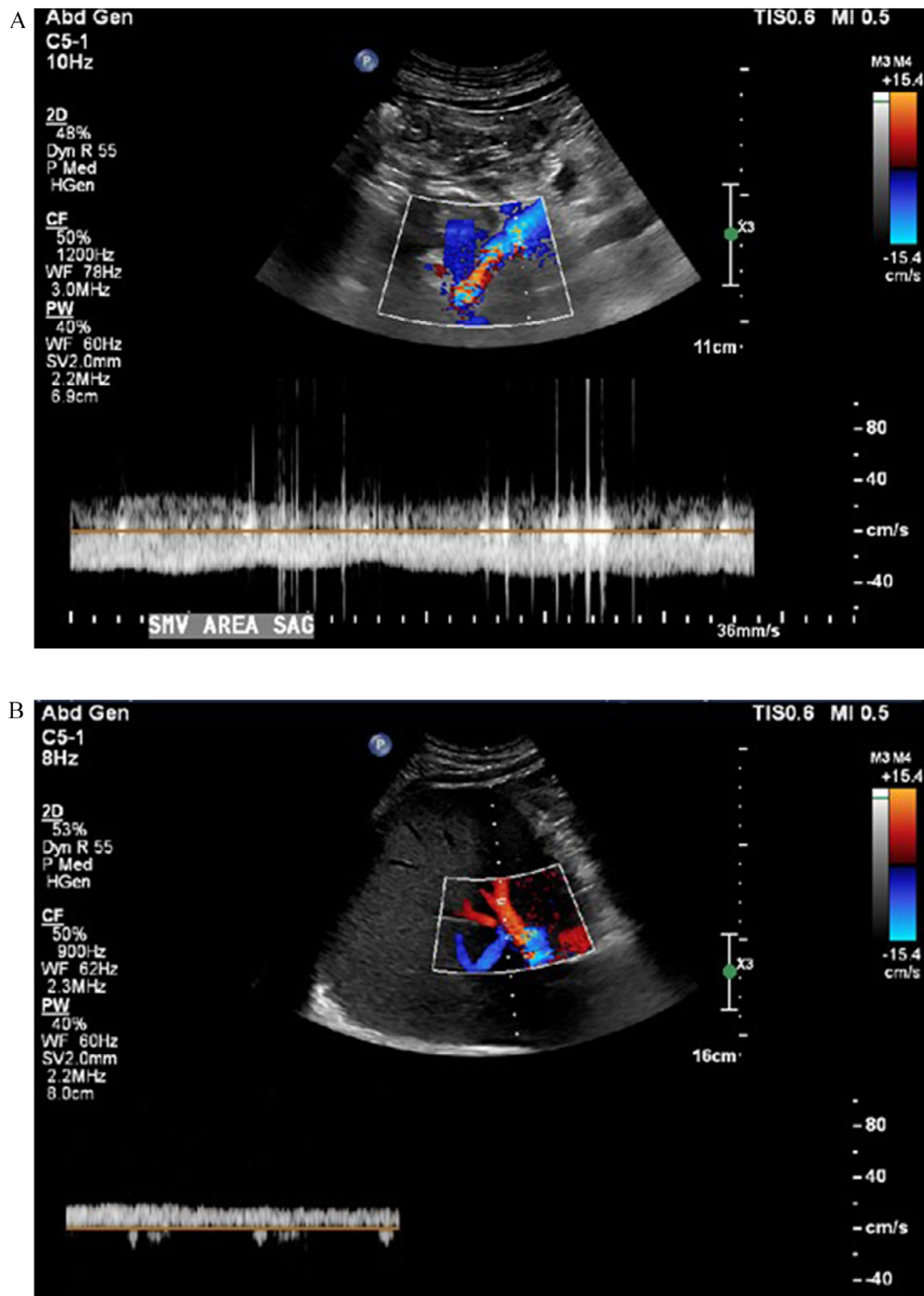


Fig. 10 – (A–C) Duplex ultrasound at 7 years post-stent placement. (A) Ultrasound demonstrating widely patent superior mesenteric vein (SMV) stent near its insertion into the portal vein with no evidence of ascites. The blood flow across the SMV portion of the stent is brisk and consistent with hepatopetal venous flow on duplex ultrasound imaging. (B) Blood flow continues in a hepatopetal flow pattern in the intrahepatic portion of the portal vein. (C) Ultrasound sagittal image of the midline deep abdomen showing no residual ascites. The imaged vessel corresponds to the infrarenal abdominal aorta near its bifurcation into the common iliac arteries.

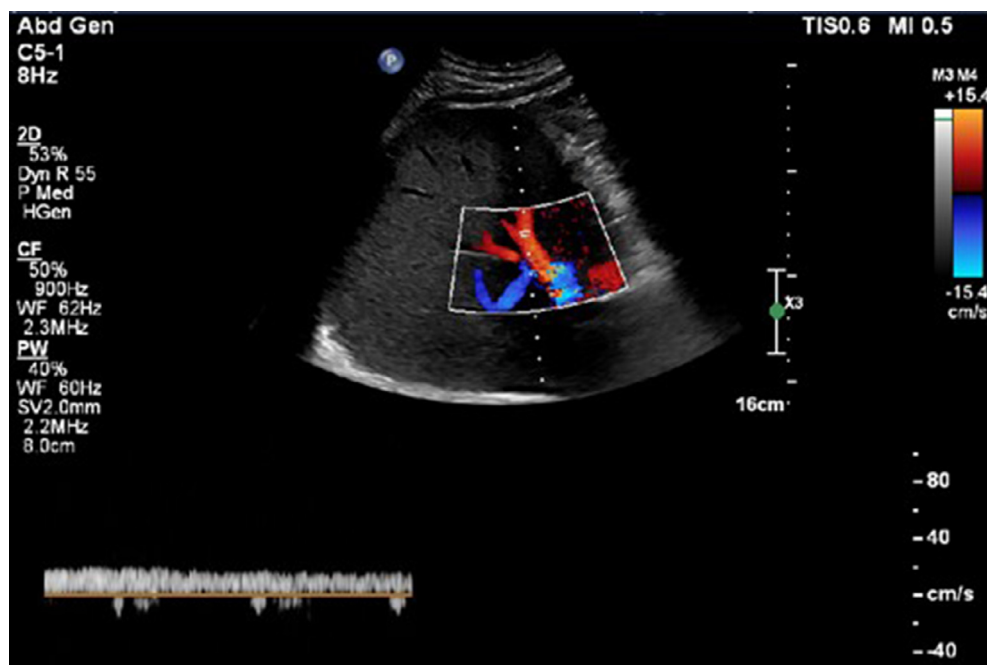


Fig. 10 – Continued

ulation was then resumed, and the patient was discharged 3 days later. The patient recovered remarkably well and was asymptomatic and free of ascites at 3 months and 7 years of clinical follow-up. Duplex ultrasound examinations at 3 months and 7 years (Fig. 10) follow-up showed a widely patent SMV and portal vein and no ascites.

Discussion

This case demonstrates the long-term efficacy of endovascular intervention in patients with vascular complications of chronic pancreatitis. This is a single case report and should not be extrapolated to infer that all patients with chronic SMV thrombosis will experience similar outcomes and longevity, but this report does shed light on the potential of splanchnic vein stent dilation in patients with chronic splanchnic vein thrombosis and demonstrates that these benefits can be long-lasting.

Additionally, a recent literature review by Khan et al suggests that portal vein stent placement following hepatobiliary and pancreatic surgery is a safe and effective treatment in patients with portal vein stenosis, with clinical success rates of 72%–100%, long-term patency of 57%–100%, and procedure-related complication rates of 0%–33.3% [12]. While this literature review included a heterogeneous population of post-surgical and liver transplant patients, we believe the technical success rates and complication rates are relatively generalizable.

While stent dilation of the chronic SMV thrombus in this case resulted in clinical resolution of the patient's symptoms,

it is poorly understood how splanchnic vein thrombus characteristics such as location and size correlate to procedural and clinical success rates. It would be helpful to delineate efficacy of splanchnic stent dilation based off location to determine if there is a difference in outcomes between portal, splenic, or superior mesenteric vein thrombosis. It is important to note that currently there is not strong evidence to suggest which subset of patients would benefit most from splanchnic vein stent after medical management has failed, and these groups should be delineated in future studies.

Furthermore, the role of long-term anticoagulation following splanchnic vein stent dilation is not well understood. Studies have varied in their usage of anticoagulation with bleeding risk and the risk of stent thrombus formation factoring into this decision [12]. Interestingly, our patient developed partial occlusion of the SMV stent 1 week following the procedure at which time anticoagulation was resumed and the patient's symptoms quickly improved. We feel that efforts should be made to better understand the role of long-term anticoagulation in these patients, and which sub-populations would benefit the most. Patient-specific factors such as inflammation in chronic pancreatitis and hypercoagulable states related to malignancy in patients in prior studies present challenges when trying to understand the risks and benefits of long-term anticoagulation.

The results at 7 years follow-up in this case report are encouraging and support the long-term benefits of pseudoaneurysm embolization and stent dilation of chronic splanchnic vein thrombosis in patients with chronic pancreatitis. Additionally, this report emphasizes the need for extended follow-up in this patient population to allow for better understanding of the longitudinal outcomes of these interventions.

Patient consent

The patient consented to this report. Additionally, patient agreed to follow-up ultrasound scan and appointment as part of this report. There is no identifiable images information in the report.

Ethical approval

All procedures performed in the 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.

Author contribution

Case concept: CM; case/literature review, manuscript drafting, and revision: all authors

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