Coil embolization of an acutely expanding spontaneous splenic artery dissection

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ABSTRACT

Symptomatic dilation of a spontaneous splenic artery dissection is a very rare and potentially catastrophic nonatherosclerotic vascular disease. Splenic artery rupture has not been reported after acute diffuse dilation, but it has been reported with celiac artery dissections. We believe treatment is mandatory if pain persists despite blood pressure control. The presentation and endovascular treatment of a spontaneous celiac trunk dissection with continued expansion of the splenic artery branch are discussed. (J Vasc Surg Cases and Innovative Techniques 2018;4:144-6.)

Spontaneous isolated celiac and splenic artery dissections are unusual nonatherosclerotic vascular entities and are frequently associated with hypertension. They are generally treated with observation, blood pressure control, and anticoagulation. Intervention, whether open or endovascular, is reserved for the rare case complicated by clinical deterioration with hypotension, progression of the dissection, or persistent abdominal pain. We describe the successful endovascular treatment of a persistently enlarging and symptomatic splenic artery dissection using coil embolization. The patient gave consent to publish his data and images.

CASE REPORT

A 44-year-old man with a past medical history of obesity, anxiety, and hyperlipidemia presented with new-onset hypertension and sudden, intense left upper quadrant and midabdominal pain. His initial blood pressure of 185/134 mm Hg responded partially to several doses of metoprolol by intravenous push, with an esmolol drip required to obtain a goal systolic pressure below 120 mm Hg. There was a family history of coronary disease but no parental history of refractory hypertension. A spontaneous celiac dissection was seen on computed tomography angiography (CTA). The celiac artery dissection extended throughout the common hepatic and splenic branches (Fig 1), causing a severe hepatic artery stenosis and several small splenic infarcts corresponding to nonopacified terminal splenic artery branches. Other than minimally elevated alanine transaminase of 71 IU/L (normal range, 5-55 IU/L) on admission, which subsequently normalized, the remaining liver enzyme levels were normal and remained normal for the remainder of the patient's hospitalization.

Initially, his symptoms responded very well to blood pressure control, and he was discharged from the hospital after 4 days of observation and decreasing pain. The patient was readmitted later the same day with an abrupt increase of severe, unremitting pain that did not respond to aggressive blood pressure control. Two repeated CTA scans showed diffuse splenic artery enlargement from 8 to 10 mm at 4 days, then to 12 mm at 6 days after the initial presentation (Fig 2). Only the splenic artery branch continued to diffusely dilate, even though the dissection involved both the common hepatic and splenic arteries. The celiac and hepatic branches remained unchanged in caliber. We recommended intervention to prevent rupture of the splenic artery.

Coil embolization was thought to be the best and least invasive way to prevent rupture, the alternative procedure being open surgical ligation of the artery. Postsplenectomy vaccine prophylaxis was given before the procedure in the event of extensive splenic infarction. Surgical splenectomy was not considered the better option, as the spleen still appeared to be viable, supplied by the short gastric branches. In addition, removal of the spleen and ligation of the origin of the splenic artery would have exposed the patient not only to operative risks, such as blood loss, pancreatitis, pulmonary complications, intra-abdominal abscess, and inadvertent injury to the stomach or colon, but also to the lifelong risk of postsplenectomy septic infection.

The celiac trunk was accessed with a visceral selective (VS2) catheter and Bentson wire, and a 5F Ansel sheath tip (Cook Medical, Bloomington, Ind) was positioned into the proximal splenic artery. A 4F Kumpe Slip-Cath was then passed easily to the terminus of the splenic dissection. Through the Slip-Cath, 18 coils were placed with the intent of thrombosis of the entire splenic artery (Fig 3). Detachable Terumo coils (Terumo Medical, Somerset, NJ) and nondetachable Cook coils were used: 11 Azure D35 (Terumo), 3 Azure CX 35 (Terumo), and 4 0.035-inch Nester (Cook) coils. Coil diameters ranged from 10 to 15 mm, lengths ranged between 14 and 34 cm, and two framing coils were used.

After coiling, completion selective splenic artery injection through the delivery catheter did not opacify the common hepatic artery (Fig 3). Withdrawal of the catheter into the aorta

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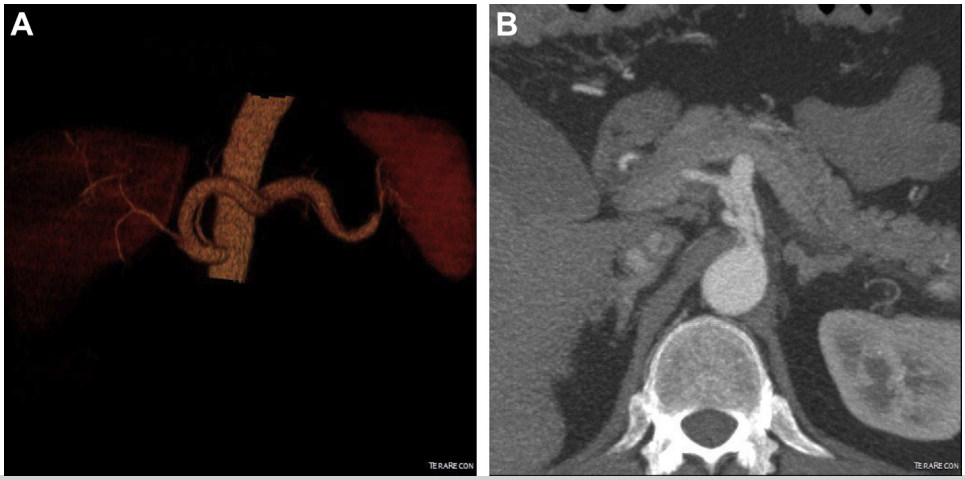

Fig 1. A, Celiac artery dissection extending throughout the common hepatic and splenic branches, causing a severe hepatic artery stenosis and diffuse dilation of the majority of the splenic artery to the level of the terminal branches in the splenic hilum. **B,** Axial image showing the celiac dissection with the entry point in the proximal celiac trunk, with no abnormality seen in the aorta at that level.

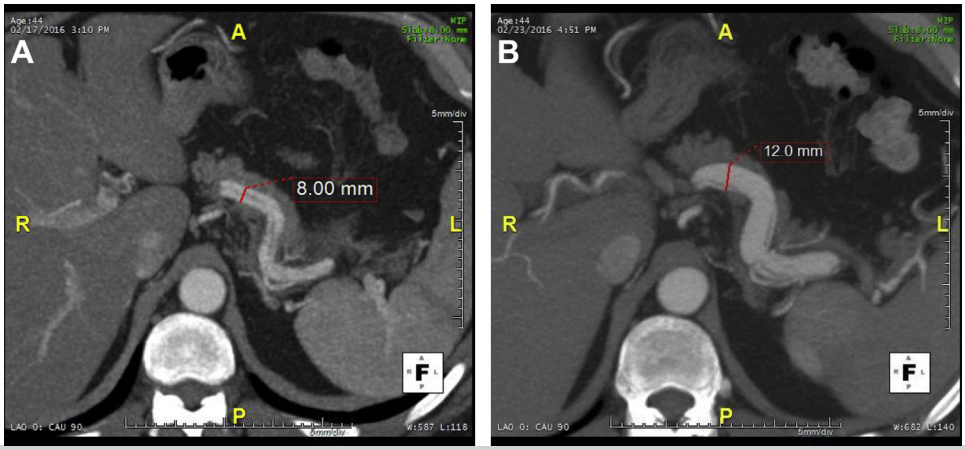

Fig 2. A and **B,** Comparison axial computed tomography angiography (CTA) images showing splenic artery diffusely dilating over 6 days from 8 to 12 mm.

for an aortogram briskly filled both the true and false lumens of the hepatic artery, suggesting that the coils were placed into the splenic artery false lumen. Before the procedure, we thought that filling either the true or the false lumen would produce the same intended result of thrombosis of the entire artery. For this reason, no intravascular ultrasound was used to attempt to discern which lumen the coils were deployed in.

The patient's abdominal discomfort completely resolved after 1 week, and he has been maintained on carvedilol and amlodipine. None of the abdominal CTA scans suggested renovascular hypertension or fibromuscular dysplasia as the cause of his new-onset hypertension, and his blood pressure has been well controlled in follow-up, with his carvedilol dosage reduced significantly as his abdominal pain subsided in the weeks after the procedure. The patient is also maintained on aspirin for the persistent celiac artery dissection.

At 9 months after the procedure, repeat CTA shows that the hepatic artery has healed without a stenosis, and the proximal splenic artery is normal in caliber, measuring 5 mm. The spleen has not changed in size or shown any new infarcts and is still perfused by a normal distal splenic artery. The celiac dissection is still visible and has not changed in its maximal craniocaudal diameter of 12 mm. The dilated celiac artery will be observed for aneurysmal degeneration in the future with duplex ultrasound as opposed to CTA, given the scatter artifact caused by the coils.

DISCUSSION

In the literature, celiac and splenic artery dissections predominantly affect hypertensive men with an average age of 55 years.[1,2] Other risk factors include pre-existing vascular disease (such as in Marfan or Ehlers-Danlos syndromes, fibromuscular dysplasia, and segmental arterial mediolysis or with atherosclerosis). They can also occur in pregnancy, have iatrogenic or traumatic causes, or be infectious in etiology. Most patients with celiac and

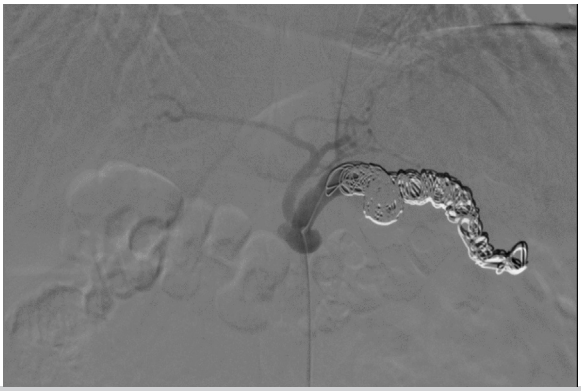

Fig 3. Celiac angiogram showing all 18 coils within the splenic artery. This selective splenic artery injection did not opacify the hepatic artery, suggesting that the coils filled the splenic artery false lumen.

splenic dissections lack these risk factors, with the cause of most spontaneous dissections thought to be mainly hypertension,[2] as in our patient.

Complications of spontaneous celiac artery dissection mandating treatment are aneurysm formation, arterial occlusion, and rupture, which are rare. The consensus in the literature is that celiac or splenic artery dissections are treatable in 75% to 90% of the cases with only observation, blood pressure control, and anticoagulation. Surgical or endovascular management is reserved for the relatively rare cases of hemodynamic instability, persistent abdominal pain, or progression of the dissection and end-organ ischemia.[2-5]

Many cases of celiac and splenic artery dissection are found in the literature, but no similar cases of refractory symptomatic dilation of only the splenic artery have been reported. However, there are at least three cases of rupture of spontaneous celiac artery dissections just distal to the celiac bifurcation in either the proximal splenic or hepatic branches,[6,7] and fatal rupture in patients with Ehlers-Danlos syndrome is also well described.[8] These cases are the basis for treating this patient aggressively.

We opted to treat this patient with standard coil embolization[9] with extensive packing to prevent recanalization, which can occur in 9% to 50% of visceral artery embolizations.[10,11] Longer detachable microcoils, up to 60 cm, are available, such as the Ruby coil (Penumbra, Inc, Alameda, Calif). Using these may have been able to reduce the number of total coils used, potentially lowering costs in this case.

Amplatzer plugs (St. Jude Medical, St. Paul, Minn) may have additional radial force that could have precipitated rupture of the weakened and actively expanding dissected artery. Nonetheless, Amplatzer plugs have also been reported in the successful treatment of symptomatic celiac dissections associated with aneurysms.[11]

This case is analogous to Sakamoto type 2 or type 5 superior mesenteric artery dissection with diffuse dilation and luminal stenosis.[12,13] Superior mesenteric artery dissections are treated conservatively in 55% to 63% of cases unless complications develop.

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