Primary intra-aneurysmal surgical repair of a celiomesenteric trunk aneurysm

R. Jason VonDerHaar, MD; Amir Shah, MD; Nicholas N. Nissen, MD; and Bruce L. Gewertz, MD

Los Angeles, Calif

We describe the surgical management of an asymptomatic 3-cm saccular aneurysm originating from a celiomesenteric trunk in a 45-year-old man. Surgical management was influenced by the location of the aneurysm, involving hepatic, splenic, and superior mesenteric arterial branches, by the young age of the patient, which made use of a synthetic graft less ideal, and by the lack of endovascular options. (J Vasc Surg Cases 2015;1:50-2.)

Visceral artery aneurysms (VAAs) are rare, occurring in 0.1% to 0.2% of the population. The most common location is the splenic artery, followed by the hepatic, superior mesenteric, and celiac arteries. A celiomesenteric trunk (CMT) is an uncommon vascular malformation in which the celiac and superior mesenteric arteries arise from a common origin on the aorta. CMT is thought to account for <1% of all visceral artery anomalies. Aneurysms of these anomalies are even rarer, with only a few dozen cases reported. Management of these aneurysms is complicated by the great number of at-risk vessels and because repair failure jeopardizes both foregut and midgut. Here, we present a case of a patient with an asymptomatic 3-cm diameter aneurysm of a CMT treated successfully with primary open surgical repair. Consent to publish was obtained from the patient.

CASE REPORT

A 41-year-old man was referred for evaluation of an asymptomatic 3-cm CMT aneurysm found on a computed tomography (CT) scan. The patient had experienced an episode of severe epigastric abdominal pain 10 years earlier, following heavy alcohol consumption, thought to be secondary to acute pancreatitis. He had been asymptomatic since then. Physical examination revealed no palpable abdominal masses or audible bruits.

The CT scan revealed a CMT with a 3-cm saccular aneurysm arising near the origin of the superior mesenteric artery (SMA). A diminutive left gastric artery arose from the aorta, but the common hepatic and splenic arteries came off the CMT. These findings were confirmed with a mesenteric angiogram (Fig 1). Endovascular repair was felt to carry excessive risk because the hepatic and splenic arteries would likely be covered by a stent. We decided that a direct autogenous reconstruction was highly preferred because it avoided the need for a conduit.

The patient underwent open surgical repair using a Mercedes incision. The duodenum and the head, neck, and proximal body of the pancreas were mobilized to allow direct anterior and posterior access to the aneurysm. The portal vein and superior mesenteric vein were controlled to allow safe retraction. The CMT was well visualized, and the aneurysm sac was delivered into the peripancreatic space inferior to the neck of the pancreas. Heparin was administered, and proximal control of the CMT and distal control of the SMA and celiac artery branches was obtained (Fig 2). The aneurysm was incised transversely and the aneurysm sac explored. The orifices to the splenic and common hepatic arteries were preserved and came off the sac immediately adjacent to the CMT, whereas the SMA came off the opposite wall of the aneurysm sac.

A primary repair was performed by direct anastomosis of the SMA at its takeoff from the sac to the combined orifice of splenic, common hepatic, and CMT. The posterior wall of the aneurysm sac was imbricated and incorporated into the posterior suture line. There was no hemodynamic instability or other reperfusion effect. A drain was left near the pancreatic body due to the extent of the dissection. A postoperative CT angiogram revealed no flow in the excluded aneurysm sac and patency of the CMT, celiac artery, and SMA (Fig 3). The patient was discharged on postoperative day 4 and experienced an uneventful recovery.

DISCUSSION

VAAs are a rare condition, with a reported incidence between 0.1% and 0.2%. Historically, the anatomic distribution of these lesions has been reported to be 60% splenic artery, 20% hepatic, 5% SMA, 4% celiac, 4% gastric and gastropiploic, and 3% jejunal, ileal, and colic arteries. Up to 22% of VAAs present with rupture, with an ensuing 8.5% mortality rate. The currently accepted threshold for treatment of an asymptomatic VAA is 2 cm. Our patient, with a 3-cm aneurysm, was deemed to be at significant risk of rupture and so warranted surgical repair despite being asymptomatic.
The visceral arteries begin forming during week 4 of embryogenesis with the creation of paired ventral segmental arteries. These segments are reduced in number by fusing until they form four single midline ventral roots connected by ventral longitudinal anastomoses. In normal development, the continuity between the third and fourth roots regresses, causing separation between the celiac trunk (roots 1-3) and the SMA (root 4). If the continuity persists, then the celiac vessels and SMA have a common origin from the aorta, which is called a CMT.1,2,7,8,11

Owing to the rarity of a CMT, the possible relationship between having this anatomical variant and developing an aneurysm is not well defined, although some have suggested that this anatomical variant may be more susceptible to developing aneurysms.1,6,8 One hypothesis is that the common origin leads to increased flow of blood across the origins of the vessels and that this may lead to aneurysm formation.3,6,8 Another theory is that a CMT may be more likely to have a congenital defect in the arterial elastic or smooth muscle layer that may lead to aneurysm formation.12 This is the proposed mechanism in patients with a persistent sciatic artery, another embryologic vascular error, who frequently develop aneurysms of those vessels.1,3,9

Surgical repair of a CMT aneurysm is inherently complex, and the choice of repair will vary with the location of the major vessels in relation to the aneurysm sac. We favored a primary repair because our patient was young and so may have had issues with longevity using a synthetic conduit. He also lived in an area remote from modern medical care.

The aneurysm was repaired primarily by creation of an end-to-end anastomosis within the aneurysm sac, functionally excluding it. Other options include resection of the aneurysm sac with reconstruction by primary end-to-end or end-to-side anastomosis.3,6,7,9,13,14 Others have also described resection of the aneurysm sac with reconstruction using a prosthetic (polytetrafluoroethylene or Dacron [DuPont, Wilmington, Del]) interposition or bypass graft.1,8,12 In rare cases, resecting the aneurysm sac or ligating its neck without vessel reimplantation may be possible if the vessels will not be compromised by the closure.2

The splenic artery can usually be ligated with impunity. Ligation of the hepatic artery as it comes off the aneurysm sac should only be considered if there is a large accessory left or right hepatic artery that will not be affected by the repair or if the gastroduodenal artery provides adequate antegrade hepatic flow. These all risk some uncertainty in hepatic perfusion, and therefore, direct hepatic arterial reconstruction is ideal.

Recently, endovascular techniques have become the preferred method to treat certain VAAs such as midspenic artery aneurysms. Endovascular repairs of SMA and celiac artery aneurysms have also been described and have...
included coiling and stenting. A literature review did not reveal any reports of endovascular repair of a CMT aneurysm, but an endovascular repair for CMT stenosis has been reported. Our patient’s anatomy was not amenable to endovascular repair given the location of the aneurysm in relation to the arterial branches. However, as endovascular techniques and stent technology continue to evolve, endovascular repair will likely become a viable option for CMT aneurysms much as it is for other VAAs today.

CONCLUSIONS

This case demonstrates several key aspects of VAA repair, including careful anatomic planning, multidisciplinary management using specialists from vascular and hepatic, pancreatic, and biliary surgery, and tailoring the approach to an individual’s anatomy and circumstance. This patient returned home to Guam after a 1-month stay in the United States and will be monitored with yearly CT angiograms for 3 years and then with less frequent examinations.

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