Giant celiac artery aneurysm

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ABSTRACT

Celiac artery aneurysms (CAAs) are rare but potentially devastating lesions. Given the high rates of mortality on rupture at large sizes, they should be treated promptly with either surgical or endovascular interventions in appropriate-risk patients. Several options exist for treatment, including surgical repair and endovascular embolization with or without stent or stent graft placement. Because of their rarity, there are few reports of successfully treated CAA lesions. Herein, we describe successful endovascular treatment of one of the largest CAAs reported in the literature. (J Vasc Surg Cases and Innovative Techniques 2019;5:447-51.)

Keywords: Visceral aneurysms; Embolization; Celiac artery aneurysm; Visceral angiography

Celiac artery aneurysms (CAAs) are exceedingly rare vascular lesions that represent <0.01% of all aneurysms.[1] Among visceral artery aneurysms (VAAs), splenic artery aneurysms are most common and account for 60% to 80% of VAAs; hepatic artery aneurysms are second in incidence, accounting for 20%. CAAs represent 4% of VAAs. However, if present, they are of clinical significance as they carry high risk for mortality on rupture. Historically, up to 87% of CAA patients presented with ruptured CAA, most of which were diagnosed at postmortem examination. Even with operative repair of rupture, mortality rate is at least 40%.[2-4] Both operative and endovascular approaches for repair of CAAs have been described, with the optimal therapeutic approach dependent on the patient's anatomy, risk factors, and underlying pathophysiologic mechanism. In this article, we present a case study featuring an otherwise healthy 87-year-old man who presented with a 10.5- × 9.7-cm CAA and underwent successful endovascular therapy. The patient consented to publication of this case.

CASE REPORT

An 87-year-old man enrolled in the Multi-Ethnic Study of Atherosclerosis (MESA) underwent scheduled non-contrast-enhanced computed tomography per study protocol. Axial imaging revealed an incidental 10.5-cm CAA with smaller aneurysms in the distal abdominal aorta and right common iliac and left internal iliac arteries. Follow-up enhanced computed tomography angiography (CTA) confirmed the presence of a 10.5- × 9.7-cm aneurysm with luminal thrombus arising from the celiac trunk. At least two outflow vessels, probably the left gastric and hepatic arteries (Fig 1), were identified. At the time of presentation, he was asymptomatic without any abdominal complaints. Given the patient's age, the large size of the lesion, and the high risk of mortality, an open repair was not offered. The patient underwent aortography and selective angiography of the celiac artery with embolization.

The procedure was performed under general anesthesia with a bilateral common femoral access approach, given the perceived difficulty of the case and anticipated case time. The patient's baseline creatinine concentration was 0.78 mg/dL with an estimated glomerular filtration rate >60 mL/min/1.73 m^2. Percutaneous bilateral common femoral artery access was used. A 6F 55-cm Ansel II sheath (Cook Medical, Bloomington, Ind) was introduced in the right femoral access. Through this, a Cobra 2 (Cook Medical) was used to engage the celiac trunk, and a microsystem consisting of an angled Maestro (Merit Medical, South Jordan, Utah) and a Headliner 16 (MicroVention, Aliso Viejo, Calif) microwire was then passed through the celiac trunk into the aneurysm. The superior mesenteric artery (SMA) was sequentially catheterized with a 5Fr Cobra 2 supported by a 45-cm Ansel II sheath from the left femoral access for the purpose of real-time visceral arteriography. Multivessel angiography was performed, revealing a large aneurysm arising from the proximal celiac trunk with three outflow vessels (splenic, left gastric, and hepatic arteries). The SMA was patent but substantially displaced with visceral vasculature draped around the aneurysm. Collateral perfusion of the hepatic, splenic, and gastric vasculature by the SMA through retrograde gastroduodenal artery (GDA) and pancreaticoduodenal arcade (PDA) flow was confirmed (Fig 2, *A*).

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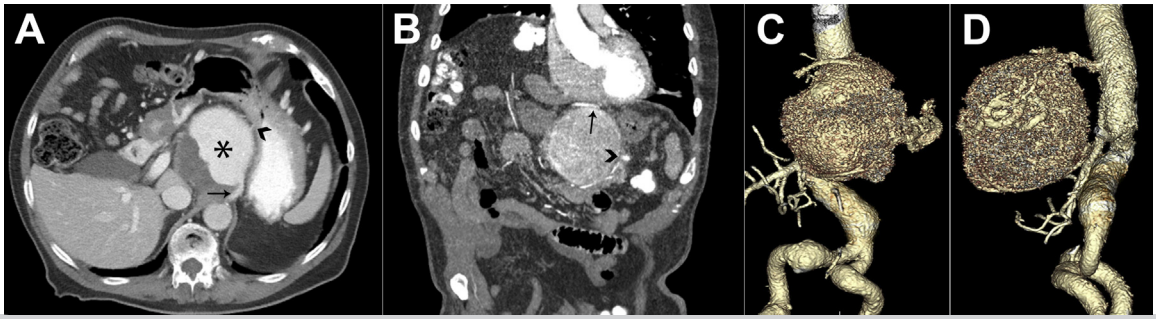

Fig 1. Computed tomography angiography (CTA) of an asymptomatic 87-year-old man. **A,** A 10.5- × 9.7-cm aneurysm (*asterisk*) with periluminal thrombus arising from the celiac trunk (*arrow*) with left gastric outflow vessel (*arrowhead*). **B,** Coronal view of aneurysm. The common hepatic artery arises from the superior aspect of the aneurysm (*arrow*). The origin of the splenic artery is not clearly visualized, but it probably arises from the aneurysm (*arrowhead*). **C,** Three-dimensional reconstruction of celiac artery aneurysm (CAA; coronal). **D,** Three-dimensional reconstruction of CAA (sagittal).

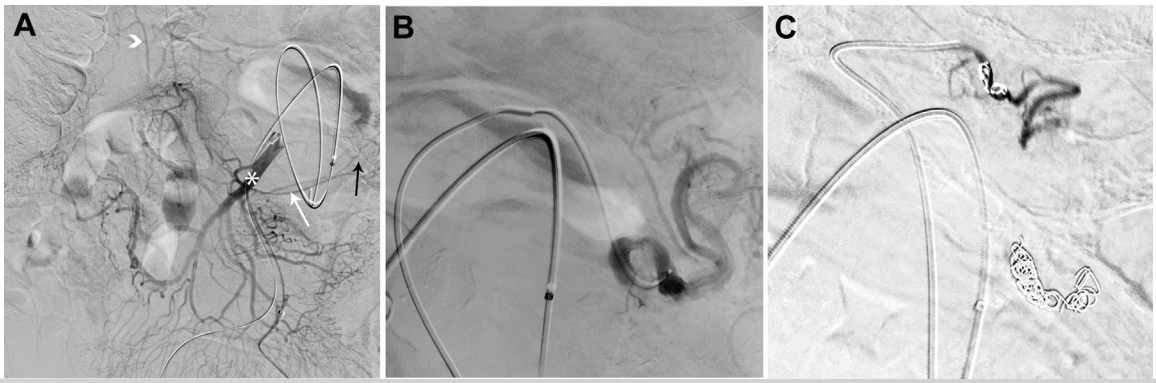

Fig 2. Angiography and embolization of two outflow tracts of celiac artery aneurysm (CAA). **A,** Superior mesenteric artery (SMA; *asterisk*) angiography demonstrating collateral perfusion of the hepatic (*arrowhead*), splenic (*black arrow*), and gastric (*white arrow*) arteries. **B,** Selective catheterization of the splenic artery outflow vessel of the CAA and subsequent coil embolization. **C,** Selective catheterization of the left gastric outflow vessel with coil embolization.

A catheter was advanced into the aneurysm and initially used to engage the outflow splenic artery. Selective arteriography demonstrated patent distal vasculature with significant tortuosity. The micro guidewire and catheter system was advanced into the splenic artery and occluded with several 6- × 7-mm micro Nester embolization coils (Cook Medical; Fig 2, *B*). Postembolization arteriography demonstrated complete occlusion of the splenic artery outflow as intended.

An angled Maestro diagnostic catheter was used to select the left gastric artery outflow. Selective angiography demonstrated patent outflow vasculature. Subsequently, multiple 3- × 7-mm Nester embolization coils were placed (Fig 2, *C*). Postembolization repeated arteriography demonstrated occlusion of the left gastric artery. A lateral projection revealed additional outflow located superiorly. By use of an angled glide catheter, this third and final outflow tract, identified as the hepatic artery, was selected (Fig 3, *A*). Angiography demonstrated filling of the proper hepatic artery retrograde from the GDA. Selective gastroduodenal arteriography was performed, revealing a large, widely patent gastroepiploic arcade with splenic artery perfusion and a large right gastric vessel arising from the proper hepatic artery with gastric perfusion. One 6- × 7-mm and two 4- × 7-mm micro Nester embolization coils were deployed in the common hepatic artery. Postembolization angiography demonstrated no inflow and significant flow stasis within the aneurysm sac (Fig 3, *B*). The catheters were withdrawn, and two 10-mm Amplatzer plugs (Abbott, St. Paul, Minn) were placed in tandem to occlude the inflow into the celiac trunk (Fig 3, *C*). Completion angiography demonstrated minimal flow into the aneurysm and reflux of contrast material into the aorta (Fig 3, *D*). Visceral arteriography from the SMA demonstrated perfusion of the hepatic, splenic, and gastric vasculature by SMA origin collaterals through PDA and GDA vessels (Fig 3, *E* and *F*). No opacification of the aneurysm was seen. The case was performed in a staged fashion, and the patient was discharged home between interventions to avoid excessive radiation doses. The splenic and gastric branches were embolized first in a single session. The hepatic outflow and celiac trunk were embolized 4 days later. Total fluoroscopy time was 120 minutes, total fluoroscopy dose was 20,495 mGy, and 300 mL of intravenous contrast material was used.

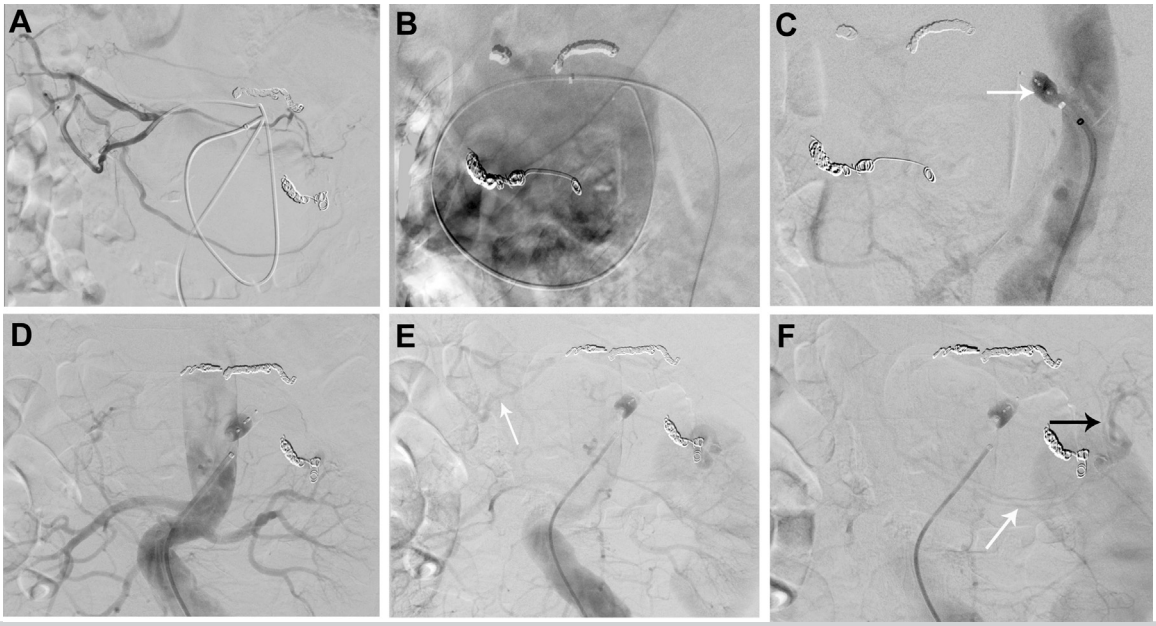

Fig 3. Angiography and embolization of hepatic outflow tracts and celiac inflow of celiac artery aneurysm (CAA). **A,** Selective catheterization and coil embolization of hepatic vessel outflow. **B,** Contrast material injected into the aneurysm sac reveals significant flow stasis and successful embolization of outflow tracts. **C,** Two Amplatzer plugs (*arrow*) used to occlude inflow into the CAA at the celiac trunk. **D,** Completion angiography demonstrates no inflow of contrast material into the CAA and reflux into the distal aorta. **E,** Visceral arteriography demonstrates collateral flow to the hepatic (*arrow*) vasculature. **F,** Arteriography demonstrating post-embolization collateral flow to the gastroepiploic (*white arrow*) and splenic (*black arrow*) arteries.

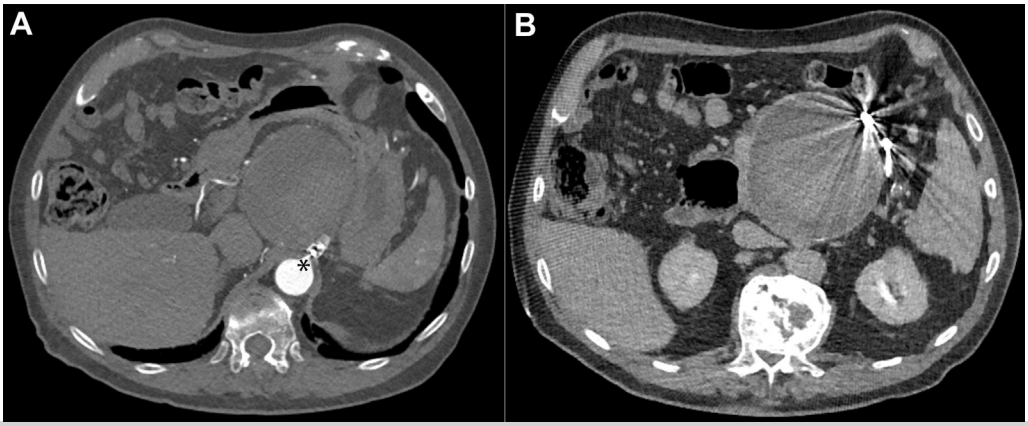

Fig 4. Computed tomography angiography (CTA) at 1-month follow-up. **A,** Arterial-phase image demonstrating no contrast material flow into the aneurysm sac from the celiac trunk (*asterisk*). **B,** Venoportal-phase image demonstrating no contrast enhancement of the aneurysm sac.

Diagnostic CTA 1 month postoperatively demonstrated no increased attenuation on post-contrast-enhanced images, consistent with persistent aneurysm exclusion and technically successful treatment (Fig 4).

DISCUSSION

Despite their rarity, the increasing use of cross-sectional imaging has greatly increased the recognition of CAAs since they were first described.[5] Historically, before the widespread use of endovascular techniques, this disease process carried an operative mortality rate of 40% for ruptured aneurysms and 5% for nonruptured aneurysms.[3,4] Of all reported cases of CAAs, rupture incidence was as high as 87% in the early 20th century, and diagnosis was often made at autopsy. This has improved to 0% to 7% in recent studies owing to early diagnosis and intervention.[5,6] Together, these data argue for swift management on discovery of a CAA. Proposed

indications for intervention include symptomatic lesions, rapidly increasing size, and aneurysms >20 to 24 mm or three times the normal reference vessel diameter.[7] These recommendations are based on rupture rates of 5% in aneurysms measuring 15 to 22 mm, which increases exponentially to 70% at diameters >30 mm.[8] Symptomatic CAAs often are manifested with vague complaints, including abdominal pain, nausea, vomiting, appetite loss, "food fear," and, rarely, a palpable mass causing compressive or obstructive disease.[9] In patients without rupture, 5% to 17% are entirely asymptomatic and diagnosed incidentally, as seen in this case study.[6] A recent study of VAAs reported a mean CAA size of 1.53 cm in 63 patients.[5] The patient in this study had no abdominal complaints despite harboring one of the largest CAAs reported to date. Before widespread treatment of syphilis was available, the etiology of one-third of these lesions was infectious, probably contributing in part to higher rupture and mortality rates.[4] In the modern day, atherosclerotic degeneration is the most common cause of aneurysm formation.[9] Interestingly, the patient in this study did not suffer from extensive atherosclerosis and had few risk factors other than advanced age, leaving the true etiology of such an impressively large lesion elusive. CAAs are frequently accompanied by aneurysms in the abdominal aorta (18%) and the splanchnic vasculature (up to 50%), thereby making solitary celiac lesions exceptionally rare.[4,10]

Whereas both surgical and endovascular approaches have been described, the optimal approach is dependent on factors including the patient's anatomy, risk factors, and underlying pathophysiologic mechanism. Operative treatment is aneurysmorrhaphy with closure or resection and revascularization of peripheral vessels that bifurcate from the aneurysm sac using either autogenous vein or prosthetic grafts.[11] When the aneurysm involves the celiac artery trifurcation, the surgical approach is especially challenging, with a mortality rate as high as 5%.[12,13]

Endovascular approaches, including stent graft exclusion and embolization, are preferred in the setting of advanced age, in patients with multiple comorbidities, or in the setting of unfavorable operative anatomy. Whereas there have been case studies detailing repair using endovascular stenting, the absence of an optimal landing zone, significant tortuosity, and evidence of an infected sac exclude the use of a stent.[14,15] In this study, we elected exclusion of the aneurysm by embolization of inflow and outflow vessels. It is important that care is taken to address all outflow vasculature, as retrograde flow through these vessels may continue to expand the aneurysm sac despite adequate inflow exclusion. The proximal neck of the aneurysm originated at the orifice of the celiac trunk, which raised the concern that embolization at this location to exclude inflow may result in visceral ischemia. Adequate collaterals from the SMA through PDA and GDA vessels were identified in this patient before intervention, thereby decreasing the risk of celiac artery distribution ischemia, and postocclusion images demonstrated preserved flow to foregut visceral organs through these collaterals. If adequate collateralization was not appreciated in all three vessels, this patient would be deemed a poor embolization candidate. A stent graft-assisted approach could be attempted to preserve flow but would be technically challenging, given the size of the aneurysm sac and the involvement of multivessel outflow. At 1 month, the aneurysm remained successfully excluded.

Stent-assisted coiling has also been described as an effective technique, especially in wide-neck aneurysms or when flow through the native artery must be preserved.[16] In the case presented herein, effective aneurysm exclusion was achieved with coiling alone because of a relatively narrow neck of inflow and outflow vessels with appropriate collateralization to distal vessels. A second approach using flow diversion devices has also been described; however, unlike coil embolization, these devices require time for the aneurysm to thrombose, which may be disadvantageous in larger sac sizes.[17-19]

This patient was treated without coil packing of the aneurysm. Previous studies of aneurysm packing demonstrate that in partially thrombosed aneurysms, even sufficient packing does not preclude recanalization of the aneurysm because factors other than compaction, such as thrombus resolution and migration of coils into the thrombus, cause the aneurysm to reopen over time.[20] Furthermore, a study of cohorts of smaller aneurysms showed that coil compaction and recanalization after coil packing occur most often after low-density packing (<24%) and in patients with larger aneurysms (as defined by >2 cm), as higher rates of packing become difficult to achieve.[21] In a giant size aneurysm with intrasac thrombus, such as the one presented here, sufficient coil packing to prevent revascularization would result in tremendous additional cost, time, and radiation exposure to the patient with marginal benefit.

A limitation of this report is the use of CTA imaging after the procedure and the absence of longer term follow-up. The presence of coil artifact may hinder detection of aneurysm revascularization. However, previous studies comparing imaging modalities with less artifact effect, such as magnetic resonance angiography and duplex ultrasound, are comparable to CTA in evaluating these aneurysms after coil embolization. Duplex or contrast-enhanced ultrasound is noninvasive and can also be used to monitor VAAs but can be limited by the operator's experience, arterial wall calcification, coil artifact, and air in the digestive tract. CTA remains the most widely used modality in these cases. Whereas selective angiography is the optimal method of assessment, CTA foregoes any morbidity associated with invasive procedures and is both more practical and feasible.[22,23]

CONCLUSIONS

We present here an exceptionally large 10.5-cm CAA that was successfully treated with embolization. Whereas more data with long-term follow-up are needed to understand the optimal approach to patients with varying anatomy, aneurysm sizes, and underlying risk factors, we show here that coil embolization of inflow and outflow vessels even without coil packing is an effective technique for large aneurysm exclusion.

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