A 54-year-old woman had chronic intermittent abdominal pain for nearly a decade. Her past medical history was significant for Charcot Marie-Tooth syndrome with mild peripheral neuropathy. Her family history was significant because both her mother and uncle died from ruptured cerebral aneurysms, and her cousin died from a ruptured abdominal aortic aneurysm. She worked actively as a law professor and was able to exercise and perform household duties without limitation. The patient had no previous operations. On examination, a nontender, pulsatile mass in the epigastrium was palpable. She had no other physical examination abnormality including abdominal bruit, skeletal or joint abnormality, or evidence of embolic lesion. Pulse examination was normal in all extremities. Genetic testing failed to reveal a discrete abnormality.

Computed tomography (CT) imaging was performed with intravenous contrast and revealed a 3.5- X 2.9-cm visceral artery aneurysm in the peripancreatic arcade (Figure 1) with an occluded celiac axis. Multiple options were considered:

1. Celiac axis revascularization followed by operative aneurysm resection.
2. Operative aneurysm resection or ligation with celiac axis revascularization depending on liver perfusion assessment.
3. Coil/glue embolization of the aneurysm sac.

Figure 1. A single axial image from a CT scan obtained before treatment (A). Note the large contrast-filled aneurysm (larger than the aorta) with partial wall calcification. The rest of the aorta was normal. A three-dimensional CT rendering of images documents proximal inflow via the superior mesenteric artery and outflow via the peripancreatic arcade (B).

Figure 2. An aortic angiogram reveals a large aneurysm arising from the superior mesenteric artery (A). The celiac axis is occluded. A selective arteriogram with catheterization of the aneurysm reveals that the celiac axis territory is filled via the gastroduodenal artery (GDA) (B). This precluded simple ligation/endovascular ablation of the aneurysm.
4. Coil/glue embolization of the aneurysm sac with celiac axis recanalization, angioplasty, and stenting.

We considered all options with the patient and decided to proceed with option 4. Femoral access was gained under local anesthesia with plans for brachial access after the diagnostic imaging. Angiography confirmed an aneurysm with inflow via the superior mesenteric artery (SMA) (Figure 2A) and outflow via the GDA supplying both the hepatic and splenic arteries (Figure 2B). Also, multiple hypertrophied collaterals around the head of the pancreas were visualized. During performance of selective cannulation of the SMA and catheter exchange, the patient developed sudden onset of abdominal pain.

Angiography confirmed an acute SMA occlusion. This was secondary to a short dissection flap in the SMA beyond the arterial inflow to the aneurysm and peripancreatic arcade (Figure 3A). Of note, given the patient's celiac axis occlusion, this resulted in profound visceral ischemia. We traversed the occlusion and performed balloon angioplasty for 90 seconds under heparinization, which restored normal prograde blood flow to the SMA (Figure 3B) and prompted resolution of the abdominal pain. The angiographic procedure was aborted. The patient was observed in an intensive care unit setting but did not have any hemodynamic instability or evidence of ongoing bowel ischemia.

Approximately 1 week later, the patient underwent an aorta-to-hepatic artery bypass with reversed greater saphenous vein (Figure 4A). Vein was chosen as conduit because of the risk of bowel/pancreatic injury. The aneurysm was intimately involved with the head of the pancreas. Extensive mobilization of the duodenum and pancreas was performed, as well as control of the infrapancreatic SMA. Interestingly, no evidence of dissection or vessel abnormality was discernable on external evaluation of the SMA. After clamping the SMA inflow and GDA outflow, the aneurysm was opened posteriorly. There were multiple collateral branches resulting in back bleeding despite SMA control. An endoaneurysmorrhaphy was performed with all feeding branches ligated, and the sac obliterated (Figure 4B). The patient was discharged without incident and went back actively work-
ing as a law professor in 6 weeks. CT imaging 6 months postoperatively showed a widely patent bypass without any aneurysm recurrence (Figure 5). The patient is doing well 1 year after the procedure and will have lifelong follow-up for aneurysm recurrence.

CONCLUSION

Visceral aneurysms are rare, and the pathophysiology remains poorly defined in contrast to other aneurysms. With large aneurysms, a focused abdominal examination can be revealing. Endovascular therapy with ablation of visceral aneurysms has been shown to be safe and durable in midterm follow-up. In this case, despite gentle catheter manipulation, a life-threatening SMA occlusion occurred, which may have been due to this patient’s fragile arteries. The remarkable family history indicates some arterial pathology despite the unrevealing genetic testing and microscopic pathological examination of the surgical specimen.

Surgical therapy for hepatic, splenic, and renal artery aneurysms has included resection of the aneurysm only or resection combined with surgical revascularization of the distal arterial bed. In this patient with a giant aneurysm, multiple collaterals, and an occluded celiac axis, restoration of the celiac axis blood flow before aneurysm resection was needed. Furthermore, with the aneurysm intimately involved with the head of the pancreas, endoaneurysmorrhaphy rather than aneurysm resection allowed ablation of the sac without damage to the pancreas or duodenum. Given the variability and anatomic constraints of visceral artery aneurysms, patients benefit from both endovascular techniques in selected cases and open vascular approaches for durable aneurysm treatment.

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