Spontaneous dissection of the celiac artery: A case report

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Spontaneous dissection of visceral arteries is rare in the absence of concurrent dissection of the aorta, iatrogenic injury from instrumentation, or trauma. We describe a spontaneous dissection of the celiac artery that was identified by computed tomographic scan in an otherwise healthy man with acute onset abdominal pain and no identifiable causes of dissection. The patient was successfully managed medically. Although endovascular treatment or surgical intervention is the procedure of choice for complicated cases, medical management with close observation is an acceptable management strategy for stable, uncomplicated cases of spontaneous celiac artery dissection. (J Vasc Surg 2007;45:1256-8.)

Spontaneous dissections of the visceral arteries and, more specifically, the celiac artery are extremely rare. Descriptions in the literature of iatrogenic injury to the celiac artery during transcatheter instrumentation and also in instances of abdominal trauma have been published. Only 55 cases of visceral artery dissection have been published since the first reported dissection in 1947. These dissections were more commonly associated with the superior mesenteric artery (SMA) and only rarely with the celiac artery. In 2000, Matsuo et al reported that only four cases of isolated celiac artery dissection had been reported in the literature. Since their report, we have been able to find seven other reports of spontaneous celiac artery dissection.

The cases that have been reviewed demonstrate similar clinical presentations and radiographic findings. Abdominal pain is the most common presenting symptom for patients with celiac artery dissections. In addition, intramural thrombus and splenic infarctions accompany the radiographic findings of celiac artery dissection. Treatments for visceral artery dissections have ranged from surgery to transcatheter embolization to close observation.

CASE REPORT

In June 2005, an otherwise healthy 53-year-old white man presented to the emergency department of an outside hospital with complaints of lower abdominal pain and nausea. The patient had no history of diabetes mellitus, hypertension, hyperlipidemia, heart disease, or smoking.

A computerized tomography angiogram (CTA) of the abdomen and pelvis revealed a normal caliber aorta. The origin of the celiac artery appeared patent; however, approximately 1 cm from the origin of the celiac artery, focal widening from 8 mm to 12 mm in diameter, with intimal flap and focal thrombus formation, was seen (Fig 1). The dissection produced 90% stenosis of the celiac artery. Focal dissection of the splenic artery was also seen, and a large splenic infarct involving approximately 50% of the splenic parenchyma was noted.

The patient was started on enoxaparin (60 mg subcutaneously every 12 hours) and his pain was managed with intravenous meperidine. His abdominal pain subsided, but he continued to have intermittent left-sided abdominal pain likely associated with the splenic infarct.

The patient’s laboratory data revealed leukocytosis with a peak of 14,100/μL and significantly elevated C-reactive protein. A repeat CTA 3 days after admission revealed a slight decrease in the amount of thrombus in the celiac artery. Again, distal patency was noted.

The patient was discharged home on hospital day 4 on enoxaparin, warfarin, and aspirin. The enoxaparin was continued until the warfarin dose was therapeutic. He was subsequently seen in vascular surgery consultation at our tertiary care center. The patient’s history was reviewed in detail, and it was decided to monitor the patient with close observation.

The patient continued on warfarin anticoagulation to keep his international normalized ratio (INR) at 3, and he had repeat CTAs to evaluate his mesenteric vasculature. We recommended warfarin for 6 months and repeat CTA at 6 months, 1 year, and yearly thereafter. He clinically continues to be free of abdominal pain.

Interval CTAs at 3 weeks, 2, 3, and 6 months showed resolution of the celiac thrombus, persistence of intimal flap with distal patency of the celiac and splenic arteries, and improvement in the splenic infarct. The patient’s most recent CTA at 9 months after the initial presentation revealed stable dissection of the celiac and splenic arteries, with persistent patency of the true lumens (Fig 2 and Fig 3).

DISCUSSION

Arterial dissection is defined as cleavage of two layers of the arterial wall caused by intramural hematoma. A spontaneous dissection of a visceral artery without associated aortic dissection is extremely rare. Extra-aortic dissections in order of decreasing frequency include the renal artery, coronary artery, cerebral artery, carotid artery, vertebral artery, and visceral arteries.

Approximately one half of all dissections involving visceral arteries are asymptomatic; occasionally, patients present with intestinal angina or hemorrhage. More
atypical symptoms have been described, depending on the location of the lesions, including malabsorption in association with dissection of the SMA, jaundice in association with dissection of the celiac and hepatic artery, or pancreatitis with involvement of the pancreatic artery.4,12-15

Causes of arterial dissection are usually atherosclerosis, trauma, iatrogenic, pregnancy, syphilis, polyarteritis nodosa, fibromuscular dysplasia, cystic medial degeneration (Marfan syndrome), and other congenital disorders of the vascular wall (eg, Ehlers-Danlos syndrome).4 Our patient demonstrated none of the these risk factors. In addition, he had no history of risk factors for atherosclerosis.

Radiographic work-up includes Doppler color ultrasound imaging and abdominal contrast-enhanced CT. Definitive diagnosis of celiac dissection is supported by selective arteriography, which also allows precise determination of the extent of involvement especially of stenotic lesions and evaluation of collateral circulation.4,16 Evaluation of the mesenteric vasculature with angiography is helpful before surgery.

In the evaluation of a patient with a newly diagnosed celiac artery dissection and progressive abdominal pain and tenderness, particularly with unstable vital signs and falling hemoglobin and hematocrit levels, one must consider ongoing dissection, aneurysm formation, hemorrhage, and ischemic bowel in the differential diagnosis. With the possible exception of ischemic bowel, a CT scan can confirm all of the above entities, and all require immediate intervention. Because of the rich mesenteric blood supply from the SMA, acute arterial occlusive complications of celiac artery dissection are often well tolerated.

Glehen et al3 suggest a treatment strategy for the acute phase of isolated symptomatic celiac artery dissections. Surgery is indicated for management of fusiform or sacculated aneurysm, occlusive lesions jeopardizing the lower digestive tract, or arterial complications, such as rupture or liver ischemia. As was the case with our patient, they further suggest that conservative medical treatment can be proposed for patients with limited dissection in whom serial

Fig 1. Computed tomography angiogram demonstrates celiac artery dissection with intramural thrombus and focal stenosis (arrow).

Fig 2. Computed tomography angiogram demonstrates stable celiac artery dissection with resolution of intramural thrombus (arrow).

Fig 3. Computed tomography angiogram demonstrates stable splenic artery dissection (arrow).
examinations have demonstrated no evidence of rupture or expansion. For patients with persistent pain, uncontrolled hypertension, hypotension, or multifocal lesions, they suggest surgical intervention.

Surgery seems to be the most reliable means of dealing with acute complications of celiac artery dissections, although embolization is also an option, particularly for those patients who are higher surgical risks. Embolization does require careful assessment of collateral blood supply.

Another possible alternative to initial management of celiac artery dissection is balloon fenestration. So et al reported recanalization of the celiac artery with balloon fenestration after iatrogenic dissection of the celiac artery during transcatheter chemoembolization for hepatocellular carcinoma. The patient had patent celiac and hepatic arteries 18 months after intervention.

Finally, endovascular stent graft placement has been performed successfully for visceral artery aneurysms. Intervention is typically suggested for aneurysmal dilatation >1.5 to 2.0 cm.

CONCLUSIONS

Isolated dissections of the visceral arteries, particularly of the celiac artery, are extremely rare. They typically present with abdominal pain, as was the case with our patient, and are occasionally associated with hemorrhage. Although treatment strategies are somewhat unclear, medical management and close observation is appropriate for uncomplicated lesions. Surgical management is the preferred treatment for those patients who have associated complications or persistent or recurrent symptoms. For those patients who are not good surgical candidates, endovascular techniques such as selective embolization and stent grafts provide other potential treatments for celiac artery dissection.

REFERENCES


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