

THE CLINICAL PICTURE

FAIZA KHALID, MD

Clinical Instructor of Medicine,
Department of Internal Medicine,
University Hospitals Cleveland
Medical Center, Beachwood, OH

SHIKHA GUPTA, MD

Assistant Professor of Medicine,
Department of Pulmonary and Critical
Care Medicine, University of Pittsburgh
Medical Center, Pittsburgh, PA

Aortic dissection presenting as ischemic limb

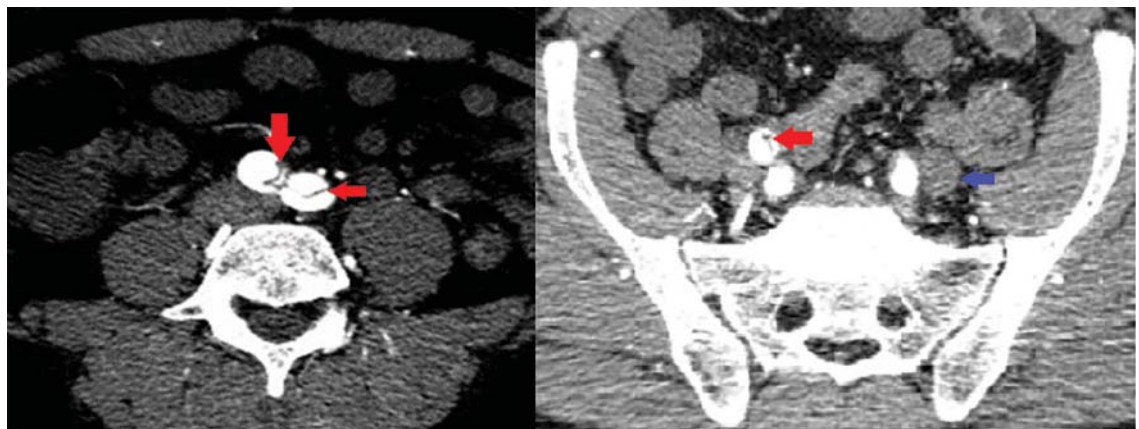


Figure 1. Left, computed tomography of the abdomen with intravenous contrast showed dissection of both the left and right common iliac arteries (red arrows). Right, dissection of the right external iliac artery (red arrow) and occlusion of the left external iliac artery (blue arrow).

With surgical repair and blood pressure control, he recovered and left the hospital 1 week later

A 40-YEAR-OLD MAN with a history of hypertension and alcohol abuse presented with acute onset of mild chest tightness, left leg pain, and increasing agitation, which prevented us from obtaining additional meaningful information from him.

On admission, his heart rate was 120 beats per minute, blood pressure 211/122 mm Hg, respiratory rate 18 per minute, and oxygen saturation 92% on room air. Given his history of alcohol abuse, we checked his blood ethanol level, which was less than 0.01%, well below the legal limit for intoxication.

We gave the patient intravenous lorazepam for possible alcohol withdrawal and started labetalol by intravenous infusion to lower his blood pressure.

On physical examination, his left lower extremity was cold and without pulses, including the femoral pulse. Suspecting acute arterial thrombosis, we ordered immediate computed

tomographic (CT) angiography of the abdomen and pelvis with left lower extremity run-off. The images showed dissection of the abdominal aorta with extension to both the left and right common iliac arteries and the origin of the right external iliac artery. There was resultant occlusion of the left external iliac artery (**Figure 1**).

Immediate CT angiography of the chest was then performed, which revealed dissection of the thoracic aorta as well, starting superior to the aortic valve annulus and involving the ascending aorta, aortic arch, and the entire descending thoracic aorta (**Figure 2**).

The patient underwent emergency surgical repair of the aortic root, ascending aorta, and aortic arch. Residual dissection of the descending aorta was managed conservatively with blood pressure control using intravenous labetalol initially, which was then switched to oral carvedilol, and the pulses returned in his left lower extremity. He had an unremarkable

doi:10.3949/ccjm.85a.17122

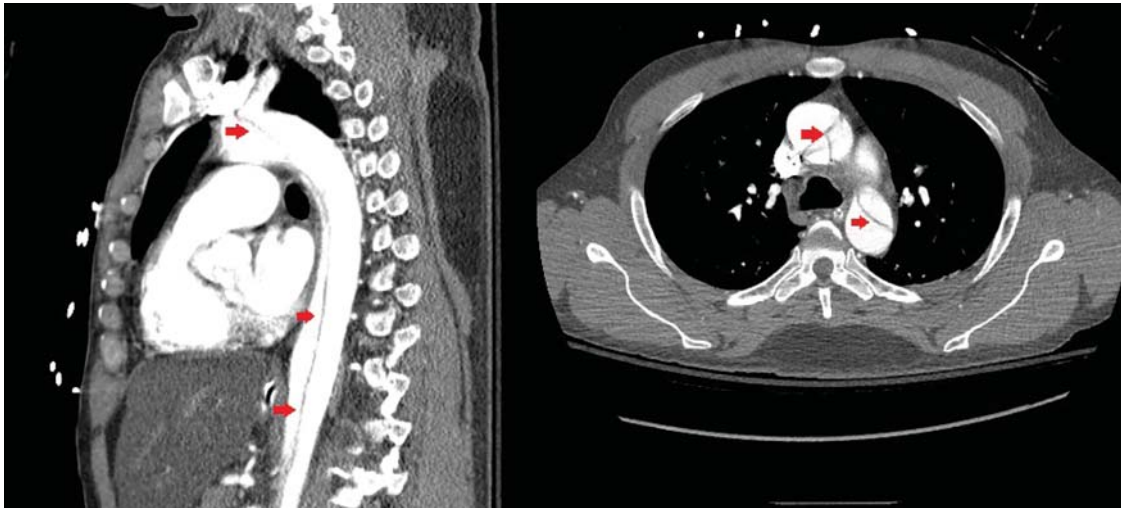


Figure 2. Computed tomography of the chest with intravenous contrast (left) showed dissection of the aortic arch, descending thoracic aorta, and abdominal aorta (red arrows). At right, dissection of the ascending and descending aorta (red arrows).

postoperative recovery and was discharged after 1 week.

■ AORTIC DISSECTION AND MALPERFUSION SYNDROME

Aortic dissection is most often associated with acute onset of sharp chest pain and upper back pain. On rare occasions, it can have an atypical presentation such as stroke, paraplegia, mesenteric ischemia, or lower limb malperfusion.¹

Extension of aortic dissection into the iliac and femoral arteries can cause impaired or absent blood flow to the lower extremity. These pulse deficits are a part of limb malperfusion syndrome. Symptoms of malperfusion syndrome vary greatly and depend on the vessels involved. Malperfusion of the branches of the aortic arch can result in stroke or altered sensorium. Compromise of intra-abdominal vessels due to dissection can involve the mesenteric bed, the renal arteries, or both, resulting in laboratory derangements such as lactic acidosis and renal failure.

How aortic dissection and malperfusion syndrome occur

Over time, shear forces on the aortic wall result in degeneration of the tunica intima and media. Dissection occurs when deterioration of the intima causes propagation of blood through a cleavage plane into the outer portion of the diseased media, forming a false lumen.

Anterograde or retrograde progression of dissection depends on the balance of the pressure gradient between true and false lumens.² With every systolic ventricular contraction, a fluid and pressure wave travels down both lumens (true and false). However, the pressure gradient between the false and true lumens allows the more pliable intimal flap to bulge into the true lumen and ostia of branch vessels, resulting in static or dynamic obstruction.

Static obstruction occurs when the false lumen projects completely into the branch vessel and there is resultant thrombosis. As the name implies, dynamic obstruction is intermittent and is responsible for 80% of the cases of malperfusion syndrome.³ Dynamic obstruction has 2 distinct mechanisms: hypoperfusion through the true lumen due to impaired flow, and prolapse of the false lumen into a branch vessel.

Factors that exacerbate hypoperfusion through the true lumen and make obliteration by the false lumen more likely include large circumference of the dissected aorta, rapid heart rate, and high systolic pressure.⁴ Therefore, it is important to control the heart rate and blood pressure using beta-blockers in cases of aortic dissection with malperfusion syndrome. This treatment may resolve the dynamic obstruction through expansion and resumption of perfusion through the true lumen.⁵

Extension of aortic dissection into the iliac and femoral arteries can cause impaired or absent blood flow to the lower extremity

MANAGEMENT OF MALPERFUSION SYNDROME

Aortic dissection can be classified as either Stanford type A (involving the ascending aorta) or type B (involving the descending aorta). Type B dissection associated with malperfusion syndrome is termed “complicated” type B aortic dissection. Our patient had both Stanford type A and complicated type B aortic dissection.

Unlike type A aortic dissection, which requires definitive open surgical repair, complicated type B aortic dissection occasionally responds to medical management alone. A plausible explanation for resolution of limb malperfusion with optimal blood pressure

control is expansion of the true lumen and obliteration of the false lumen, as was likely the case in our patient.

In most cases, however, limb malperfusion persists despite optimal medical management. In such patients, endovascular graft stenting or open surgical repair may be needed. Open surgical repair procedures like bypass grafting or surgical fenestration are associated with significant rates of mortality and morbidity.⁵ Therefore, an endovascular approach rather than conventional surgical repair for complicated type B aortic dissection is advocated after optimal medical management.⁶ Endovascular repair also promotes favorable aortic remodeling without the morbidity associated with open surgical repair.

REFERENCES

1. Namana V, Balasubramanian R, Kariyanna PT, Sarasam R, Namana S, Shetty V. Aortic dissection with hemopericardium and thrombosed left common iliac artery presenting as acute limb ischemia: a case report and review. *Am J Med Case Rep* 2015; 3(10):338–343. doi:10.12691/ajmcr-3-10-9
2. Crawford TC, Beaulieu RJ, Ehlert BA, Ratchford EV, Black JH 3rd. Malperfusion syndromes in aortic dissections. *Vasc Med* 2016; 21(3):264–273. doi:10.1177/1358863X15625371
3. Williams DM, Lee DY, Hamilton BH, et al. The dissected aorta: percutaneous treatment of ischemic complications—principles and results. *J Vasc Interv Radiol* 1997; 8(4):605–625. PMID:9232578
4. Chung JW, Elkins C, Sakai T, et al. True-lumen collapse in aortic dissection: part II. Evaluation of treatment methods in phantoms with pulsatile flow. *Radiology* 2000; 214(1):99–106. doi:10.1148/radiology.214.1.r00ja3499
5. Gargiulo M, Bianchini Massoni C, Gallitto E, et al. Lower limb malperfusion in type B aortic dissection: a systematic review. *Ann Cardiothorac Surg* 2014; 3(4):351–367. doi:10.3978/j.issn.2225-319X.2014.07.05
6. Dake MD, Kato N, Mitchell RS, et al. Endovascular stent-graft placement for the treatment of acute aortic dissection. *N Engl J Med* 1999; 340(20):1546–1552. doi:10.1056/NEJM199905203402004

ADDRESS: Faiza Khalid, MD, Clinical Instructor of Medicine, Department of Internal Medicine, University Hospitals Cleveland Medical Center, 2663 South Green Road, Beachwood, OH 44122; Faizakhalid24@gmail.com