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Evolving Treatments for Aortic Dissection

A review of classifications and endovascular repair for this challenging clinical presentation.

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Aortic dissection is the most common aortic catastrophe, with dynamic pathology and high rates of morbidity and mortality if left untreated. Successful management mandates high clinical suspicion, prompt diagnosis, accurate assessment of aortic anatomy, and long-term surveillance. Patients with uncomplicated dissections have traditionally been treated with medical therapy that involves managing blood pressure and controlling pain. Operative intervention is reserved for complicated scenarios, including rupture, malperfusion syndrome, rapidly enlarging aortic diameter, progression of the dissection, intractable pain, and/or hypertension. In this article, we present the case of an 82-year-old woman with a Stanford type B (DeBakey type IIIB) aortic dissection who failed medical therapy and required aortic endografting.

CASE STUDY

An 82-year-old woman with a past medical history of hypertension and breast cancer presented to an outside hospital with back and abdominal pain. A CT angiogram (CTA) showed a Stanford type B (DeBakey type IIIB) aortic dissection with an entry tear in the proximal descending thoracic aorta (Figure 1A and 1B). She was transferred to our facility for higher-level care. Upon arrival, she was admitted to the intensive care unit and treated with antiimpulse medical therapy. Her back and abdominal pain initially resolved, but she was unable to tolerate a regular diet. A review of her CTA showed a celiac artery originating primarily from the false lumen without any appreciable flow (Figure 2A). The superior mesenteric artery was patent (Figure 2B). The patient complained of vague postprandial abdominal pain despite adequately controlled blood pressure. We diagnosed her with malperfusion secondary to mesenteric compromise and offered her a thoracic endovascular aortic repair (TEVAR).

After induction of general anesthesia, we performed bilateral percutaneous femoral artery cannulation. Intravascular ultrasound (IVUS) confirmed that the guidewire was in the true lumen. Interrogation of the aorta revealed a dissection extending from the left subclavian artery (LSA) to the aortic bifurcation, with a proximal entry tear 2 cm distal to the takeoff of the LSA. Interrogation of the mesenteric vessels revealed a compressed true lumen at the level of the celiac artery (Figure 3A). The proximal landing zone

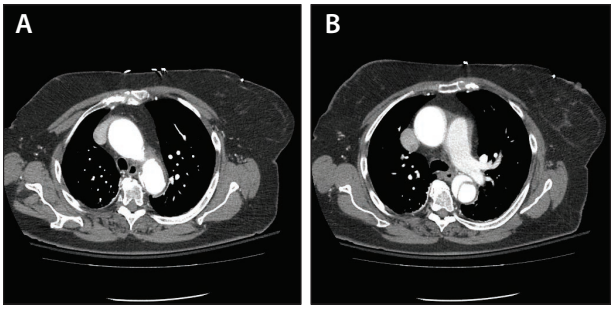

Figure 1. A CTA showing a Stanford type B (DeBakey type IIIB) aortic dissection with an entry tear in the proximal descending thoracic aorta (A) and the subsequent dissected descending aorta with a true and false lumen (B).

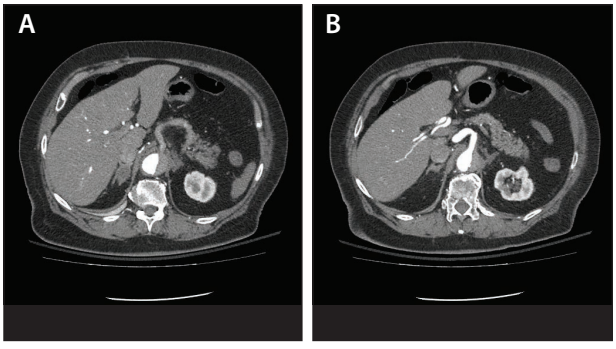

Figure 2. A CTA showing a celiac artery originating primarily from the false lumen without any appreciable flow (A). The superior mesenteric artery was patent (B).

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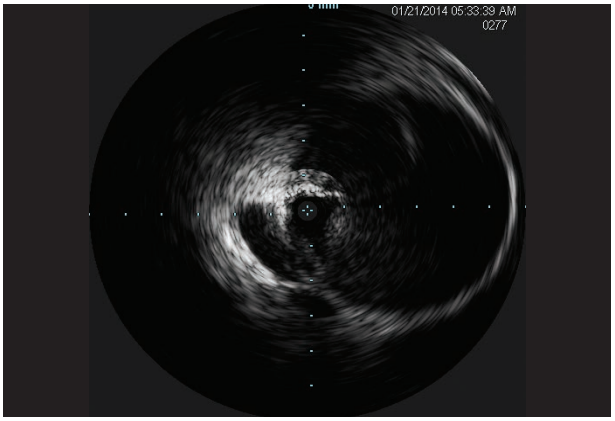

Figure 3. A compressed true lumen at the level of the celiac artery.

was selected, and the neck diameter was measured with IVUS. A 32-mm X 20-cm Medtronic Valiant stent graft was selected for placement with < 10% oversizing. The device was advanced into the proximal descending thoracic aorta over a Lunderquist extra-stiff guidewire (Cook Medical). A thoracic angiogram was obtained, and the location of the LSA was identified (Figure 4A). The device was deployed immediately distal to the takeoff of the LSA. A postdeployment angiogram showed successful coverage of the proximal entry tear (zones 3 to 5) and minimal flow in the false lumen (Figure 4B). An abdominal aortogram after the deployment of the stent graft confirmed flow to the mesenteric and renal vessels (Figure 4C). Postdeployment IVUS confirmed reexpansion of the true lumen and patency of the celiac artery (Figure 5). Femoral artery puncture sites were treated with percutaneous closure.

AORTIC DISSECTION CLASSIFICATIONS

Stanford and DeBakey

In the United States, there are roughly 5 to 30 cases of aortic dissection per million people each year.[1] It occurs more frequently among men, and its incidence peaks in the sixth and seventh decades of life. Established risk factors for aortic dissection include hypertension, smoking history, connective tissue disorders (eg, Marfan syndrome, Loeys-Dietz syndrome, Ehlers-Danlos syndrome), preeclampsia, and cocaine abuse. Aortic dissection is pathologically characterized by a tear in the intima, resulting in blood dissecting into the medial layer of the aortic wall and creation of a false lumen.

The original description by DeBakey in 1965 defined type I aortic dissections as those originating in the ascending aorta with further involvement of the descending thoracic and abdominal aorta.[2] Type II dissections differ in that they are confined to the ascending aorta. Type III dissections are distinct from proximal aortic dissections, originate at the level of the subclavian artery, and are either entirely confined to the descending thoracic aorta (type IIIA) or are found to extend to the abdominal aorta (type IIIB). This classification was further refined in 1970, with the introduction of the Stanford classification.[3] Proximal (DeBakey types I and II) aortic dissections are classified as Stanford type A dissections. Distal aortic dissections (DeBakey type III) are described as Stanford type B dissections.

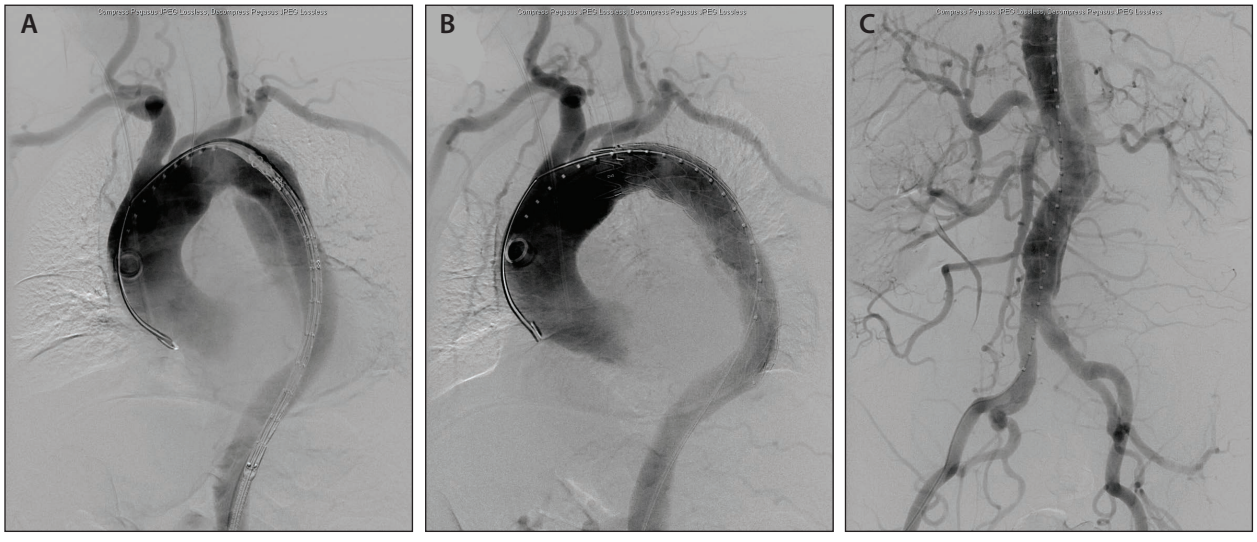

Figure 4. The location of the LSA is identified via a thoracic angiogram (A). A postdeployment angiogram showing successful coverage of the proximal entry tear (zones 3 to 5) and minimal flow in the false lumen (B). An abdominal aortogram after the deployment of the stent graft confirms flow to the mesenteric and renal vessels (C).

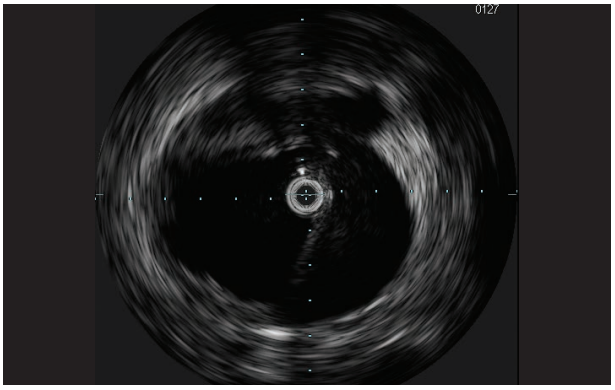

Figure 5. A postdeployment IVUS confirming reexpansion of the true lumen and patency of the celiac artery.

Although the Stanford classification is the more widely used description in the literature, the DeBakey classification has re-emerged in recent years as a more applicable schema as endovascular strategies for treatment of distal aortic dissection have rapidly evolved. This is particularly important when considering the pathophysiology of aortic dissection. Proximal (DeBakey types I and II) aortic dissections typically occur because stress on the aortic wall is greatest in the ascending aorta. Proximal descending thoracic aortic dissection occurring within 2 cm of the origin of the LSA represents the second most common pattern of dissection, as this segment of the aorta is similarly exposed to a high degree of wall stress. Understanding of this location and its relationship to the LSA are essential when selecting a proximal landing zone for endovascular treatment of dissection.

Acute and Chronic

Aortic dissections are further classified according to symptom onset. Historically, *acute* aortic dissections were defined as those dissections that occurred within 14 days of symptom onset; dissections lasting longer than 14 days were defined as *chronic* aortic dissections. More recent nomenclature has introduced subacute dissections as those occurring from 2 to 6 weeks, and chronic dissections as lasting longer than 6 weeks.

Complicated and Uncomplicated

Finally, aortic dissections have been stratified based on their natural history and clinical sequelae. Complicated aortic dissections include ruptured cases; result in malperfusion of the brain, spinal cord, abdominal viscera, kidneys, or lower limbs; or cause intractable pain or pain refractory to optimal medical therapy. Complicated dissections represent 25% to 40% of DeBakey type III aortic dissections and require urgent or emergent intervention, either by open surgical or endovascular approaches. Uncomplicated aortic dissections do not have these complications and are frequently managed with anti-impulse therapy—frequently beta-blockade—titrated to a systolic blood pressure 90 to 110 mm Hg. Despite optimal medical therapy, late mortality is as high as 30%, and up to 40% of patients will require intervention for aneurysmal degeneration or failed medical management (often within 12 months of symptom onset).

This was the case in our patient, who had persistent pain 8 days after symptom onset, despite optimal medical therapy. She underwent TEVAR with coverage of the entry tear in the proximal descending thoracic aorta. TEVAR has rapidly become the preferred approach at most centers for treatment of distal aortic dissections. The treatment goals are to cover the entry tear, promote thrombosis of the false lumen, and facilitate aortic remodeling. Technical success in most cases reestablishes flow to the true lumen and restores perfusion to the viscera, kidneys, and lower limbs. In our case, the stent graft restored patency to the patient's celiac artery, as demonstrated by IVUS, and her symptoms improved.

CONCLUSION

Although aortic dissections are associated with considerable morbidity and high rates of mortality, advancements in technology have improved management of this challenging problem. Endovascular repair has become the initial choice of treatment for acute uncomplicated and complicated DeBakey type III dissections and may have the potential to further shift treatment paradigms for ascending aortic dissections as the technology evolves. We have presented our management approach of an acute, complicated, DeBakey type IIIB dissection using IVUS and the Medtronic Valiant stent graft to reexpand the true lumen, promote thrombosis of the false lumen, and restore flow to the mesenteric vessels. These technologies should be in the armamentarium of endovascular surgeons and interventionists who treat aortic dissections. ■

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