Morphologic Characteristics for Treatment Guidance in Uncomplicated Acute Type B Aortic Dissection

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Case
A 79-year-old woman with an eventless cardiovascular medical history presented at the emergency department with acute-onset chest pain radiating to the back, accompanied by nausea, vomiting, and profuse sweating. The patient was hemodynamically stable, with a blood pressure of 130/70 mm Hg in both arms and heart rate of 60 beats per minute; the ECG showed no signs of cardiac ischemia, and cardiac biomarkers were not elevated. A computed tomography scan of the chest and abdomen showed a Stanford type B dissection of the aorta (Movies I and II in the online-only Data Supplement), which extended to the suprarenal aorta, not involving the renal arteries. The celiac trunk and superior mesenteric artery originated from the false lumen, without signs of mesenteric ischemia. The patient was treated medically with intravenous labetalol, targeted at a heart rate <60 beats per minute (with systolic blood pressure <120 mm Hg), and analgesia. A second computed tomography scan 3 days after admission showed a stable dissection with progressive thrombosis of the false lumen. In the following days, the patient’s heart rate and blood pressure remained within the target range with IV labetalol, pain was adequately treated with opioids, and the labetalol dose was lowered gradually. At day 6 after admission, the patient suddenly developed excruciating thoracic and abdominal pain, became hemodynamically unstable shortly thereafter, and died because of rupture of the dissection.

Diagnosis and Work-Up
Acute type B aortic dissection is the most common aortic catastrophe and is associated with high morbidity and mortality rates.1,2 Patients with acute aortic dissection (AoD) can present with a variety of signs and symptoms that mimic other more common disease modalities. Therefore, a high index of suspicion is warranted in patients at risk for AoD, such as those with hypertension, use of cocaine or other stimulants, a family history of thoracic aneurysm or AoD, presence of genetic collagen-vascular disorders (eg, Marfan or Ehlers-Danlos syndrome), and inflammatory vasculitides (eg, Takayasu or giant cell arteritis).3 Most patients present with sudden severe chest, back, or abdominal pain, which may have a radiating or migratory pattern. These symptoms sometimes resolve, whereas dissection-related complications, such as heart failure, pulse deficit, neurologic deficits, or other signs of vascular insufficiency, persist.

Modalities to diagnose AoD include echocardiography, abdominal ultrasound, and computed tomography scan. Of these modalities, computed tomography scan has the highest sensitivity and specificity.4 However, these imaging modalities should not be considered as competitive but rather as complementary. The goal of imaging is not exclusively diagnostic but also to elucidate AoD anatomy, including involvement of the ascending aorta; location and number of the intimal tear(s); size of the aortic root; state...
of perfusion of limbs and end organs; extent, trajectory, and patency of the false lumen; and presence of re-entry. Together with patient characteristics and clinical symptoms, elucidation of AoD anatomy assists clinicians in making therapeutic decisions.

**Treatment of Acute Type B Aortic Dissection and Risk Factors to Guide Therapeutic Choices**

Initial treatment of AoD, irrespective of its actual type, consists of stabilizing the patient and instituting adequate blood pressure and heart rate control to reduce shear stress on the aortic wall. β-Blockers modulate the 3 parameters that influence aortic wall stress: (1) velocity of ventricular contraction, (2) rate of ventricular contraction, and (3) blood pressure. Therefore, IV β-blockade is considered the first-line agent in the medical treatment of AoD.2 Heart rate should be targeted at ≤60 beats per minute. When β-blockers are contraindicated, nondihydropyridine calcium channel-blocking agents should be used. If systolic blood pressure remains >120 mmHg, intravenous angiotensin-converting enzyme inhibitors or vasodilators should be administered.4

If imaging studies show signs of involvement of the ascending aorta, hence type A AoD, surgical intervention is indicated. Open surgical graft replacement is the gold standard, but endovascular treatment by stent-graft placement is evolving. When a dissection of the descending aorta is present, hence type B AoD, the choice for initial treatment is based on differentiating high-risk complicated cases from lower-risk less complicated cases. If an acute type B aortic dissection is accompanied by signs of impaired organ perfusion or impending rupture, or if pain or hypertension cannot be adequately controlled, the presentation is considered complicated.4,6 In cases of inadequate perfusion, surgical or endovascular fenestration or thoracic endovascular aneurysm repair (TEVAR) can be used. In cases of impending rupture or noncontrollable pain or hypertension, TEVAR (or open graft replacement) is indicated.4,6 Traditionally, uncomplicated acute type B aortic dissection is treated medically. However, despite adequate antihypertensive treatment, aortic-related complications may occur during follow-up, with high mortality rates.6–10 Therefore, interest in surgical management of uncomplicated acute type B aortic dissection has increased in recent years. However, initial results in selected groups, mainly from the Investigation of Stent Grafts in Aortic Dissection trial, failed to show benefit of elective stent-graft placement versus medical therapy on survival (2-year cumulative survival rate 95.6±2.5% with optimal medical therapy versus 88.9±3.7% with TEVAR; P=0.15) or adverse events during an observational period of 2 years, despite favorable effects on aortic remodeling.11 Nevertheless, more recent evidence suggests that TEVAR in the acute phase may improve survival and reduce aortic dissection over the long term (5 years).12,13

Prognostic predictors in patients with acute type B aortic dissection encompass patient characteristics, biomarkers, and clinical symptoms.6,8,14 An increasing number of morphologic predictors have been proposed.15–17 First, it is critical to differentiate classic acute type B aortic dissection from intramural hematoma type B, which, in contrast to classic acute type B aortic dissection, is not characterized by the presence of an intimal flap or entry tear. Intramural hematoma type B can often be treated medically, and aortic enlargement is less common during follow-up, which suggests that intramural hematoma type B may have a slightly more benign course than classic acute type B aortic dissection in the acute setting.14 Saccular configuration of the false lumen is associated with increased aortic dilatation, whereas an increased number of entry tears, location of the false lumen on the outer aortic curvature, and circular configuration of the true lumen are morphologic signs associated with decreased aortic dilatation.16,17 Furthermore, Evangelista et al18 showed that a greater size of the entry tear, a greater maximum diameter of the descending aorta, and a proximal location of the entry tear were morphologic predictors of mortality and need for intervention in the descending aorta because of complications. The ongoing acute uncomplicated aortic dissection type B: evaluating stent-graft placement or best medical treatment alone (ADSORB) trial19 is a multicenter, prospective, randomized, controlled trial comparing endoluminal stent grafting with best medical treatment in uncomplicated acute type B aortic dissection. The ADSORB trial is expected to provide us with further insights into the optimal treatment of uncomplicated acute type B aortic dissection.

The dissection in our patient had an elliptic configuration of the true lumen. The diameter of the descending aorta was relatively large, and she had a single and relatively large entry tear, factors related to increased aortic dilatation or adverse event rates. Therefore, especially in hindsight, it might have been beneficial to perform a TEVAR in this patient to prevent aortic rupture.

Disclosures

None.

References

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