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A 55-Year-Old Man With an Aortic Dissection

A 55-year-old man presented to a community hospital with a chief complaint of possible seizure and altered mental status. Emergency medical services (EMS) had found him as the sole occupant of his car, which had been pulled over to the side of the road with no sign or report of trauma. On EMS arrival, the patient was noted to be bradycardic into the 50s with a weak, thready radial pulse and unobtainable blood pressure.

EMS arrived at the community hospital with the patient who was noted to be very drowsy, actively vomiting, mumbling incoherently, and was not spontaneously moving his left lower extremity. His initial vitals were as follows: heart rate of 60, blood pressure of 82/61, and oxygen saturation 98% room air; he was also afebrile. A secondary survey was performed, which showed no evidence of trauma, no chest wall tenderness, no cardiac murmurs, a tender abdomen, movement of both of his upper extremities, movement of his right lower extremity, and limited, prompted movement of his left lower extremity.

Over the next several minutes, the patient became increasingly agitated with ongoing vomiting and confusion. Fluid resuscitation was initiated, and the patient was intubated. After this, a focused assessment with sonography in trauma examination was performed, which showed no evidence of cardiac effusion or hemoperitoneum. After the intubation was completed, a chest x-ray was performed, which showed a poorly defined aortic arch (Fig. 1).[1] The patient, with ongoing resuscitation, was taken for a computed tomographic (CT) scan.

A noncontrast head CT scan was noted to be negative. Because of his chest x-ray findings as well as his constellation of symptoms, which included neurologic deficit, altered mental status, vomiting, hypotension, and abdominal tenderness, CT scans of the chest, abdomen, and pelvis with intravenous contrast were performed. It showed a large, Stanford type A aortic dissection extending from the aortic root (Fig. 2) through the aortic arch (Figs. 3 and 4) and the descending aorta and involving the right iliac artery (Fig. 5). The dissection involved the celiac trunk and superior mesenteric artery but preserved the renal arteries.

The patient was sedated aggressively with propofol and fentanyl. A position of comfort and synchrony with the ventilator were also strictly maintained. After initial fluid resuscitation, his blood pressure had increased to 139/70 with an ongoing heart rate of 61. Thus, a right-sided internal jugular central venous catheter was placed, and the patient was started on an esmolol drip for a goal heart rate of 60 and systolic blood pressure of 100 to 120. A local rotor wing critical care transport service was contacted for rapid transport to a facility capable of emergent surgical intervention.

On arrival to the tertiary care facility, the patient was immediately taken to the operating room where he underwent surgical repair with a 34-mm Hemashield graft (Atrium Medical Corporation, Hudson, NH) under circulatory arrest. He was subsequently transferred to the intensive care unit where he was extubated on postoperative day 0. His care was advanced over the next several days with excellent functional improvement but did have a residual left gaze preference with slight expressive aphasia. Thus, neurology was consulted and magnetic resonance imaging/magnetic resonance angiography was performed, which showed small, scattered areas of acute infarct.

The patient underwent inpatient physical therapy and was discharged to an inpatient rehabilitation facility alert, oriented, and able to ambulate on hospital day 10.

Discussion

The aorta is the body's largest blood vessel, and all oxygenated blood must pass through the aorta in order to reach the systemic circulation. Anatomically, it travels from its root just beyond the left ventricle and ends at its bifurcation into the right and left common iliac arteries.

Histologically, the aorta consists of 3 layers: the tunica intima, media, and adventitia. The innermost layer, the tunica intima, consists of a thin endothelial layer and is easily damaged. The middle layer, the tunica media, consists of elastic tissue. The outer layer, the tunica adventitia, consists of collagen fibers, sensory nerve fibers, and a network of small blood vessels supplying the aorta known as the vasa vasorum. The inner 2 layers do not have sensory innervation, so pain is primarily perceived from the adventitia and tends to follow the anatomic course of the portion of the aorta involved.

In aortic dissection, the easily damaged intima is violated, which allows blood to enter the medial layer. Depending on the force and frequency of pulsatile blood against the dissection point, blood may dissect in this layer and create a "false lumen" between the tunica intima and the tunica adventitia. Depending on the location and extent of the dissection, a variety of signs, symptoms, and sequelae develop.

Aortic dissections are the most common aortic emergency, occurring with an average incidence of 10,000 per year in the United States.[1] They carry an in-hospital mortality of up to 58%[2] and a mortality of 0.25% to 1% per hour for the first 48 hours.[3] Rapid diagnosis and management with transport to definitive care are essential in the management of aortic dissection. Prehospital and transport medical crews should have an understanding of how to recognize and treat these complex patients.

Figure 1. A post-intubation chest X-ray was complete in the supine view. Note the poorly demarcated aortic knob as indicated by the red arrow.

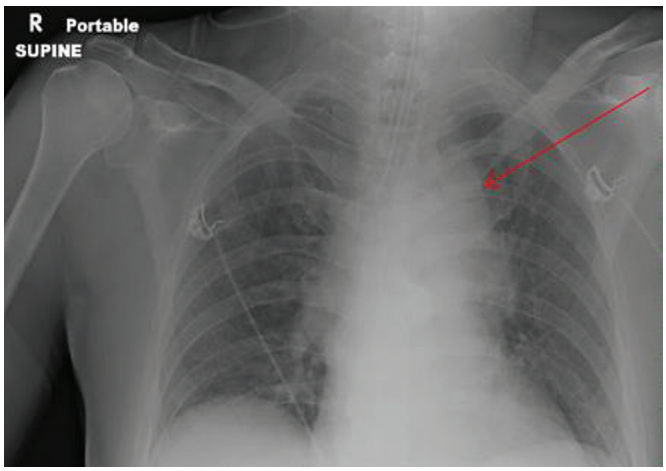


Figure 2. Arrows indicating dissection involving the aortic root (top left) and the thoracic descending aorta (bottom right).

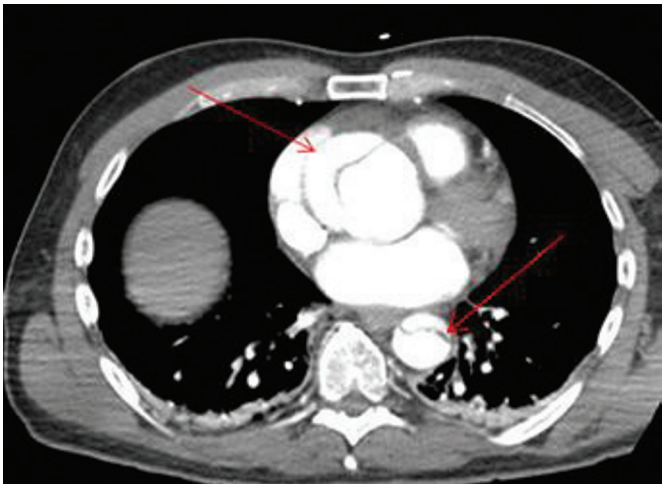
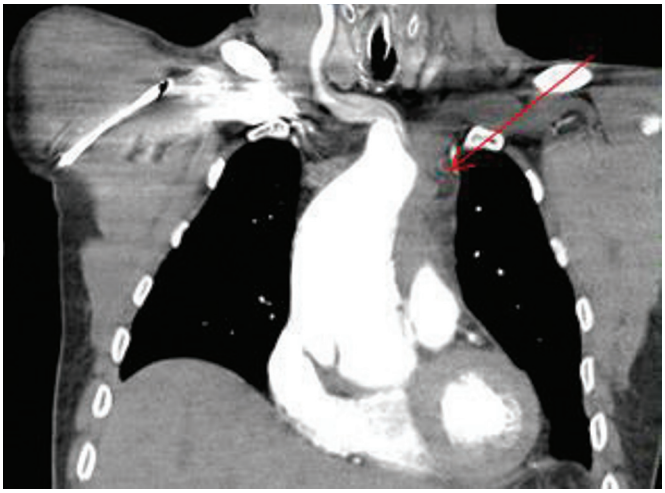


Figure 3. Coronal image of the thoracic aorta. The arrow indicates dissection at the aortic arch with loss of the lumen of the left internal carotid artery.



Atraumatic aortic dissections have a bimodal age distribution. Younger patients will often have conditions that predispose them to an intimal tear in areas of the aorta that are vulnerable to sheer stress. Examples of this are collagen vascular disease (Marfan syndrome and Ehlers-Danlos syndrome), bicuspid aorta, aortic coarctation, and vasculitis.[4] Furthermore, patients may report an activity inducing an acute elevation in blood pressure, such as cocaine use[5] or recent history of intense resistance exercise.[6] Before cardiac surgery is also associated with an increased risk.[4] The largest group of those diagnosed with aortic dissection is composed of patients $>$ 50 years old with chronic hypertension.[1]

Aortic dissection is classified using 2 different systems (Stanford and DeBakey), which help delineate treatment strategies. Stanford type A dissections involve the ascending aorta, although they may involve the arch and descending aorta as well. They are typically considered surgical emergencies because of the proximity of the false lumen to multiple structures essential for cardiac output. Stanford type B dissections are limited to the descending aorta (distal to the left subclavian artery) and are more often managed medically because of equal or higher mortality with surgical management.[7] The less commonly used DeBakey system divides dissections into 3 categories. DeBakey type I dissections involve the ascending aorta, the arch, and the descending aorta. Type II involves the ascending aorta only. DeBakey type III dissections begin distal to the left subclavian artery but may dissect proximally and distally (type IIIa) or distally alone (type IIIb).[2,8] For simplification, the Stanford classification system is more commonly used; type A dissections are those that involve the ascending aorta (and are therefore surgical emergencies), and type B dissections are those that do not.

Depending on the location of the dissection, patients can present with a variety of symptoms, which, similar to many vascular events, begin abruptly.[6] Often, signs and symptoms will follow the anatomic course of the aorta and localize to sites of impaired circulation (Fig. 6). Involvement of the aortic root may affect the pericardium, aortic valves, and the coronary arteries.[19] This may result in syncope and hypotension caused by a number of different etiologies, including pericardial tamponade, severe aortic insufficiency, aortic rupture with massive hemothorax (because of dissection through the adventitia), bradyarrhythmia caused by occlusion of the coronary arteries, or sudden increase in vagal tone because of stimulation of aortic baroreceptors. Syncope is a presenting symptom in 9% to 14% of Stanford type A aortic dissections, and hypotension/shock is present on presentation in up to 27%.[1,9] Both are associated with increased mortality.[4,10]

If the ascending aorta is involved, patients often present with chest pain radiating to the left shoulder and back as the dissection moves toward the arch. If the aortic arch is involved, patients may complain of pain in the jaw, neck, and upper back. Furthermore, involvement of the arch may result in obstruction of the carotid arteries, leading to acute stroke, Horner Syndrome, or altered mental status. If the descending