Clinical Case of the Month

A 68-Year-Old Man With Chest Pain and Left-Sided Weakness

Juan D. Ramirez, MD; Asad Chaudhry, MD; David Litner; Shannon Alwood, MD; D. Luke Glancy, MD; Herman A. Heck, MD; and Fred A. Lopez, MD (Section Editor)

CASE PRESENTATION

A 68-year-old man came to the emergency department (ED) after awakening from sleep with an episode of sharp chest pain which was 10/10 in intensity, radiated to the back, and was associated with shortness of breath, diaphoresis, weakness of the left side of his body and difficulty speaking. His past medical history was significant for hypertension and Parkinson´s disease.

The patient was allergic to sulfonamides, and was taking lisinopril daily for management of his blood pressure.

Visibly agitated, his vital signs included: pulse of 56 beats per minute; blood pressure in the right arm of 170/56 mm/Hg; respiratory rate 18 breaths per minute; and pulse oximetry of 100 percent on room air.

On physical exam the patient's pupils were equal, round, and reactive to light, and extra-ocular movements were intact. Cardiovascular, pulmonary, and abdominal exam revealed no abnormalities. The extremities were not edematous, and pulses were 2+ bilaterally in both upper and both lower extremities. The patient was oriented to place, space, and time. He was able to follow simple commands and demonstrated coherent language. Motor strength was 3/5 in the left upper extremity, 5/5 in the right upper extremity, 4/5 in the left lower extremity, and 5/5 in the right lower extremity. The patient's Glasgow coma scale was reported as 14 (eyes: 3; verbal: 5; motor: 6).

Laboratory data revealed no elevation in cardiac enzymes. His white blood cell count was 17.1x103/uL (4.5-11.0) with a differential that included 86% neutrophils, 8% lymphocytes, and 6% monocytes. Hemoglobin was 13.9 gm/dL (13.5-17.5); hematocrit was 40.1% (40-51); and platelet count, 236x103/uL (130-400). Coagulation profile included a PT of 10.3 sec (9.5-12.5); an INR of 1.0 (0.9-1.1); and a PTT of 26.6 sec (24.0-36.0). Serum sodium was 138 mmol/L (135-146); potassium, 4.2 mmol/L (3.6-5.2); chloride, 104 mmol/L (96-110); bicarbonate, 24 mmol/L (24-32); glucose, 110 mg/dL (65-99); blood urea nitrogen, 31 mg/dL (7-25); creatinine, 1.4 mg/dL (0.8-1.5); and calcium, 9.1 mg/dL (8.4-10.3)

Urine and blood toxicology studies were unrevealing. With the patient breathing two liters/min of supplemental oxygen, an arterial blood pH was 7.31 (7.35-7.45); pCO2, 41 mmHg (35-45); pO2, 124 mmHg (90-100); HCO3, 20 mmol/L (22-26); and oxygen saturation, 97% (95-100).

An electrocardiogram revealed evidence of left ventricular hypertrophy but no changes suggestive of ischemia or injury.

A plain chest radiograph revealed a tortuous aorta, moderate cardiomegaly with left ventricular predominance and cephalization of the pulmonary vasculature without overt congestive changes. A computed tomographic (CT) scan of head without contrast revealed atherosclerotic calcifications within the vertebral arteries. A CT angiogram of the chest and abdomen with contrast revealed a Stanford type A aortic dissection extending down to the level of the caudal-most renal artery origin and up into all three cephalic vessels with occlusion or extremely limited flow observed in the right common carotid artery (RCCA). The RCCA originated from the false lumen of the dissection while all

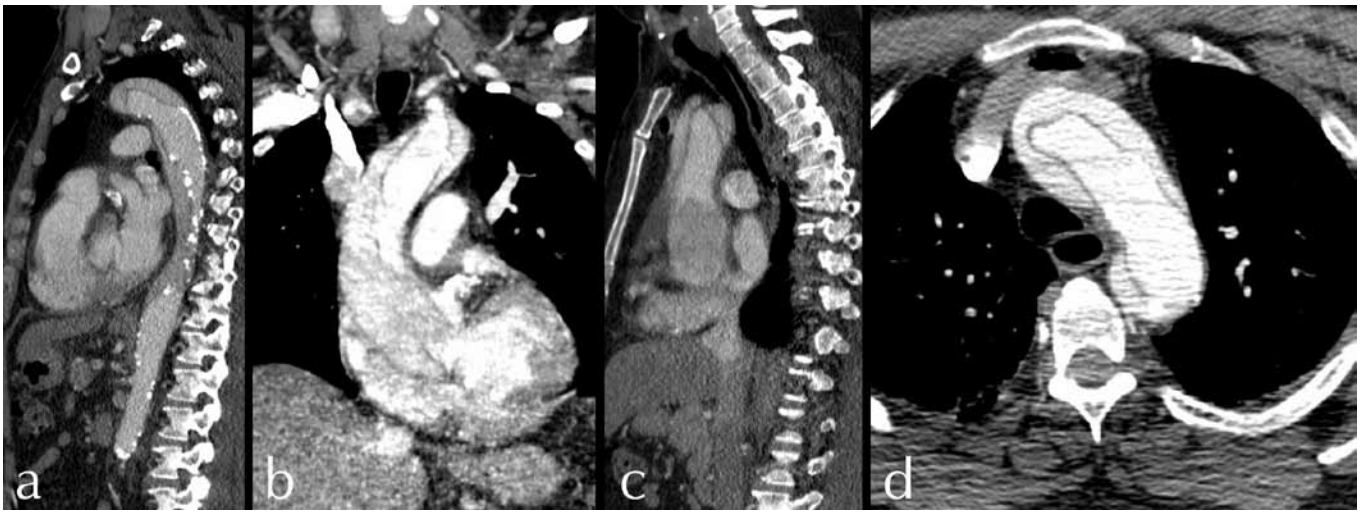

Figure. Chest computed tomograms. (a) Sagittal view of the aortic dissection at the level of the aortic arch and descending aorta. Multiple calcific deposits are noted in the descending aorta. (b) Coronal view of the aortic dissection at the level of the ascending aorta. (c) Sagittal view of the aortic dissection at the level of the ascending aorta. (d) Transverse view of the aortic dissection at the level of the aortic arch.

other major vessels arising from the arch and visceral vessels arising from the abdominal aorta were supplied by the true lumen (Figure).

An echocardiogram revealed mild aortic regurgitation, dissection of the aorta extending from the ascending into the descending aorta and a left ventricular ejection fraction of 55%.

The patient was immediately started on an esmolol drip and required intubation due to the development of a decreased level of conciousness and decorticate posturing. He became hemodynamically unstable and was taken to the operating room where he developed pulseless electrical activity and ultimately expired secondary to tamponade and exanguination.

An autopsy revealed a ruptured aortic dissection involving the ascending and descending aorta with extension into the right common carotid artery, as well as severe aortic atherosclerosis.

DISCUSSION

An aortic dissection is defined as a separation in the wall of the aorta, usually in the outer portion of the media, allowing blood to create a false lumen. It is relatively uncommon but when unrecognized and left untreated it can quickly lead to death. The mortality rate of an untreated ascending aortic dissection may be as high as 1% to 2% per hour after the symptoms develop, and 40% to 50% of patients die within 48 hours.[1] Aortic dissections usually occur in patients in their sixth and seventh decades of life and have a male: female predominance of 2:1.

Systemic arterial hypertension is the single most important predisposing factor in the development of an aortic dissection. Dissections occur in the range of 2.6 to 3.5 per 100,000 person-years. In 2000, the International Registry of Acute Aortic Dissection (IRAD) reported that 72% of patients with aortic dissections had a history of hypertension, and 65% were men with a mean age of 60 years. Other predisposing risk factors include atherosclerosis, a pre-existing aortic aneurysm, underlying collagen vascular disease such as the Marfan syndrome, bicuspid aortic valve, coarctation of the aorta, diabetes mellitus, cocaine use, Turner's syndrome, and trauma.[2]

The initiating event in an acute aortic dissection usually is a tear in the intima that extends into the media of the aorta. This provides a conduit for blood to pass into the media and there to separate the wall of the aorta into two layers. Once this separation has occurred it can propagate proximally or distally as an additional or false aortic lumen. The direction of the separation determines the clinical presentation and the subsequent treatment of the patient. The false lumen usually communicates with the true lumen at a site distant from the initial tear, but rarely no entry or exit tear is found.

Aortic dissections are classified based on their anatomical location. In the Stanford classification type A dissections, which comprise about 60% of all dissections, involve the ascending aorta independent of the site of the tear and can also involve any other part of the aorta. Type B dissections involve only the transverse and/or the descending aorta. In the older DeBakey system type 1 dissections involve both the ascending and the descending aorta. Type 2 dissections are limited to the ascending or transverse aorta, and type 3 dissections involve only the descending aorta.[3]

Typical symptoms include sudden onset of excruciating sharp and tearing chest pain that may radiate posteriorly and to the abdomen or lower back. Clinical complications can include development of acute aortic regurgitation,

pulmonary edema, pericardial rub/tamponade, loss of pulses, and neurologic deficits including syncope and cerebrovascular accidents. Signs of aortic insufficiency include a decrescendo diastolic murmur, widened pulse pressure, and the new onset of pulmonary edema.

The electrocardiogram and plain chest radiograph are not very sensitive diagnostically. Diagnosis is generally made with CT angiography, magnetic resonance angiography or transesophageal echocardiography (TEE). Bedside TEE is utilized in patients who are hemodynamically unstable. Magnetic resonance imaging (MRI) can potentially detect blood flow thereby illustrating the direction of the dissection as antegrade or retrograde. All three of these modalities will confirm the diagnosis by demonstrating a separation of the true lumen from the false lumen.[4]

It is critically important to begin medical therapy as soon as the diagnosis of an aortic dissection is considered. Therapy is aimed at reducing the sheer stress on the aortic wall. The patient should be admitted to the intensive care unit for close hemodynamic monitoring. Initial therapy with parenteral beta-adrenergic blockers is typically utilized with goals of heart rate of approximately 60 beats per minute and a systolic blood pressure of 120 mmHg or less, the latter commonly requiring the subsequent addition of an intravenous infusion of sodium nitroprusside. Direct vasodilators such as hydralazine should not be used as they may worsen or propagate the dissection by increasing hydraulic shear.[5]

If a patient has a Type A aortic dissection, immediate surgical correction is required in addition to medical therapy. The goal of surgery is to reconstruct the aortic wall to prevent further dissection.

After the patient is stabilized, long-term therapy consists of blood pressure control and reduction of cardiac contractility. Patients who have chronic Type B dissections need to be monitored every 6 to 12 months with a contrast-enhanced CT scan or an MRI to detect propagation of the dissection.[6] Avoidance of extreme physical activity is advised.

REFERENCES

1. Desanctis RW, Doroghazi RM, Austen WG, et al. Aortic dissection. *N Engl J Med* 1987;317:1060-1067.
2. Spittell PC, Spittell JA Jr, Joyce JW, et al. Clinical features and differential diagnosis of aortic dissection: experience with 236 cases (1980- 1990). *Mayo Clin Proc* 1993; 68:642-651.
3. Guilmet D, Bachet J, Goudot B, et al. Aortic dissection: anatomic types and surgical approaches. *J Cardiovasc Surg* 1993; 34:23-32.
4. Dzau V, Creager M. Diseases of the aorta. In: Fauci A, et al, (editors). *Harrison's Prinicple's of Internal Medicine*, 14th edition. New York: Mc-Graw-Hill; 1998:1394–1408.
5. Vongpatanasin W, Victor R. Vascular diseases and hypertension. *Cecil Essentials of Medicine*, 7th edition. Philadelphia, PA: Saunders Elsevier;2007:167-180.
6. Nienaber CA, von Kodolitsch Y, Nicolas V, et al. The diagnosis of thoracic aortic dissection by noninvasive imaging procedures. *N Engl J Med* 1993;328:1-9.

Dr. Ramirez is a house officer in the Department of Internal Medicine/Emergency Medicine training program at Louisiana State University Health Sciences Center in New Orleans, Louisiana. **Dr. Chaudhry** is a first year cardiology fellow at Drexel University/ Hahnemann in Philadelphia. **Dr. Alwood** is a staff member in the Emergency Department at Our Lady of the Lake Hospital in Baton Rouge, Louisiana. **Mr. Litner** is a summer senior college student in the Department of Neuroscience Center of Excellence at Louisiana State University Health Sciences Center. **Dr. Glancy** is a professor in the Section of Cardiology, Department of Medicine, at Louisiana State University Health Sciences Center. **Dr. Heck** is a faculty in the Section of Cardiothoracic Surgery, Department of Surgery, at Louisiana State University Health Sciences Center. **Dr. Lopez** is a professor and vice chair in the Department of Medicine at Louisiana State University Health Sciences Center.