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Syed Saad, MD, Samiya Yasin, MD, Neeraj Jain, MD, Avaneesh Jakkoju, MD, Ryan Chauffe, DO, Fred A. Lopez, MD

CASE PRESENTATION

A 72-year-old man without significant past medical history presented to the emergency department with complaints of worsening lower extremity weakness and paresthesias for two days. Associated symptoms included nausea, vomiting, diarrhea and dysuria. He returned from Mexico one week prior to the presentation. The physical examination was remarkable for decreased muscle strength in both lower extremities. Initial data indicated a urinary tract infection for which intravenous (IV) levofloxacin was administered. A nerve conduction study was consistent with Guillain-Barré Syndrome (GBS), but his neurologic abnormalities resolved rapidly. On the third hospital day, he developed acute onset chest discomfort and dyspnea requiring supplemental oxygen. Physical examination revealed a new blowing, grade III/VI diastolic murmur which was loudest at onset and heard best in the third left intercostal space. Bibasilar crackles were auscultated. Discrepant blood pressures in the upper extremities (109/56 mm Hg from right arm and 154/66 mm from Hg left arm, respectively) were also noted. Additional data demonstrated an acute kidney injury with a creatinine of 3.0 mg/dL (baseline 1.1 mg/dL). Serial troponin measurements were unremarkable. A chest radiograph revealed alveolar infiltrates and bilateral pleural effusions. Transthoracic and transesophageal echocardiograms demonstrated aortic insufficiency and possible aortic dissection. Aortic computed tomography angiogram (CTA) revealed an extensive Stanford type A aortic dissection extending to the left renal artery (Images 1-6). Cardiothoracic surgery was consulted and an urgent ascending aortic root dissection repair with root replacement was performed. The patient improved clinically and was discharged in stable condition.

Image 1. Cardiac cross-sectional view showing separation of true and false aortic lumens with an intimal flap (arrow) at the level of the aortic valve. (AV=aortic valve; RA=right atrium; RV=right ventricle; LA=left atrium).
DISCUSSION

Introduction

Acute aortic syndrome is a range of severe, painful, and potentially life-threatening abnormalities of the aorta including aortic dissection, intramural hematoma, penetrating aortic ulcer, and ruptured thoracic aortic aneurysm.¹ The incidence is around 30 cases per million individuals annually.² Acute aortic dissection is the most catastrophic and frequent. If left untreated, type A aortic dissection (AD) has a mortality rate of about 1% per hour; half of such patients expire by the third day, and up to 80% by the end of the second week.³

Diagnosis

The classic clinical presentation of AD includes the sudden onset of severe “tearing” or “ripping” chest pain. However, the presenting symptoms may vary, making the diagnosis challenging. On initial evaluation, 38% of acute AD can be missed.⁴ The diagnosis requires a high clinical suspicion. A thorough patient history and physical examination are critically important as well as imaging studies for confirmation. A history of hypertension and connective tissue disease are well described risk factors for an acute AD.⁵ Physical exam findings may include hypertension or hypotension (cardiac tamponade), blood pressure differential greater than 20 mm Hg in both arms,
Emergent surgical intervention is the preferred treatment for Stanford type A ascending AD. Expedited intervention is also preferred for complicated Stanford type B dissections which have one or more of the following characteristics: increasing aortic diameter, increasing hematoma size, involvement of major branches of the aorta, impending rupture, refractory pain and a developing saccular aneurysm.

All other descending aortic dissections are managed medically by reducing the blood pressure and the shearing forces of myocardial contractility. Antihypertensive therapy, including beta blockers, is the first line of treatment for all stable chronic aortic dissections.¹¹

Pain management with narcotics and opiates is also an integral part of painful dissections.¹²

Fluoroquinolones are commonly used antibiotics.¹³ These drugs are implicated in upregulation of matrix metalloproteinases (MMPs), the main extracellular matrix enzymes in collagen degradation. Collagen, the most abundant protein in the body, is found in tendons, muscles, kidneys, vessel walls and cornea. Fluoroquinolone use is associated with potentially disabling diastolic murmur of aortic insufficiency, wide pulse pressure, bounding pulse and neurologic deficits. Laboratory findings depend on the level and extent of dissection and may include leukocytosis, anemia, elevated creatinine, elevated blood urea nitrogen, and elevated lactate dehydrogenase.⁹ A chest radiograph is the initial imaging study. It may be normal or reveal a widened mediastinum. Transesophageal echocardiogram is more sensitive than transthoracic echocardiogram, but the aortic arch is not typically adequately visualized. Though highly operator dependent, ultrasound modalities are helpful for ascending aortic dissections especially in cases with aortic valve involvement. Contrast enhanced CTA of the thoracic aorta typically offers more definitive diagnostic assessment in patients who are hemodynamically stable. Magnetic resonance angiography (MRA) offers high test sensitivity for AD with a specificity similar to CTA.⁷,⁸ Invasive aortography has historically been the gold standard, but its use is being challenged by non-invasive imaging primarily due to patient safety and outcomes data.⁹

Classification and management

Management of acute aortic dissection can be medical or surgical and is usually determined by the anatomic classification according to the Stanford classification. Stanford type A includes dissection of the ascending aorta and arch whereas Stanford type B includes the descending aortic dissection distal to the left subclavian artery.¹⁰

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Aortic syndromes and fluoroquinolone use

Fluoroquinolones are commonly used antibiotics.¹³ These drugs are implicated in upregulation of matrix metalloproteinases (MMPs), the main extracellular matrix enzymes in collagen degradation. Collagen, the most abundant protein in the body, is found in tendons, muscles, kidneys, vessel walls and cornea. Fluoroquinolone use is associated with potentially disabling...
side effects involving tendons, muscles, nerves, joints and central nervous system, and the FDA has recommended that these agents only be used for treatment of acute exacerbations of acute bronchitis, uncomplicated urinary tract infections, and acute bacterial sinusitis when no alternative antibiotic options exist.¹⁴ Recent studies have investigated the risk of aortic aneurysm and dissection with the use of fluoroquinolones. Daneman et al. performed a population based study and found that fluoroquinolones were associated with an increased hazard of aortic aneurysm with a calculated hazard rate of 2.72 (95% CI 2.53 to 2.93).¹⁵ In another case control analysis, Lee at al. reported an increase in the risk of aortic aneurysm or dissection in patients treated with fluoroquinolones (rate ratio [RR], 2.43; 95% CI, 1.83-3.22).¹⁶ After evaluating reports of cases and the medical literature, the FDA announced in a Drug Safety Communication (dated May 10, 2017) that that the available evidence does not support that the use of fluoroquinolones may result in aortic syndromes (or retinal detachment).¹⁷ The FDA will continue to monitor for fluoroquinolone-associated safety issues.

REFERENCES


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