Intractable Vomiting: A Dissection in Disguise

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Abstract

Aortic dissection describes the formation of a tear in the intima of the aorta, allowing the formation of a false lumen that dissect below the layers of the aortic wall. It is a dangerous disease with mortality shown to be 25-30%\textsuperscript{18}. Dissections are categorized per their anatomic location. The most common and simple classification system is the Stanford system which classifies any dissection involving the ascending aorta as class A and all others as class B. Class A dissections require open surgical treatment whereas class B are typically treated medically\textsuperscript{4,11,19}. Patients with dissection are typically 60-80-year-old males with a history of hypertension\textsuperscript{4,5,12}. Aortic dissection is classically associated with sudden onset of severe sharp or tearing chest pain in a hypertensive adult. The diagnosis of acute aortic dissection has been shown to be associated with some pain up to 95.5% of the time\textsuperscript{4}. However, with increasing age the percentage of painless dissection likewise increases\textsuperscript{4}. This case involves a 68-year-old female who presented to the emergency department with an uncommon presentation of aortic dissection. This patient presented with 4 days of intense nausea and vomiting without any pain. This presentation will discuss the variable presentations of dissection and highlights the importance of maintaining a broad differential diagnosis. X-ray may not show the classic mediastinal widening expected with class A dissection up to 20-28% of the time\textsuperscript{12}. CT scan has been shown to have 100% sensitivity, with MRI and transesophageal echocardiography approaching those numbers. Serologic markers have begun to show promise\textsuperscript{12}. D-dimer is the most studied of these with a 95.2% median sensitivity and 60.4% median specificity\textsuperscript{17}. Other serologic markers including soluble elastin fragments, smooth muscle myosin heavy chains, and others have been studied but more research is still needed\textsuperscript{12}.
Introduction

The aortic wall consists of three main layers, the tunica intima, tunica media and tunica adventitia. Aortic dissection occurs when an intimal tear allows the accumulation of blood within wall and subsequent “dissection” of these layers. This creates a false lumen within the tunica media alongside the true lumen of the aorta\(^8,19\). Dissections can be acute or chronic, with less than 14 days being classified as acute\(^4\). Traditionally, dissections are classified per their anatomic location. The most succinct and common classification system for aortic dissection is the Stanford classification. In this system, any dissection involving the ascending aorta is class A and any without ascending involvement are class B. The other common classification system, the DeBakey system, uses class III as a synonym to the Stanford B. A DeBakey class I then describes a dissection originating in the ascending aorta and extending distally with a DeBakey II remaining localized to the ascending\(^4,8\). The importance of dissection classification lies in the treatment. All acute dissections require aggressive impulse and blood pressure control, typically with intravenous beta-blockers\(^17\). Dissections involving the ascending aorta are a surgical emergency\(^11,17\). In contrast, dissections only involving the descending aorta can usually be treated medically\(^11,12\). However, patients that are not hemodynamically stable, with end organ damage, rapid expansion, risk of rupture or intractable pain are indicated for surgical intervention\(^17\). Recently, another classification system referred to by the mnemonic DISSECT has been proposed in order to describe dissections with more information to assist in decisions related to endovascular treatment going forward. DISSECT stands for the duration of the disease; the location of the tear in the intima, the dissection size, the segmental extent of the aortic involvement; the clinical complications, and whether there is a thrombus in the false lumen\(^1\).

Aortic dissection is a dangerous condition with overall mortality shown to be between 25-30%\(^4\). Overall, dissection is about twice as common in men than in women\(^4,5,12,13\). However, women typically have dissections later with an average age of 67 with the male average of 60\(^13\). A history of hypertension is the most common risk factor for dissection, present in about 70% of patients\(^4,12,15\). But several other important risk factors exist, including atherosclerosis, connective tissue disorders, cocaine use, pre-existing aortic aneurysm, bicuspid aortic valves, and pregnancy\(^4,5,8,13\). One study showed that Marfan’s was present in 50% of dissection patients less than 40 years old\(^6\).

Classically dissection is associated with sudden onset of sharp or tearing chest pain. Studies report this is the case 60% of the time with some form of chest pain present in 80% of cases\(^4,12\). Dissection has also been known to be associated with back and abdominal pain, and the International Registry of Acute Aortic Aneurysms (IRAD) has shown that “any pain” has been associated with diagnosed dissection up to 95.5% of the time\(^4,12\). However, other studies have shown that dissection can present painlessly between 5-15% of the time\(^2\). Dissection can be associated with various symptoms depending on the location of the dissection and any associated blood flow disruption to various organs. For instance, a dissection extending into the coronary arteries could lead to cardiac ischemia, and one extending into the carotids could have neurologic effects\(^8\). Neurologic symptoms have been shown to present with acute dissection 17-40% of the
time and are commonly present in patients with painless dissection\textsuperscript{2}. Other signs and symptoms associated with type A dissection, include mitral regurgitation with its associated murmur (44%), a pulse deficit (18.7%), syncope (12.7%) and pericardial tamponade (13%)\textsuperscript{12,14}. Those have also been reported with type B dissection, however they are less common. Type B dissections are more likely to have hypertension on presentation (70%) whereas type A are more likely to present either normo- or hypotensive (64.3\%)\textsuperscript{12}.

**Case Report**

LG is a 68-year-old female with a past medical history of hypertension that presented to the emergency department with 4 days of continuous nausea and intractable vomiting. The patient’s symptoms had a sudden onset and were continuous prior to coming into the ED. She was seen at a local urgent care and given a prescription for Zofran which she reports only made her nausea worse. Her symptoms were exacerbated by drinking fluids. The patient also reported feeling generally weak and uncomfortable but denied any pain, and specifically any chest or abdominal pain. Her review of systems was negative for any fever, shortness of breath, cough, syncope, diaphoresis, diarrhea, dysuria, hematuria, back pain, headache or any focal neurological deficits.

The patient’s history was positive for hypertension, hypothyroidism secondary to a thyroidectomy and left bundle branch block. She was not currently taking medications for her chronic hypertension, but was on levothyroxine. She denied tobacco, alcohol or other drug use. Her family history was non-contributory.

Examination in the emergency department revealed an oral temperature was 36.5\textdegree{}C, heart rate 65 beats per minute, blood pressure 115/81, respiratory rate 18 breaths per minute, and room air pulse ox of 91\%. The patient was alert and oriented, appeared uncomfortable, but in no acute distress. Pupils were reactive and extraocular movements were intact. Her ENT exam was negative other than a dry oral mucosa. Her neck was supple without jugular venous distention. Cardiac exam noted a regular rate and rhythm without murmurs, gallops or rubs, and her extremities were without edema. Lungs were clear to auscultation bilaterally, and her respirations were non-laborered with symmetric chest wall expansion. Her abdomen was soft, non-distended, without palpable masses and bowel sounds were present. She did have some mild upper abdominal tenderness without any guarding or rebound. There was no focal neurological deficits, with normal motor and sensation throughout.

Due to the patient’s intractable vomiting for several days, a CBC, CMP, lipase and lactic acid were ordered to evaluate for electrolyte imbalance as well as potential abdominal pathology. Laboratory results revealed an elevated white blood cell count at 19,000/uL, mildly decreased sodium and chloride at 135mmol/L and 97mmol/L respectively, and increased BUN at 30mg/dL with a creatinine of 1.11mg/dL. An EKG, chest x-ray and troponin were ordered to evaluate for pulmonary and cardiac pathology. The EKG revealed a sinus rhythm, rate of 63, with a first degree AV block as well left bundle branch block. No evidence of ischemia or any satisfaction of
sgarbossa’s criteria, and her initial troponin was negative. Chest X-ray revealed a right sided pleural effusion. CT abdomen and pelvis with IV contrast was ordered at the same time to rule out bowel obstruction or other abdominal pathology, which incidentally showed an aortic dissection starting just above the root of the aorta with a moderate pericardial effusion of up to 2.4cm in width (figure 1).

The patient continued to deny any chest, abdominal, or back pain. Due to the finding of ascending aortic dissection, the patient was transferred to a facility with cardiothoracic surgical intervention. A CT-angiogram of the chest performed at the receiving facility showed a Stanford type A aortic dissection (figure 2,3,4). The patient’s type A aortic dissection was repaired by replacement of the aortic valve, ascending aorta, and the patient was discharged home eight days after the initial presentation to the ED.

**Discussion**

Aortic dissection is a dangerous condition, and even with the advancements in diagnosis and treatment, overall mortality has been shown to be 25-30%. Type A dissection is especially dangerous with mortality up to 50% in 48 hours without intervention. From this case, we can see that acute aortic dissection does not always “play by the rules”. As noted before, aortic dissection is typically painful, and generally involves men in their 60s. Studies have shown over 80% percent of patients with dissection will present with acute onset chest pain and over 60 percent of patients will present with “classic” sharp, tearing chest pain. IRAD has shown that “some pain” is related to dissection over 95% of the time. However, painless dissection has been shown by some studies to be between 5-15% of diagnosed cases. Research has shown that the percentage of painless dissection goes up with increasing age, as well as in patients with diabetes, previous aneurysm and previous procedures involving the aorta. Painless dissections have also been shown to more likely involve the ascending aorta. Dissection can involve neurological symptoms 17-40% of the time, and in some cases these neurological symptoms can mask pain or even be present with truly painless dissection. These masquerading symptoms can lead to inaccurate diagnosis which could have potentially catastrophic consequences for the patient. An example being a dissection patient receiving thrombolytic therapy for what is believed to be a cerebrovascular accident. In 2012 a study performed at Baylor University revealed that during a 3-year period 23% of ascending dissections were not discovered until surgery for another condition or until post mortem examination. This information highlights the importance of a broad differential and maintaining a high index of suspicion for dissection. Although dissection is more common in men, over age 70 just greater than 50% of dissection patients are female. Elderly patients are more likely to have lower blood pressures at presentation, less likely to have neurologic symptoms, less likely to have mitral regurgitation and less likely to have pulse deficits. In addition to being older at presentation, women are also more likely to present later for treatment and less likely to have an abrupt onset of pain. Women are more likely to have pericardial or pleural effusions, hypotension and pericardial tamponade. Women also have higher surgical mortality than men. With all this in mind it still begs the question, how does one reliably rule out aortic dissection?
Chest x-ray imaging can show mediastinal widening with type A dissection, a JAMA review in 2016 showed that 20-28% of the time this is not the case\textsuperscript{12}. This same review reported that CT was shown to have a sensitivity of 100%, MRI had a sensitivity of 95-100%, and transesophageal echocardiography was 86-100% (with transthoracic performing more poorly with a median sensitivity of 86.9%)\textsuperscript{12}. Several serologic markers have shown promise regarding the diagnosis of dissection. The most studied of these is d-dimer, with a recent meta-analysis showing a sensitivity of 95.2% and specificity of 60.4\textsuperscript{10,18}. As such, d-dimer may be helpful in ruling out dissection with atypical presentation\textsuperscript{10}. Other serologic markers that have been studied include soluble elastin fragments, smooth muscle myosin heavy chain, matrix metalloproteinase and soluble lectin-like oxidized low density lipoprotein receptor\textsuperscript{10,12}. Further research remains to be done on these various tests before recommendations are made for their use, but in the future these may prove beneficial in diagnosing a deadly disease that may be in disguise.

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### References


Figures

Figure 1. The original CT abdomen and pelvis showing the dissection, pericardial effusion and pleural effusions.
Figure 2. CT angiogram of the chest performed at the receiving facility.
Figure 3. CT angiogram of the chest performed at the receiving facility.
Figure 4. CT angiogram of the chest performed at the receiving facility.