Chest Pain in a 60-Year-Old Man

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From the Case Records of the Alpert Medical School of Brown University Residency in Emergency Medicine

DR. HEATHER RYBASACK-SMITH: The patient is a 60-year-old male who presented to the hospital with the sudden onset of non-radiating anterior chest pain after eating dinner. He reported associated nausea and mild shortness of breath. The pain was described as moderate in intensity, intermittent, and alleviated by resting and remaining still. He had never experienced chest pain prior to this episode. His past medical history was significant for hypertension (treated with lisinopril), alcohol abuse, and type II diabetes managed with oral glipizide and metformin. He took a daily aspirin, disulfiram, a multi vitamin and ibuprofen and smoked ½ pack per day. He was married, employed as a truck driver, and lived with his wife. Initial vital signs included blood pressure of 133/63 with a pulse of 72. Physical exam revealed clear lungs, normal heart sounds without a murmur or gallop, equal pulses and blood pressures bilaterally and a soft and non-tender abdomen. He had no edema. The patient was alert and in no acute distress. On his initial evaluation he was pain-free. The initial impression was acute coronary syndrome (ACS) and the patient was given an aspirin and a workup was initiated. The patient’s initial ECG (figure 1) was non-ischemic and an initial CXR showed no acute abnormalities—there was no effusion, widened mediastinum or abnormal aortic knob, and there was no evidence of congestive heart failure. Lab results were significant only for mild hyperglycemia (glucose 125 mg/dl), a normal creatinine and a negative troponin. Plans were made to admit the patient to the medical service for a rule out myocardial infarction.

Several hours into his hospital stay, the patient developed persistent, sharp chest pain and had a blood pressure of 175/70 mm Hg. A repeat ECG was unchanged. He received sublingual nitroglycerin with minimal affect. Approximately 3 hours after initial evaluation, the patient became hypotensive to 75/52 mm Hg with a pulse of 79 beats per minute. He received fluid resuscitation along with blood products and a dopamine infusion, and a surgical consult was requested.

An emergency CTA chest/abdomen/pelvis (figure 2) showed an extensive Type A aortic dissection beginning at the root of the aorta and continuing to the bilateral iliac arteries. It involved the bilateral subclavian and right brachiocephalic arteries, the celiac axis, and the left renal artery. There was a large amount of hemopericardium, and the left kidney had complete absence of contrast dye.

DR. MARK GREVE: This patient had none of the classic signs of aortic dissection. What led you to suspect the diagnosis?

DR. RYBASACK-SMITH: Initially, the patient’s presentation was concerning for acute coronary syndrome (ACS). However, the sudden recurrence of severe pain with hypotension led clinicians to quickly reevaluate the cause of his unremitting pain and consider other life threatening etiologies, including aortic dissection. Acute aortic dissection (AAD) is a
notoriously difficult diagnosis to make, and is often missed in the emergency setting. Data from the international registry of aortic dissection (IRAD) revealed that classic findings such as aortic regurgitation and pulse deficits were only seen in 31.6% and 15.1% of patients, respectively.\textsuperscript{1,2} A normal chest x-ray was noted in about 12.5% of cases and a widened mediastinum or an abnormal aortic contour can be seen as infrequently as 20%–60% of cases.\textsuperscript{3,4} While acute onset of severe chest pain is the most common presenting complaint, data from IRAD and other sources note painless dissection in 4.5%–12% of cases, and in one older study up to 27%.\textsuperscript{1,3,5} These patients are likely to be older, chronic steroid users, patients with Marfans, or those who present with syncope (9–13% of type A dissections), stroke or congestive heart failure.\textsuperscript{1,5} While the majority of patients do report chest pain, it is usually distinguished as abrupt and sharp, rather than tearing.\textsuperscript{1,4,7}

The ability of EM physicians (EMPs) to diagnose AAD has been evaluated in several studies. In 33–57% of cases practitioners do not suspect the diagnosis on initial presentation and some studies have indicated an overall rate of missed diagnosis between 40–55%.\textsuperscript{3,8,9,10} When patients present with classic findings of chest and back pain, an accurate diagnosis was made in 86% of cases studied.\textsuperscript{9}

There is a great deal of overlap between the clinical symptoms and signs of ACS and AAD. ACS is the most common misdiagnosis and age and anterior chest pain are confounding factors often leading to premature closure.\textsuperscript{2} Additionally, evidence of acute ST elevations occur in 1–7% of patients with proximal aortic dissection. This most commonly occurs in the right coronary circulation as the dissection can extend into the right coronary ostium leading to posterior-inferior injury.\textsuperscript{11} CK-MB and troponin have been shown to be insensitive in distinguishing between AAD & ACS, and have been implicated in missed diagnoses.\textsuperscript{8}

Consequences of missed diagnosis are potentially catastrophic. The mortality for Type A dissections without treatment approaches 1–2% per hour in the first 48 hours after the event.\textsuperscript{10} Predictors of death and poor outcome include age >70 years, abrupt onset of chest pain, hypotension/shock and tamponade, kidney failure, pulse deficit and an abnormal ECG.\textsuperscript{12} In modern EM, there is a push toward aggressive treatment for ACS due to worsening outcomes from delayed reperfusion.\textsuperscript{13} Yet, the consequences of inappropriate treatment with antithrombotic agents are great and can lead to delayed diagnosis, major bleeding, greater hemodynamic instability, increased risk of aortic rupture and an overall trend toward increased mortality.\textsuperscript{2} Given the risks of inappropriate antithrombotic treatment in AD and the potential for misdiagnosis, a low threshold for imaging is appropriate.

**DR. ROBERT TUBBS:** What type of patient typically presents with aortic dissection?

**DR. WILLIAM BINDER:** Numerous primary and acquired risk factors for AAD have been identified through IRAD and other studies. These are noted in table 1 and include: male gender, age >50 years, tobacco use, DLD, cocaine/stimulant use, pheochromocytoma, atherosclerosis, connective tissue disorders such as Marfan Syndrome and vascular Ehlers-Danlos Syndrome, infectious diseases, inflammatory diseases such as Takayasu and Behcet’s diseases, trauma, chronic corticosteroid use and polycystic disease.\textsuperscript{1,14,15} Systemic hypertension is the most common...
Table 1.

<table>
<thead>
<tr>
<th>Risk Factor</th>
<th>Comments</th>
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<tr>
<td>Male gender</td>
<td>&gt; 50 years old</td>
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<tr>
<td>Long Standing Arterial Hypertension</td>
<td>Tobacco use Dypsipidemia Cocaine/Crack/stimulants Pheochromocytoma</td>
</tr>
<tr>
<td>Atherosclerosis</td>
<td>Can lead to penetrating ulcer</td>
</tr>
<tr>
<td>Connective tissue disorders/genetically triggered thoracic aortic diseases and congenital disorders</td>
<td>Marfan Vascular Ehlers-Danlos syndrome (type 4) Bicuspid aortic valve Coarctation of the aorta Hereditary thoracic aortic aneurysm Loeys-Dietz Noonan syndrome Turner syndrome</td>
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<tr>
<td>Infectious Diseases</td>
<td>Syphilis Salmonella Tuberculosis</td>
</tr>
<tr>
<td>Inflammatory Disorders</td>
<td>Takayasu’s arteritis Behcet’s disease Ormond’s disease Systemic Lupus Erythematosis</td>
</tr>
<tr>
<td>Pregnancy</td>
<td>*controversy exists as to whether this is a risk factor</td>
</tr>
<tr>
<td>Iatrogenic causes</td>
<td>Cardiac surgery/Valvular Surgery Vascular surgery Catheter/instrumentation</td>
</tr>
<tr>
<td>Trauma</td>
<td>Deceleration injury MVA Air bag Fall from height</td>
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<tr>
<td>Other</td>
<td>Weight Lifting Polycystic Disease Chronic corticosteroids</td>
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risk factor for both Stanford A and B types of dissection.16 However, up to 25% of all patients who present with acute aortic syndrome, including thoracic aortic dissection, have a positive family history of aortic disease. Mutations in greater then a dozen genes are related to syndromic and nonsyndromic forms of familial thoracic aortic aneurysm and dissection.17

Acute aortic syndromes, whether genetic or acquired, appear to share a common pathophysiologic pathway: degradation of the medial wall and subsequent stiffening and fibrosis of the vessel intima. The initial insult can be a result of genetic or acquired disease such as atherosclerosis, vasculitis, connective tissue or inflammatory diseases, or a bicuspid valve. In hypertension, chronically high pressures lead to intimal thickening, fibrosis, calcification and fatty acid deposition, the walls become stiff and weakened. Often a rupture of a plaque is the nidus for intimal tear, which can spread anterograde or retrograde to the tear.3,18

DR. MEHRI KAMAT: What was the patient’s clinical course?

DR. RYBASACK-SMITH: Upon arrival to the operating room (OR), the patient’s exam had changed markedly. He was noted to have a ruddy colored face and chest, a non-palpable left radial pulse and 1+ trace right radial pulse. He had bilateral edema and mild expiratory wheezes. In the OR he was noted to have a large pericardial effusion with tamponade physiology, which was relieved through creation of a pericardial window and extraction of clot. He underwent repair of the dissection with a tube graft. Post-operatively his ejection fraction was estimated to be 45–50%. The patient was extubated post-operatively day 0, developed atrial fibrillation requiring amiodarone, and was discharged to home on hospital day 9 with VNA services. He had minimal blood flow to his left kidney at time of discharge but had normal right kidney perfusion. His peak creatinine was slightly elevated at 1.64 mg/dl, but normalized at the time of discharge.

FINAL DIAGNOSIS: Type A Aortic Dissection

References


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