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Painless Aortic Dissection in a Patient with Atrial Fibrillation: A Case Report

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Abstract

Thoracic aortic dissection is a potentially life-threatening condition with an associated high mortality rate if unrecognized. Historically, medical training and clinical practice have emphasized having a high index of clinical suspicion for thoracic aortic dissection when patients present with ripping or tearing chest or back pain that may be coupled with unequal pulses or blood pressures in the upper extremities. The sensitivity for this presentation approaches 90% and is possibly improved by the addition of a new neurologic deficit or widened mediastinum on chest x-ray [1]. Painless aortic dissections, however, present a particularly challenging subset of aortic dissections because they do not present with these classic symptoms. Yet, they are just as deadly and comprise a notable portion of all aortic dissection cases. We present a case involving painless aortic dissection identified serendipitously while evaluating a patient presenting with atrial fibrillation and dyspnea.

Keywords: Aortic Dissection; Painless Aortic Dissection; Diagnostic Heuristics; Atypical Presentations.

Introduction

Aortic dissections occur when a tear in the aortic intima allows blood to pool within a newly formed "false" lumen. As blood flows through the aorta, the constant pressure causes the dissection to expand, sometimes affecting branching arteries and resulting in a variety of clinical symptoms. Each year, dissections occur in 5 to 30 people per million, accounting for approximately 10,000 deaths annually in the U.S. [2,3]. The Stanford system classifies aortic dissections into Type a (TAAD) and Type B (TBAD). TAAD involves the ascending aorta up to the aortic isthmus, while TBAD affects the descending aorta distal to the isthmus. Both types carry a high mortality rate without proper treatment. TAAD mortality increases by 0.5% to 2% per hour [3-5], peaking at about 50% [6,7]. TBAD dissections have an in-hospital mortality of 9-10% [8,9]. Given the high mortality risk, rapid diagnosis and management are crucial. While a cross-sectional CT scan remains the gold standard for diagnosis [10], clinicians must quickly and accurately determine who needs a scan. Effective heuristics are essential for this determination and require assessing risk factors and presenting symptoms.

Non-modifiable risk factors include age, male sex, genetic connective tissue disorders (e.g., Marfan syndrome), bicuspid aortic valve, and a family history of aortic disease. The main modifiable risk factors are hypertension, smoking, and atherosclerosis [11]. TAAD typically presents with severe, tearing, ripping, or pulsating chest pain radiating to the back, while TBAD often presents with severe abdominal pain and a wider array of symptoms. Unfortunately, many dissections don't present

in the stereotypical manner and, notably, up to 10% of patients experience painless dissections [6]. As a result of the heterogeneity in clinical presentation, only 15-43% of acute aortic dissections are immediately recognized [6]. Because of this, clinicians need a broader notion of common TAAD and TBAD symptoms to prevent delays in diagnosis.

This is challenging because the symptoms of an aortic dissection can be a function of the specific vascular territory affected. For example, instead of pain, a dissection crossing the carotid arteries present as syncope, as an altered mental status, or with focal neurologic deficits due to decreased cerebral blood flow [12], all of which mimic cerebrovascular events. This can lead to inappropriate anticoagulant therapy, which can be fatal in aortic dissection cases. Therefore, clinicians must use diagnostic heuristics beyond the "classic" symptoms to raise their suspicion for aortic dissections, as delays and misdiagnosis can be life threatening.

Case Report

A 90-year-old female was referred to the Emergency Department after her allergist noted an irregular heart rate on examination. The patient had been short of breath for the past three days and had difficulty walking short distances. She also endorsed episodic pleuritic chest tightness and a "chest fluttering," although she denied these symptoms at that present moment. She denied any history of atrial fibrillation, atrial flutter, or any other dysrhythmias; though she was told, she had a heart murmur but was unsure if it was ever checked out. Review of systems was otherwise positive for fatigue, chest tightness, palpitations, and dysuria. In the ED, the patient was tachycardic to 107 but had a pulse rate of 90. Her BP was 110/90 and SPO2 was 92% on room air. Her temperature was normal at 36.4ºC and she denied any current pain. The physical examination was notable for an irregularly irregular rhythm and ECG confirmed presence of atrial flutter. Atrial flutter in conjunction with shortness of breath raised concern for a pulmonary embolism and acute coronary syndrome, so a troponin and D-Dimer were obtained. The troponin was elevated at 29 ng/L and the D-Dimer was markedly elevated at 6512 ng/mL, which prompted a CT-PE study. While no PE was found, there was an unexpected ascending aortic aneurysm that began near the aortic root and extended nearly 47 mm in length. Concerning, irregularities of the root and a moderate pericardial effusion suggested a presence of a ruptured dissection leaking into the pericardial sac (Figures 1&2).

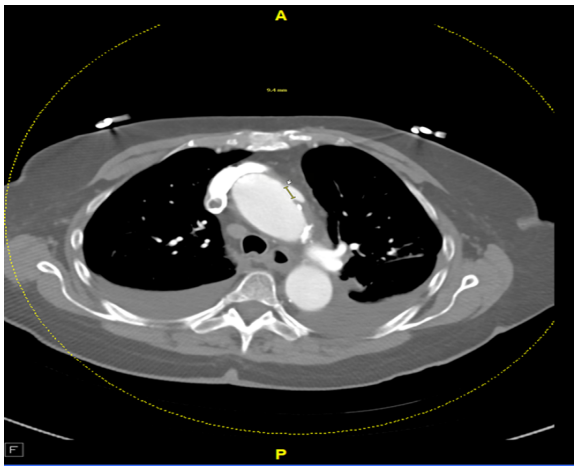

Figure 1: CT-PE with contrast showing ascending aortic aneurysm with aortic root irregularities concerning for aortic dissection versus pseudo aneurysm formation.

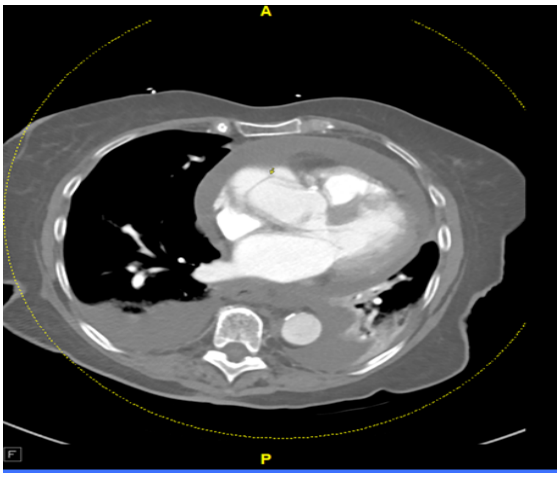

Figure 2: CT-PE with contrast illustrating pericardial effusion secondary to ruptured aortic dissection leaking into the pericardial sac.

The ED provider consulted cardiothoracic surgery, who decided the patient wasn't a candidate for surgery given her age and comorbidities, and she was subsequently admitted to the ICU for medical management. In the ICU, she remained on esmolol and a nicardipine drip to maintain permissive hypotension. After consulting family and discussing with the patient, she was transitioned out of the ICU and into hospice care. Two days after presenting to the ED, the patient was discharged with instructions to follow up with her PCP and palliative care.

Discussion

Painless aortic dissections (PAoD) represent a particularly challenging subset of aortic dissections due to the absence of the classic symptoms of severe chest pain, which can delay diagnosis and treatment. Up to one-third of dissections are painless and, while the lack of pain could be caused by a combination of factors unique to each patient, several leading hypotheses have emerged to explain this phenomenon. First, the adventitia is the only layer of the aorta that is innervated [13], such that if the dissection does not affect the adventitia, there will be no pain. Second, 76% of PAoDs cause some form of neurologic deficit [3] that is likely secondary to either hypotension or dissection of the arteries supplying the brain. Cerebral ischemia in aortic dissections is a well-documented phenomenon and produces stroke-like symptoms in about 30% of all aortic dissections [14, 15, 16]. If this cerebral ischemia affects areas of the brain involved in pain perception (e.g., thalamus, somatosensory cortex), a patient may not be able to perceive the classic tearing chest pain, which explains why roughly two-thirds of all PAoDs with neurologic deficits present without pain [17]. Notably, PAoDs can occur in the descending aorta if the dissection causes spinal cord ischemia, and there are multiple documented cases of painless TBAD affecting the anterior spinal artery and presenting as painless paraplegia [18]. Finally, many patients with PAoDs have an underlying chronic condition or previous aortic aneurysm that degrades the vessel wall and alters pain perception. This is most commonly caused by some mix of hypertension (62%), previous aortic aneurysm (9%), diabetes (7%), and smoking (7%) [3].

While often non-specific, clinicians can use a combination of clues found on history, physical examination, labs, and chest x-rays and bedside ultrasound to help make the diagnosis of PAoD. The most common abnormalities in physical examinations are asymmetric peripheral pulses and a heart murmur. Twenty-nine percent of patients have a right-left asymmetry, and 24% have an upper-lower asymmetry. Additionally, 88% of PAoDs are TAADs [3], and the physical examination findings mirror this. On auscultation, 30% of patients have an aortic regurgitation murmur as the aortic valve is commonly affected. Additionally, the proximity to the heart in TAADs increased the risk of cardiac tamponade, which is seen in about 12% of PAoDs, and, if present, carries an 83% mortality rate [3]. As such, 11% of patients have diminished heart sounds, and 2% present with a pericardial rub. In total, abnormal heart sounds are seen in 52% of patients with PAoD [3], which is much greater than the general population, in which only 10% have abnormal heart, sounds [19]. While there are no labs specific for aortic dissections, the most sensitive is a D-Dimer. As blood pools in the false lumen, stasis allows coagulation to occur. Because of this, D-Dimers have a negative predictive value of 97% when assessing for aortic dissections [3]. On chest x-ray, PAoDs can show a wide array of abnormalities, including mediastinal widening (35%), tortuous aorta (23%), and cardiomegaly (12%).Finally, while ECGs are normal in 71.4% of PAoDs, dissections can be associated with atrial dysrhythmias, supraventricular tachycardia, and complete heart block [20].

Conclusion

Patients with aortic dissections may not present with the classic tearing chest pain, and as shown by this case, some may not experience any pain at all. Recognizing the pathology's diverse array of symptoms is crucial for timely diagnosis and treatment. Clinicians must maintain a high index of suspicion for aortic dissections in the appropriate clinical context, even in the absence of specific historical or physical examination findings, as any delay or misdiagnosis could be catastrophic.

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