CLINICAL VIGNETTE

Acute Aortic Dissection Diagnosis in the Cath Lab

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Introduction

Aortic dissection, the detachment of the intimal layer of the aorta, is a life-threatening condition with an incidence of 3-5 cases per 100,000 people in the United States annually.[1] Aortic dissections are most commonly classified as Stanford "type A", indicating that the ascending aorta is involved, or "type B", which does not involve the ascending aorta. While both types may be life-threatening, type A is typically a surgical emergency. In type A dissections, separation of the intimal layer of the aorta may result in obstruction of blood flow in the proximal aorta and its branches. This has high in-hospital mortality of 24% for patients presenting to the hospital. Morbidity is also high, with only 55% of surviving patients returning to independent living.[2]

Early identification and treatment of aortic dissection is essential in improving outcomes. However, diagnosis may be challenging as it is uncommon and the presenting symptoms — chest pain, back pain and hemodynamic instability—significant overlap with other, more common, life-threatening diagnoses. Correct diagnosis on initial presentation is missed in more than 30% of cases.[2] When dissection is suspected, laboratory testing and chest x-ray may be helpful, but are often nondiagnostic. Coronary artery ischemia caused by dissection flap obstruction may present as an ST-elevation myocardial infarction (STEMI) on electrocardiogram (ECG). This often prompts emergent angiography, delaying the correct diagnosis of aortic dissection.

Due to the catastrophic consequences of acute aortic dissection and the difficulty in prompt diagnosis, a high index of suspicion is needed.

Case

A 66-year-old woman with untreated hypertension presented with chest and neck discomfort. These symptoms, along with malaise, had started in the morning and persisted for hours, when she presented to the emergency department at 7pm.

The patient was in distress, agitated, with difficulty breathing, and required pressors and transcutaneous pacing. Her vital signs were included blood pressure of 85/67 mmHg and heart rate of 41 bpm. Her cardiopulmonary physical exam was otherwise unremarkable. Initial ECG showed a junctional rhythm at 37 bpm (Figure 1A) with biphasic and inverted T-waves in the anterior, anterolateral and lateral leads.

The patient was intubated for airway protection and required transcutaneous pacing. Vasopressors were started and initial labs were significant for troponin-I of 19 ng/mL and potassium of 2.8 mmol/L. Pacing was stopped when her native heart rate increased to 105 bpm and repeat ECG showed sinus tachycardia, premature atrial complexes and inferior STEMI (Figure 1B).

She was promptly transferred to the cardiac catheterization laboratory. Once on the table, she experienced ventricular fibrillation arrest, necessitating defibrillation. A Judkins Left coronary catheter was placed in the aortic root and the first contrast injection revealed contrast stasis under a flap in the left coronary sinus. (Figure 2A) and the procedure was aborted. A computed tomography (CT) angiogram demonstrated a Stanford type A aortic dissection. (Figure 2) and cardiac surgery was consulted for emergent surgical repair. She underwent aortic root and arch replacement with aorta to innominate and carotid bypass grafting. Postoperatively, the patient remained in cardiogenic shock despite inotropes and pressors. She was placed on an extracorporeal membrane oxygenation circuit and a percutaneous microaxial left ventricular assist device was inserted for mechanical circulatory support. Unfortunately, she did not recover and died from shock on postoperative day three.

Discussion

Our patient experienced a type A aortic dissection. Correct diagnosis was initially missed. She presented with acute chest discomfort with dynamic ECG changes meeting STEMI criteria. The most likely diagnosis was STEMI, which required emergent intervention.[3] Her hemodynamic instability delated CT imaging and may have been unsafe. Chest radiographs are not diagnostic for aortic dissection but may have provided suspicious clues leading to the correct diagnosis, such as a widened mediastinum. Performing routine chest x-ray prior to presuming a diagnosis of STEMI remains a subject of debate.[4] ECG findings can also be confusing. In our patient an intimal flap from her dissection would transiently obstruct blood flow to her coronary artery, leading to myocardial ischemia and dynamic ECG changes (Figure 1). Although this finding has been well described in acute aortic dissection, it may also occur in STEMI from coronary plaque rupture.

In our case, the diagnosis of aortic dissection was made in the cath lab by angiography. This is not ideal for several reasons.

First, the transportation, set up and operator time required for this invasive procedure costs valuable time, during which arrangements for necessary surgery could have been started. Second, the introduction and manipulation of wires, catheters and other devices into the aortic root has the potential to exacerbate a dissection. Finally, although coronary angiography is routine prior to most cardiac surgeries, it is not indicated before emergent repair of the aortic root.

Ideally, the diagnosis of the aortic dissection could be suspected based on risk factors and presentation. Although a chest x-ray may be informative, a definitive diagnosis is typically made with CT imaging or transesophageal echocardiography. A high index of suspicion must be maintained to consider aortic dissection. When suspected, emergent noninvasive imaging should be performed prior to invasive angiography.

Type A aortic dissection have high mortality and morbidity, even under ideal circumstances with best management. Low clinical threshold is needed to evaluate patients presenting with chest discomfort. Although the diagnosis may be made by angiography, this is not ideal. As with most patients with type A aortic dissection, our patient did poorly.

Figures

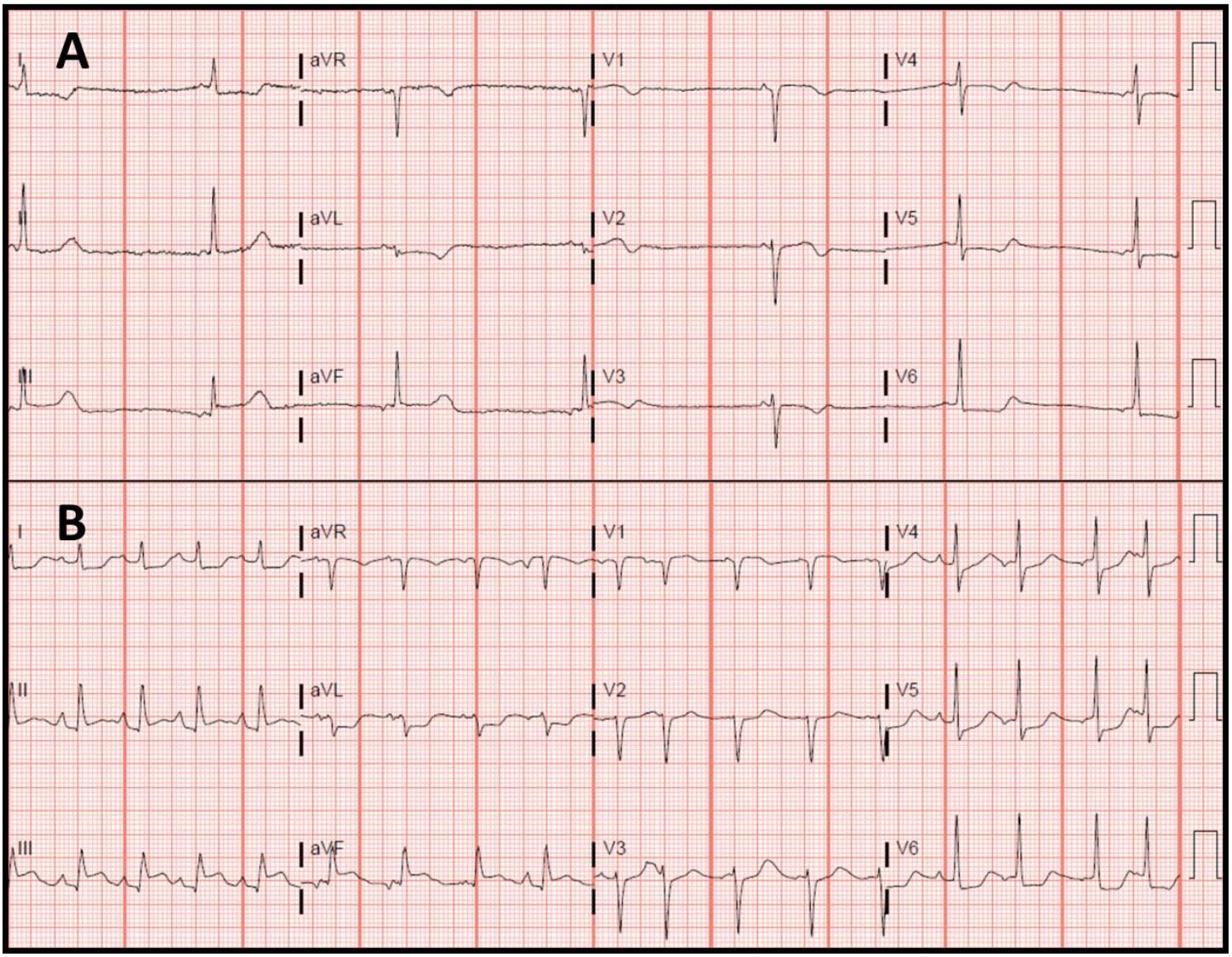

Figure 1. The patient's ECG findings. The presenting rhythm (A) was junctional bradycardia with ST changes. The patient then entered a rhythm of sinus tachycardia with inferior STEMI.

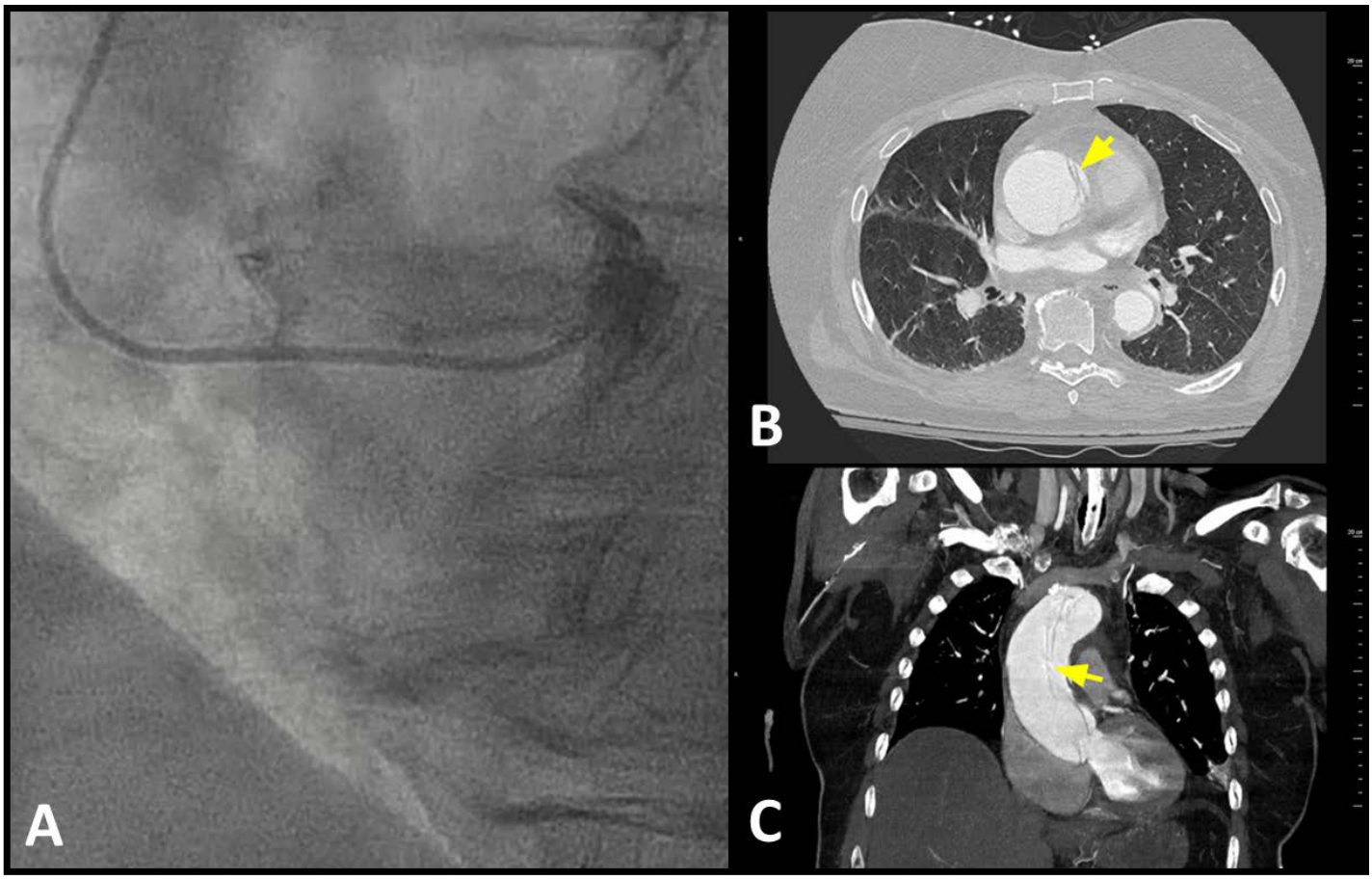

Figure 2. Imaging findings in acute Stanford type A aortic dissection. Initial diagnosis in this case was made during invasive angiography (A), which shows contrast behind an intimal flap after injection through a Judkins Left catheter. The aortic dissection flap (arrow) is clearly seen on CT imaging, shown here in axial (B) and coronal (C) reconstruction.

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