Stanford Type a Aortic Dissection Presenting with Dyspnea: A Case Report

Parinaz Ayat, Bridget Ayinbono Azera, Suzette Blondelle graham-Hill, Andrea Trimmingham, Samy I. McFarlane*

Department of Internal Medicine, State University of New York: Downstate Medical Center, Brooklyn, New York, United States- 11203

*Corresponding author: Smcfarlane@downstate.edu

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Abstract Acute Aortic dissection is relatively uncommon but can lead to fatal outcome due to misdiagnosis and/or delay treatment [1]. In this report we present a case of a 45-year-old man presenting with chief complaint of substernal chest pain with no remarkable laboratory and echocardiography finding. He was admitted to the cardiology service with clinical suspicion of acute coronary syndrome (ACS). However, further evaluation led to the diagnosed of acute aortic dissection and referral for urgent repair. Aortic dissection could mimic other disorders such as ACS and pulmonary embolism due to variation in the presenting symptoms [1]. Therefore, high clinical suspicious could lead to timely diagnosis and initiation of life-saving therapeutic interventions.

Keywords: chest pain, aortic dissection, acute coronary syndrome, atypical presentation, Stanford classification type A

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1. Introduction

Acute aortic dissection is relatively uncommon condition and could represent a challenge due to its varying initial clinical presentations with symptoms and signs that overlap with other entities, while at the same time it is crucial to make a prompt diagnose and management [2]. Abrupt onset chest pain is the most common presenting symptoms and mostly describes as sharp, tearing, ripping and excruciating with the maximum severity at the time of onset [1]. However, atypical presentations such as painless aortic dissection, and cardiac arrhythmias have also been also reported3. Here, we presented a case of Stanford type A aortic dissention with dyspnea and atypical chest pain.

2. Case Presentation

45-year-old obese man with hypertension, type 2 diabetes mellites and coronary artery disease presented to the emergency department with complain of shortness of breath started about 12 hours before presentation. It was accompanied by retrosternal chest pain described as sharp pain with the scale of 8-9/10, with no radiation, no alleviating or aggravating factors. He also had complained of nausea, but denied vomiting, abdominal pain, dizziness, diaphoresis, back pain, hematuria, or headache and no fever/chills. In the emergency department (ED), he was hemodynamically stable with the following vital signs:

- Blood pressure (BP) 150/95
- Heart rate (HR) 40-50bpm, sinus bradycardia
- Temperature (T) 98F
- Respiratory rate (RR) 22
- Oxygen saturation (O2S): 98-100% on room air

He did not have any remarkable finding on the physical exam:

- General: awake, no acute distress, lying on bed
- Cardiovascular: S1, S2 regular, no murmur or gallop
- Pulmonary: no accessory muscle use, clear to auscultation bilaterally, no wheezing or crackles.
- Abdomen: obese, bowel sounds positive in four quadrants, soft, not distended, not tender, negative McBurney’s and Murphy’s sign.

- Extremities: radial and posterior tibialis pulses are full and symmetric bilaterally. Capillary refill is less than 2-3 seconds.
- Neurology: No focal neurology sings, motor and sensory are grossly intact.

Laboratory test including complete blood count (CBC), comprehensive metabolic panel (CMP), and troponin level are as below:
Chest x-ray (CXR) taken as portable, with report of enlarged cardiac silhouette, pulmonary vascularity within normal limits and no consolidation or effusion (Image 1).

Electrocardiograph (ECG) showed sinus bradycardia with ST segment elevation (STE) in leads aVR and V1. (Image 2)
Cardiology service consulted immediately, after evaluating the patient, bedside Echocardiogram showed hypokinetic infero-septal wall with preserved LVEF and flattening of the septum and patient brought to the catheterization laboratory to rule out acute coronary syndrome.

Left heart catheterization (LHC) showed clean coronaries with left ventricle end diastolic pressure (LVEDP) 39mmHG and left ventriculogram (LV) showed 40-45%. Patient received Aspirin (ASA) 324mg, Plavix 600mg and Heparin drip for full dose anticoagulation therapy. Post procedure patient still complaining of chest pain with no change, also he had been having multiple non-bloody, non-bilious vomiting. Zofran for vomiting and Morphine for pain provided, and patient sent for chest computed tomography angiography (CTA) to rule out possible pulmonary emboli (PE). PE was ruled out but study showed Type A aortic dissection extending from the aortic root through the entire visualized portion of the aorta. Dilated ascending aorta to 4.3 cm. The right brachiocephalic artery is perfused by the false lumen. The left common carotid left subclavian and left vertebral artery are perfused by the true lumen. (Image 3 - Image 4)

Heparin drip immediately discontinued, as patient found to be hypertensive with SBP 160, Nitroglycerin drip started, and patient transferred to outside facility for further urgent management.

3. Discussion

Aortic dissections are classified based on the Stanford Classification system. This system divides dissections based on anatomical evolvement into two groups, Type A and B. The Stanford type A means dissection involves the ascending aorta and/or aortic arch which is associated with higher mortality [4]. Type A is seen more with painless presentation [5], with syncope, congestive heart failure (CHF), and stroke being more frequent presenting symptoms in this group [6]. Most of the aortic dissection with painless presentation has normal electrocardiogram (ECG) [7], but different types of arrhythmias such as atrioventricular block, atrial fibrillation, supraventricular tachycardia, ventricular bigeminy and even complete heart block has been reported [2].

Type A aortic dissection can cause coronary perfusion impairment due to mechanical obstruction in the coronary ostia, without extension of the dissection into the coronary arteries [8].

Treatment for ascending aorta and aortic arch aneurysms could be challenging and progression to more severe complications like acute aortic dissection and aortic rupture are common [9].

To have a high clinical suspension and to avoid missed diagnosis, the first step is to assess risk factors for aortic dissection through history taking [1]. We should consider patient's underlying disease such as hypertension, or atherosclerosis, and history of collagen diseases such as Marfan syndrome and Ehlers-Danlos syndrome, bicuspid aortic valve, aortic coarctation, and Turner syndrome, also iatrogenic causes of aortic dissection include cardiac catheterization, angioplasty, or cardiac surgery could be the precipitating factor. It is also crucial to keep in mind that family history may play a role [10].

In this case, patient presented with retrosternal chest pain, with the picture more similar to ACS, although pain in acute aortic dissection may change its location with further extension of dissection, his pain location did not change.

In contrast, the pain associated with acute ACS starts slowly, gains intensity with time, and its character is usually more oppressive and dull [11]. In this case patients’ initial symptom was dyspnea started twelve hours prior to presentation, so a physician must always suspect aortic dissection because atypical presentations can be the case, and also aortic dissection could be accompanied by ACS. Physician must keep in mind that painless aortic dissection has been reported with an incidence of 6.4% to 17% [1,12]. Uncommon symptoms include syncope, CHF symptoms, cardiac tamponade, lower extremity weakness, paraplegia, mesenteric ischemia, or peripheral ischemia may also happen [1].

Physical findings on examination are important, blood pressure (BP) should be taken from both left and right arms, or legs. Thirty five percent on the proximal dissections and Seventy percent of distal dissections found to have high BP [13], also less than twenty percent of patients could have pulse deficit or pulse difference due to intimal flap or compression by hematoma [13], and about fifty percent of patients with proximal aortic dissection have diastolic murmur due to aortic regurgitation [1].
Neurologic examinations are also important, to evaluate neurological deficits such as loss of consciousness and ischemic paresis. These findings may happen in about forty percent of patients with proximal aortic dissection [14].

In this case, we provided intravascular nitroglycerin to lowers preload and therefore reduces wall stress, resulting in a decrease in myocardial oxygen demand, decreased chest pain and blood pressure.

ECG cannot be used as a diagnostic tool for aortic dissection, that is because nearly thirty one percent of aortic dissection could present with normal ECG. However, an ECG must be performed on all patients with chest pain for the differential diagnosis of acute myocardial infarction of aortic dissection, and to diagnose possible combined aortic dissection and myocardial infarction [1,15].

As we expect to see mediastinal widening on chest X-ray (CXR) in the presence of aortic dissection, just in around sixty to ninety percent of cases, abnormal CXR findings such as abnormal aortic contour, mediastinal widening, or aortic shadow widening can be seen [13].

Diagnostic imaging such as computerized tomography (CT), magnetic resonance imaging (MRI), transthoracic echocardiography (TTE), or aortography must be performed to have definitive diagnosis, with CT being the most often used modality to diagnose aortic dissection because of its availability, high specificity and sensitivity, however it has its limitations since it cannot detect aortic regurgitation. MRI also has high specificity and sensitivity, but the test is time consuming and less available, and TEE has this benefit that it can be used for hemodynamically unstable patients since it can be performed at the bedside or in an operating room for emergency [11].

In this report, we emphasize the importance of clinical suspicion of aortic dissection, patient could present in the emergency department, or even to the outpatient clinics, with various kinds of presenting symptoms as we explained. The clinical key is that always be aware of aortic dissection even though the patient presents only with chest pain that might be similar to chest pain of ACS.

To have a better approach, American Heart Association presented guidelines with a risk assessment tool which could be used to stratify patients from low risk to high risk [16], therefore physician can apply additional diagnostic methods based on the probability of the disease.

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