American Journal of Medical Case Reports, 2021, Vol. 9, No. 3, 180-183
Available online at http://pubs.sciepub.com/ajmcr/9/3/12
Published by Science and Education Publishing
DOI:10.12691/ajmcr-9-3-12

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@downstte.edu

Received December 02, 2020; Revised January 05, 2021; Accepted January 14, 2021

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*

Cite This Article: Parinaz Ayat, Bridget Ayinbono Azera, Suzette Blondelle graham-Hill, Andrea Trimmingham, and Samy I. McFarlane, "Stanford Type a Aortic Dissection Presenting with Dyspnea: A Case Report." *American Journal of Medical Case Reports*, vol. 9, no. 3 (2021): 180-183. doi: 10.12691/ajmcr-9-3-12.

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
Hear 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:

CBC		CMP			
WBC	6.81	Total protein	6.1	Na	140
RBC	4.92	Albumin	3.84	K	2.1
Plt	175	ALT	22	Cl	93
Hb	15.5	AST	28	HCO3	36
HCT	46.3	Alk	54	BUN	17
		Calcium	9.5	Cr	1.5

COVID-19 negative	T. Bili	1.3		
Miscellaneous				
Blood glucose	150	Troponin		0.08
BNP	84	Lactic acid		3.8

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).

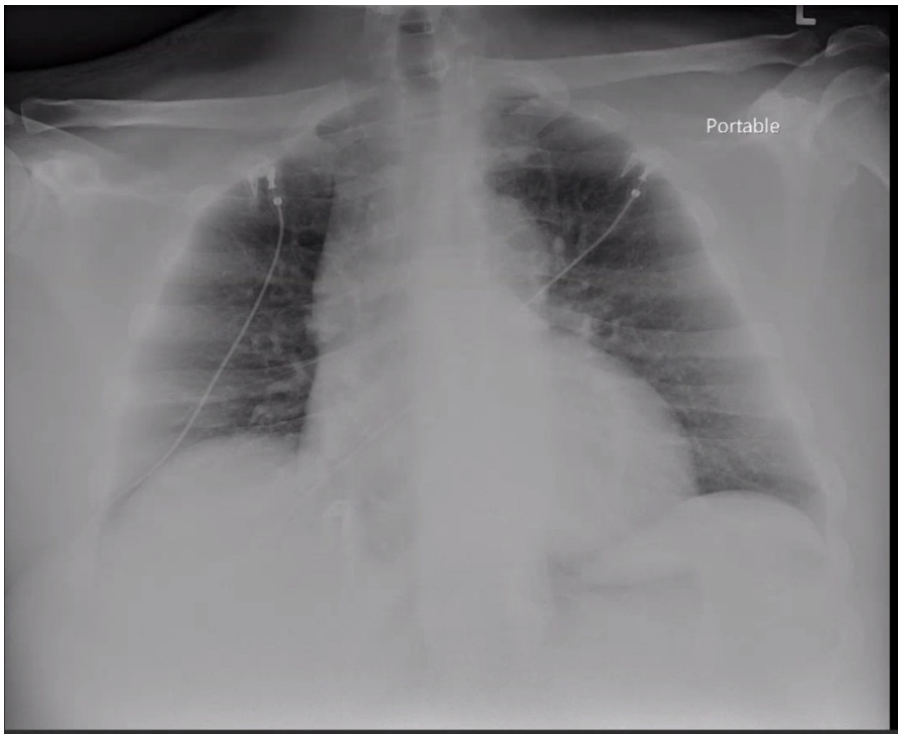

Image 1. CXR: enlarged cardiac silhouette and widen mediastinum.

Electrocardiograph (ECG) showed sinus bradycardia with ST segment elevation (STE) in leads aVR and V1. (Image 2)

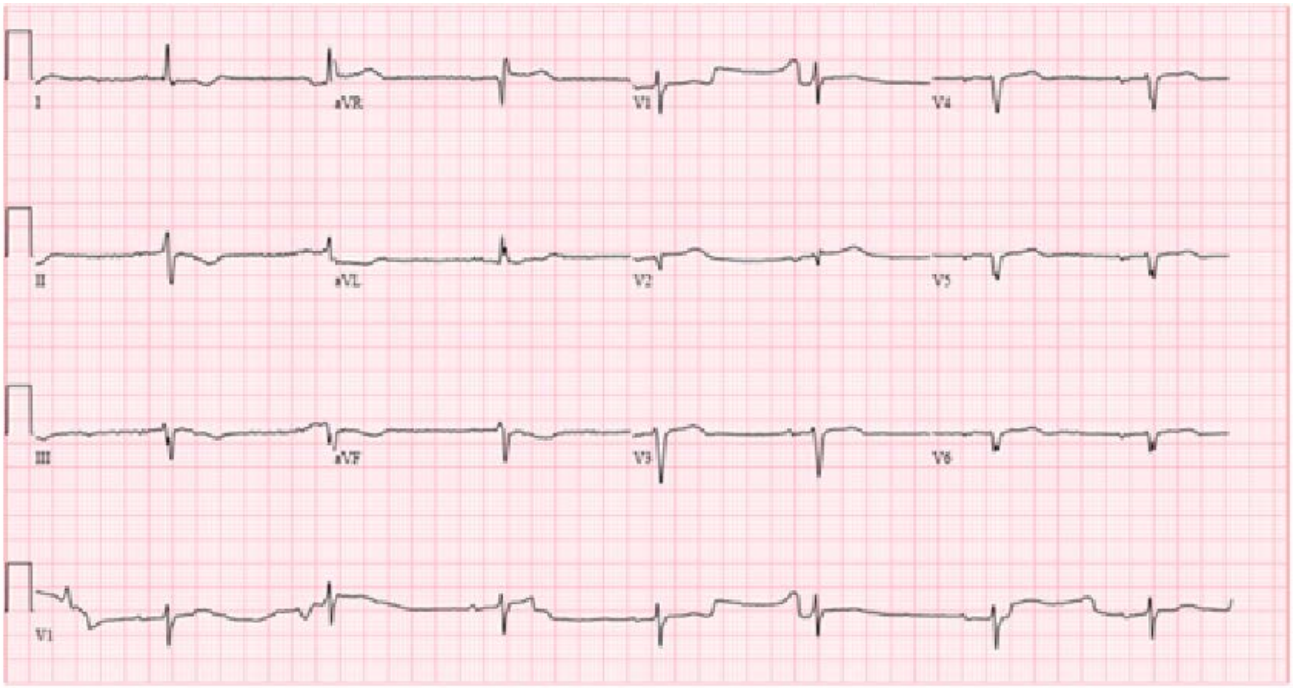

Image 2. Patient's first ECG at the ED, with STE on lead aVR and V1.

Cardiology service consulted immediately, after evaluating the patient, bedside Ecocardiogram 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 transfer to outside facility for further urgent management.

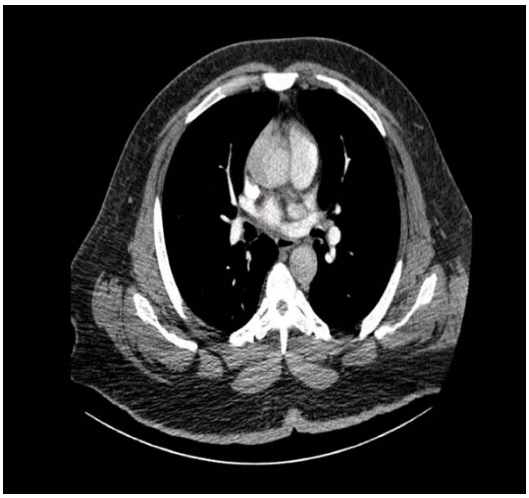

Image 3. Dissection in ascending aorta indicating Stanford type A dissection

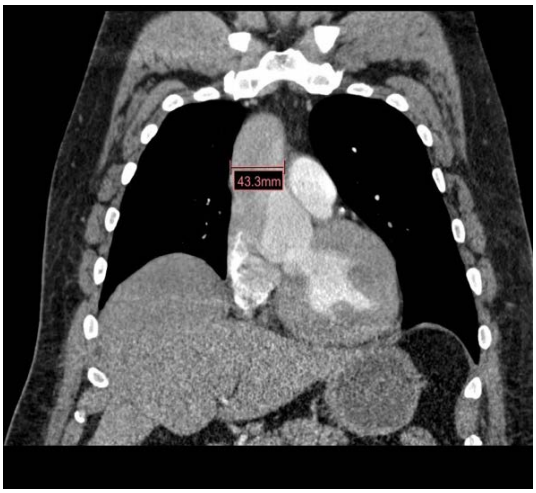

Image 4. Dissecting aorta with increased diameter

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 factior[1]. 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), transesophageal 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.

Acknowledgements

This work is supported, in part, by the efforts of Dr. Moro O. Salifu MD, MPH, MBA, MACP, Professor and Chairman of Medicine through NIMHD Grant number S21MD012474.

References

[1] Hyun Joo Kim, Hyun-Ki Lee, and Belong Cho, A Case of Acute Aortic Dissection Presenting with Chest Pain Relieved by Sublingual Nitroglycerin, Korean J Fam Med. 2013 Nov; 34(6): 429-433. PMID: 24340165.

[2] Chun-Hsien Chen, et al. A case report of painless type A aortic dissection with intermittent convulsive syncope as initial presentation, Medicine (Baltimore). 2017 Apr; 96(17): e6762. PMID: 28445305.

[3] Hagan PG, Nienaber CA, Isselbacher EM, et al. The International Registry of Acute Aortic Dissection (IRAD): new insights into an old disease. JAMA 2000; 283: 897-903.

[4] Daily PO, Trueblood HW, Stinson EB, et al. Management of acute aortic dissections. Ann Thorac Surg1970; 10: 237-47.

[5] Park SW, Hutchison S, Mehta RH, et al. Association of painless acute aortic dissection with increased mortality. Mayo Clin Proc 2004; 79: 1252-7.

[6] Gaul C, Dietrich W, Friedrich I, et al. Neurological symptoms in type A aortic dissections. Stroke2007; 38: 292-7.

[7] Liu ZY, Zou YL, Chai BL, et al. Analysis of clinical features of painless aortic dissection. J Huazhong Univ Sci Technol Med Sci 2014; 34: 582-5.

[8] Lardi C, Lobrinus JA, Doenz F, et al. Acute aortic dissection with carotid and coronary malperfusion: from imaging to pathology. Am J Forensic Med Pathol 2014; 35: 157-62.

[9] Leontyev S, et al. Aneurysms of the ascending aorta and aortic arch, Chirurg. 2014 Sep; 85(9): 758, 760-66.

[10] Hasham SN, Lewin MR, Tran VT, Pannu H, Muilenburg A, Willing M, et al. Nonsyndromic genetic predisposition to aortic dissection: a newly recognized, diagnosable, and preventable occurrence in families. Ann Emerg Med. 2004; 43:79-82.

[11] Erbel R, Alfonso F, Boileau C, Dirsch O, Eber B, Haverich A, et al. Diagnosis and management of aortic dissection. Eur Heart J. 2001; 22: 1642-1681.

[12] Park SW, Hutchison S, Mehta RH, Isselbacher EM, Cooper JV, Fang J, et al. Association of painless acute aortic dissection with increased mortality. Mayo ClinProc. 2004; 79: 1252-1257.

[13] Hagan PG, Nienaber CA, Isselbacher EM, Bruckman D, Karavite DJ, Russman PL, et al. The International Registry of Acute Aortic Dissection (IRAD): new insights into an old disease. JAMA. 2000; 283: 897-903.

[14] Fann JI, Sarris GE, Mitchell RS, Shumway NE, Stinson EB, Oyer PE, et al. Treatment of patients with aortic dissection presenting with peripheral vascular complications. Ann Surg. 1990; 212: 705-713.

[15] Spittell PC, Spittell JA, Jr, Joyce JW, Tajik AJ, Edwards WD, Schaff HV, et al. Clinical features and differential diagnosis of aortic dissection: experience with 236 cases (1980 through 1990) Mayo Clin Proc. 1993; 68: 642-651.

[16] Rogers AM, Hermann LK, Booher AM, Nienaber CA, Williams DM, Kazerooni EA, et al. Sensitivity of the aortic dissection detection risk score, a novel guideline-based tool for identification of acute aortic dissection at initial presentation: results from the international registry of acute aortic dissection. Circulation. 2011; 123: 2213-2218.