Case Report

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A Case of Aortic Dissection Presenting as CVA with Pulmonary Embolism

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Abstract

Aortic dissection is a life threatening condition of the aorta. Early diagnosis and management can reduce the patient's morbidity and mortality; hence one should have a high index of suspicion for this condition. This case reports a 53 yrs old male presenting with headache, slurring of speech, Rt leg weakness with dyspnoea and sweating. A patient of aortic dissection can present in many ways depending on the site involved and may mimic other disorders like ACS, PE or CVA. Therefore, clinicians must always be aware of aortic dissection and its different clinical manifestations.

Keywords: Aortic dissection; Pulmonary Embolism.

Introduction

Acute aortic dissection is a rare but potentially life-threatening condition. It is more prevalent in men and in patients with advanced age, with approximately 75% occurring in patients between 40 and 70 years of age. Younger patients with aortic dissection usually have a history of an underlying connective tissue disease. Of note, about half of all aortic dissections in women under the age of 40 years occur in the third trimester or early postpartum period.

Risk factors for acute aortic dissection include chronic hypertension, a bicuspid aortic value, coarctation of the aorta, or inherited connective tissue disorders such as Ehlers- Danlos and Marfan syndromes. Vascular inflammatory disorders such as giant cell arteritis or Takayasu arteritis are additional risk factors for dissection.

Aortic dissection results from a tear in the intimal layer of the vessel wall. Common inciting factors include the chronic conditions listed previously, as well as illicit drug use or blunt thoracic trauma.

The classic presentation of an acute thoracic aortic dissection is that of a 55- to 65-year-old male with chronic hypertension who develops a sudden onset of severe sharp or tearing chest pain radiating to the intrascapular area. Keep in mind that this is a fairly rare condition that often presents in an atypical manner.

Case Report

Pt a 53 yrs old came to ER with c/o headache with Rt lower limb weakness with excessive sweating and breathlessness 1 hr before arrival to the ER.

Pt on arrival had a BP of 70/40 in rt arm with a pulse of 152/min, the rhythm being Sinus Tachycardia and an SpO_2 of 72% on room air with a Respiratory rate of 34/min.The chest on auscultation had B/L basal crepts . Pt was immediately started on IV Fluids and put on Non-Re breather mask with O_2 at 15 L/min.

SpO_2 increased to 85% with a BP of 80/50. ECG at this time showed ST Elevation in aVr with ST depression in lateral leads. (Figure 1). Decision to intubate the pt was taken due to his increasing respiratory efforts.In Suspicion of PE and ACS urgent cardiology consultation was taken and 2D ECHO revealed No RWMA ,Normal LV and RV contractility with trace MR, No AR and EF-60%. Chest X-ray should widening of Mediasfinum with prominent Aortic knuckle and pulmonary edema. (Figure 2).

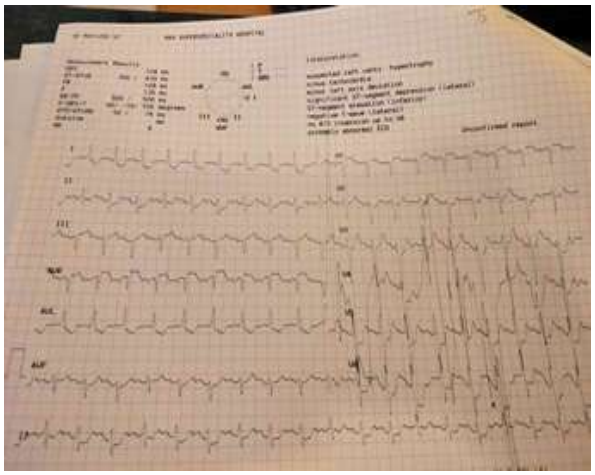

Fig. 1: Showing ST elevation in AVR & ST depression in Lat Leads

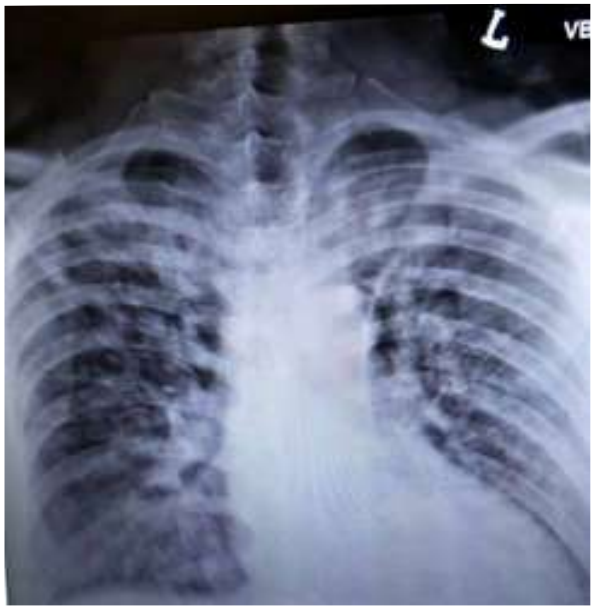

Fig. 2: X-ray showing widened mediasfinum

Pt was shifted for CT Brain to rule out any CVA which showed no lesion in the brain. While shifting the pt back from the CT Room to ER we noticed a pulsatile mass in his abdomen. Pt was Immediately brought to ER and his B/L vascular status and vitals were reassessed.

The pulses in both the upper limbs were different,with left being more pulsatile than right and BP on rt side was 80/50 mm hg, and left side was 130/80 mm hg, A femoral arterial line was secured on rt side, which showed BP-110/70 mm hg and patient was given 10 mg metoprolol.

In strong suspicion of Aortic dissection, patient was shifted to CT room for CT Aortogram, which showed:

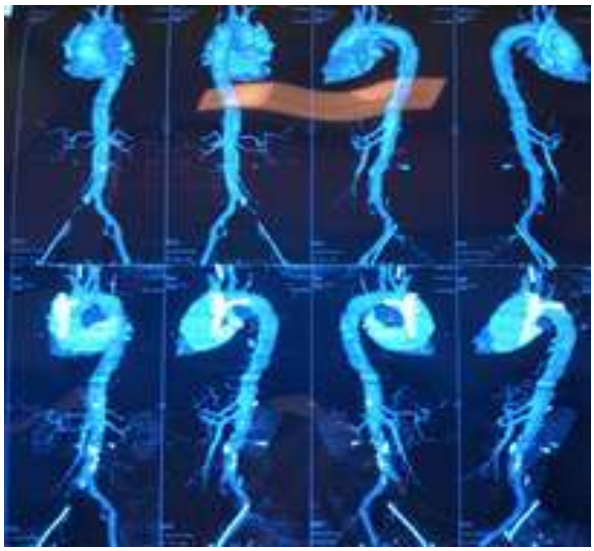

Fig. 3: Sagittal section showing extent of dissection

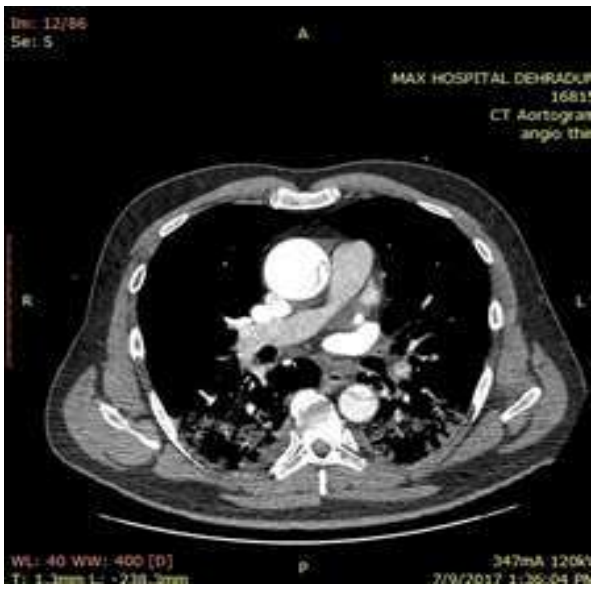

Fig. 4: Dissection starting from Aortic Root

Aortic dissection,involving aortic root,ascending aorta, arch in descending aorta, extending cranially into the brachiocephalic trunk and left subclavian artery and extending caudally into b/l external illiac arteries (DEBAKEY TYPE 1 and STANFORD A).

Patient was immidiately was brought back to ER,with vitals were:

BP-100/70 MM HG

PR-120/MIN

SPO2-96% ON 100% FIO2

RR-20/MIN with full ventilatory support.

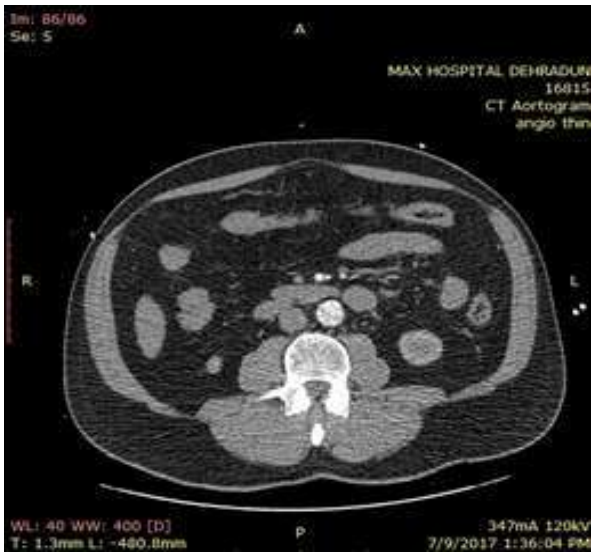

Fig. 5: Last slide on aortogram showing extent of dissection

The criticality of the case was discussed with the relatives of the patient and was advised to shift the pt to a cardiac surgery capable centre for further management but the pt expired on the way.

Discussion

Aortic dissection is one of the most rae and life threatening conditions of the aorta. The International registry of aortic dissection (IRAD) estimates its incidence to be 3 in a 1000 cases. If left untreated many die within the first 48 hrs [1]. Aortic dissection results from a tear in the intimal layer of the vessel wall. Common inciting factors include the chronic conditions listed previously, as well as illicit drug use or blunt thoracic trauma [2] High-pressure pulsatile blood will travel through this tear into the media layer of the aorta, thereby separating the intima from the adventitia. This creates a false lumen for aortic blood flow that can extend distally (antegrade) , proximally (retrograde) , or in both directions. Rarely, the false lumen will rupture through the adventitia, resulting in immediate hemodynamic collapse. The majority of aortic dissections originate in the ascending aorta (65%), the aortic arch (10%), or just distal to the ligamentum arteriosum (20%). The Stanford classification system divides aortic dissections clinically into types A and B. Type A dissections involve the ascending aorta, whereas type B dissections involve only the distal aorta (origin of the intimal tear is distal to the left subclavian artery) .

- Stanford type A (proximal) dissections typically require surgical intervention, whereas Stanford type B (distal) dissections are managed medically.
- Complications of acute dissection include myocardial infarction, cardiac tamponade, aortic valve insufficiency, stroke, renal failure, paralysis, limb ischemia, and death.

According to an IRAD review, the most common presenting complaint was pain. This pain is typically abrupt and intense from the onset. The site of the pain is often indicative of the site of dissection. Anterior chest pain is typical in ascending aorta dissection; neck and jaw pain may indicate dissection involving the arch and carotid arteries. Interscapular tearing pain suggests dissection of the descending aorta. (Figure 6).

Other symptoms are attributed to decreased organ perfusion. Focal neurological symptoms, including limb paresthesia and weakness, indicate involvement of spinal arteries. Evidence of limb ischemia or mesenteric ischemia suggests compromise of their respective arterial supplies. Symptoms can also be caused by organ compression by an expanding hematoma. Dyspnea suggests tracheal or bronchus compression, dysphagia results from esophageal

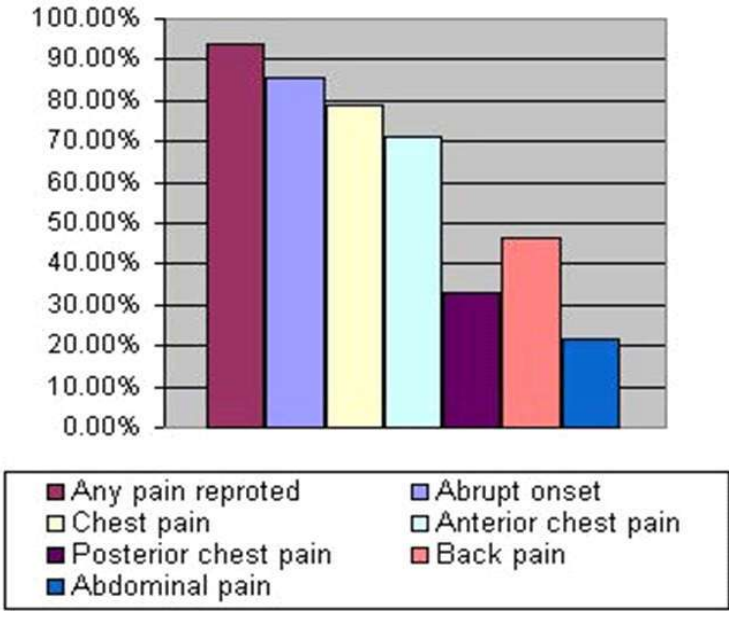

Fig. 6: Different characteristics of pain associated with Stanford type A dissection [1]

compression, and hoarseness indicates recurrent laryngeal nerve compression. A superior vena caval syndrome may be precipitated by compression of the superior vena cava.

Physical examination may reveal hypertension or hypotension, the latter being an ominous sign. Hypotension may be a sign of critical complications (e.g. cardiac tamponade, rupture of the aorta), but it can also be due to hypervagotonia. Pulse deficit (defined as weak or absent pulse in the carotid, brachial or femoral arteries) is more common in acute Stanford type A dissection compared to type B (19% versus 9% respectively) [3]. Pulse deficit is identified in 15% of cases and is generally associated with greater complications and mortality rates. A disparity of greater than 20 mm Hg in the systolic blood pressure of each arm also raises suspicion for aortic dissection.

A plain chest radiograph may provide important clues for the diagnosis. However, from 12% to 37% of patients have no abnormality, and this study should not be used to exclude dissection. The most common radiographic abnormality is a widened mediastinum or abnormal aortic contour. Other possible findings include pleural effusion, displacement of aortic intimal calcification, and deviation of the trachea, mainstream bronchi, or esophagus .

CT (especially multidetector-row CT) is the imaging modality of choice for diagnosis of dissection.CT can reliably identify a false lumen and can provide additional details such as the anatomy of the dissection, the location of the dissection flap, extension of the flap into great vessels, signs of aortic rupture, and signs of end-organ damage. CT protocols should be both with and without IV contrast. Invasive catheter angiography is rarely necessary.

CT may also diagnose intramural hematoma and penetrating atherosclerotic ulcer. Penetrating atherosclerotic ulcer can be difficult to distinguish from large atheromatous plaques. CT diagnosis of penetrating atherosclerotic ulcer depends on extension of the ulcer past the intima. Ulcers often have overhanging edges and focal outpouchings of the aorta itself. Intramural hematoma is often identified by a high-signal mass in the aorta on CT. This often appears as a crescent and is best seen on noncontrasted images.

In experienced hands, transesophageal echocardiography may be as sensitive and specific as CT. The procedure generally has to be performed under moderate sedation or even general anesthesia. Known esophageal disease is a relative contraindication. Sound transmission is disrupted by air in the trachea or left bronchia, which may make evaluation of the ascending aorta difficult. The accuracy and precision oftransesophageal echocardiography are highly operator dependent. MRI has been used to evaluate stable patients with suspected aortic disease.

Initial management of aortic dissection includes control of blood pressure and heart rate to decrease the shear forces on the dissected aorta. Intravenous beta blockers (e.g. labetalol or metoprolol) are the mainstay of medical treatment. If needed, vasodilatation with agents such as sodium nitroprusside or intravenous calcium channel blockers can be used. The goal of surgical treatment with type A aortic dissection is to alleviate symptoms, control complications and prevent aortic rupture.

Table 1: Comparison between imaging modality used for diagnosis of Type A aortic dissection [1]

Imaging technique	Sensitivity	Specificity
TEE	98 (95–99)	95 (92–97)
Helical CT	100 (96–100)	98 (87–99)
MRI	98 (95–99)	98 (95–100)

Conclusion

Aortic dissection is an uncommon but potentially life threatening condition. Reliable diagnostic modalities are available but High suspicion index remains key for early diagnosis and management. In our case the only possible explanation for such wide symptomatology could have been its extension into brachiocephalic trunk and subclavian artery. This case reminds us of how serious diseases can masquerade in vague complaints. Patients should never be profiled and all complaints should always be revisited.

References

1. Clouse WD, Hallett JW, Jr, Schaff HV, Spittell PC, Rowland CM, Ilstrup DM, et al. Acute aortic dissection: population-based incidence compared with degenerative aortic aneurysm rupture. Mayo Clin Proc. 2004;79:176–180. [PubMed].
2. Olsson C, Thelin S, Stahle E, Ekbom A, Granath F. Thoracicaortic aneurysm and dissection: increasing prevalence and improved outcomes reported in a nationwide population-based study of more than 14,000 cases from 1987 to 2002. Circulation. 2006; 114:2611–18. [PubMed].

3. Shirakabe A, Hata N, Yokoyama S, Shinada T, Suzuki Y, Kobayashi N, et al. Diagnostic score to differentiate acute aortic dissection in the emergency room. Circ J. 2008;72:986–90. [PubMed].
4. Braverman AC. Aortic dissection: prompt diagnosis and emergency treatment are critical. Cleve Clin J Med. 2011;78:685–96. [PubMed].
5. 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: 642–51. [PubMed].
6. 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. [PubMed].
7. Erbel R, Alfonso F, Boileau C, Dirsch O, Eber B, Haverich A, et al. Diagnosis and management of aortic dissection. Eur Heart J. 2001;22:1642–81 [PubMed].
8. Harris KM, Strauss CE, Eagle KA, Hirsch AT, Isselbacher EM, Tsai TT, et al. Correlates of delayed recognition and treatment of acute type A aortic dissection: the International Registry of Acute Aortic Dissection (IRAD) Circulation. 2011;124:1911–18. [PubMed].
9. Klompas M. Does this patient have an acute thoracic aortic dissection? JAMA. 2002;287:2262–72.[PubMed].
10. Hansen MS, Nogareda GJ, Hutchison SJ. Frequency of and inappropriate treatment of misdiagnosis of acute aortic dissection. Am J Cardiol. 2007;99:852–56. [PubMed].