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DISASTER WITHOUT A WARNING – AORTIC DISSECTION – CASE REPORT

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Abstract

Aortic dissection is a life threatening clinical entity. Aortic dilatation may lead to sudden tear of the layers in the aorta wall causing a dissection and thereby allowing blood to flow abnormally between the layers. These aortic abnormalities are potentially life threatening because they can decrease blood flow to other parts of the body such as the brain, heart, limbs or other vital organs, leading to symptoms similar to that of thrombo-embolic disease or even cause the aorta to break open (rupture).
We hereby present a case of 61 years old lady diagnosed to have a thoracic aorta aneurysm and developed an extensive Aortic dissection within 12 hours of detection of the aneurysm.

Keywords: *Aortic dissection and management, Thoracic Aortic aneurysm and Dissection, Asymptomatic aortic dissection, DeBakey classification, Stanford classification.*

Introduction

Acute aortic dissection is an uncommon clinical entity which pose a threat to life, whereby blood leaves the normal aortic channel through a discrete point causing an intimal layer tear and rapidly dissects into the inner layer of media from the outer layer, thereby producing a false channel. The extent and nature of aortic dissection (described below) and also the involvement of the branches determines the clinical symptoms in which the patients may present with stroke (cerebral hypo perfusion), Myocardial Infarction (cardiac hypo perfusion), loss of peripheral pulses, sensory dysfunction, or motor dysfunction (ilio-femoral or brachiocephalic hypo perfusion), acute kidney injury (renal hypo perfusion); paraplegia (spinal hypo perfusion), or abdominal tenderness, bowel ischemia (mesenteric hypo perfusion). Organ hypo perfusion caused by the dissection is responsible for substantial postoperative morbidity and mortality and therefore requires early, accurate diagnosis and prompt treatment which may prove to be life saving.

Case Report

A 61 years old lady presented to out patient clinic with complains of dyspnea on exertion (NYHA Class II) for the past 7 months. She had no complains of chest pain, heaviness, palpitations or radiating pain to nape of neck, epigastrium or shoulders. She had no complains of any thromboembolic events (stroke, MI, amaurosis fugax, limb ischemia, DVT, pulmonary embolism) in any other systems in the past. She had a past history of restrictive lung disease, chronic gastritis and hypertension for which she was on irregular medications.

General examination of the patient revealed a conscious, oriented and afebrile patient, with heart rate 85/min, B.P-150/70 mmHg in both the upper limbs and respiratory rate of 20/min. Cardiac examination showed regular sinus rhythm with S1, S2 heard in all areas and no murmurs, gallops or rubs were heard. Abdominal examination revealed soft, no rebound tenderness, no guarding, no masses, and positive bowel sounds. On palpation 2+ symmetrical pulses were felt in Carotid, Subclavian, Brachial, Radial, Ulnar Femoral, Popliteal, Anterior tibialis and Posterior tibialis with no bruit or thrill. Other systemic examinations were unremarkable.

Lab investigations were within normal limits. 2D Echo revealed aortic root dilatation measuring 4.6cm with no regional wall motion abnormality and LVEF of 50%. To verify the extent of the Aortic root dilatation she underwent a CT Angiogram, which revealed aneurysmal dilatation of the ascending aorta with Aorto-ventricular junction measuring 27.0mm, Aortic sinus measuring 42.2mm, Sino-tubular junction measuring 36mm, and ascending aorta-

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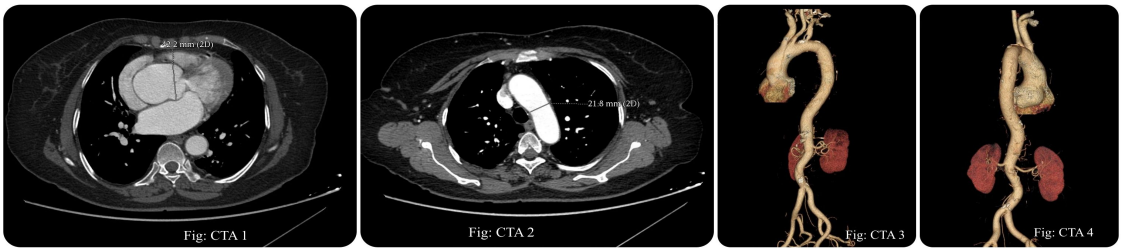
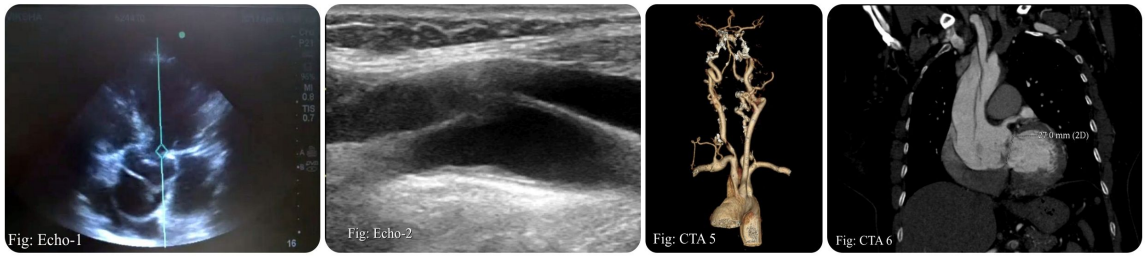
measuring 45.6mm. The Arch of Aorta (AOA) measuring 21.8mm and proximal descending thoracic aorta measuring 24.4mm were normal in course and calibre with no intimal flap or peri-aortic hematoma. The Celiac trunk and its branches, Superior Mesenteric artery (SMA) and its branches, Inferior Mesenteric artery (IMA) and its visualized branches, Renal arteries on both side, bilateral Common Iliac arteries (CIA), bilateral External & Internal iliac arteries were normal. In view of the ascending aorta aneurysm, she was admitted for further evaluation and was planned for aneurysmal repair.



(Fig: CTA 1 - Aneurysmal dilatation of the ascending aorta with Aorto-ventricular junction measuring 27.0mm, Fig: CTA 2 – Normal Arch of Aorta measuring 21.8mm, Fig: CTA 3, CTA4 – AOA, proximal descending thoracic aorta, Celiac trunk and its branches, SMA and its branches, IMA and its branches, b/l Renal arteries, b/l CIA, b/l External & Internal iliac were normal in course and calibre with no intimal flap or peri-aortic hematoma.)

Within 12 hours of hospital stay, she developed sudden onset throat discomfort, colicky abdominal pain associated with profuse sweating. General examination showed an agitated patient with bradycardia (45/min), B.P (70/40 mmHg), tachypnea (36/min) and hypoxia (oxygen saturation on room air 85%). Cardiac examination showed regular sinus bradycardia with S1, S2 heard in all areas and no murmurs. Neck examination revealed 2+ symmetrical Carotid pulses.

ECG revealed sinus bradycardia with fresh T wave inversion in V1-V4 and hence was managed with Inj. Atropine and other supportive care to which she responded well. 2D Echo revealed Aortic dissection flap in the Sino-tubular junction with aortic root measuring 4.0cm, Aortic Annular measuring 2.0cm, Sino-tubular junction measuring 5.1cm and Ascending Aorta measuring 4.8cm. There was moderate Aortic regurgitation. The Aortic dissection flap was extending till right Common Carotid artery to the bulb (Fig: Echo1 and Echo 2).



(***Fig: Echo 1- Aortic dissection flap in the Sino-tubular junction with aortic root measuring 4.0cm, Fig: Echo 2 - Aortic dissection flap extending till right Common Carotid artery to the bulb, Fig: CTA5- dissection involving brachiocephalic trunk with origin of right Common Carotid artery from false lumen, Fig: CTA6 - Dissection extending up to aortic sinus and the arch vessels)***

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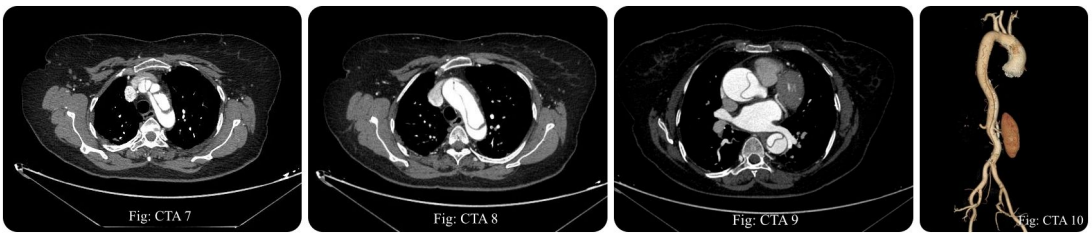
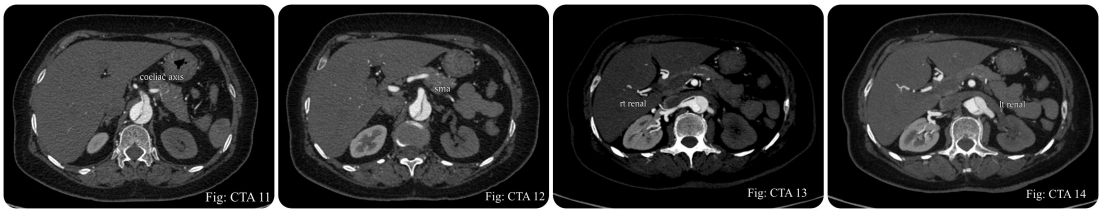
CT angiogram revealed Stanford type-A Aortic dissection (Fig: CTA 5, CTA 6, CTA 10). The intimal flap was extending up to aortic sinus; there was extension of dissection in the arch vessels, involving the brachiocephalic trunk with origin of right Common Carotid artery that was completely thrombosed from the false lumen (Fig: CTA 5, CTA 6). The dissection was also involving left Subclavian artery and was arising from the true lumen (Fig: CTA 7-CTA10). The origin of left main coronary artery was from true lumen with normal LAD, LCX and RCA. The dissection involved the entire length of thoracic and abdominal aorta up to the iliac bifurcation on the right side.



(Fig: CTA 7-10 –The intimal flap was extending up to aortic sinus, in the arch vessels and involved the entire length of thoracic and abdominal aorta up to the iliac bifurcation on the right side)

Dissection was also seen extending into osteal celiac axis, splenic artery, Superior Mesenteric artery (SMA), Inferior Mesenteric artery (IMA), bilateral renal arteries and right common iliac artery (CIA) and were arising from the false lumen, while left gastric artery, hepatic arteries, right renal arteries were arising from the true lumen. Hypo perfused spleen and left kidney was noted as the false lumen was thrombosed and there was compromised blood flow (Fig: CTA 11- CTA14).



(Fig: CTA 11-Dissection extending into osteal celiac axis, Fig CTA 12 - Dissection extending into SMA, Fig: CTA 13 - Dissection extending into right renal artery, Fig: CTA 14 - Dissection extending into left renal artery)

Based on the severity of the findings, she was immediately planned for a Bental procedure and aortic arch replacement depending on the extent of the intimal tear. However, after explaining the disease modalities, procedure details, postoperative complications and life expectancy outcome, the patient and the relatives refused to undergo the procedure, and she was being discharged on best possible medical management.

Discussion

Acute aortic dissection (AD) is one of the leading causes of death among aortic pathologic conditions. In this condition a column of circulating blood separates the layers of the media layer with variable proximal and distal extension throughout the length of the aorta (1) mostly due to degeneration of collagen and elastin in the aortic intima media. It is thereby classified as Stanford type-A (Prevalence: 70-75%), (Fig: 1) which involves the ascending aorta and Stanford type-B (Prevalence: 25-30%), (Fig: 1) which involves all other dissections regardless of the primary intimal tear. Studies have demonstrated that ascending aortic dissection is twice as common as

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descending aortic dissection. Other system of classification is by DeBakey system, which states that, type I (60%) - originates in the ascending aorta and propagates at least to the aortic arch and often beyond it distally, type II (10-15%) – originates in and is confined to the ascending aorta only and type III (25-30%) – originates in descending aorta, rarely extends proximally but will extend distally.

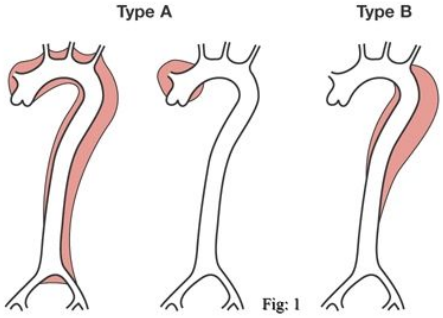

Fig: 1

Percentage	60%	10-15%	25-30%
Type	DeBakey I	DeBakey II	DeBakey III
	Stanford A		Stanford B
	Proximal		Distal

Classification of aortic dissection Fig: 2

Literatures demonstrated that the incidence of acute AD in general western population is estimated to range from 2.6 to 3.5 per 100,000 person/year (2). 20% of this patients dies before reaching the hospital and 30% die during hospital admission (3). Stanford type- A AD have a mortality of 1.2% per hour during the 1st 24-48 hours of the presentation and if left untreated the mortality rate rises up to 50% in one week (3). The peak incidence is in the sixth to seventh decade and men are twice more commonly affected than women.

The most common predisposing factor for AD is systemic hypertension, coronary artery bypass graft surgery, previous aortic valve replacement, cocaine use, strenuous resistance training, trauma and collagen fiber disorders such as Marfan syndrome, Ehlers-Danlos syndrome, annulo-aortic ectasia, bicuspid aortic valve, aortic coarctation, and Turner syndrome (4-7).

In acute AD, lab investigations are generally non-diagnostic. There are many imaging modalities for AD but are only performed after medical stabilization of a patient. Bedside trans-esophageal echocardiography (TEE) is the investigation of choice in hemodynamically unstable patient who present with symptoms of thrombo embolic events in any system. It has a sensitivity of 98% and specificity of 95%. The main advantage of TEE lies in the fact that esophagus is in close proximity with the thoracic aorta and hence true and false lumen can be easily identified. It is a bedside procedure, and useful for patients not stable for MRI. Magnetic resonance imaging (MRI) is a preferred imaging modality for patients with chronic chest/abdominal pain who are hemodynamically stable, as it has more sensitivity and specificity than CT angiogram. It has a specificity and sensitivity of 98% respectively and can visualize the whole extent of the aorta in multiple planes, can assess branch vessels, but this modality is time consuming and hence cannot be used in a hemodynamically unstable patients. Both TEE and MRI have excellent sensitivity however; MRI is more specific and is the investigation of choice for stable patient. CT scan with contrast is reserved for situations in which both TEE and MRI are unavailable or contraindicated and has a sensitivity and specificity of 83% and 90-95% respectively. The main disadvantage with CT angiogram is that intimal flap is seen in less than 75% and cannot reliably delineate branched vessels. (8-11).

	Angiography	CT	MRI	TEE
Sensitivity	Poor	Average	Excellent	Excellent
Specificity	Good	Good	Excellent	Good
Site of tear	Good	Poor	Excellent	Good

Summary of specialized imaging techniques

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Acute dissections involving the ascending aorta are considered surgical emergencies. The surgical options includes excision of the intimal tear, obliteration of entry into the false lumen proximally, and reconstruction of the aorta with interposition of a synthetic vascular graft. However, dissections that are confined to the descending aorta are usually medically treated unless the patient demonstrates progression of dissection or continued hemorrhage into the pleural or retroperitoneal space.

The most common clinical presentation of a aortic dissection is that of the sudden onset of "tearing" chest pain which will radiates to the back and mimics that of a Myocardial ischemia, with associated pulse or BP deficit between the upper limbs, and an abnormal chest X-ray which is seen in only one-third of the patients. At the time when the acute aortic dissection takes place, symptoms and signs are usually produced due to rupture or occlusion of major vessels. Pain is usually the most common symptom. However, dissection can be painless in 2-5% of the patients and symptoms can be less severe. Patients with suspected aortic dissection should be immediately admitted in an intensive care unit and diagnosis should be confirmed as soon as possible. Initial management would include pain control, reduction of systolic blood pressure in between 100 mmHg and 120 mmHg, adequate renal perfusion (urine output more than 30ml/hour), monitor evidences of cerebral hypo perfusion and minimizing stress (heart rate less than 60). Anti hypertensive preferred are IV Beta blockers (Propanolol/Labetalol); Nitroprusside can be used only if SBP is still more than 100mmHg only after using beta-blockers. Patients who are hemodynamically unstable should be intubated and start on supportive management.

Intraoperative mortality rate of Type-A dissection varies around 7-35% and postoperative mortality is around 27%. All patients should receive lifelong therapy of oral beta-blocker to reduce systemic blood pressure and the rate of rise in systolic pressure, thereby minimizing aortic wall stress. The long-term outcome rate of Type-A dissection at 5 years and 10 years is 68% and 52% respectively and that of type-B dissection is 60-80% and 40-80% respectively (12-14). Spontaneous resolution is rare.

Conclusion

This case report emphasizes the importance of clinical suspicion of this potentially life threatening condition. CTA, MRI, TEE are the imaging modalities available in these patients. Despite optimal medical and surgical approaches, prompt diagnosis is extremely important. Patients already diagnosed to have thoracic aortic aneurysm have an increased fold of developing aortic dissections. AD should always be considered as a differential diagnosis in any symptomatic presence of sudden onset of back/ chest/ abdominal pain or throat discomfort, and should be thoroughly evaluated and investigated for other features of AD.

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Conflicts of interest

No potential conflict of interest relevant to this article was reported.

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