A RARE CASE OF AN ACUTE ON CHRONIC THORACOABDOMINAL AORTIC DISSECTION

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Abstract
Aortic dissection (AD) is a rare cardiovascular emergency with a high rate of mortality. Most patients present with typical signs and symptoms of AD. However, atypical presentation increases risk of delay in diagnosis and treatment. Patients may present with syncope, seizures or other neurological symptoms which may deceive the diagnosis of AD. Dissection can be in the acute or chronic phase, but there has been no documentation of an acute dissection which occurs in a chronic dissection. We hereby to report a case of an acute on chronic thoraco-abdominal dissection.

Keywords: Aortic Dissection, Seizures, Syncope

Introduction
Aortic dissection (AD) is a tear that happens in the inner layer of the aortic wall, resulting in the formation of a false lumen. The false lumen will cause decreased blood flow to the vital organs causing failure. Aortic dissection further weakens the aortic wall and this may lead to rupture which would be fatal (1). It is one of the rare cardiovascular emergencies with a mortality rate approaching 50% in the first 48 hours of onset in diagnosis delayed or missed (2). Patients may present with typical symptoms of severe back pain, chest pain or abdominal pain. However, there are several reported cases presented with neurological symptoms (3). Thus, making a diagnosis of AD is difficult and involves thorough examination of the history of the patient and risk assessment. Aortic dissection can happen in an acute setting (within 2 weeks of onset) or in chronic condition (after 2 weeks of onset). In this paper, we would like to report a case of an acute on chronic thoraco-abdominal aortic dissection in which the patient presented with syncope and transient resolved chest pain.

Case Background
A 52-year-old man, who is an active smoker with an underlying history of hypertension, presented to the emergency department (ED), complaining of abdominal and chest pain for two days. The pain was constant and tight in nature, with radiation to the back. Prior to presentation, he experienced syncopal attack which lasted for 2 minutes. There was no tonic-clonic movement over all four limbs associated with the syncope.

Upon arrival at ED, he was conscious and alert. There was no chest pain, no shortness of breath and no giddiness. His blood pressure was 135/85 mmHg, heart rate 71 beats per minute, air saturation 97%, pain score was 5/10 and he was afebrile. Electrocardiography showed normal sinus rhythm with a rate of 68 bpm with no T and ST segment deviation. Blood investigations showed a hemoglobin level of 11.3, a white cell counts of 10.9 and a platelet count of 280. Urea and electrolytes were within the normal ranges.

Plain posterior anterior chest radiograph showed widen mediastinum with left pleural effusion (Figure 1). Bedside ultrasound revealed no free fluids in the abdomen and confirmed left pleural effusion. Bedside echocardiography showed poor cardiac window. The history taking was brief as the patient deteriorated rapidly in the ED; hence, the detailed social history taking regarding smoking and past medical history was not achieved.

While in the ED, the patient developed generalized tonic-clonic convulsions which lasted 2 minutes. Postictally he was confused, complaining of chest discomfort and shortness of breath. His blood pressure transiently dropped to 50/35 mmHg with a heart rate initially 30 bpm, but subsequently his blood pressure rebounded to 140/79 mmHg with a heart rate of 114 bpm, and his
air saturation level was 75%. A bolus of IV labetolol, 5 mg, was given to lower the systolic blood pressure to less than 120 mmHg. The patient was intubated due to the worsening of his respiratory distress. Post intubated portable chest radiograph, showed a complete white out of the left lung. Repeated bedside ultrasonography (USG) showed dilatation of the abdominal aorta measuring 5x6 cm, with the presence of a false lumen in the abdominal aorta. There were no free fluids in the abdomen and the echocardiogram showed no pericardial effusion.

Urgent computed tomography angiography (CTA) (Figures 2 & 3) revealed that the ascending thoracic aorta measured 4 cm in diameter, with the descending aorta dilated throughout its entire length, and its widest diameter was 7.4 cm. The entry point of the false lumen was seen at the level of T6 vertebra with a thrombus present within the false lumen at the proximal aspect, and at the exit point of the left common iliac artery. Another flap was seen at the lateral aspect of this false lumen, which bare a suspicion of recanalization. There was no contrast extravasation and no evidence of intrathoracic contrast pooling. There was evidence of left pleural effusion with layering seen. Circumferential thrombus was seen within the false lumen in the abdominal aorta with the coeliac trunk, superior mesenteric artery and left renal artery arising from the false lumen. There were no hyperattenuating crescent, break in calcification or aortic drape sign which to suggest an impending rupture of the dissection.

Figure 1: Chest radiograph shows widen mediastinum (10 cm), calcium sign presence, left pleural effusion, trachea deviation to the right side

Figure 2: CTA showing Stanford B Dissecting Aorta from the (A) anterior view and (B) posterior view

Figure 3: Dissection over Descending Aorta. (A) Upper thorax (B) Lower thorax
A diagnosis of acute on chronic ‘Stanford B’ aneurysm dissection was made. However, due to the complexity and severity of the dissection, the patient was managed conservatively by maintaining ventilation support. Left pleural effusion was not drained as its consistency was similar to clots. Drainage would have been risky as the patient may lose the tamponade effect and this could lead to rupture of the aneurysm. He was admitted into the intensive care unit (ICU) and eventually succumbed to death on day 12 of his admission.

**Discussion**

This case highlights the complex presentation of an acute on chronic aortic dissection with underlying thoracoabdominal aneurysm. An extensive literature review did not reveal any similar cases of an acute on chronic aortic dissection. Clinical presentation of an acute aortic dissection may be atypical as seen in this patient. He initially presented with chest and abdominal pain which had resolved on arrival to the ED. He also complained of syncope which can be attributed to complications of the Acute Aortic Dissection (AAD) which may cause neurological deficits, vascular insufficiency, syncopeal and aortic regurgitation in 10% of cases (4). Development of seizure in this patient is likely due to global cerebral hypoperfusion which causes hypoxia in the brain and is seen in 3% of cases of AAD (5).

Chest radiograph (CR) has a sensitivity of 64% with specificity of 86% for AAD. Radiological features in the chest radiograph typical for AAD are a widened mediastinum of more than 8 cm, loss of aortic knob, deviation of mediastinal structures, opacified aorto-pulmonary window, left pleural effusion and calcium sign. Calcium sign is defined as separation of the intimal calcification from outer aortic soft tissue of more than 10 mm in the CR. This sign is more frequently (14%) seen in patients with descending aorta dissection (6). In this case, the patient had a widened mediastinum and worsening left pleural effusion which could be indicative of haemothorax due to rupture. Haemothorax occurs in 10% of descending aortic rupture following a dissection and usually occurs in the left side of the pleura (7).

Transthoracic echocardiography (TTE) has low sensitivity (31-50%) in diagnosing Stanford B aortic dissection. However, it is useful to detect any pericardial effusion and any retrograde involvement of the ascending aorta or arch of the aorta. In this case, the use of bedside echocardiography was limited due to the poor cardiac window. Therefore, other imaging modalities should be considered to aid in the diagnosis of AAD.

CT angiography is a goal standard imaging to diagnose and identify the type of dissection, which reported its sensitivity and specificity of nearly 100% (4). This patient’s CTA shows a Stanford B acute on chronic dissection. Findings that suggest chronic dissection in this patient are presence of thrombi within the false lumen at the proximal aspect and at the exit point of the left common iliac artery. In addition, the flaps are already opacified which hinted of a chronic rather than an acute dissection. False lumen thrombus is significantly common due to the increase in stasis by aneurysmal degeneration and atheromatous changes of neo-intima (8). The features are suggestive of acute on chronic dissection as there is another flap seen within the thrombosed false lumen (Figure 2).

Stanford B dissection (TBAD) happens approximately in 3 per 100,000 persons per year, with a mortality rate of 30-40% within 5 years. The prognosis is predicted in between complicated and uncomplicated types of TBAD. This patient had a complicated TBAD in which he was hypotensive, and exhibited the presence of neurological signs and signs of aortic rupture. Complicated TBAD happens in 30% of cases and its survival rate is only 50%. The goal of treatment in TBAD is to restore perfusion and to prevent further dissection or rupture of the aneurysm. All TBAD patients should be treated medically with a β-blocker or a calcium channel blocker to reduce hemodynamic forces so as to maintain systolic blood pressure below 120 mmHg to limit the risk of rupture.

In complicated TBAD, endovascular surgery reduces mortality compared to an open surgery, with a 2.8% to 29.3% mortality rate. It is shown that endovascular surgery for complicated TBAD is superior compared to optimal medical therapy only (9). However, our patient had not been able to progress to surgery as he remained unstable throughout his ICU stay. Moreover, he had a massive left hemotorax which could not be drained out due to it loss of tamponade effects once decompression was done. He finally succumbed to death after 12 days in the ICU.

**Conclusion**

Aortic dissection (AD) is a life-threatening medical emergency, which carries a high rate of mortality. To diagnose AD in the emergency department is challenging. Even though a majority of patients presented with typical severe chest pain, a few of them may present with atypical symptoms. Thus, the long-standing risk factors and a high index of suspicion toward the presenting complaints should suggest the diagnosis of AD. Chest radiograph and echocardiography are the best bedside modalities in diagnosing AD. However, the goal standard of diagnostic imaging is computed tomography angiography (CTA) to confirm the stage of AD. This is an important guide for the clinician to manage the patient medically or surgically so as to improve the mortality rate of AD.

**Competing interests**

The authors declare that they have no conflict of interest.

**Ethical Clearance**

Informed consent was taken from patient’s daughter for case discussion and publication.
Reference


