Extensive Chronic Type B Aortic Dissection Extending To The Left Common Femoral Artery, A Rare Case Report.

Abstract:

Background: Aortic dissection is a potentially fatal condition, defined as an injury in the innermost layer of the aortic wall leading to high-pressure blood flow between the layers of the aorta, creating a true and false lumen. CT scan is the gold standard for diagnosing AD due to its high sensitivity and specificity. Treatment of acute type A aortic dissection (TAAD) is by surgery while acute type B aortic dissection (TBAD) is managed by medical therapy.

Aim: Recognition of aortic dissection and its different clinical presentations.

Methods: Herein we report a 47-year-old man presenting repeatedly for years to the Emergency Department of various hospitals in Saudi Arabia with symptoms of chest pain that were thought to be from Acute coronary syndrome (ACS), but later diagnosed as aortic dissection type B.

Conclusion and Results: The incidence of TBAD is under-reported and might be misdiagnosed for years.

Keywords; Aortic dissection, Type B aortic dissection, acute coronary syndrome, Misdiagnosed TBAD

Introduction:

Aortic dissection (AD) is a relatively uncommon, life-threatening medical emergency, caused by the separation of the inner layers of the Aorta, interrupting normal blood flow [1,2,3,]. The incidence of acute AD is estimated to range from 2,6 to 3,5 per 100,000 persons/year [4].

The Stanford classification defines Type B Aortic Dissection (TBAD) as the involvement of the descending thoracic aorta with the absence of ascending aortic involvement [5]. TBAD presents in its acute form with a sudden onset of tearing chest pain and may mimic common conditions, such as acute coronary syndromes, pulmonary

embolism, and acute abdominal illness, posing a high risk of misdiagnosis [4,5,6]. According to large clinical studies, systemic hypertension is present in about 80% of patients with acute TBAD. Thus, it is one of the most important risk factors, along with increasing age and atherosclerosis [3,4,7]. Some patients with TBAD present with subtle symptoms or signs, causing a delay in diagnosis [2]. The most validated imaging modality for TBAD is Computed Tomography (CT), the presence of a false aortic lumen establishes AD diagnosis.

Regarding treatment paradigms, TBAD is controlled medically with antihypertensive medication, while Type A aortic dissection (TAAD) is usually corrected surgically [5,12]. Moreover, the acute phase is followed by a chronic phase distinguished by varying degrees of aneurysmal dilatation, and false lumen thrombosis [12].

Case report:

A 47-year-old physically active male, known for hypertension and heavy smoking on Lisinopril 10 mg od for hypertension. Presented to the Emergency Department at King Abdulaziz Hospital in the eastern region of Saudi Arabia, with a few-hours duration of severe, on and off, retrosternal chest pain radiating to his back. He had a similar episode two weeks earlier while he was on a visit to a different region in Saudi Arabia, requiring hospital admission with a provisional diagnosis of ACS, and was discharged 5 days later with a negative ACS workup including cardiac enzymes, ECG series, and Echocardiogram. An outpatient Stress ECG was recommended. Pain upon presentation was present on rest worsened by exertion. There was no abdominal pain, nausea, or vomiting, and no history of limb numbness, claudication, or weakness. The initial examination was unremarkable, apart from high blood pressure (BP) of 190/110 mmHg, of note resting heart rate was 60bpm. All peripheral pulses were intact, with no deficit or difference in the upper or lower limbs. There was no radio-radial radial or radio-femoral delay or BP differences in both arms. ECG was remarkable for left ventricular hypertrophy (Figure 1). All initial labs were unremarkable and there was no Troponin I leak, apart from a moderately elevated D-dimer of 1.3 μg/ml. Furthermore, no ischemic ECG changes, and a normal Echocardiogram.

Since he had similar presentations in the past and had a resting heart rate of 60 bpm, we elected to perfume directly a cardiac CT scan with contrast to rule out obstructive coronary artery disease. The reported was normal coronaries with an incidental finding of aortic dissection, the study was followed with a CT aortogram revealing dissection involving the whole thoracic, abdominal aorta, left common iliac, left external iliac up to the left common femoral artery. The origin of the left subclavian artery, celiac trunk, and left renal artery seems to be involved in the

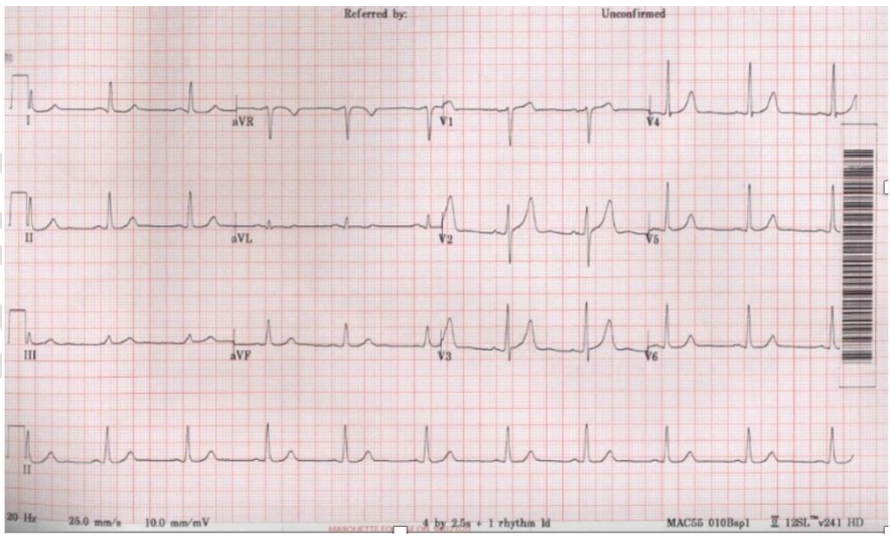
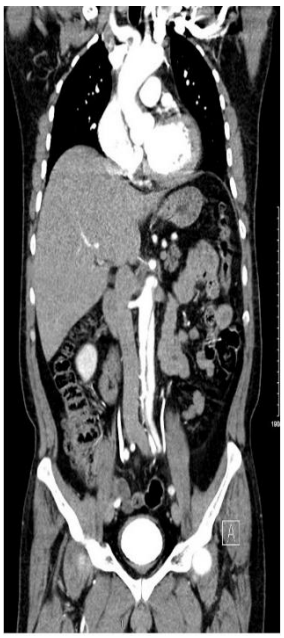


Figure 1: ECG showing left ventricular hypertrophy by voltage criteria.

dissection with a false lumen, and a mural thrombus was seen in the false lumen of the

descending thoracic aorta measuring 1.8 x 2.1 x 3.3 cm suggesting chronicity of the dissection (Figure 2A-B-C-D). In addition, a subtle hypodense area was seen around the descending thoracic aorta along with mild left pleural collection with relatively high-density contents indicating probably degraded blood.



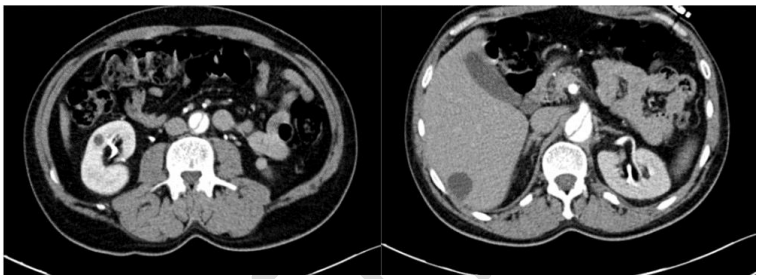


Figure 2A: Aortic dissection as seen on axial CT angiogram images.

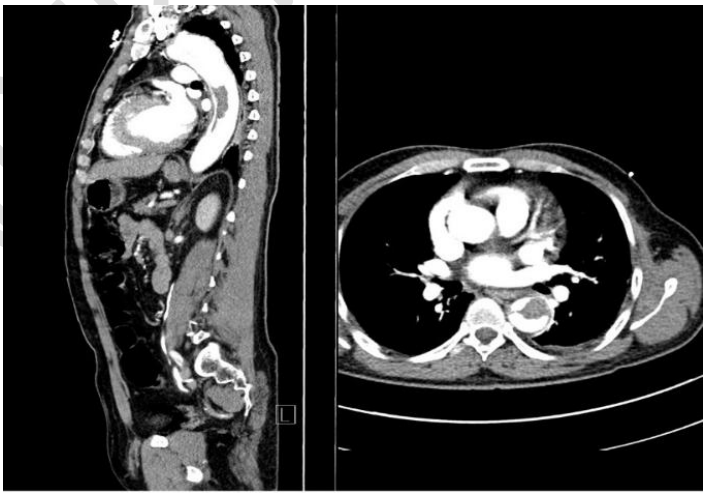


Figure 2B: Aortic dissection with intramural thrombus as seen on CT axial-sagittal angiogram images.

Figure 2C-D: (Figure 2C) is on the left showing extensive aortic dissection seen on coronal CT angiogram images. (Figure 2D) is on the right showing a CT Aortogram 3D reconstruction image with intimal flap, extending to the left renal artery, left common lilac, external lilac, and left common femoral artery.

Upon breaking the bad news, he recalled having similar episodes of chest pain necessitating emergency room visits 4 and 12 years back, where ACS was ruled out each time. A thorough chart review revealed repeated visits to ER and Primary health care clinics with very high BP readings exceeding ~200/120mmHg caused by various painful triggers. The in-hospital course of management included optimal anti-hypertensive medications, immediate cessation of anticoagulants, and vascular surgery advice to continue optimum pharmacological treatment. He was closely monitored, with daily physical examinations, labs, and ECGs, all were unremarkable. He remained, asymptomatic and was discharged home with close follow-up as an outpatient with both Cardiology and Vascular surgery. In this case, we postulate based on history and CT aortogram findings, that the timing of Aortic dissection onset was more than two weeks, and probably was years ago, and uncontrolled blood pressure was a major contributing factor, in addition to smoking.

Discussion:

According to Stanford's classification of Aortic dissection, type A involves the ascending aorta and type B indicates a dissection limited to the descending aorta [8]. Type B aortic dissection (TBAD) presents more commonly as a chronic ailment and occasionally presents acutely, while type A usually presents as a disastrous fatal event, with a mortality of 1.2% per hour in the first 24-48 hours, and half of presenting patients eventually die within a week if untreated, furthermore, 20% of patients with AD die before reaching the hospital and 30% die during hospital admission [6,13].

Aortic dissection (AD) results from a tear in the intimal layer of the aorta, allowing blood to flow between the intima and media, resulting in the formation of a false lumen, disrupting normal blood flow and perfusion to vital organs or eventually causing aortic rupture [1,2,3,]. Booher et al, proposed 4 distinct periods for AD: hyperacute (symptoms from onset to 24 hours), acute (2-7 days), subacute (8-30 days), and chronic (>30 days), overall survival was progressively lower through the 4 time periods. [13]

Misdiagnosing AD with other medical emergencies, such as ACS, pulmonary embolism, or acute abdominal ailments has been expressed in 30-50% of AD patients [6-12]. Chest pain accounts for 79% of TAAD and 63% of TBAD presentation respectively [6], therefore thrombolytic therapy for ACS may be fatal in AD, resulting in hemodynamic instability, hemorrhagic pleural and pericardial effusions, increased hemorrhagic postoperative complications, and a trend toward increased mortality [2,12]. Hence our patient was mistakenly diagnosed with ACS/Unstable angina repeatedly, the maximum severity of chest pain was at two weeks, 4-8 years intervals, and was placed on anticoagulants and antiplatelets, but luckily not thrombolytics. Therefore, one must maintain a high index of suspicion for AD, as it may be misdiagnosed as ACS or present combined with ACS.

Confirmed associations in connection with AD are hypertension, atherosclerosis, congenital bicuspid aortic valve, pregnancy, and strenuous activities, while connective tissue disorders (CTD) were particularly more with TBAD [4,5]. In several International Registry of Acute Aortic Dissection (IRAD) high blood pressure was documented in 70% of TBAD, and 35% of TAAD, in addition to a pulse deficit of 20-30% respectively, as a sequel to intimal flap or hematoma compression [2,6].

While Stanford classified AD to type A which involves the ascending aorta and type B which involves only the descending aorta [9], Lempel et al proposed a new classification; TAAD starting in the ascending aortic artery with or without aortic arch involvement, and TBAD distal to the subclavian artery with or without proximal extension into the transverse arch between the innominate and left subclavian artery which may or may not extend distally into the descending aorta [10].

For a definitive diagnosis of AD, cardiac CT/angiography is the most available and widely used modality to diagnose AD. Magnetic resonance imaging is an alternative, while transesophageal echocardiography (TEE) is considered for hemodynamically unstable patients. AD should be suspected on abnormal chest x-ray since 60–90% elicited abnormal aortic contour or mediastinal widening [6]. ECG must be performed to differentiate acute myocardial infarction from aortic dissection [12].

TAAD typically necessitates surgical intervention due to its tendency to cause coronary artery occlusion, rupture into the pericardium leading to tamponade, or aortic valve dissection resulting in aortic insufficiency, while TBAD is usually addressed less emergently with medical treatment to reduce hemodynamic forces and mitigate the risk of dissection extension. Outcome studies have shown that the weight of the evidence for TBAD favors nonsurgical treatment [10]. Acute TBAD is more aggressive than chronic expanding TBAD, with a reported 30-day mortality of 19% compared to 0%, respectively post endovascular repair, with significantly higher complication rates [6].

Conclusion:

Chronic aortic dissection may remain asymptomatic despite the widespread extension. This case highlights the importance of maintaining a high index of clinical suspicion for aortic dissection, prompting early diagnoses in a hypertensive patient who presents with typical or atypical symptoms.

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