INTRODUCTION

Aortic dissection is uncommon condition, with high mortality rate if untreated. Stanford type A aortic dissection is considered to be an emergency. Around one third of patients ultimately diagnosed with acute aortic dissection are first thought to have another diagnosis. The true incidence of acute aortic dissection is therefore difficult to determine, but is estimated to be 5-20/1,000,000 [1]. Imaging studies such as CT, MRI chest, or Trans-Esophageal Echocardiography (TEE) are necessary to confirm the diagnosis as well as to differentiate the type of aortic dissection, which has very significant implications on the treatment options and the threshold to intervene. We report a very challenging and unusual case of long segment aortic dissection where the type of the dissection was very difficult to identify due to limitations of the available imaging studies. Most classic aortic dissections begin at 3 distinct anatomic locations: the aortic root; 2 cm above the aortic root; and just distal to the left subclavian artery. Ascending aortic involvement may result in death from wall rupture, hemopericardium and tamponade, occlusion of the coronary ostia with myocardial infarction, or severe aortic insufficiency [2-4].

Aortic intramural hematoma (AIH) is a more recently described entity in which no intimal flap is present. It results in a spontaneous medial hematoma that may be secondary to an infarction of the vasa vasorum of the adventitia. Aortic intramural hematoma accounts for approximately 25% of aortic dissections. Involvement of the ascending aorta, especially if the overall aortic diameter is greater than 5 cm, should be treated surgically to prevent rupture or progression to a classic dissection with intimal tear. Conservative management is indicated for AIH of the descending aorta [5-7].

Early diagnosis and treatment are essential for improving the prognosis. Patients may present with the classic history of acute-onset tearing central chest pain that radiates to the back. Malperfusion symptoms may result from dissection-related side branch obstruction. It has been reported that up to 20% of patients with acute aortic dissection may present with syncope without a history of typical pain or neurologic findings [8]. Syncope can result from hypotension secondary to cardiac tamponade [9] or from obstruction of cerebral vessels. Aortic regurgitation gives rise to a diastolic murmur, which has been reported in 40%-50% of patients with proximal dissections [3], and severe regurgitation can result in heart failure [10].

CASE REPORT

A 66-year-old Caucasian male presented to our facility with 3 days history of chest pain and shortness of breath. He has a past medical history significant for hypertension, dyslipidemia, chronic kidney disease and old ischemic stroke with residual right hemiparesis. In the emergency room, patient was found to be hypertensive with a systolic blood pressure >190. His labs were remarkable for elevated creatinine and BUN. Chest X-ray
showed widening of mediastinum which was suggestive of possible aortic dissection. Contrast CT angiography of abdomen was done for further evaluation and it showed an acute dissection of the thoracic aorta that extends from the mid ascending aorta to the infra-renal aorta without evidence of extension into the common iliac arteries suggestive of Stanford type A aortic dissection. The celiac artery and superior mesenteric artery were involved, 4.7 cm aortic aneurysm in transverse aorta, and large pericardial effusion was present with no evidence of extravasation of contrast into the pericardial space. Transthoracic echocardiography revealed a calcified intimal flap just beyond the distal arch. Moreover, large partially calcified intimal flap was present in the abdominal aorta with no proximal clear cut extension. Normal ejection fraction was reported with dilated aortic root, dilated aortic arch, and large pericardial effusion with no evidence of cardiac tamponade. Trans-esophageal echocardiography was done later, and it revealed a partially calcified intimal flap in the distal portion of the arch and in the descending thoracic aorta in concordance with the trans-thoracic echocardiogram findings and no abnormalities were reported in the ascending aorta. These findings were significantly consistent with Stanford type B aortic dissection. Based on that, he was treated medically with nicardipine drip for blood pressure control. Due to the discrepancy of these tests, imaging studies were reviewed, and repeated CT chest with contrast was obtained and surprisingly confirmed that he had Stanford type B aortic dissection, with the dissection starting distal to the left subclavian artery and intact ascending aorta (figures 1-3). Based on the family preference medical management was continued, and 3 weeks later patient was discharged to a rehabilitation facility.

One week after discharge, patient was readmitted again due to severe neck pain and shortness of breath. CT neck was done and it was unrevealing. CT chest without contrast was done and showed increase in the size of aortic aneurysm to 6 cm with grossly stable appearance of Stanford type A dissection consistent with the first CT angiography. At that stage, cardiothoracic surgery reevaluated the situation and recommended surgical intervention due to the significant increase in the size of the aortic arch and proximal descending aorta, which might eventually lead to rupture. He underwent emergent Hemahield ascending aortic and hemiarch replacement and mechanical aortic valve replacement without major complications.

**DISCUSSION**

Aortic dissection is considered to be a relatively rare condition with reported estimated incidence of 5 - 27 cases per million per year. The Stanford classification differentiates 2 types: type A involving the ascending aorta, and type B involving the descending aorta, regardless of the site of the intimal tear or the distal extent. Acute aortic dissection is readily diagnosed using CT scanning, with the reported diagnostic accuracy ranging from 88-100 % [11]. Older spiral CT scanners reported to have accuracy >95%, but they require a very thorough technique for optimum sensitivity and accuracy [12]. Conventional CT scanners without spiral acquisition have less satisfactory results. For example, there is a much
higher risk of missing rapidly moving dissection flaps while using incremental scanners [4]. Commonly, with older CT scanners, it is usually necessary to complete another diagnostic test (such as echocardiography) to ensure satisfactory diagnostic accuracy [13,14]. Contrast CT chest is one of the tests of choice to make diagnosis of aortic dissection because of its availability, but it has major disadvantages such as the fact that intimal flap is seen in less than 75% of cases and the site of entry is rarely identified. These side effects may lead to serious delay in the diagnosis of the correct type of dissection leading to significant delay in management, and our patient was a fair example of these drawbacks. Additionally, there is potential nephrotoxicity due to contrast agent exposure. In our patient, the origin of flap was intermediate between Stanford type A and B based on the CT scan findings and this actually postponed the appropriate treatment. Based on this example, we believe that Spiral CT or multi-slice CT can improve the accuracy of diagnosis substantially.

CONCLUSION

Despite the wide use of contrast CT scan as the gold standard for aortic dissection diagnosis and classification, it has its own limitation and diagnostic discrepancies between different imaging modalities should always be confirmed by repeat imaging before management line is decided.

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REFERENCES