



Dissection of ascending aorta in a young fellow during gym heavy weightlifting: Interesting case report

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Abstract

Aortic dissection is a rare but often catastrophic condition if not diagnosed and managed acutely. Although rare, aortic dissection needs to be in the differential when a young weightlifter presents with chest pain as a delay in diagnosis may be fatal. A young otherwise healthy 23-year-old male who has been using anabolic steroids for a long time developed Type I aortic dissection associated with heavy weightlifting. The patient did not have a recent history of trauma to the chest, no history of hypertension, and no illicit drug use. He presented with severe chest pain radiating to back and syncopal event with exertion during heavy weightlifting 90 kg at a time. Initial vitals were significant for blood pressure of 82/54 mmHg, pulse of 95 beats per minute, respirations of 22 per minute, and oxygen saturation of 93% on room air. Bedside echocardiogram showed aortic root dilatation with dissection of ascending aorta and cardiac tamponade. Computed tomography (CT) scan of chest revealed dissection of ascending aorta was confirmed. Cardiothoracic surgery was done successfully with repair of ascending aorta.

Keywords: aortic, gym, weightlifting

1. Introduction

Aortic dissection is a clinical emergency that commonly presents with tearing chest pain and hemodynamic instability. The immediate mortality rate in aortic dissection is as high as 1% per hour over the first several hours, making early diagnosis and treatment critical for survival. Therefore, high index of clinical suspicion is important as delay in diagnosis can have dreadful consequences. Aortic dissection in young healthy individuals has been reported in the literature but is relatively rare. In present review, we report a case of healthy male athlete with heavy weightlifting and use of anabolic steroids. He presented with chest pain in ER and was found to have aortic dissection.

2. Case Report

A 23-year-old young healthy male was admitted to cardiac emergency department with complaint of acute chest pain. On the day of admission, he went to the gym to lift heavy weights, which he routinely does on a daily basis. During the workout, he developed severe chest pain with radiation to his back. He initially thought that he had pulled his muscle in his chest. After completing his weightlifting at the gym, he reportedly had a syncopal event with exertion earlier in the day, so emergency medical services was called to bring him to the hospital. He described the chest pain as sharp, radiating to the back. The patient was cold, clammy, and short of breath. Prior to this incident, the patient had been healthy all his life

without any medical problems. He never smoked, did not have hypertension, and had no family history of sudden cardiac death or any collagen vascular disease. There was no predisposing risk factor for aortic dissection other than heavy weightlifting. To develop and maintain a muscular body, he recently started injecting intramuscular anabolic steroids for the last 3 months and was going to the gym every day, lifting around 90 kg at a time.

On examination upon arrival to the hospital, the patient's blood pressure was 82/54 mmHg, pulse was 95 beats per minute, respirations were 22 per minute, and oxygen saturation was 93% on room air. He was a well-developed, well-nourished, 6'1" muscular young male with a normal color of his lips and extremities. Both upper and lower extremities were cold and clammy. The patient had diminished palpable radial pulses in his upper extremities but no palpable pulse in his lower extremities. His jugular venous pulse was elevated. Auscultation of his heart revealed distant, muffled heart sounds without any murmur. Lungs were clear to auscultation, and the patient was in mild respiratory distress. Abdominal examination was normal, nontender and no pulsatile and non palpable mass. The patient was visibly uncomfortable, restless.

The patient's ECG revealed sinus tachycardia, nonspecific ST Depression with T-wave inversion in leads V5–V6, and voltage criteria suggesting LVH (Figure 1).

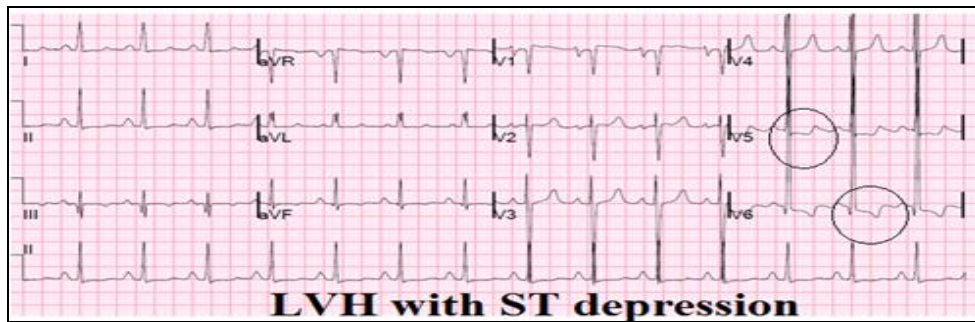


Fig 1: ECG suggests LVH and T-wave inversion in leads V4–V6.

The chest X-ray revealed clear lungs with cardiomegaly and a wide mediastinum. 2D Echocardiogram revealed moderate left ventricular hypertrophy and a mild to moderate circumferential

pericardial effusion with cardiac tamponade grade 1 and aortic root dilatation with ascending aortic dissection (Figure 2).

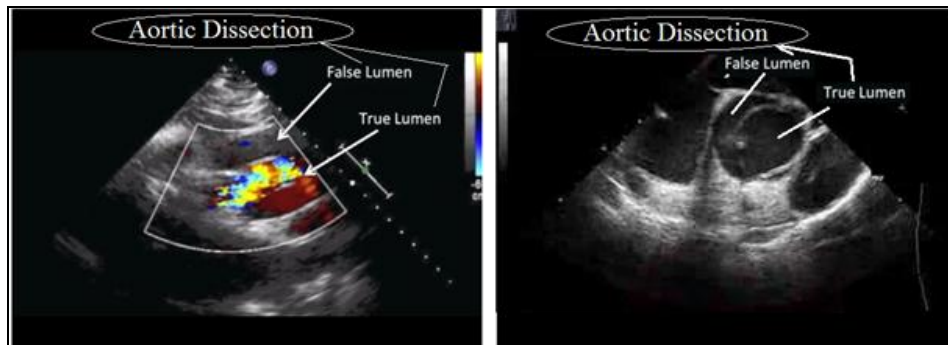


Fig 2: Echocardiogram showing left ventricular hypertrophy and ascending aortic dissection with pericardial effusion.

A chest computed tomography (CT) immediately followed by echocardiogram revealed an ascending aorta measured as 5.6 cm in diameter with an aortic dissection suggesting rapid

expansion of the dissection (Figure 3). Hemopericardium and cardiac tamponade were present with 2 cm thick fluid in the pericardium.

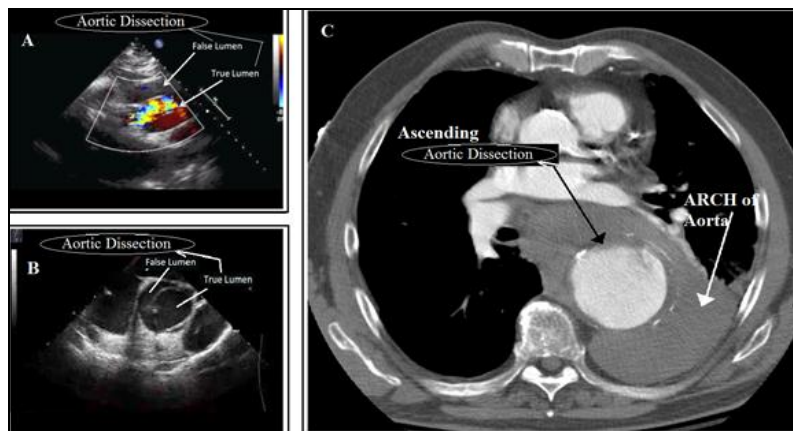


Fig 3: CT scan of chest with contrast showing dissection flap in ascending aorta (red arrow).

He was shifted to cardiothoracic surgery unit subsequently in operating room where a 3 cm aortic graft was placed. The aortic dissection had extended to the noncoronary cusp without involvement of the left main or right coronary arteries. It was challenging to repair the noncoronary cusp and keep the native valve intact. Intraoperatively, it was thought that the moderate aortic regurgitation was acceptable given his age and to avoid lifelong anticoagulant for this young patient. Although the patient’s initial echocardiogram prior to surgery

showed mild aortic regurgitation, the echocardiogram performed following the emergent repair of the ascending aortic dissection showed severe aortic regurgitation. The patient’s hospital course was complicated by persistent severe aortic regurgitation requiring a second procedure to repair the native aortic valve. Following the second surgical procedure, the patient’s severe aortic regurgitation was minimized to moderate aortic regurgitation without necessitating aortic replacement. Both surgeries were successful for the patient.

3. Discussion

Aortic dissection is a fatal condition with high morbidity and mortality. Reported incidence is 16 per 100,000 men and 7–9 per 100,000 women with the mean age at presentation of 63 years [1, 3]. There is a genetic predisposition for aortic dissection in cases of Marfan, Loey-Dietz, and Ehler-Danlos syndromes, familial aneurysms, bicuspid aortic valve, and coarctation of the aorta. Apart from genetics, the most common risk factors contributing to aortic dissection include hypertension, smoking, dyslipidemia, illicit drugs including cocaine and amphetamine, inflammatory disorders such as Takayasu, Behcet, and giant-cell arteritis, and trauma to the chest wall [4]. Cystic medial degeneration is a non-inflammatory loss of elastic fibers in the aortic media, and it is not pathognomonic for any of the abovementioned etiologies of dissection. Dissection usually starts with an intimal tear and is not always aneurysmal initially. Intramural hematoma and penetrating atherosclerotic ulcer can cause aortic dissection as well. Elevated blood pressure further propagates the intimal tear precipitating aortic dissection [5, 6].

Chest pain with radiation to the back is the most common complaint of a patient with aortic dissection, which was the initial presentation of the patient in this case report. During an aortic dissection, there may be involvement of side branches of the aorta, which may cause malperfusion of the brain leading to a cerebral vascular accident, decrease blood to the heart muscle producing a myocardial infarction, or can involve adjacent structures and valves which may cause cardiac tamponade or aortic regurgitation. CT angiography scan is the preferred modality for diagnosis [7], and Transesophageal Echocardiogram may be needed if the patient is unstable or is not able to be transferred for a CT scan. Magnetic resonance angiography is another good alternative modality.

Surgical treatment is recommended for Type a dissection. Overall in-hospital mortality is around 25%, and mortality for medically managed patients is 58%. Mortality is 1-2% per hour for the first day in patients who do not qualify for surgery [3, 7]. Surgery involves the placement of a synthetic interposition graft to reconstitute the true lumen. Surgery may or may not involve preimplantation of coronary arteries and resuspension of the native aortic valve, depending upon the level of dissection. Resuspension of the native aortic valve is preferred to replacement of the valve if possible [8].

Aortic dissection in younger individuals is very rare. Excluding the patient discussed in this case study, as per our knowledge, there are only a few reported cases of aortic dissection in weightlifters with a history of anabolic steroid use, and one of these cases also had a history of cocaine and heroin use. The following list summarizes the six reported cases of aortic dissection in male weightlifters less than 38 years old [9, 13].

Our patient has been using anabolic steroids chronically that might be a contributing factor to aortic dissection but there is no clear association found between use of anabolic steroids and aortic root dilatation and dissection in the literature. More importantly he was likely having underlying mild aortic enlargement that concomitant with hemodynamic changes of heavy weightlifting raised aortic wall stress to a level that begets aortic dissection. Of the five reported cases of Type I

aortic dissection in male weightlifters less than 38 years old, four of the patients confirmed use of anabolic steroids and the fifth patient had no known history of anabolic steroid use. Interestingly there are no reports of aortic dissection in anabolic steroids users that are not weightlifters. In the opposite there are quite a few reports of aortic dissection in weightlifters that do not use anabolic steroids. More importantly the pathological analysis does not suggest any scientifically proven relationship of steroids and atherosclerosis or of anabolic steroids and aortic dilatation or anabolic steroids and aortic dissection. Anabolic steroids theoretically could increase a patient's low density lipoprotein and decrease high density lipoprotein, thus promoting atherosclerotic deposition of the aortic intima and leading to weakening of the aortic wall. Therefore, atherosclerotic aortic walls are prone to dissection and rupture [9, 11].

The patient presented in this case had moderate ventricular hypertrophy, which is difficult to explain in the absence of long standing hypertension. Anabolic steroid use may be the cause of his ventricular hypertrophy. A dilemma faced in the operating room for this patient was how to fix the aortic valve as the aortic dissection involved the noncoronary cusp. In this situation, the aortic valve could have been either replaced or repaired. Replacing the aortic valve would have committed this young patient to a lifetime anticoagulation therapy. On the other hand, repairing the noncoronary cusp would not require anticoagulants, but there would continue to be an acceptable degree of aortic regurgitation that has a potential to worsen over time, which was the initial option selected for this patient. Prior to aortic valve repair, the patient had severe aortic regurgitation, which was minimized to moderate aortic regurgitation following aortic valve repair, and the patient continues to remain asymptomatic to present day. He will likely need an aortic valve replacement in the future if he becomes symptomatic. The patient has been followed up as an outpatient with serial echocardiograms to reassess the degree of aortic regurgitation. He was strongly encouraged to avoid weightlifting and all anabolic steroid use. He continues to take Lisinopril as an outpatient to decrease his afterload and therefore mitigate the effects of aortic regurgitation. He would have also been started on metoprolol had he not been bradycardic. He has been able to return to aerobic exercise without recurrence of symptoms and is seen regularly in the outpatient cardiology clinic. Aortic dissection is categorized based on the location and origin of the dissection. The patient has been followed up as an outpatient with serial echocardiograms to reassess the degree of aortic regurgitation. He was strongly encouraged to avoid weightlifting and all anabolic steroid use.

4. Conclusion

Weightlifting is known to elicit profound hemodynamic stress on the walls of the aorta and weightlifting alone even without anabolic steroids use may predispose young patients to aortic dissection, particularly Type I aortic dissection. Although aortic dissection in this age group is very rare, it needs to be considered as a possible differential diagnosis in younger patients who are weightlifters with or without history of anabolic steroid use as the mortality and fatal morbidity are very high if left untreated.

5. Conflict of Interests

The authors do not have any conflict of interests.

6. Consent

The authors have taken consent by this patient.

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