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Case report

## ACUTE AORTIC DISSECTION IN A YOUNG HEALTHY ATHLETE WITH ANDROGENIC ANABOLIC STEROID USE: A CASE REPORT

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### ABSTRACT

**Background:** Acute aortic dissection can occur at the time of intense physical exertion in strength-trained athletes like weight lifters, bodybuilders, throwers, and wrestlers. Rapid rise in blood pressure and history of hypertension are the most common causes of aortic dissection in athletes. It is a very tragic event because of its high mortality rate of about 32% in young patients. We report a case of aortic dissection in a young weightlifter with a history of anabolic steroid usage with an extensive intimal tear of the aorta at Sino tubular junction and arch. All athletes must be assessed for predisposing factors for aortic dissection, and all patients should be encouraged to undergo appropriate diagnostic studies, like echocardiography and blood pressure monitoring while weightlifting to recognize possible predisposing factors for aortic dissection. Athletes who do have a problem should be encouraged to avoid or limit their exercise or activity by their cardiologist. It is vital that this disastrous event be prevented in young people. In conclusion, although a rare occurrence, AD should be considered in symptomatic patients with any family history of early cardiac deaths, a history suggestive of a connective tissue disorder (that is, multiple joint surgeries) or who practice weightlifting.

**Keywords:** Acute aortic dissection; Athlete; Anabolic steroids.

### INTRODUCTION

Acute aortic dissection results from a tear in the intima and media of the aortic wall, with the subsequent creation of a false lumen in the outer half of the media and elongation of this channel by pulsatile blood flow. Dissection of the aorta is associated with a high degree of morbidity and mortality despite continuing improvements in diagnostic and surgical techniques<sup>1</sup> and hypertension is present as the most common cause in 70–90% of patients with aortic dissection.<sup>2</sup> A number of normal daily and athletic activities require isometric or static exercise. Sports such as weightlifting and other high-resistance activities are used by power athletes to gain strength and skeletal muscle bulk. These exercises significantly increase blood pressure, heart rate, myocardial contractility, and cardiac output.

Hypertension has long been recognized as an important risk factor for the development of aortic aneurysms and dissections.<sup>1,3</sup> Also, it has been speculated that the very high blood pressure generated during the lifting of weights, particularly with straining accompanied by a Valsalva maneuver, may be the cause of an aortic intimal tear.<sup>3</sup> Pre-participation cardiovascular evaluation of young competitive athletes is warranted on the basis of the available evidence.<sup>4</sup> Any person, regardless of age with predisposing conditions to aortic dissection, including hypertension, should be sturdily encouraged to refrain from weightlifting. We present a case of aortic dissection in a young athlete with no history of hypertension.

## CASE PRESENTATION

Mr. A, 34 Year old athlete an active runner and weightlifter was seen by cardiologist on referral request from Internal Medicine for evaluation of cardiac murmur in emergency section of our hospital.

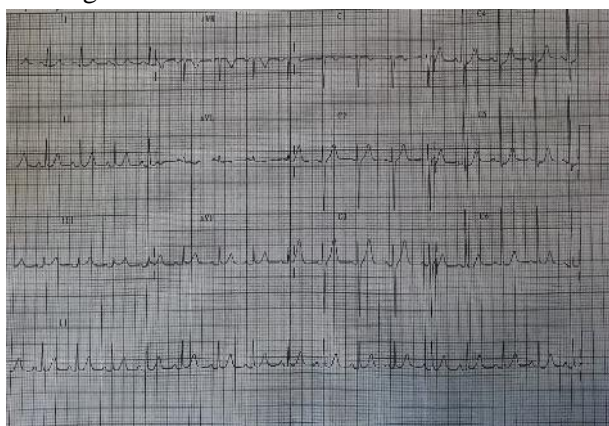
**Relevant History:** Mr. A. visited ER with complaints of cough with expectoration [blood tinged], low grade fever with gradual onset shortness of breath and orthopnea since 2 days. Generalized fatigue and body aches. Right upper abdominal pain, No chest pain. No syncope/No palpitations. He gave history of daily exercises in the gym, bodybuilding, takes protein supplements and anabolic steroids.

**Past History:** None significant except recently seen two days ago In ER with pain abdomen which was diagnosed as renal colic.

**Risk Profile:** No hypertension, diabetes, dyslipidemia or cardiovascular disease. Anabolic steroids for body building. Non smoker and no alcohol consumption

**Physical Examination:** BP: 120/65 mmHg [R], 114/65 mmHg [L], PR: 95/min regular, Peripheral pulses palpable normal bilaterally symmetrical. No radio-femoral delay. No edema. CVS- mid-late relatively loud diastolic murmur. Chest- bilateral scattered R>L coarse repitation with wheeze and tubular breath sounds at Right infra scapular region.

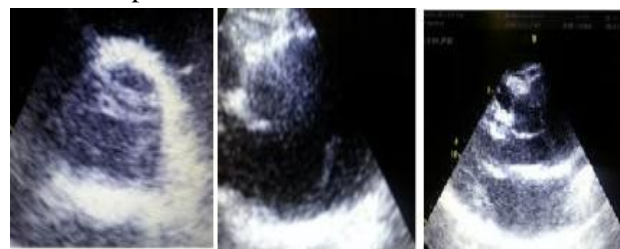
**Investigations: ECG:** NSR 95/minute. No acute ST-T changes.



**Fig 1: ECG at presentation.**

**Labs:** CBC, WBC $13.63 \times 10^3$ /ul, Hb 15.7G/dl, N 10.72[78.6%] L 11%, M 8.5%. D-dimer 1.76 mg/l [n<0.5], CRP 85. BNP 7853. RFT deranged, Normal LFT. **CX-ray,** R>L lower lobe consolidation. Heart, Mediastinum normal. No pneumothorax or pleural effusion. No hilar or Mediastinal lymphadenopathy.

CT ratio <0.5. **ECHO:** Normal LV dimensions and systolic function. Dilated Aortic root with visible intimal flap in Aorta. Mod-severe AR.



**Fig 2: Echocardiogram shows intimal tear/flap in different views.**

**Course:** Mr. A was diagnosed with acute aortic dissection with mod-severe AR and acute heart failure in context with Lower respiratory tract infection. He was immediately referred to cardiothoracic surgeon and was operated the same day. Operative findings revealed ascending aortic aneurysm 8 cms. Dissection within the aneurysm and tears at Sino tubular junction and arch. Severe aortic incompetence due to dilated root and normal leaflets. Surgery included tube graft replacement of root, ascending and arch with preservation and reimplantation of valve leaflets within the tube graft [David's Procedure]. Postoperative period was complicated with bleeding renal impairment requiring temporary dialysis and hepatic impairment. He was subsequently discharged with normal renal and hepatic function.

## DISCUSSION

Hypertension is a main risk factor of aortic sclerosis and subsequent aortic aneurysm formation and aortic dissection. Smoking and hypercholesterolemia are additional risk factors. 15%–20% of death secondary to high speed accidents are related to aortic trauma, frequently associated with myocardial contusion. Iatrogenic aortic dissection is often related to cardiac catheterization, angioplasty, or surgery. Inflammatory diseases can affect the aorta as in Takayasu arteritis and syphilis as well as in Behcet's or Ormond's disease. Cocaine and amphetamine associated with aortic aneurysm formation and dissection are newly detected etiologies.<sup>10</sup>

### **Aortic dissection -common presenting symptoms**<sup>10</sup>

Pain: Pain alone, Pain with syncope, Pain with signs of congestive heart failure, -Pain with cerebrovascular accident (stroke), Congestive heart

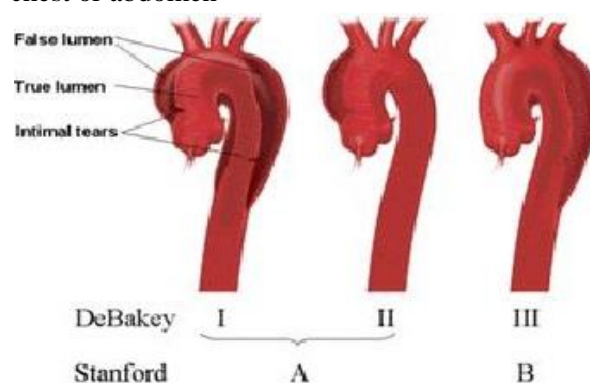
failure without pain, Cerebrovascular accident without pain, Abnormal chest roentgenogram without pain, Pulse loss without pain.

#### **Aortic dissection: deferential diagnosis**<sup>10</sup>

Acute coronary syndrome with and without ST-elevation, Aortic regurgitation without dissection, Aortic aneurysms without dissection, Musculoskeletal pain, Pericarditis, Mediastinal tumors, Pleuritis, Pulmonary embolism, Cholecystitis, Atherosclerotic or cholesterol embolism

The cardiovascular system adapts to exercise. Top-level training is often associated with morphological changes in the heart including increases in the left ventricular chamber size, wall thickness, and mass. The increase in the left ventricular mass as a result of training is called "athletes' heart".<sup>5</sup> Morgan Roth and his colleagues<sup>6</sup> distinguished two different morphological forms of athletes' heart: a strength-trained heart and an endurance-trained heart. According to their theory, athletes involved in endurance training, sports with a high dynamic component like running, are presumed to demonstrate eccentric left ventricular hypertrophy, characterized an unchanged relationship between left ventricular wall thickness and left ventricular radius (i.e. ratio of wall thickness to radius), which means an increased left ventricular chamber size with a proportional increase in wall thickness. On the other hand, strength-trained athletes involved in mainly static or isometric exercise like weightlifting, bodybuilding, and wrestling, are presumed to demonstrate concentric left ventricular hypertrophy, which is characterized by an increased ratio of wall thickness to radius, which means an increased left ventricular wall thickness with an unchanged left ventricular chamber size. In addition to the aforementioned changes, in weight lifters as strength-trained athletes, cardiac output, heart rate, and blood pressure tend to increase. A rapid increase in the systemic arterial blood without a decrease in the peripheral vascular resistance, in combination with aortic medial degeneration, may contribute to the development of the aortic dissection<sup>7</sup>; this is an event that may occur in non-trained weightlifters or those with predisposing factors for aortic dissection, like hypertension, congenital cardiovascular disease (e.g. coarctation of aorta, congenital stenotic aortic valve, and unicuspid and bicuspid aortic valve),

supravalvular aortic stenosis, connective tissue disorders (e.g. the Marfan syndrome and familial cystic medial degeneration syndromes), and fibro muscular dysplasia. Also in athletes who have mild-to-moderate aortic enlargement, an increased blood pressure due to heavy weightlifting, raises aortic wall stress to a level that begets aortic dissection.<sup>8</sup> Aortic dissection is a very tragic event because of its high mortality rate of about 32%, and the most common causes of death after aortic dissection involving the ascending aorta include the rupture into the pericardial cavity with resultant tamponade, occlusion of the coronary arteries, and free rupture into the chest or abdomen<sup>2</sup>



**Fig 3: Classification of Aortic dissection**

The majority of reports describes ascending Aortic dissection (the area of greatest hemodynamic stress), which is also the most common location for dissection secondary to connective tissue disorders and congenital anomalies.<sup>2</sup> In these cases, the medial portion of the aorta is weakened not from hypertension induced degeneration (as is the case with the older population<sup>1</sup>, but instead is secondary to a congenital defect.

Perhaps the most well-known connective tissue disorder is Marfan's syndrome. However, this entity represents only one end of a spectrum of conditions that stem from defective fibrillin-1 synthesis, collectively known as fibrillinopathies. Fibrillin-1 is the lipoprotein that serves as the framework for elastin, the major elastic component of the aortic wall. While Marfan's syndrome is a dominantly inherited condition, other fibrillinopathies vary in penetrance and expression, and familiar non-Marfan's dissections have been described. Recent work suggests that aortic involvement may be related to premature termination codon mutations, and to other

mutations in the gene for fibrillin-1 (chromosome 15q21.1)<sup>9</sup>

All athletes must be assessed for predisposing factors for aortic dissection, and all patients should be encouraged to undergo appropriate diagnostic studies, like echocardiography and blood pressure monitoring while weightlifting to recognize possible predisposing factors for aortic dissection. Athletes who do have a problem should be encouraged to avoid or limit their exercise or activity by their cardiologist. It is vital that this disastrous event be prevented in young people.

#### **Prevention of aortic dissection in inherited diseases (Marfan's Syndrome, Ehlers-Danlos Syndrome, Annuloaortic ectasia)<sup>10</sup>**

1. Life-long beta-adrenergic blockade
2. Periodic routine imaging of the aorta
3. Prophylactic replacement of the aortic root before diameter exceeds 5.0 cm in patients with a family history of dissection
4. Prophylactic replacement of the aortic root before diameter exceeds 5.5 cm
5. Moderate restriction of physical activity

#### **CONCLUSION**

In conclusion, although a rare occurrence, AD should be considered in symptomatic patients with any family history of early cardiac deaths, a history suggestive of a connective tissue disorder (that is, multiple joint surgeries) or who practice weightlifting. The investigation and surveillance of fibrillinopathies patients is ill defined, but prompt referral and/or admission for further investigation is merited. Cessation of weight lifting or isotonic stress activities until a definitive investigation has been obtained is prudent. Data for Anabolic steroid usage and acute aortic dissection is inadequate till date so an alert and suspicious mind in the emergency room should be always welcome.

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