

# Anabolic Steroid Use and Aortic Dissection in Athletes: A Case Series

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## ABSTRACT

The use of anabolic steroids in supraphysiologic doses has grown in the last decade as doping drugs in athletes. The high dose of anabolic-androgenic steroids (AAS) causes cardiomyopathy, hypertension, thrombosis, myocardial infarction (MI), weakness of connective tissue, and its sequelae such as tendon injury and aortic dissection. Dissection of the ascending aorta is an uncommon injury that has been recognized with increasing frequency in bodybuilders in recent years. It has been proposed that such cases commonly accompany the weakening of connective tissue and must be actively evaluated in the presence of anabolic steroid usage. We present a case series of isolated ascending aorta dissection in athletes who were bodybuilders. All cases were evaluated by transthoracic echocardiography (TTE) and laboratory exams. These cases also served as a reminder of the risks of ascending aorta dissection with AAS, especially in strength athletes who place high demands on their musculoskeletal structures. The results of the current study suggested that anabolic steroid abuse may be associated with detrimental effects on the myocardium represented as cardiomyopathy or atherosclerotic changes in the coronary artery as MI. These findings also strongly suggest that anabolic steroid treatment predisposes the individual to aortic dissection, especially when the patients are exercised.

Athletes have always been looking for solutions to improve their strength and muscle. Doping in athletes refers to the use of drugs that increase their power and strength.<sup>1</sup>

Anabolic-androgenic steroids (AAS) are testosterone-derived compounds and constitute the primary sex hormone of men.<sup>2</sup> Doped doses of AAS have a wide spectrum of clinical complications.<sup>3</sup> Through atherogenic, thrombotic, and vasospastic effects, AAS damages the cardiovascular system.<sup>3</sup> Previous studies demonstrated that cardiac death, myocardial infarction (MI), and stroke could occur in healthy bodybuilders who use supraphysiologic doses of AAS.<sup>4,5</sup>

In many studies, atherosclerosis as a side effect of long-term AAS consumption has been reported.<sup>6-8</sup> AAS elevates low-density lipoprotein and reduces high-density lipoprotein, causing increased chances of atherosclerotic sediment and aortic wall prone to dissection.<sup>9</sup> Meanwhile, rapid hemodynamic changes in weightlifters, especially in systemic blood pressure, leads to aortic dissection.<sup>10</sup> In most cases, the aortic dissection induced by hypertension is generally related to the deposition of atherosclerotic plaques and thinning of the vascular wall.<sup>11</sup> Aortic dissection

in young, healthy individuals has been reported in the literature but is relatively rare.<sup>11</sup>

We report five cases of ascending aortic dissection in patients with a professional weightlifting background diagnosed in the Imam Ali Hospital of Kermanshah University of Medical Sciences, Iran.

## CASE REPORTS

### Case one

A 27-year-old man (75 kg) presented to our emergency department and was monitored by a cardiologist. Dissection occurred following weightlifting across his chest and shoulders. He described multiple episodes of faintness over the preceding hours. These were associated with sweating, dyspnea, and temporal right eye vision deficit. The first episode began during heavy weight lifting (180 kg) and lasted 10 minutes before resolving with rest and drinking fluids. The second episode began while lifting weights again, which was more intense in severity and lasted for 20 minutes. The third fainting episode occurred when he left the gym, whereby he fell over the floor outside the club. The patient was a coach and a professional bodybuilder. During the

first lifting, he remembered weight training heavier than his normal regimen involving squatting with weights of around 140 kg in the supine position across the anterior chest wall. He initially denied taking illicit drugs but, upon insistence, he confessed that he took intramuscular 250 mg testosterone twice per week and human growth hormone 20 mg/day for two years. In the emergency room, the patient's vital signs were a blood pressure of 90/60 mmHg, heart rate of 120 beats/min, respiratory rate of 26 breaths/min, and temperature of 36 °C. Blood chemistry revealed increased levels of cardiac troponin (up to 7.6 ng/mL) and creatine kinase (CK) (up to 597 IU/L). Transthoracic echocardiography (TTE) revealed ascending aorta dissection with severe aortic regurgitation. The patient underwent emergency cardiac surgery.

#### **Case two**

A 33-year-old man presented with an ascending aorta dissection. The patient was referred to the cardiology department with stabbing, retrosternal chest pain, which had appeared after weight lifting in the gym some hours before. The pain was consistent and was not similar to any pain that occurred previously in his chest wall, at the time when he was having difficulties in lifting at the gym. The history showed that the patient had been doing bodybuilding combined with weightlifting, professionally for 10 years. The patient did not deny using steroids and other illicit drugs. He used anabolic steroids comprised of human growth hormone 30 mg/day and testosterone 400 mg/day twice a week. He presented at the emergency room in cardiogenic shock. In the physical examination, the patient was confused with cold sweating, where his pulse was not palpable. The musculature of his shoulder girdle, pectoralis, rectus, and quadriceps was remarkably developed. The electrocardiogram (ECG) revealed 3 mm ST-segment elevation in inferior and precordial leads 3, but previous ECG tracings were not available for comparison. Coronary angiography was not performed preoperatively. TTE performed on admission revealed right ventricular hypokinesia, left ventricular hypertrophy with reduced global left ventricle ejection fraction (EF = 45%). The large flap was observed in the anterior wall of the ascending aorta. His chest radiograph was normal. The laboratory data were unremarkable except for increased biochemical evidence of myocardial damage: CK = 1.387 U/L, CK myocardial-bound (CK-MB) = 108 U/L, and

troponin I > 50 mmol/L. The patient was initially treated with intravenous nitroglycerin and scheduled for an emergency operation. A median sternotomy was performed, and a large hematoma was removed from the pericardial cavity, and arterial blood leaked to the pericardium from the ruptured aortic wall. The postoperative period was complicated by low cardiac output, which was managed by inotropic drugs and intra-aortic balloon pump (IABP). The ECG change disappeared within several hours postoperation, and the patient was weaned from inotropic drugs and IABP during the next day of operation. Echocardiography performed on the fifth day confirmed hypokinesia of the right heart wall motion where ECG abnormalities were not normalized over the following two months and beyond. The patient was followed-up for 24 months and remained asymptomatic until today. He stopping using illicit drugs.

#### **Case three**

A 30-year-old man (71 kg) who was a bodybuilder was admitted to the emergency department for evaluating a troublesome retrosternal pain of 30-minute durations. The pain started abruptly without any preceding symptoms after lifting weight more than his usual norm. He experienced severe retrosternal pain that was not radiating to the neck, arm, or interscapular region. Examination of the patient was unremarkable except for a well-formed and tortuous musculature in the anterior chest wall and shoulder girdle, with severe anxiety, cold sweating, and delirium. His medical history revealed growth hormone (25 mg/day) and testosterone (250 mg/week) usage. The patient had no clinical features of Marfan syndrome. His pulse was 140 (beats/min), and his blood pressure was 60/45 mmHg. A chest X-ray revealed mild mediastinal widening. Laboratory tests indicated increased levels of myocardial necrosis markers CK (1987 IU/L) and troponin (> 50 mmol/L). TEE revealed aortic dilatation with the possible presence of hematoma in the aortic wall or a small intimal flap in the proximal ascending aorta. There was also severe aortic regurgitation, and pericardial hematoma noted. The patient was scheduled for an emergency operation. His recovery was uneventful recovery, and he was discharged on the seventh postoperative day. Pathology revealed no specific findings in the resected aortic segment. At the six-month follow-up, he was well, and no recurrence of dissection was observed.

### Case four

A 31-year old male bodybuilder was admitted to our center with chest pain and dyspnea. Past medical history was unremarkable except for consistent intramuscular testosterone (250 mg/week) and methenolone acetate (200 mg/week) for three years for bodybuilding purposes. In physical examination, the patient had anxiety, sweating, and low blood pressure (70/20 mmHg) with a systolic 3/6 murmur heard along the left sternal border. TTE showed severe aortic valve regurgitation and ascending aortic dissection with the tearing site of the flap in the sinutubular junction with dissection flap extending to the abdominal aorta. Coronary arteries angiography was not performed. The patient underwent cardiac surgery. The operation was complicated with delirium and acute renal failure in the postoperative course. The patient was discharged on the 23<sup>rd</sup> postoperative day in good condition.

### Case five

A 33-year-old male who was a professional bodybuilder was referred to our emergency department. He reported the use of AAS for two months before his admission. Specifically, he took intramuscular testosterone 250 mg twice per week. His blood pressure was 155/90 mmHg, and heart rate was 106 beats/min. His laboratory blood analysis revealed cardiac troponin 8.5 ng/mL, CK = 700 IU/L, CK-MB = 302 IU/L. After the last lifting of a heavy weight, which exerted tremendous pressure on his chest wall, the patient suddenly complained of a heavy chest and back pain, irradiating to the shoulders. In the emergency room, an instant drop in blood pressure to 80/30 mmHg was documented, and the patient developed a cardiogenic shock because of cardiac tamponade. The patient was immediately transferred to the operating room. Transesophageal echocardiography showed a tricuspid aortic valve with moderate aortic regurgitation and an aortic annulus, which was in the normal upper limit (44 mm). After implantation of the supracoronary graft, TEE demonstrated a mild residual tricuspid aortic valve regurgitation that could be left in situ. Further postoperative course was uneventful. A TTE in the outpatient clinic, more than two months after aortic surgery, could not demonstrate mild residual aortic regurgitation.

## DISCUSSION

One of the side effects of anabolic steroid use is aortic dissection, which is associated with hypertension and atherosclerosis. Chronic steroid use may cause fragility of the vessel because of the negative effect of the anabolic steroids on collagen formation and connective tissue strength. Several studies have reported that one of the adverse effects of long-term steroid therapy is the occurrence of atherosclerotic changes in the arterial system.<sup>6-8</sup>

Elevation of blood pressure induces macrophages directly as stress and indirectly as an inflammatory factor. Studies have shown that in people with high blood pressure, inflammatory factors such as interleukin-6, vascular endothelial growth factor, macrophage chemoattractant protein-1 (MCP-1), MMP-2, and MMP-9 rise.<sup>12,13</sup>

The pathology of aortic dissection is not well-defined. Possible mechanisms include intimal tear due to hemodynamic changes. High blood pressure is believed to exacerbate atherosclerosis plaque formation, which may cause intimal tear formation.<sup>10</sup>

Connective tissue injury has been found as a complication of anabolic steroid use in athletes. Note that only skeletal muscle training in bodybuilding causes rapid adaptations to connective tissue.<sup>14</sup> These hypertrophy and strength growths of skeletal muscles take place rapidly, but connective tissues (vessel walls, tendons, ligaments) remain weak and predisposed to trauma in conditions where vigorous force are applied to them. Therefore, connective tissue injuries of the aortic wall in bodybuilders are thought to occur from a rapid rise in training intensity, volume, and hypertension.<sup>15</sup> Experimental study of animal models has suggested that anabolic steroids may alter biomechanical properties of connective tissue of the aortic wall, though ultrastructural evidence supporting this claim is lacking.<sup>15</sup> Supraphysiologic doses of androgenic steroids reduce the degradation and promote the synthesis of type I collagen, which has no role in collagen strength in the aortic wall.<sup>16</sup> Weakened connective tissue of great vessels has presented itself in the dissection of ascending aorta as Laplace's law render ascending aorta as a most predisposed area for dissection.<sup>17,18</sup>

Illicit drug users, such as bodybuilders, have a chance of MI, dissection, cardiomyopathy, and ischemic chest pain, which rarely occur in other young patients and are not caused by coronary

artery disease. Instead, they are usually related to an abnormal course of the coronary artery in the retro pulmonary artery, supraventricular tachyarrhythmia, myocarditis, aortic stenosis, dissection, or coarctation. Young bodybuilders presenting with chest pain have an equal chance to have dissection or MI where fibrinolytic therapy may be indicated, yet these patients need precise preoperative diagnosis of the disease to prevent catastrophic events. In non-illicit drug users, fibrinolytic therapy is associated with a catastrophic event as MI occurrence is exceedingly rare.

Several factors can affect aortic dissection, such as age, hypertension, and congenital abnormalities such as Marfan's syndrome, Turner's syndrome, bicuspid aortic valve, and aortic coarctation.<sup>19</sup> We reported five cases of young bodybuilders with ascending aortic dissection who were referred to the Imam Ali Hospital of Kermanshah University of Medical Sciences. All had no congenital abnormality. Their initial symptoms of dissection developed while they were lifting weights. All patients had a history of hypertension and previously used anabolic steroids. They were successfully treated surgically, and histopathology revealed aortic medial changes in all of them.

Although information about the relationship between the aortic dissection and the use of anabolic steroids was associated with a case report study, awareness of the relationship between them is effective. Following-up patients with long-term steroid use is important to prevent aortic dissection.

## CONCLUSION

Hemodynamic stresses of weight lifting, including a rapid rise in systemic arterial blood pressure without a drop in total peripheral vascular resistance, in combination with aortic medial degeneration, may have contributed to the development of aortic dissection. Successful treatment included aortic fenestration and aortoiliac bypass. The findings suggested that a new type of aortic dissection in young weightlifters may be emerging.

### Disclosure

The authors declared no conflicts of interest.

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