CASE REPORT

Cardiomyopathy induced by anabolic-androgenic steroid abuse

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SUMMARY

Bodybuilders use anabolic-androgenic steroids to increase muscle mass, but abuse of these hormones has been related to cardiomyopathy in the past. A 60-year-old Caucasian male bodybuilder with medical history of male hypogonadism and on testosterone replacement therapy, allegedly preparing for a weightlifting competition and receiving stem cell infusions from his trainer, is transferred to the intensive care unit for worsening shortness of breath after failing treatment for community-acquired pneumonia. Chest X-ray on transfer was suggestive of pulmonary oedema, and transthoracic echocardiography showed an ejection fraction of 25%–30%. The patient was taken for cardiac catheterisation, which yielded non-ischaemic cardiomyopathy. His testosterone levels were supratherapeutic. Anabolic-androgenic steroid abuse can be a cause of cardiomyopathy in patients who have no other risk factor for such disease.

BACKGROUND

Anabolic-androgenic steroid (AAS) is the synthetic derivative of the male hormone testosterone that is often used by athletes to increase muscle mass and strength, thereby increasing their performance in competitive events.¹ The Food and Drug Administration has approved AAS for use in the treatment of male hypogonadism, wasting syndrome in HIV infection, and anaemia due to bone marrow or renal failure, but it continues to be a drug of abuse in spite of it being a controlled substance, with at least three million abusers in the USA.² There has been speculation that prolonged abuse of AAS is associated with life-threatening conditions such as sudden cardiac death (SCD), myocardial infarction (MI), increased serum lipoproteins, congestive heart failure and polycythaemia.³ We present a case of AAS-associated, non-ischaemic cardiomyopathy with associated polycythaemia.

CASE PRESENTATION

A 60-year-old Caucasian man with medical history of primary male hypogonadism and on testosterone cypionate replacement therapy, 200 mg intramuscularly every 3 weeks, was transferred to the intensive care unit 1 day after admission due to acute hypoxic respiratory failure after being admitted for community-acquired pneumonia, which is treated with azithromycin and ceftriaxone. The patient denies any fever, chills, diaphoresis, chest pain, recent sick contacts or long periods of immobilisation, no other

medical history, and no family history of cardiac disease. He also denies smoking or illicit drug abuse. He was advised, a month prior to admission, by his primary care physician to discontinue use of testosterone since his last level was 2872 ng/dL, which a year ago was 388 ng/dL, with haemoglobin of 17 mg/dL; given that he was training for a weightlifting competition, he continued testosterone plus stem cell infusions, which he was getting illegally from his trainer. Vital signs on transfer were remarkable for tachycardia of 130 beats per minute, respiratory rate of 45 breaths per minute, blood pressure of 82/64, temperature of 37°C and an oxygen saturation of 89% on the bilevel positive airway pressure mask on 100% fractional inspired oxygen. Physical examination disclosed an agitated, muscular man, with heart and lung examination disclosing S3 and S4 with bilateral inspiratory crackles, and accessory muscle use. He was oliguric and there were no rashes noted. The rest of the exam was otherwise within normal limits. Remarkable laboratory findings are shown in [table 1](#), while chest X-ray ([figure 1](#)) and CT ([figure 2](#)) showed findings suggestive of pulmonary oedema and cardiomegaly. Electrocardiogram demonstrated sinus tachycardia plus T wave inversions in leads V4, V5 and V6 accompanied with negative troponins ([figure 3](#)), while transthoracic echocardiography (TTE) ([figure 4](#)) yielded an ejection fraction (EF) of 25%–30% (volumes are shown in [table 2](#)). Cardiac MRI was not ordered because it was not available in our centre, and the patient was too unstable to be transferred to a tertiary centre for the study to be done.

Cardiac angiogram was preferred over coronary CT due to it being therapeutic and diagnostic. Also, the patient was unstable haemodynamically to receive high doses of beta-blocker for reparation. After performing angiogram, which denoted non-ischaemic cardiomyopathy ([figure 5](#)) and an EF of 15%–20%, an intra-aortic balloon pump (IABP) was placed and a dopamine infusion was started for haemodynamic support. A procalcitonin, full viral, inflammatory and drug panel was sent, which showed negative results. The patient's urine drug screen was negative and his testosterone level was >1500 ng/dL (reported as too high to read on lab comments). His lipid panel showed total cholesterol and low-density lipoprotein (LDL) within normal limits, but low high-density lipoprotein (HDL) at 26 mg/dL. The patient was discharged home with aspirin 81 mg daily, carvedilol 12.5 mg orally twice daily, digoxin 0.25 mg orally daily, valsartan 40 mg orally twice daily and furosemide

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Table 1 Initial laboratory findings

Test	Values
White cell count	$14 \times 10^9/L$
Haemoglobin	17 mg/dL
Haematocrit	55%
Platelet	519×10^3
Creatinine	1.6 mg/dL
Lactate	4 mmol/L

20 mg orally daily. A wearable cardioverter-defibrillator was indicated, but due to financial constraints the patient was unable to acquire. At 6-month follow-up, the patient remains symptomatic and his recent TTE shows an improved EF of 50%–55% (volumes in [table 2](#)). Repeat testosterone was decreased to 346 ng/dL.

INVESTIGATIONS

1. Chest X-ray.
2. TTE.
3. EKG.
4. Testosterone level.
5. Complete blood count.
6. Comprehensive metabolic panel.
7. Viral panel.
8. C-Reactive protein.
9. Erythrocyte sedimentation rate.
10. Procalcitonin.
11. Lipid panel.

DIFFERENTIAL DIAGNOSIS

1. Non-ischaeamic cardiomyopathy.
2. Viral cardiomyopathy.
3. Ischaemic cardiomyopathy.
4. Stress-induced cardiomyopathy.
5. Community-acquired pneumonia.

TREATMENT

1. Azithromycin 500 mg intravenously daily.
2. Ceftriaxone 2 g intravenously daily.
3. Dopamine continuous infusion.
4. IABP.
5. Aspirin 81 mg orally daily.
6. Carvedilol 3.125 mg orally daily.
7. Digoxin 0.25 mg orally daily.
8. Valsartan 160 mg orally daily.
9. Furosemide 20 mg orally twice daily.

Figure 1 Two-view chest X-ray showing cardiomegaly and pulmonary oedema.

Figure 2 CT of the chest showing pulmonary oedema, bilateral pleural effusions and cardiomegaly.

OUTCOME AND FOLLOW-UP

The patient was discharged from the hospital, and on his 6-month follow-up with cardiology he had recovery of left ventricular function ([table 2](#), 6-month follow-up). Cardiac MRI was not performed due to financial struggles and problems with transportation from the patient's side.

DISCUSSION

This case was significant for AAS abuse with polycythaemia in a patient who developed non-ischaeamic cardiomyopathy. AAS possesses anabolic and androgenic properties that can directly harm the myocardium in a dose-related manner. This is thought to be caused by fibrosis mediated by aldosterone-like effects and by growth-promoting effects on the cardiac muscle. These effects are followed by apoptosis caused by a sudden influx of intracellular calcium.^{4,5}

Other mechanisms proposed for AAS-associated cardiotoxicity are changes in lipid metabolism, which involve decrease in HDL and increase in LDL. AAS promotes a hypercoagulable state due to increased thromboxane A2 and platelet thromboxane A2 receptor, accompanied by polycythaemia. This can increase viscosity, which has deleterious effects in patients with underlying coronary artery disease.⁶ Although our patient had a normal total cholesterol and LDL, his HDL was significantly decreased and he had polycythaemia with thrombocytosis, supporting the diagnosis of AAS-associated cardiotoxicity.

Figure 3 EKG showing T wave inversion in leads V4, V5 and V6.

Figure 4 Transthoracic echocardiography displaying an ejection fraction of 25%–30%.

The Endocrine Society Clinical Practice Guidelines recommend a dose of 75–100 mg/week of testosterone in male hypogonadism, but AAS abusers report using 5–29 doses greater than recommended. This can produce side effects such as gynecomastia, testicular atrophy, liver adenomas and aggressive behaviour, but the most worrisome are severe cardiac effects. It is well established that the use of AAS can cause hypertension and left ventricular hypertrophy. There have also been reported cases associating it to SCD, atrial and ventricular arrhythmias, stroke, and MI.^{7–9}

AAS is generally consumed in blocks or ‘cycles’ at a time, usually lasting 8–16 weeks and then going through a drug-free period that can last months or years. Users become dependent when withdrawal symptoms appear after abrupt cessation of AAS. This can be accompanied by tolerance, leading patients to

higher doses and employing a pyramid schedule. Progressively increasing the dose in a stepwise manner close to the first half of the cycle before reducing them symmetrically in the second half; or patients can become abusers when they develop an uncontrolled urge to take AAS despite adverse health effects.¹⁰

Recent studies suggest that AAS users have impaired left ventricle (LV) systolic function impairment denoted by their EF when compared with non-AAS users. This finding was mostly seen in subjects who were currently on AAS at the time of the study, which suggests LV dysfunction may be related to drug use pattern.¹¹ Cardiopathological studies have found that the most common abnormality is left ventricular hypertrophy, associated with fibrosis and myocytolysis.¹²

Most patients reported to have adverse cardiac effects while consuming AAS have been young individuals with low risk for cardiovascular disease. The effect of AAS on older populations has not been well described, with the age range of case reports being 20–51 years. Older age groups may have an increased sensitivity to myocardial injury, which in turn may translate to noxious cardiovascular events in AAS users. There are no set guidelines on the treatment of AAS-induced cardiomyopathy, so we extrapolated treatment from other recommendations to treat our patient.^{13 14}

Learning points

- ▶ Anabolic-androgenic steroid (AAS) should be considered in the differential of non-ischaemic cardiomyopathy in men without any risk factors.
- ▶ AAS can directly harm the myocardium by causing tissue fibrosis and apoptosis.
- ▶ Other cardiotoxic events are changes in lipid metabolism, hypercoagulable states and polycythaemia.
- ▶ Cardiac MRI is the standard of care in the evaluation of non-ischaemic cardiomyopathy, which was not available in our centre but should be in our work-up always.¹³
- ▶ There is no guided therapy for AAS-induced cardiomyopathy, but extrapolation of management of other types of cardiomyopathies can be done to treat patients, as was done in this case.

Table 2 Echocardiogram volumes

Volumes	Values		Reference
	Admission	6-month follow-up	
Left ventricular end-diastolic volume (mL)	160	104	69–185
Left ventricular end-systolic volume (mL)	122	58	22–78
Right ventricular end-diastolic diameter (cm)	3.6	2	–
Right ventricular systolic pressure (mm Hg)	49	24	–

Figure 5 Cardiac angiography of the right and left coronary arteries displaying non-ischaemic cardiomyopathy and non-obstructive coronary artery disease.

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