Steroid-induced cardiomyopathy

"Awareness of the harmful cardiac effects of anabolic steroid use must be promoted within the medical profession and among potential users"

Clinical record

In December 2012, a 30-year-old man was admitted via the emergency department of our tertiary hospital with atrial fibrillation (AF), new-onset biventricular cardiac failure, acute renal failure and elevated liver function test results.

He presented with a 2-week history of dyspnoea, palpitations and epigastric discomfort. An electrocardiogram confirmed AF with a rapid ventricular response, and he was subsequently admitted to hospital. His initial heart rate varied between 120 and 140 beats/min and his blood pressure was 140/90 mmHg. He had distended jugular veins and cardiac examination revealed a gallop rhythm and an apical pansystolic murmur. His lungs were clear to auscultation and he had no peripheral oedema.

The patient was a successful bodybuilder and strongman. Over the past 12 months, he had taken testosterone 1.5 g per week, trenbolone 500 mg per week, methandrostenolone 40 mg daily, anastrozole 0.5 mg daily and naproxen 1.1 g daily in preparation for a national championship competition. The products were obtained through other users at the gym where the patient trained. He had ceased all the above supplements about 6 weeks before his admission. He was 141 kg at the time of presentation.

Further questioning elicited that he had taken anabolic steroids for about 7 years leading up to his presentation. He stated that he had only recently started taking trenbolone. Further examination did not reveal any evidence of gynaecomastia, testicular atrophy or acne.

His social history was otherwise unremarkable. There was no history of heavy alcohol use, smoking or illicit drugs. There was no family history of cardiomyopathy. There were no signs and symptoms of a viral illness.

Fifteen months before presentation, he had a transthoracic echocardiogram for hypertension, which revealed normal biventricular size and systolic function, normal biatrial size, normal diastolic function and normal valve function. At the time, he also underwent a treadmill stress echocardiogram, for which he exercised for 8 min 50 s on a 2-minute Bruce protocol, achieving 100% maximum predicted heart rate and 14.5 metabolic equivalents. There was no evidence of inducible ischaemia.

Initial laboratory tests showed an increased haemoglobin level (192 g/L [reference interval (RI), 130–180 g/L]) with a normal haematocrit level (0.54 L/L [RI, 0.40–0.54 L/L]); renal dysfunction (creatinine level, 138 µmol/L [RI, 62–106 µmol/L]) with normal electrolytes; a mildly increased level of high-sensitivity troponin T (26 ng/L [RI, < 15 ng/L]) with no subsequent increase; and increased liver enzyme levels (alanine aminotransferase [ALT], 207 U/L [RI, < 41 U/L]; aspartate aminotransferase [AST], 116 U/L [RI, < 40 U/L]). However, his albumin and bilirubin levels and international normalised ratio were normal. Initial therapy included metoprolol and anticoagulation with low molecular weight heparin.

The patient underwent transoesophageal echocardiography on Day 3 of his admission. This showed severe global biventricular dysfunction, moderate to severe mitral regurgitation as a result of annular dilatation, biatrial enlargement, and the presence of spontaneous echo contrast in the left atrial appendage without thrombus. Electrical cardioversion was performed, resulting in sinus tachycardia; however, AF recurred within 24 hours.

Pharmacological therapy to promote sinus rhythm included intravenous amiodarone (300 mg immediately, followed by 1200 mg over 24 hours) followed by oral loading (400 mg three times a day).

Our patient was given carvedilol and ramipril. However, he deteriorated on Day 4, developing hypotension (blood pressure, 80/50 mmHg) and renal dysfunction (creatinine level, 267 µmol/L), and a worsening of his liver function (ALT, 1857 U/L; AST, 1697 U/L). Low-dose dobutamine infusion was started and continued for 72 hours, resulting in excellent diuresis and improvement in his clinical condition with recovery of liver and kidney function.

Investigations to exclude a secondary cause of cardiomyopathy included thyroid function tests, iron studies and plasma metanephrine tests, which all returned normal results.

Our patient continued to improve and was discharged after a 15-day admission with outpatient follow-up. During the subsequent 18 months, he received the following medical therapy:

- angiotensin-converting enzyme inhibitor: ramipril 5 mg daily;
- β-blocker: carvedilol uptitrated to 50 mg twice daily;
- aldosterone antagonist: initially spironolactone 25 mg daily, subsequently changed to eplerenone 25 mg daily because of gynaecomastia;
- amiodarone to maintain sinus rhythm: initially 200 mg daily, reduced to 100 mg daily, and eventually stopped because of hyperthyroidism;
- anticoagulation for AF: initial warfarin therapy now changed to aspirin; and
- testosterone replacement (125 mg per week) for rebound low serum testosterone.

He had serial transthoracic echocardiograms, with improvement documented in left ventricular structure and function (Box).

Our patient was treated for a dilated cardiomyopathy as a result of anabolic steroid use. He has now stopped taking anabolic steroids for 18 months. He weighs 122 kg; however, through a different training regimen, he can lift the same weight as he did when he was 141 kg. ◆

Echocardiogram results

Date	LVEF	LVEDD (mm)	LVMI (g/m^2)
17/12/12*	< 15%		
30/01/13	40%	64	185
08/07/13	54%	61	165
02/12/13	60%	60	152
25/03/14	63%	59	147

LVEF = left ventricular ejection fraction (reference interval [RI], > 55%). LVEDD = left ventricular end diastolic diameter (RI, < 55 mm). LVMI = left ventricular mass index (RI, < 127 g/m^2). * Transoesophageal echocardiogram. ◆

Previous work has shown that the use of supra-physiological testosterone doses results in increased fat-free mass, muscle size and strength in men.[1] Studies have also shown that despite education about the potential side effects of anabolic steroids, many users will continue their practice.[2]

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Cardiomyopathy, including ventricular hypertrophy and dilatation, is a complication of anabolic steroid use that has previously been described.[3,4] Anabolic steroids are thought to cause changes in heart muscle structure through their effect on androgen receptors expressed on cardiac myocytes.[3]

Of note also is the regimen of anabolic steroid use in our patient. The amount of testosterone used was about 15–20 times that used for testosterone replacement therapy, and methandrostenolone is not recommended owing to its potential for hepatotoxicity.[5] Oestrogen blockade with anastrozole aims to prevent gynaecomastia resulting from anabolic steroid misuse, while also increasing serum testosterone levels.[6] Trenbolone is a veterinary grade anabolic steroid used for cattle growth, but has been used in a hazardous way by sports competitors and bodybuilders.[7]

Our case highlights an interesting presentation of a dilated cardiomyopathy with acute decompensated heart failure 6 weeks after cessation of anabolic steroids in a patient who had performed physically at an elite level only 2 weeks before admission. Further inhospital decompensation may have been precipitated by the acute effect of β-blocker therapy on cardiac output in this context, reducing the heart rate when stroke volume was extremely low. Definitive management involved cessation of the offending agents, exclusion of other reversible causes of heart failure, and initiation of conventional heart failure therapy. Awareness of the harmful cardiac effects of anabolic steroid use must be promoted within the medical profession and among potential users so that such cases can be prevented.

Lessons from practice

- Anabolic steroid use and misuse is an important issue in the bodybuilding community.
- Anabolic steroid use and misuse is an important potential cause of dilated cardiomyopathy.
- The mainstay of treatment involves abstinence from the offending agent, as well as initiation of conventional heart failure therapy.
- The recent addition of trenbolone to the patient's steroid regimen potentially contributed to his presentation. ◆

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