

Androgenic anabolic steroid abuse causing cardiomyopathy.

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Introduction

Androgenic anabolic steroids (AAS) are synthetic derivatives of testosterone and their analogs, such as testosterone enanthate, nandrolone, and androstenediones [1]. Androgenic anabolic steroid has been used in the management of hypogonadism and cachexia associated with HIV and cancer, but it is also abused especially by athletes to improve performance and muscle mass [2]. In physiologic doses, AAS have a protective effect on the cardiovascular system while supra-physiological doses are associated with adverse cardiovascular outcomes [3]

Multiple possible mechanisms have been studied in explaining the effects of AAS on the cardiovascular system including but not limited to potentiating dyslipidemia, stimulating cardiomyocyte hypertrophy, increasing cardiac stress, impairing coronary circulation and accelerating calcifications, reducing sensitivity of beta-adrenoreceptors, lowering arrhythmic thresholds and inducing myocyte apoptosis and fibrosis [4]. It has been linked with increased incidence of dilated cardiomyopathy, myocardial infarction, sudden cardiac death, and arrhythmias. Regardless of the mechanism of action, AAS associated cardiovascular disease remains a cause for concern due to the prevalence of AAS use in the general population [5]. Our case highlights a case of dilated cardiomyopathy and polycythemia associated with the use of AAS.

Case presentation

A 50-year-old male with a past medical history of hyperlipidemia, obesity, asthma, and hypogonadism on weekly testosterone injections and anastrozole came with the complaint of progressive shortness of breath over one month. A week before this presentation, the patient attributed his symptoms to an asthma exacerbation and went to the ED, where he received albuterol and was discharged on a course of prednisone with minimal improvement. However, his symptoms worsened, and he was brought to the hospital again. Physical exam and vitals were normal except for tachycardia and trace bilateral edema. Initial labs were significant for polycythemia (17.9g/dL), acute kidney injury (creatinine 1.29 mg/dl), mildly elevated liver enzymes, elevated BNP (561 pg/mL), and negative troponin. EKG was negative for ischemic ST-T changes. Chest x-ray revealed cardiomegaly. Further blood work showed elevated total testosterone level (> 1500 ng/dL) and free testosterone level (> 432 pg/mL).

An echocardiogram showed severely decreased EF at 20%, severe global hypokinesis, mild left ventricular dilatation and right ventricular dilatation, and moderately reduced right ventricular global systolic function. Cardiac catheterization did not show any significant coronary blockage. The patient was started on guideline-directed medical therapy with significant improvement in his symptoms [6]. The patient was discharged, and an outpatient cardiac MRI showed severely dilated left and right ventricle suggestive of dilated cardiomyopathy. The patient had a repeat echo seven months after the initial echo and showed a nondilated left ventricle with mildly reduced systolic function, EF 45%, normal right ventricular size, and systolic function.

Discussion

This case was significant for presenting anabolic steroid abuse with polycythemia in a patient who developed nonischemic cardiomyopathy. Androgenic anabolic steroid abuse can cause cardiomyopathy in multiple ways- direct toxic to myocardium due to increased production of reactive oxidative species, activating apoptotic pathways, and increasing the activation of the renin-angiotensin-aldosterone system, fibrosis mediated by aldosterone-like and growth-promoting effects on the cardiac muscle [7]. The endocrine Society clinical practice guidelines recommend a dose of 75-100 mg/week of testosterone in male hypogonadism, but this patient was taking 200 mg/week [8]. Androgenic anabolic steroid-induced cardiomyopathy is usually seen in young males and is reversible with cessation of androgenic anabolic steroid, which is seen in this patient.

Conclusion

Androgenic anabolic steroid abuse is one of the reversible causes of nonischemic cardiomyopathy. It is important to monitor androgenic anabolic steroid abuse in primary care.

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