CLINICAL VIGNETTE

Recognizing Anabolic Steroid-Induced Cardiomyopathy in At-Risk Populations

Jody Tai, DO and Matthew McCullough, MD

Introduction

Anabolic steroids (AS) are defined as any drug or hormonal substance chemically and pharmacologically related to testosterone other than estrogens, progestins, and corticosteroids which bind to androgen receptors in the body to promote growth and regulate metabolism.[1] AS are classified as DEA schedule III drugs due to their potential for misuse for enhancing muscle mass. An estimated 1 million Americans are actively experiencing AS use disorder.[2] Typical populations at risk include athletes and those with body dysmorphic disorder, although non-athletes and all genders and ages can be affected.[3] As such, it may be difficult to identify at-risk populations with increased risk of misdiagnoses. We present a patient with anabolic steroid-induced cardiomyopathy leading to hospitalization with decompensated heart failure.

Case Report

A 47-year-old male with no known past medical history presented to the emergency department for dyspnea on exertion that started six months ago, which correlated with the start of his first cycle of ingesting anabolic steroids to build muscle mass. The symptoms that prompted admission included shortness of breath, paroxysmal nocturnal dyspnea, orthopnea, bilateral lower extremity edema, and testicular swelling that all worsened in the past two weeks coinciding with the start of his second cycle of anabolic steroids. He denied cough, fever, chills, chest pain, or palpitations. Social history includes remote tobacco use and history of methamphetamine use, with last exposure 16 years prior. The patient was born and raised in South America and immigrated to the United States as an adult.

Upon presentation to the emergency room, his vitals included tachycardia to 117bpm and elevated blood pressure of 170/116mmHg. Physical exam was remarkable for a systolic murmur over the left lower sternal border, jugular venous distension to 12cm, bilateral 3+ pitting edema to the knee, distended abdomen, and large scrotal swelling. Labs were significant for leukocytosis of 13.9K/cumm, creatinine of 1.84mg/dL, ALT 71U/L, troponin of 0.084ng/mL, and B-natriuretic peptide of 1587pg/mL. Initial chest x-ray (Figure 1) showed moderately severe cardiac decompensation with severe cardiomegaly and pulmonary vascular congestion. Electrocardiogram did not show any ischemic changes. Transthoracic echocardiogram was significant for a reduced ejection fraction of 30-35%, severe hypokinesis in the right coronary artery (RCA) distribution, with right ventricular pressure overload and moderate pulmonary hypertension (Figure 2).

The patient was admitted with acute decompensated heart failure for intravenous diuresis with a loop diuretic. The differential for this new case of heart failure with reduced ejection fraction (HFrEF) included ischemia, particularly with hypokinesis in the RCA territory. However, left-heart coronary angiogram showed only mild luminal irregularities and no obstructive coronary artery disease (Figure 3). Given the patient's history of amphetamine use, a well-known cause of HFrEF, substance use disorder was also considered. However, urine toxicology screen was negative, and the patient reported sobriety from amphetamines and alcohol for many years. Chagas disease was also considered given his South American birth. *Trypanosoma cruzi* IgG was nonreactive. Other test included negative HIV screen, TSH, and echocardiogram with no valvular abnormalities. Ultimately, anabolic steroid use was the most likely cause of this patient's heart failure.

The patient received intravenous diuresis with total decrease of 25 kilograms in standing weight. He started guideline-directed medical therapy (GDMT) for his heart failure which included losartan, carvedilol, spironolactone, and empagliflozin during hospitalization. The patient was discharged on this regimen along with oral diuretics with a plan to transition to sacubitril/valsartan after discharge. Four months post-discharge, the patient was on maximal GDMT and reported total abstinence from anabolic steroids. Repeat echocardiogram showed increase in ejection fraction from 30-35% to 50-55%. He continues to follow closely with cardiology.

Discussion

The prevalence of AS abuse has increased over the past two decades to 6.4% in males and 1.6% in females, with male athletes at highest risk.[4] There are three main methods which people abuse both oral and injectable AS. These are classified as cycling, pyramiding, and stacking.[1] Cycling is characterized by use followed by cessation, in the belief that the drug-free cycle allows the body to recover normal hormone levels. Pyramiding involves ingestion of steroids in cycles of six to 12 weeks, starting with a low dose, slowly increasing, and then decreasing the amount to zero. This is believed to allow the body time to adjust to the high doses. Finally, stacking involves

taking two or more forms of steroids in the hopes of a positive synergistic effect.

Side effects of using AS involve the reproductive, cardiac, endocrine, and psychiatric systems.[5] Many side effects are due to secondary hypogonadism from suppression of the hypothalamic–pituitary axis.[5] Reduced gonadotropin secretion causes atrophy of the testis as well as decrease of sperm production. Gonadal function usually returns but recovery can take several months in both men and women.[6] Specific to this case, cardiovascular risk includes myocardial dysfunction, coronary atherosclerosis, arrhythmia, and concentric left ventricular hypertrophy. The pathophysiology behind AS-induced cardiomyopathy is not well understood, but a prevailing theory includes the induction of tissue fibrosis and apoptosis via androgen receptors present on cardiac myocytes.[7]

Although there is no specific AHA guidelines for the management of AS-induced cardiomyopathy, this patient responded well to extrapolation of GDMT. Other management includes discontinuation of anabolic steroids, addressing cardiovascular risk factors, and lifestyle modifications.[8] Prognosis is variable, with some showing improvement in cardiac function after discontinuation of anabolic steroids.[7]

Conclusion

AS abuse can lead to cardiovascular complications, and unlike other drug-induced causes of cardiomyopathy such as amphetamines, it is unclear whether cessation of AS can also lead to reversal of the cardiomyopathy.[7] Although no specific AHA management guidelines have been developed, most patients respond well to management extrapolation of guideline-directed medical therapy. When building a differential for patients with new HfrEF in at-risk populations, healthcare providers should maintain a high clinical suspicion of AS use. In addition to athletes and men, non-athletes and patients of all genders and ages may present with signs of hyper- and hypogonadism. Ultimately, AS-induced cardiomyopathy may be a diagnosis of exclusion after ruling out viral, ischemic, substance-induced, infiltrative, or medication-induced causes. Recognizing the cardiovascular impact of AS use and a multidisciplinary management approach should be implemented.

Figures

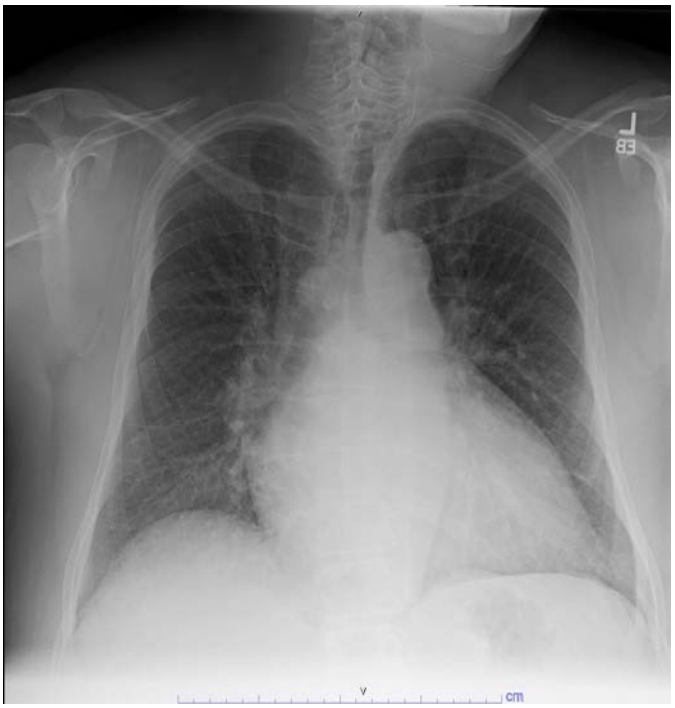

Figure 1: Chest x-ray showing moderately severe cardiac decompensation with severe cardiomegaly and pulmonary vascular congestion.

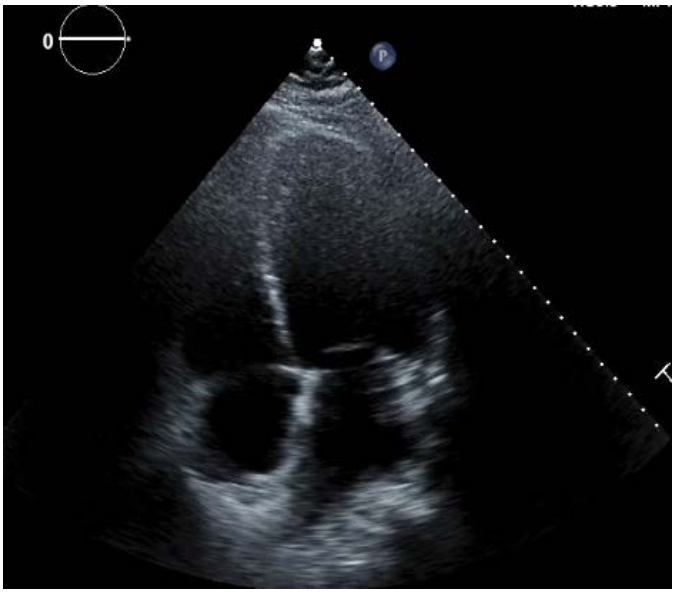

Figure 2: Transthoracic echocardiogram with reduced ejection fraction of 30-35%, severe hypokinesis in the distribution of the right coronary artery (RCA) with right ventricular pressure overload and moderate pulmonary hypertension.

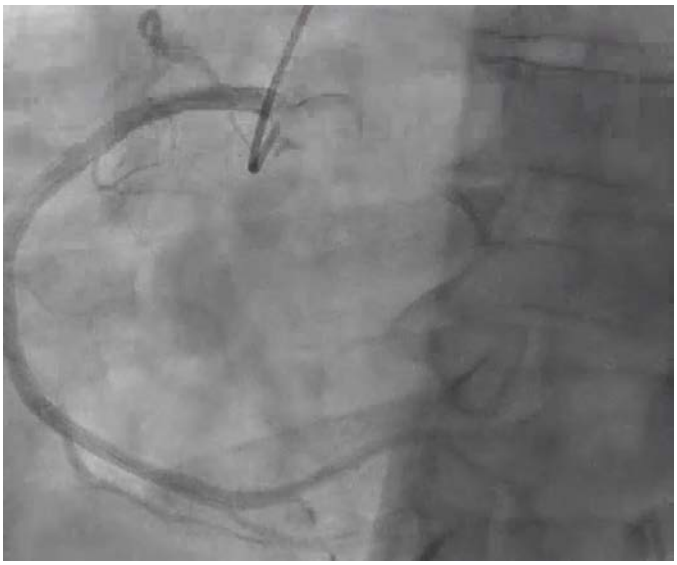

Figure 3: Left-heart coronary angiogram that showed mild luminal irregularities and no obstructive coronary artery disease.

REFERENCES

1. NIH National Institute on Drug Abuse. Anabolic Steroids and Other Appearance and Performance Enhancing Drugs (APEDs). Available at: https://nida.nih.gov/research-topics/anabolic-steroids. Retrieved 7/10/24.
2. **Graham MR, Davies B, Grace FM, Kicman A, Baker JS**. Anabolic steroid use: patterns of use and detection of doping. *Sports Med*. 2008;38(6):505-25. doi: 10.2165/00007256-200838060-00005. PMID: 18489196.
3. **Baron DA, Martin DM, Abol Magd S**. Doping in sports and its spread to at-risk populations: an international review. *World Psychiatry*. 2007 Jun;6(2):118-23. PMID: 18235871; PMCID: PMC2219897.
4. **AlShareef S, Gokarakonda SB, Marwaha R**. Anabolic Steroid Use Disorder. 2023 Jun 20. In: *StatPearls* [Internet]. Treasure Island (FL): StatPearls Publishing; 2024 Jan–. PMID: 30844201.
5. **Rahnema CD, Lipshultz LI, Crosnoe LE, Kovac JR, Kim ED**. Anabolic steroid-induced hypogonadism: diagnosis and treatment. *Fertil Steril*. 2014 May;101(5):1271-9. doi: 10.1016/j.fertnstert.2014.02.002. Epub 2014 Mar 14. PMID: 24636400.
6. **El Osta R, Almont T, Diligent C, Hubert N, Eschwège P, Hubert J**. Anabolic steroids abuse and male infertility. *Basic Clin Androl*. 2016 Feb 6;26:2. doi: 10.1186/s12610-016-0029-4. PMID: 26855782; PMCID: PMC4744441.
7. **Bhattad PB, Roumia M**. Building Body With Anabolics Is Weakening the Heart: Anabolic Steroid Induced Cardiomyopathy. *Cureus*. 2022 Jul 5;14(7):e26579. doi: 10.7759/cureus.26579. PMID: 35936132; PMCID: PMC9352142.
8. **Bhopalwala H, Hussain A, Turab M, Dewaswala N**. Anabolic steroid-induced cardiomyopathy in middle age: a case report and literature review. *J Am Coll Cardiol*. 2024 Apr;83(13_Supplement):2968.