

CLINICAL VIGNETTE

A “Swole” Heart – The Detrimental Cardiovascular Effects of Anabolic Steroids

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Case

A 61-year old male with past medical history consistent for paroxysmal atrial fibrillation (not on anticoagulation) presented with 1 week of progressive abdominal distention, orthopnea and intermittent dyspnea on exertion. Social history was remarkable for a regimented lifestyle that included several hours of weightlifting daily, monthly cycles with weekly injections of “EQ” (an anabolic horse steroid with brand name “Equipoise”, active ingredient *Boldenone undecylenate*), frequent dabbling of homeopathic remedies involving nutritional supplements and herbs, and intermittent use of intranasal cocaine to “push his workouts to the next level.” This behavior had led to at least one instance where he “lost his cool and went too far” in a public altercation.

On presentation to the ED, the patient was found to be in atrial fibrillation with rapid ventricular response with rates in the 110s-140s (confirmed with EKG) but with stable blood pressure at his baseline. Physical exam was remarkable for a muscular male, BMI 30, with pressured cadence of speech, jugular venous distention to the mandible at 45 degrees elevation, an irregularly irregular heart rhythm, lungs with mild basilar crackles, and 2+ pitting edema to the mid tibia. Admission labs revealed an AKI vs. CKD with Cr 1.5 mg/dL, transaminitis with a hepatocellular pattern (AST 105 units/L, ALT 254 units/L), WBC 6.0 g/dL, Hgb 16 g/dL, Plt 108k/uL, A1c 7.96 mg/dL, lipid panel with cholesterol 105 mg/dL, HDL 22 mg/dL, triglycerides 100 mg/dL, and LDL 63 mg/dL, Urine drug screen was positive for cocaine and troponin peaked at 0.035, with pro-BNP 6730 ng/L. He was also noted to have non-sustained ventricular tachycardia on telemetry for 5 seconds, symptomatic with rigors and acute delirium, subsequent imaging included chest radiograph with cardiomegaly, mild central vascular congestion and patchy opacities at the lung bases, a CT A/P showing mild ascites and gall bladder thickening, a renal US with normal parenchyma, and a transthoracic echocardiogram with left ventricular hypertrophy, reduced ejection fraction to 30-35%, and moderate mitral regurgitation.

The patient was admitted and started on antibiotics for pneumonia, rate control with digoxin per cardiology, and optimal medical management (OMT) and diuretics for newly diagnosed acute heart failure exacerbation. Secondary workup for cardiac and hepatic dysfunction including TSH, iron studies, HIV, RPR, COVID nasal screen and hepatitis serologies were all unremarkable. Notably, patient refused many of his scheduled medications, including his DOAC, insisting that he

would only agree to take “baby aspirin” as a blood thinner despite multiple attempts at counseling and education.

On hospital day 3, a rapid response was called when the patient was found at bedside with depressed sensorium awakening only to hard sternal rub, weakness in his extremities (4/5 strength of his right upper and lower limbs), a drooping left face and expressive aphasia. He was rushed to the ICU for serial neurologic monitoring. Immediate CT imaging including CT brain, CTA neck did not show acute intracranial abnormalities, and patient returned to neurologic baseline within 2 hours, consistent with a transient ischemic attack.

The rest of the patient’s hospitalization was characterized by stricter medication adherence, titration of OMT, and aggressive diuresis. He continued to exhibit cluster B personality traits with headstrong insistence of continuing his resistance band exercises in his hospital room, and even pre-ordered “liver detox meds” and beta-hcg from his personal trainers to better expedite his recovery. He was discharged after achieving euvolemia with close outpatient cardiology follow-up.

Discussion

Anabolic-Androgenic steroids (AAS) are synthetic testosterone derivatives taken to increase muscle mass and improve athletic performance. These hormones pass through target cell membranes and bind with intracytoplasmic androgen receptors, setting off a signaling cascade that ultimately increases protein synthesis in muscle cells (with hypertrophy of type I and type II muscle fibers) and stimulates erythropoiesis.^{1,2} Illicit use of AAS using exogenous formulations can allow users to achieve blood concentrations of testosterone more than 100 times normal physiologic levels.²

Their notoriety was brought to light in professional sports during the 1954 Vienna Olympics, with first confirmed use documented in Russian weightlifters.³ Since then, these drugs have spread to the general public recreationally for purposes of enhanced strength training, reducing recovery times in between sessions and cosmetic doping. AAS regimens typically fit one of several patterns: 1) cycling (taking steroids over a period of 6-12weeks, with abstinence period between “cycles” for a reprieve from side effects), 2) pyramiding (gradually increasing and then decreasing steroid doses over a period to mitigate withdrawal), and 3) stacking (adopts either the cycling or

pyramiding framework, with use of more than one steroid at a time to avoid tolerance and reaching a “plateau”).⁴ Stacking, in particular, illustrates most users tend to administer other drugs simultaneously and are also more likely than non-steroid users to take additional supplements of unknown purity from commercial vendors. These include protein powders and creatine, estrogen blockers, human growth hormone and ephedrine.⁵ As such, it is often difficult to find direct causal relationships between AAS and observed complications of abuse.

Longitudinal use of AAS has been tied to a variety of adverse consequences. Aside from the more well-known direct effects upon the hypothalamic-pituitary-gonadal axis including hairline recession with testicular atrophy, infertility, and aggression,⁶ they can also cause hepatic and renal impairment. They have been linked to transitory increases in liver enzymes, cholestatic jaundice, hepatic cysts and rare instances of progression to hepatocellular carcinoma. Kidney dysfunction may result as a complication of rhabdomyolysis, and focal segmental glomerulosclerosis with tubular atrophy and interstitial fibrosis can be found.²

There may also be significant detriment to the cardiovascular system. AAS can accelerate atherosclerosis by worsening dyslipidemia (increasing LDL and reducing HDL) and upregulating the activity of thromboxane-A2 receptors and platelet aggregation, thereby inducing a state of hypercoagulability and increasing risk of thrombosis.^{1,2} Arrhythmogenic thresholds are also lowered, with studies showing significantly higher incidence of abnormal post-exercise electrocardiograms (e.g., extension of QRS-complex >114 milliseconds, arrhythmias, including atrial fibrillation, ventricular fibrillation, ventricular tachycardia, supraventricular and ventricular ectopic beats) compared with non-AAS using controls.² In addition, cardiac pump was compromised. In a cohort study of gymnasium weight lifters by *Baggish et al.*, researchers obtained echocardiography of exposed (AAS-using) and non-exposed (non-AAS-using) men. Compared to non-users, AAS users demonstrated higher LV mass indexes, relatively reduced LV systolic function (mean±SD LVEF = 52±11% vs. 63±8%; P<0.001) and diastolic function (E' = 9.3±2.4 cm/s vs. 11.1±2.0 cm/s; P<0.001). In subgroup analysis of AAS users who were “off-cycle,” while systolic function showed recovery, diastolic dysfunction remained, suggesting a more permanent form of acquired pathology, postulated to be secondary to myocardial fibrosis.^{2,7,8}

Finally, our patient developed transient ischemic attack during his hospitalization. His underlying atrial fibrillation, without anticoagulation for stroke prophylaxis, as well as chronic AAS use likely elevated his risk of a cerebrovascular accident. AAS can cause platelet aggregation and increase atherosclerosis, thus increasing the risk for atherothrombotic events. Santamarina et al reported a young male athlete with no cardiovascular risk factors other than chronic use of the anabolic steroid stanozolol who suffered a posterior territory ischemic stroke.⁹ Laboratory, radiographic, and cardiovascular evaluation did not reveal any underlying chronic illness such as hypertension, dyslipidemia,

or diabetes mellitus, that would increase his risk for a cerebrovascular accident. Thus, it is important to take into consideration the risk for acute neurologic decompensation in patients with chronic AAS use, even if they do not have other cardiovascular risk factors.

Conclusion

Our patient was a chronic AAS user admitted for multiorgan dysfunction, all strongly linked to his substance abuse. Perhaps the most concerning and deserving of attention, however, are the toxic effects on the cardiovascular system. Heavy AAS use has been associated with prothrombotic states, dysrhythmias, left ventricular hypertrophy, diastolic dysfunction, and even fulminant heart failure and cerebrovascular accidents.

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