



Case Report

Anabolic-androgenic steroid use in a young body-builder: A case report and review of the literature



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ARTICLE INFO

Keywords:

Anabolic steroids

Steroid abuse

Dilated cardiomyopathy

Polycythemia

Bodybuilders

ABSTRACT

Introduction: Anabolic-androgenic steroid (AAS) abuse is routine in athletes to enhance their overall physique. It often leads to detrimental effects, including cardiovascular diseases, hormonal imbalances, and cancer. Our case presentation emphasizes two important aspects: the first is the importance of thorough history taking in correctly diagnosing diseases with multiple etiologies. The second one relates to the reversible and preventable hazards of the increasing incidence of usage of illicit drugs, mainly androgenic anabolic steroids in young adults.

Case presentation: We present a case of a 30-year-old male bodybuilder with presenting complaints of increased anxiousness, excessive anger, and dyspnea on minimal exertion. Echocardiogram showed a dilated cardiomyopathy with left ventricular ejection fraction (LVEF) of 20%. The patient was counseled for quitting AAS and symptomatically treated on heart failure management guidelines. He responded well to the management plan and now enjoying a healthy life.

Conclusion: It is imperative to raise awareness regarding the substantial adverse effects of AAS abuse that might precipitate severe cardiovascular system complications leading to morbidity and eventual mortality. Most of the times, the pathological changes due to AAS abuse are reversible. This shows a good prognosis and better compliance with the management plan advised to the patients.

1. Background

Anabolic-androgenic steroids (AAS) are synthetic drugs produced to imitate the male sex hormone testosterone [1]. They have anabolic effects more than androgenic effects as compared to testosterone. Trained athletes commonly misuse these performance-enhancing supplements. Most of these agents have no established virtue and are associated with adverse clinical outcomes [2]. To augment muscle mass, strength, growth, and athletic performance, AAS, such as boldenone, are commonly exploited. Many countries have prohibited AAS due to its significant adverse effects [3]. Multiple adverse outcomes have emerged due to the nonselective use of boldenone to enhance physical performance. International Agency for Research on Cancer classifies

boldenone as "class 2A" (growth promoter and steroid; probable human carcinogen with a high carcinogenic index) [4]. Apart from causing acne, stretch marks, hair growth, voice changes, pain, and liver changes (e.g., cholestasis, adenoma, and carcinoma), AAS can also cause notable cardiac events, such as hypertension, thrombosis, arrhythmias, systolic and diastolic dysfunction, left ventricular hypertrophy, and myocardial infarction [5]. According to a meta-analysis including 187 studies, worldwide AAS lifetime use was 3.3% and higher in men (6.4%) than women (1.6%) [1].

We herein present a case of a 30-year-old weight lifting enthusiast who had abused AAS for four years and developed dilated cardiomyopathy with a 20% ejection fraction. We report this case to wake awareness among the physicians and healthcare workers to watch out

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<https://doi.org/10.1016/j.amsu.2022.104567>

Received 5 July 2022; Received in revised form 29 August 2022; Accepted 4 September 2022

Available online 17 October 2022

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for young patients presenting with symptoms of adverse effects of AAS and establish a strong patient-physician relationship to help them recover from this illness. This case report has been reported in line with the SCARE 2020 criteria [6].

2. Case presentation

A 30-year-old male bodybuilder and weight-lifting enthusiast presented to our cardiology outpatient department with complaints of increased anxiousness, hair loss, excessive anger, and shortness of breath on minimal exertion. His past medical history was unremarkable for any recent bacterial or viral infection, coronary artery disease, and diabetes. He was a non-smoker and non-alcoholic. His drug history was significant for using androgenic anabolic steroids (AAS). He disclosed that he had been using anabolic steroids, mostly Equipoise (Boldenone) and, less commonly, Nandrolone for the past four years. He had elevated blood pressure, recorded at 160/110 mmHg. The patient was afebrile with oxygen saturation at room air within normal limits. On physical examination, the patient appeared to be excessively muscular with a ruddy appearance, an S3 gallop on auscultation of the heart, and a displaced apex beat.

The patient's laboratory investigations were unremarkable except for markedly elevated haemoglobin of 20.9g/dL, hematocrit of 57.9%, and an increased alanine aminotransferase measuring 83 U/L. His chest X-ray showed an enlarged cardiac silhouette and prominent upper lobe pulmonary vessels (Fig. 1). An echocardiogram showed a left ventricular ejection fraction (LVEF) of 20% along with a dilated left atrium and aortic root, and an enlarged left ventricular cavity with global hypokinesia; overall, severely impaired left ventricular systolic function, and a grade I diastolic dysfunction. Mitral and aortic regurgitation were also noted on the echocardiogram (Fig. 2). The ultrasound scan of both kidneys was unremarkable.

Table 1 demonstrates the laboratory and echo findings at admission, at 12 months follow-up and his most recent follow-up visit.

The possible causes of dilated cardiomyopathy (DCM) in our patient, in addition to steroid induced cardiomyopathy, were as follows: non-ischemic cardiomyopathy, ischemic cardiomyopathy, viral cardiomyopathy, and stress-induced cardiomyopathy. They were all ruled out with a thorough history, complete physical examination, and laboratory and imaging tests. Considering the given history of AAS abuse, and significant negative clinical, laboratory and echocardiogram parameters for other causes of DCM, we diagnosed AAS-induced dilated cardiomyopathy.

We counseled the patient about his current state of health and explained the potential consequences of androgenic anabolic steroid

use. We also initiated antihypertensive and heart failure maintenance therapy. During one year of follow-up, he was treated with a combination of beta-blockers, angiotensin converting enzyme inhibitors (ACEi) and aldosterone antagonist according to recommended heart failure treatment guidelines for long-term management. He also got nine phlebotomy sessions initially to normalize his hemoglobin level. Subsequent phlebotomies were done after every two months. The patient showed steady but continuous improvement in symptomatology.

As we can see in Table 1, there is a remarkable improvement throughout treatment. Left ventricular ejection fraction (LVEF) has been improved from 20% to 55%. The hematocrit came within the normal range. The patient is still being followed up regularly and is enjoying good health. He is determined not to use any steroids and works out every day and eat a balanced and healthy diet.

3. Discussion

Anabolic-androgenic steroids (AAS) abuse is a common practice nowadays, especially in young adults. These illicit performance-enhancing supplements are used by both professional athletes and high school students. Although the incidence of AAS abuse is high among professional athletes, a meta-analysis on the global epidemiology of AAS abuse has shown that the principal goal of most men who abuse AAS is to look more muscular, rather than an increase in their athletic efficiency [1].

Boldenone undecylenate, an AAS testosterone analogue, is used in veterinary medicine. Our patient used it as a part of his bodybuilding work out and developed dilated cardiomyopathy as its cardiovascular side effect. After a thorough review of the literature, we found a spectrum of clinical manifestations with which an AAS abuser can present. Some patients only had cardiovascular side effects like cardiomyopathy and acute MI; others had developed complications like stroke, liver failure, etc.

Clark and Schofield noted dilated cardiomyopathy with an ejection fraction of 10–15% in a bodybuilder taking anabolic steroids [7]. Echocardiography findings revealed four-chamber dilation with global hypokinesia. The patient responded well to the treatment given and became asymptomatic after 18 months with LVEF of 50–55%. Similarly, Ahlgrim and Guglin noted DCM with LVEF of 18% in an athlete [8].

Kalmanovich et al. reported a similar case of boldenone-induced dilated cardiomyopathy (DCM) [9]. The patient had an ejection fraction (EF) of 20%. Echocardiography findings revealed DCM with mitral and tricuspid regurgitation. Like our case, the patient showed improvement in EF after appropriate treatment. In another case report published by Garner et al., the authors found that besides reduced EF and DCM, the patient also had polycythemia [10]. This associated finding is also present in our case. Our patient also had polycythemia as per his initial CBC reports.

All these cases mentioned above are consistent with the reversible nature of non-ischemic AAS-induced cardiomyopathy without complications. The patients showed significant improvement in symptoms as well as clinical parameters at their subsequent follow-up visits.

Contrastingly, there are cases reported previously in which the patients developed complications of AAS abuse. For example, Bispo et al., Youssef et al., and Shamloul et al. noted mural thrombi formation in their respective cases [11–13]. Shamloul and his colleagues reported a stroke in their patient, and unfortunately, the patient did not survive [13].

This case presentation's uniqueness lies in two important aspects: the first thing is the cooccurrence of polycythemia with DCM due to AAS abuse, which is seen only in one case before [10]. The second thing is the reversibility of the underlying pathology that is also endorsed by the cases described above.

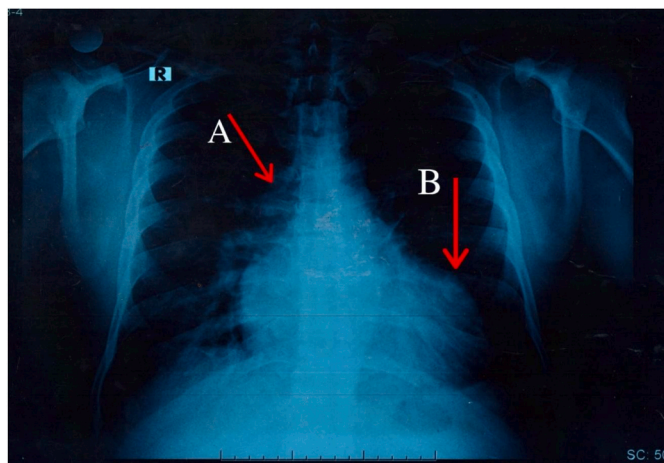


Fig. 1. Initial Chest X-ray showing prominent upper lobe pulmonary vessels (A) and an enlarged cardiac silhouette (B).

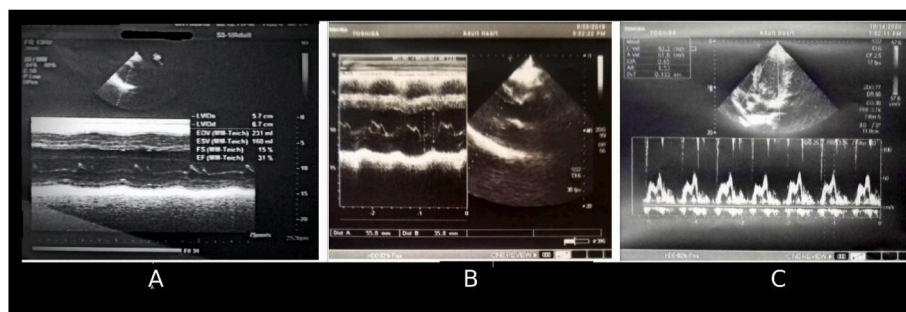


Fig. 2. Echocardiography images at admission (A), at one-year follow-up (B), and the most recent visit (C).

Table 1

Laboratory and Echocardiographic investigations of the patient at first day of presentation, follow-up after one year, and most recent hospital visit.

Significant Laboratory and Echocardiogram Parameters				
	Reference Values	Patient (At Presentation)	Patient (12 months after diagnosis)	Patient (At the most recent visit)
Laboratory				
Hemoglobin (Hb)	13–18g/dL	20.4	19.5	16.2
Hematocrit (Hct)	38–52%	57.9	57	51.9
Alanine aminotransferase (ALT)	9–40 U/L	83	157	73
LDL cholesterol	<130 mg/Dl	153	140	136
Echocardiographic Findings				
Aortic Root Diameter	20–37mm	39	36	36
Left Atrium (End Systole)	15–40mm	45	40	39
Left Ventricle Systolic Diameter (LVSD)	25–41mm	57	35	40
Left Ventricle Diastolic Diameter (LVDD)	35–56mm	67	55	55.5
Left Ventricle Ejection Fraction (LVEF)	55%–75%	20	50	55
Associated Findings		Global Hypokinesis, Trace MR, Mild AR	Mild Apical Hypokinesis, Mild AR, Thin Rim of pericardial effusion	Mild AR
MRI measured LVEF	56–78%	26	–	–
MRI measured Myocardial Mass	118–238 g	237	–	–

4. Conclusion

Meticulous history taking helps draw significant clues for the diagnosis, especially in obscure cases. It is imperative to raise awareness regarding the substantial adverse effects of AAS abuse that might precipitate severe cardiovascular system complications leading to morbidity and eventual mortality. Most of the times, the pathological changes due to AAS abuse are reversible. This shows a good prognosis and better compliance with the management plan advised to the patients.

Availability of data and materials

Not applicable.

Ethical approval

N/A.

Sources of funding

No funding was received for this case report.

Author contribution

MJ conceived and designed the study. MJ and HAS were responsible for data collection and acquisition of data. HAS, MJK, HM, UAK, and R analyzed and/or interpreted the data. HAS and HM performed the literature review. MJK, HAS, and HM wrote the initial manuscript. MJ,

UAK, and RNS critically revised the manuscript. All authors have approved the final manuscript.

Registration of research studies

1. Name of the registry: NA
2. Unique Identifying number or registration ID: NA
3. Hyperlink to your specific registration (must be publicly accessible and will be checked): NA

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Provenance and peer review

Not commissioned, externally peer reviewed.

Consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

Declaration of competing interest

No conflict of interest.

Acknowledgment

None.

Appendix A. Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.amsu.2022.104567>.

Abbreviations

AAS	Anabolic-androgenic Steroid
LVEF	Left Ventricular Ejection Fraction
DCM	Dilated Cardiomyopathy
ACEi	Angiotensin Converting Enzyme inhibitors

References

- [1] D. Sagoe, H. Molde, C.S. Andreassen, T. Torsheim, S. Pallesen, The global epidemiology of Anabolic androgenic steroid use: a meta-analysis and meta-regression analysis, *Ann. Epidemiol.* 24 (5) (2014 May 1) 383–398.
- [2] D.M. Jenkinson, A.J. Harbert, Supplements and sports, *Am. Fam. Physician* 78 (9) (2008 Nov 1) 1039–1046.
- [3] E. Tousson, M. El-Moghazy, A. Massoud, A. El-Atrash, O. Sweef, A. Akel, Physiological and biochemical changes after boldenone injection in adult rabbits, *Toxicol. Ind. Health* 32 (1) (2016 Jan) 177–182.
- [4] H.F. De Brabander, S. Poelmans, R. Schilt, R.W. Stephany, B. Le Bizec, R. Draisci, S. S. Sterk, L.A. Van Ginkel, D. Courtheyn, N. Van Hoof, A. Macri, Presence and metabolism of the anabolic steroid boldenone in various animal species: a review, *Food Addit. Contam.* 21 (6) (2004 Jun 1) 515–525.
- [5] J. Van Amsterdam, A. Opperhuizen, F. Hartgens, Adverse health effects of anabolic-androgenic steroids, *Regul. Toxicol. Pharmacol.* 57 (1) (2010 Jun 1) 117–123. Page 4 of 8.
- [6] R.A. Agha, T. Franchi, C. Sohrabi, G. Mathew, A. Kerwan, SCARE Group, The SCARE 2020 guideline: updating consensus surgical CAse REport (SCARE) guidelines, *Int. J. Surg.* 84 (2020) 226–230, <https://doi.org/10.1016/j.ijsu.2020.10.034>.
- [7] C. Ahlgrim, M. Guglin, Anabolics and cardiomyopathy in a bodybuilder: case report and literature review, *J. Card. Fail.* 15 (6) (2009 Aug 1) 496–500.
- [8] B.M. Clark, R.S. Schofield, Dilated cardiomyopathy and acute liver injury associated with combined use of ephedra, γ -Hydroxybutyrate, and anabolic steroids, *Pharmacotherapy* 25 (5) (2005 May) 756–761.
- [9] E. Kalmanovich, S. Minha, M. Leitman, Z. Vered, A. Blatt, Androgenic-anabolic steroid (Boldenone) abuse as a cause of dilated cardiomyopathy, *J. Gen. Prac.* 2 (3) (2014 Apr 3) 153.
- [10] O. Garner, A. Iardino, A. Ramirez, M. Yakoby, Cardiomyopathy induced by anabolic androgenic steroid abuse, *Case Rep.* (2018 Jun 29), 2018:bcr-2017.
- [11] M. Bispo, A. Valente, R. Maldonado, R. Palma, H. Glória, J. Nóbrega, P. Alexandrino, Anabolic steroid-induced cardiomyopathy underlying acute liver failure in a young bodybuilder, *World J. Gastroenterol.: WJG* 15 (23) (2009 Jun 21) 2920.
- [12] M.Y. Youssef, A. Alqallaf, N. Abdella, Anabolic androgenic steroid-induced cardiomyopathy, stroke and peripheral vascular disease, *Case Rep.* (2011 Jul 4), 2011:bcr1220103650.
- [13] R.M. Shamloul, A.F. Aborayah, A. Hashad, F. Abd-Allah, Anabolic steroids abuse-induced cardiomyopathy and ischaemic stroke in a young male patient, *Case Rep.* (2014 Feb 26), 2014:bcr2013203033.