Impaired Physical Performance and Clinical Responses after a Recreational Bodybuilder's Self-Administration of Steroids: A Case Report

Article · December 2015

DOI: 10.5534/wjmh.2015.33.3.209

5 authors, including:

Katherine Veras

University of São Paulo

10 PUBLICATIONS **5** CITATIONS

SEE PROFILE

Fernando L Silva-Junior

Federal University of Piauí, Parnaíba, Piauí,…

14 PUBLICATIONS **13** CITATIONS

SEE PROFILE

Adriano Eduardo Lima-Silva

Federal University of Pernambuco

115 PUBLICATIONS **457** CITATIONS

SEE PROFILE

Flávio O Pires

University of São Paulo

86 PUBLICATIONS **257** CITATIONS

SEE PROFILE

pISSN: 2287-4208 / eISSN: 2287-4690
World J Mens Health 2015 December 33(3): 209-213
http://dx.doi.org/10.5534/wjmh.2015.33.3.209

Case Report

Impaired Physical Performance and Clinical Responses after a Recreational Bodybuilder's Self-Administration of Steroids: A Case Report

Katherine Veras[1,2], Fernando Lopes Silva-Junior[2], Adriano Eduardo Lima-Silva[3], Fernando Roberto De-Oliveira[4], Flávio Oliveira Pires[2]

[1]Biomedical Sciences Institute, University of São Paulo, [2]Exercise Psychophysiology Research Group, School of Arts, Sciences and Humanities, University of São Paulo, São Paulo, [3]Sport Science Research Group, Department of Physical Education and Sports Science (CAV), Federal University of Pernambuco, Recife, [4]Departament of Physical Education, Federal University of Lavras, Lavras, Brazil

We reported clinical and physical responses to 7 weeks of anabolic-androgenic steroid (AAS) self-administration in a male recreational bodybuilder. He was self-administrating a total of 3,250 mg of testosterone when his previous and current clinical and physical trials records were revisited. Body shape, performance, and biochemistry results were clustered into three phases labeled PRE (before the self-use), POST I (immediately at the cessation of the 7-week administration), and POST II (12 weeks after the cessation). Elevated testosterone and estradiol levels were observed in the POST I phase, while hepatic and renal functions remained altered in the POST II phase. Body mass and body fat percentages increased throughout the three phases. When adjusted according to body mass, drops in aerobic and anaerobic power and capacity (2.1% to 12.9%) were observed across the phases. This case report shows that overall performance decreased when a bodybuilding practitioner self-administered AAS.

Key Words: Doping in sports; Lipid metabolism; Strength training; Testosterone

According to the position stand of the American College of Sports Medicine [1], administration of anabolic-androgenic steroids (AAS) improves strength in both experienced and inexperienced weight trainers with or without a controlled diet. AAS is used to increase body dimensions and body weight, although increases in lean body mass are less evident. On the other hand, the use of AAS has been associated with altered endocrine, sexual, and hepatic functioning [2].

Legally or illegally, athletes may use AAS while being clinically monitored by a team of physicians and physiologists [2]; however, bodybuilding practitioners do not typically rely on a multidisciplinary team but instead self-administer the drug, so they end up taking greater risks when using AAS. Informal testimonials suggest that their decision making is based on anecdotal reports of an attitude that health risks are limited and offset by great performance gains [3,4]. In this regard, the lack of data obtained di-

Received: Jun 9, 2015; Revised: Aug 4, 2015; Accepted: Sep 14, 2015

Correspondence to: Flávio Oliveira Pires
Exercise Psychophysiology Research Group, School of Arts, Sciences and Humanities, University of São Paulo, Arlindo Béttio, 1000, 1524 - Ermelino Matarazzo - São Paulo/SP 05508-000, Brazil.
Tel: +55-11-30917246, Fax: +55-11-95335777, E-mail: piresfo@usp.br

rectly from bodybuilding practitioners that combines clinical and performance measures may contribute to this misperception. In fact, most studies have focused on either physical (performance and body shape) or clinical effects in isolation [5,6]. Thus, we report here the clinical, performance and body shape outcomes, in combination, in a bodybuilding practitioner that had self-administered AAS for 7 weeks.

CASE REPORT

A 28-year-old Caucasian male was a bodybuilding practitioner experienced in strength training (~10 years) while he was participating in a local community program for promotion of health and well-being. This program offered exercise and physical activity orientation as well as support for those engaged in clinical and physical trials. However, individuals were free to include different exercise modes and routines in addition to the recommendations provided. During a visit for a clinical trial, the individual reported dizziness, headaches, and mood disturbs while he was filling out a questionnaire. After mentioning his AAS self-use, the individual was advised to end the drug administration immediately. Thereafter, it was recommended that he avoid the use of medication and maintain his habitual diet for a 3-month wash-out period. The records of his previous and current clinical and physical trials were revisited, and new clinical and physical trials were performed. The individual provided written informed consent for the use of these data, in conformity with the Helsinki declaration. Thus, clinical, performance, and body shape data were clustered as PRE (before the AAS self-administration), POST I (immediately at the cessation of the 7-week AAS self-administration), and POST II phases (12 weeks after the cessation of self-administration). The PRE phase refers to measures obtained within 1 month before the AAS self-use was begun.

For the purpose of this study, the individual provided details on the drug administration and his bodybuilding training schedule. He had self-administered 3,250 mg of a commercial steroid which contained Propionate, Phenylpropionate, Isocaproate and Caproate (Duratestom®; Organon, São Paulo, Brazil) for 7 weeks (465 mg/wk), while performing strength training for muscle hypertrophy (daily training volume of ~836 repetitions).

1. Clinical variables

The results of the clinical investigation are presented in Table 1. Elevation of free testosterone (T) concentrations was observed in the POST I phase, after the 7 weeks of AAS self-administration. However, the T levels tended to match the PRE values in POST II phase, 12 weeks after the cessation of AAS-self administration. Follicle stimulating hormone (FSH), luteinizing hormone (LH), and estradiol concentrations remained at normal range throughout the period (Table 1).

Total cholesterol ($CHOL_T$) and low-density lipoprotein (LDL) were elevated in PRE phase, so that decrements were observed after AAS discontinuation. Furthermore, when compared to the PRE phase, the very-low-density lipoprotein and triglycerides (TG) values were increased by more than 10% in the POST I and by more than 130% in the POST II. In contrast, comparing the same phases, high-density lipoprotein (HDL) levels decreased by 23% and 13% in the POST I and POST II, respectively. Table 1 summarizes these results.

Renal and hepatic biomarkers were not altered in the POST I and POST II phases, at the cessation and 12 weeks after the end of AAS self-administration, respectively (Table 2). The exception was the urea levels, which were already high in the PRE phase.

Table 1. Hormonal and lipid profile before AAS self-administration (PRE), immediately at the cessation of 7-week AAS self-administration (POST I), and 12 weeks after cessation (POST II)

Variable	PRE	POST I	POST II	Normal range
LH (IU/L)	5.1	4.1	3.7	1.0~8.4
FSH (IU/L)	2.8	2.2	2.3	<10.5
Testosterone (pg/mL)	624	1,303	802	271~965
Estradiol (pg/mL)	3.17	5.04	3.68	ND~52
$CHOL_T$ (mM/L)	285	227	267	<200
TG (mM/L)	112	125	259	<150
HDL (mM/L)	59	45	51	>50
LDL (mM/L)	204	157	164	<130
VLDL (mM/L)	22	25	52	2~30

AAS: anabolic-androgenic steroids, LH: luteinizing hormone, FSH: follicle stimulating hormone, $CHOL_T$: total cholesterol, TG: triglycerides, HDL: high-density lipoprotein, LDL: low-density lipoprotein, VLDL: very-low-density lipoprotein, ND: non-detectable.

2. Performance variables

A maximal incremental cycling test (1600 EC, Ergociser; Cateye, Osaka, Japan) was used to assess aerobic power (peak power output; W_{PEAK}) and capacity (lactate threshold; $LT_{3.0}$), and a Wingate test assessed the anaerobic power (AnP) and capacity (AnC). The one-repetition maximum (1RM) test indicated the maximal strength. Performance results were expressed relative to the current body mass, as AAS induces increases in body mass.

The POST I phase showed a decrease in the W_{PEAK} and slight variation in the $LT_{3.0}$ when compared to the PRE phase. Furthermore, both the W_{PEAK} and $LT_{3.0}$ showed additional decreases in POST II, so that values recorded 12 weeks after the cessation of AAS self-administration were 5.5% and 2.1% lower than those observed in the PRE phase, respectively. Accordingly, when compared to the PRE phase, a 3.1% and 12.9% decrease in AnP and AnC, respectively, was observed at the POST II phase. However, there were 7.8% (AnP) and 2.7% (AnC) increases in the POST I phase. Thus, the aerobic and anaerobic performance 12 weeks after the end of AAS self-administration was lower than the performance measured before the AAS administration (Table 3). The 1RM leg press test results showed an increase throughout the period of AAS administration. Therefore, when compared to the PRE phase, the 1RM load increased 2.2% and 7.0% in POST I and POST II phases, respectively (Table 3).

Table 2. Renal and hepatic serum biomarkers before AAS self-administration (PRE), immediately at the cessation of 7-week AAS self-administration (POST I), and 12 weeks after cessation (POST II)

Variable	PRE	POST I	POST II	Normal range
Creatinine (mg/dL)	1.1	1.1	1.0	0.7～1.4
Urea (mg/dL)	29.1	31.3	30	5～20
AST (U/L)	21	18	23	14～59
ALT (U/L)	13	15	26	10～55
ALP (U/L)	79	-	72	45～150

Normal ranges: values adapted from the User's Guide, Associated Regional and University Pathologists Laboratories. AAS: anabolic-androgenic steroids, AST: the aspartate transaminase concentration, ALT: the alanine transaminase concentration, ALP: the alkaline phosphatase concentration, –: missing data.

3. Body shape variables

Measures of height, body mass, circumferences of the neck, chest, waist, abdomen, hips, arms, forearms, thighs, and legs, and six skinfolds (triceps, subscapular, chest, suprailiac, abdomen and thigh; Harpenden Skinfold Caliper®; Harpenden, Harpenden, England) were performed to calculate the body fat percentage (%BF), body mass index (BMI), and waist-to-hip ratio (WHR). When compared to the PRE phase, the body shape results revealed an increase of 5.6% and 6.8% in the POST I and POST II phases, respectively. This increased body mass was followed by elevations in %BF and the sum of skinfolds in POST I (4.6% and 10.0%, respectively) and POST II (24.6% and 27.9%, respectively). Accordingly, the BMI and WHR indexes increased throughout the period investigated, both the POST I and POST II phases (Table 4).

DISCUSSION

1. Clinical variables

Although the T concentration was elevated above the

Table 3. Performance variables before AAS self-administration (PRE), immediately at the cessation of 7-week AAS self-administration (POST I), and 12 weeks after cessation (POST II)

Variable	W_{PEAK} (W/kg)	$LT_{3.0}$ (W/kg)	AnP (W/kg)	AnC (W/kg)	1RM (kg/kg)
PRE	3.7	2.5	5.8	4.2	4.7
POS I	3.6	2.5	6.2	4.3	4.8
POS II	3.5	2.4	5.6	3.7	5.1
%⊿PRE-POS I	−2.8	+0.7	+7.8	+2.7	+2.2
%⊿PRE-POS II	−5.5	−2.1	−3.1	−12.9	+7.0

Variables are expressed relative to body mass changes.
AAS: anabolic-androgenic steroids, W_{PEAK}: peak power output during incremental test, $LT_{3.0}$: lactate threshold, AnP: anaerobic peak power output, AnC: relative anaerobic capacity, 1RM: one-repetition maximum test.

Table 4. Body shape variables before AAS self-administration (PRE), immediately at the cessation of 7-week AAS self-administration (POST I), and 12 weeks after cessation (POST II)

Variable	∑SF (mm)	BF%	BM (kg)	BMI (kg/m^2)	WHR
PRE	45.2	6.5	66.5	25.0	0.85
POST I	49.7	6.8	70.2	26.4	0.89
POST II	57.8	8.1	71.0	26.7	0.87
%⊿PRE-POST I	+10.0	+4.6	+5.6	+5.6	+4.7
%⊿PRE-POST II	+27.9	+24.6	+6.8	+6.8	+2.4

AAS: anabolic-androgenic steroids, ∑SF: sum of all skinfolds, BF%: body fat percentage, BM: body mass, BMI: body mass index, WHR: waist-to-hip ratio, %⊿: percent change.

normal range at POST I, T levels returned to the normal range in the POST II phase. Perhaps the dosage and duration of administrationallowed a fast restoration of T levels. Furthermore, the LH and FSH were maintained throughout the AAS administration period. Some studies have reported pituitary-hypothalamus axis suppression after AAS administration; thus further research is required to elucidate the dose-response and time course relationship to AAS [4,5].

The HDL results were in agreement with the literature. The 23.7% drop we observed in HDL after 7 weeks of AAS agreed with the consistent, remarkable reduction in HDL levels previously reported after 3 to 26 weeks of AAS administration [2]. It has been postulated that supra-physiological doses of AAS are related to a decrease in HDL levels, partially due to the higher HDL catabolism induced by increases in hepatic lipoprotein lipase activity [7]. Studies have reported conflicting results on TG, $CHOL_T$, and LDL levels. For example, although LDL is expected to increase as a response to oral administration of stanozolol, studies have reported reductions in LDL after injections of T [2] Thus, it is possible that results of TG, $CHOL_T$, and LDL were related to the specificity of the AAS used. Further, these results may reflect excessive food consumption as individuals may prefer hyper-caloric diets when combining AAS and strength training [5]. However, we have not manipulated variables such as hormones, diet, and strength training, so it is difficult to specify what changed the lipoprotein metabolism [2].

Importantly, the bodybuilding practitioner had reduced HDL levels throughout the AAS self-administration, in addition to the increased TG levels. These results are alarming, as cardiovascular risk is expected to increase in individuals with low levels of HDL [8-10]. Thus, this case report serves to highlight the harmful effect of AAS misuse on lipoprotein metabolism related to cardiovascular risk.

2. Performance responses

Results of aerobic and anaerobic performance (adjusted for body mass alterations) agreed with earlier results expressed in absolute terms and may challenge the use of AAS to improve aerobic and anaerobic performance in activities or sports that are dependent on body mass [2]. In contrast, the 1RM load increased in the POST I and POST II phases, and these results agreed with increases between 5% and 20% reported for bodybuilders using AAS. The increase in strength may be related to the incorporation of new muscle fibers derived from satellite cells or a faster recovery after the training sessions [2].

3. Body shape responses

Increases in body mass between 2 and 5 kg have been reported with AAS misuse and may relate to increases in fluids, muscle cross-sectional area, and fat-free mass [2,5]. However, it is possible that the bodybuilding practitioner increased his body mass through increases in body fat (∑SF and body fat %), even though the changes in extracellular fluids and muscle cross-sectional area cannot be ruled out [4]. In fact, the BMI and WHR indexes were raised at POST I and POST II. Although the BMI and WHR indexes must be cautiously interpreted in bodybuilding practitioners, these results deserve attention when self-administrating AAS is combined with a high caloric intake.

In summary, we report a case of altered hormonal and lipid profile, together with impairments in overall performance and body shape (with an exception of maximal

strength performance) in a bodybuilding practitioner who had self-administrated AAS for 7 weeks. This case report serves to clarify the misperception of a minimal health risk–major performance gain relationship when administering AAS, discouraging AAS misuse by strength sports and bodybuilding practitioners.

ACKNOWLEDGEMENTS

The authors are grateful to Frederico Marchizotti and Edilamar Menezes de Oliveira, University of São Paulo, and Cindy George, University of Cape Town for the technical assistance.

CONFLICT OF INTEREST

No potential conflict of interest relevant to this article was reported.

REFERENCES

1. American College of Sports Medicine position stand on the use of anabolic-androgenic steroids in sports. Med Sci Sports Exerc 1987;19:534-9.
2. Hartgens F, Kuipers H. Effects of androgenic-anabolic steroids in athletes. Sports Med 2004;34:513-54.
3. Haupt HA, Rovere GD. Anabolic steroids: a review of the literature. Am J Sports Med 1984;12:469-84.
4. Kutscher EC, Lund BC, Perry PJ. Anabolic steroids: a review for the clinician. Sports Med 2002;32:285-96.
5. Alén M, Häkkinen K. Physical health and fitness of an elite bodybuilder during 1 year of self-administration of testosterone and anabolic steroids: a case study. Int J Sports Med 1985;6:24-9.
6. van Breda E, Keizer HA, Kuipers H, Wolffenbuttel BH. Androgenic anabolic steroid use and severe hypothalamic-pituitary dysfunction: a case study. Int J Sports Med 2003;24:195-6.
7. Hislop MS, St Clair Gibson A, Lambert MI, Noakes TD, Marais AD. Effects of androgen manipulation on postprandial triglyceridaemia, low-density lipoprotein particle size and lipoprotein(a) in men. Atherosclerosis 2001;159:425-32.
8. Hajimoradi B, Kazerani H. Echocardiographic findings in power athletes abusing anabolic androgenic steroids. Asian J Sports Med 2013;4:10-4.
9. Nottin S, Nguyen LD, Terbah M, Obert P. Cardiovascular effects of androgenic anabolic steroids in male bodybuilders determined by tissue Doppler imaging. Am J Cardiol 2006;97:912-5.
10. Santos MA, Oliveira CV, Silva AS. Adverse cardiovascular effects from the use of anabolic-androgenic steroids as ergogenic resources. Subst Use Misuse 2014;49:1132-7.