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Review Article

Anabolic-Androgen Abuse: A Practical Guide to The Primary Care Provider

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Abstract

Abuse of anabolic-androgen steroids (AAS) is common among young men to promote muscle growth. Exact prevalence of AAS abuse is unknown because most abusers do not disclose such information to their physicians. AAS abuse results in hypogonadotropic hypogonadism and infertility that may last for several months and years after cessation of androgens. Moreover, AAS abuse is associated with dependence, increased cardiovascular (CV) events and mortality. These complications are likely due to elevation of blood pressure, heart rate, polycythemia, and low high-density-lipoprotein-cholesterol (HDL-C) levels, and polycythemia. Once AAS abuse is suspected from physical exam and laboratory findings, a non-judgmental approach should be pursued by the medical provider to clarify adverse effects of androgens, and counselling about their cessation after gradual tapering or abruptly if possible. For men electing to continue AAS, close monitoring of adverse effects and their treatment are necessary. Randomized trials are urgently needed for guidance of the optimum management of complications and withdrawal of AAS.

Key words: androgens; testosterone; abuse; dependence; complications; polycythemia; cardiovascular events

Introduction

Epidemiology

AAS intake outside medical supervision is common. A meta-analysis of 187 studies suggested that lifetime global prevalence of AAS use to be 6.4% for men and 1.6% for women [1]. More recent studies showed that AAS are mainly abused by men between 20-40 years of age, who constitutes 98% of users [2]. Most AAS users are non-competitive athletes and body builders found in gyms. Thus, it is estimated that 4-6% of fitness center men visitors use AAS [2]. However, precise prevalence of AAS abuse is hard to determine as significant number of subjects do not disclose such information to their providers (see below). The main reason for AAS abuse is increasing muscle bulk and enhancement of appearance [3]. In fact, in the landmark trial of Bhasin et al in 1996, administration of supraphysiological doses of testosterone equivalent to 6 times the doses given for treatment of hypogonadal men, caused significant increase in muscle size and strength after 10 weeks [4]. In addition, testosterone increased fat-free mass, particularly when associated with exercise [4]. In a subsequent trial, the same group showed that these changes were dose-related [5]. In a recent prospective study of real-world androgen abuse, Verdegal et al [6] found that AAS for approximately 8 weeks increased fat free mass by 4.4 kg. Most of this gain was lost 3 months after cessation of androgens [6].

Patterns of AAS abuse

There are 2 main patterns for AAS abuse. The first used by approximately 80% of men is taking androgens in cycles lasting several weeks to several months, during which multiple androgens are abused in high doses reaching 5-12 times therapeutic doses given to hypogonadal men [7]. Each cycle is followed by a post-cycle period during which subjects take accessory drugs to help recovery of endogenous testosterone levels such as clomiphene citrate (CC) orally and human chorionic gonadotropins (hCG) subcutaneously [3,7]. The second less common pattern is called blast-and-cruise method, in which men use continuously natural and synthetic testosterone usually in lower doses compared with those taken during a cycle [7,8]. In both patterns, men may take an aromatase inhibitor (e.g. letrozole, anastrazole) to decrease conversion of testosterone to estradiol for prevention of gynecomastia [3,7]. It should be emphasized that most androgens and accessory drugs are obtained from the internet and black market and are therefore prone for adulteration. In an analysis of 19 studies, Magnolini et al [9] have estimated that 38% of anabolic steroids found on the black market were fake or counterfeited. Moreover, the quality of androgens in the black market is very poor with less than 20% of the products containing the claimed substance in the respective amount and other products contain undeclared ingredients [10]. The use of other illicit drugs among AAS abusers is common. In one study, 46% of AAS abusers were also abusing amphetamines and 32% cocaine [7]. In a large survey, the most frequently used androgens were testosterone enanthate (80% of users) followed by testosterone cypionate, metandione and trenbolone (50% of users), and oxandrolone and nandrolone (46% of users) [11]. Trenbolone, a synthetic androgenic steroid originally designed to promote growth in cattle, has high affinity to androgen

receptors and is associated with more psychological problems (irritability, depression, mood fluctuations, aggression) versus other abused androgens [12].

Attitudes of androgen abusers toward physicians

In a personal interview and questionnaire, Pope et al [13] and Bonnecaze et al [14] found that 56% of androgen abusers never disclosed their history of abuse to any physician. Similar proportions were recorded in another large multinational survey (n=2384) [14]. In the latter survey, 55% of those who had admitted androgen use to the providers felt discriminated against by their physicians [14]. Indeed, androgen abusers have negative attitudes towards physicians and believe medical providers are not knowledgeable about effects of androgens. Thus, 40% of androgen abusers trusted information from their drug dealers at least as much as they trusted information from any physician that they had seen [13]. Indeed, physicians were rated the lowest, whereas coaches/gurus and body building websites received the most favorable ratings with respect to knowledge about AAS [14].

Consequences of AAS abuse

1. Androgen-induced hypogonadism

By binding to androgen receptors, exogenous androgens cause feedback inhibition of the hypothalamic-pituitary-testicular axis leading to inhibition of the 2 pituitary gonadotropins: follicular stimulating hormone (FSH) and luteinizing hormone (LH) with subsequent inhibition of endogenous testosterone, testicular atrophy, oligospermia/azoospermia and infertility [3]. In most cases, this type of iatrogenic hypogonadism resolves slowly within 12 months after cessation of AAS. However, in few cases of prolonged and high-dose AAS abuse, it may last for several years or is even irreversible.

2. Anabolic-androgen dependence

Approximately, one-third of AAS abusers develop dependence [15]. Thus, shortly after discontinuation or lowering doses of androgens, these men experience withdrawal symptoms such as increased irritability, anxiety, in addition to hypogonadal symptoms including fatigue, depression, low-libido and erectile dysfunction (ED). For this reason, approximately 77-80% of subjects are reluctant to discontinue AAS [7,16].

Cardiovascular complications

A. Effects of AAS on lipid panel

The effects of androgens on plasma lipids are complex. Thus, androgens significantly decrease HDL-C levels and increase levels of the atherogenic apolipoprotein B (Apo B) [7]. On the other hand, AAS decrease levels of lipoprotein a, an independent risk factor for CVD disease [7]. Therefore, whether the net effect of androgens on lipids is beneficial or harmful in terms of CV events is unclear at present and require further studies.

B. Effects of AAS on blood pressure and heart rate

Androgen abuse causes increase in blood pressure, possibly due to water and sodium retention [7]. In one cross-sectional study, Abdullah et al [17] reported higher systolic blood pressure (SBP) in men formerly or currently abusing AAS compared with non-androgen users weightlifting control men; 141 mmHg and 133 mmHg, respectively (P<0.001). In another cross-sectional study using 24 h BP monitoring, Rasmussen et al [18] showed that current AAS abusers exhibited higher mean 24-h SBP (132 mmHg) than former AAS abusers (126 mmHg) and control men (126 mmHg). SBP did not differ between former AAS abusers and controls, suggesting that the hypertensive effect of AAS may be reversible. Further, diastolic blood pressure (DBP) and arterial pressure did not differ among all three groups. Notably, both current and former AAS abusers exhibited higher heart rate than controls [18]. An increase of heart beats of 10/min following AAS abuse was also demonstrated in the prospective study of Smit et al [19].

C. Polycythemia

Erythrocytosis is the most common adverse effect of testosterone therapy [20]. The underlying mechanism is likely stimulation of erythropoietin and inhibition of the main iron regulator by testosterone [21]. Hepcidin inhibits iron absorption in the gut. Therefore, its suppression by testosterone increases iron availability [21]. The degree of erythrocytosis, reflected by the hematocrit value, correlates with testosterone dosage and circulating testosterone concentrations, and is more common with high baseline hematocrit values, and in old age [20]. The average increase in hematocrit with T therapy in hypogonadal men is 1.4-4.3% but may be higher, approximately 5%, among users of high dose of androgens [7,22]. In a meta-analysis including hypogonadal men, the oral testosterone undecanoate and the intramuscular testosterone enanthate/cypionate formulations (the most abused androgens) caused the highest rise in hematocrit 4.3% and 4.0%, respectively [22]. This was followed by the testosterone gel 3.0% and patch 1.4% [22]. Polycythemia may be implicated in increased arterial and venous thrombosis as result of increased blood viscosity. A large retrospective study (n=5,842) found that hypogonadal men who developed polycythemia (defined as hematocrit > 52%) to testosterone therapy had higher rates of CV events and venous thromboembolism compared with those who did not have polycythemia (odds ratio 1.35; 95% CI 1.13 to 1.61) [23].

D. Cardiac adverse effects of AAS

Smit et al [19] performed cardiac 3D echocardiography in 25 men on 3 occasions: before starting androgen abuse, last week of their androgen cycle (median duration of cycle was 16 weeks), and 1 year after starting their cycles i.e. approximately 8 months after stopping androgen intake. Thus, at the end of AAS use cycle, the authors found a mild but statistically significant decrease in left ventricular ejection fraction of 4.9 percentage points (95% CI, 7.2 to 2.5; P<0.001) [19]. In addition, there was increase in left ventricular mass by 28.3 g (95% CI, 14.2 to 42.4; P<0.001) that correlated with androgen weekly dose [19]. Fortunately, these changes returned to baseline approximately 8 months after stopping androgen abuse [19]. Mean SBP and DBP increased by 6 mmHg and 5 mmHg and heart rate increased by 10 beats/min [19]. Again, these changes in blood pressure returned to baseline after cessation of androgen intake [19]. Abdullah et al [17] found even greater difference in ejection fraction between AAS abusers and control subjects, 49% and 59%, respectively.

E. Increased cardiovascular events and mortality associated with AAS abuse

In a cohort study from Denmark including 1198 recreational athletes who abused androgens and were followed for a mean of 11 years, mortality was increased compared with control individuals, hazard ratio (HR) 2.81 (95% CI, 1.98 to 3.99; P< 0.001) [24]. Moreover, androgen abusers demonstrated increased risk of acute myocardial infarction (HR 3.00), percutaneous coronary intervention or coronary artery bypass graft (HR 2.95), venous thromboembolism (HR 2.42), arrhythmia (HR 2.26), heart failure (HR 3.63), and cardiomyopathy (HR 8.90) [25]. Post-mortem studies of young men with a history of androgen abuse found significant cardiac abnormalities such as left ventricular hypertrophy, coronary thrombosis, and dilated cardiomyopathy [26].

Approach to subjects suspected of AAS abuse

As mentioned earlier, most AAS abusers will not disclose such information to their medical providers. However, there are several findings in the physical exam, vital signs, and laboratory results that strongly suggest AAS abuse. These findings are summarized in table 1. In terms of laboratory findings, high hematocrit is a common clue to AAS abuse. In addition, very low or undetectable levels of LH and FSH suggest current or past (within 3-12 months) androgen abuse. Shankara et al [27]

have shown that a single suppressed serum LH was the best reliable laboratory parameter for identification of androgen abuse with an overall accuracy of 89% followed by suppressed FSH with 74% accuracy [27]. Finally, if testosterone levels are elevated, current intake of exogenous testosterone is confirmed. Once the diagnosis of AAS abuse is suspected, every attempt should be made to build trust between the provider and abuser. Confrontation of the diagnosis should be gradual and non-judgmental. Then, explanation of the adverse effects of AAS should be done to counsel patients to discontinue AAS. If patient agrees to stop AAS, there are no guidelines to guide physicians about management of AAS withdrawal consequences. In fact, in one survey of 100 endocrinology health providers in the UK (16% consultants, 48% trainees, 23% endocrine nurses) 84% advised men with AAS-induced hypogonadism to wait until symptoms resolved with time without treatment [27]. Moreover, only 20% were confident treating AAS-induced hypogonadism [27]. Studies using CC, hCG, and tamoxifen to treat withdrawal symptoms and accelerate recovery of the reproductive function are of low-quality and conflicting [28,29]. The author prefers gradual tapering of androgen doses over 6-12 months. If depression is a prominent withdrawal symptom, referral to psychiatry is required.

1. Vital signs	2. Physical exam	3. Laboratory findings
Hypertension	Muscular habitus	Elevated hematocrit
Tachycardia	Frontal baldness	Subnormal or undetectable LH and FSH
	Acne in the back and chest areas	High plasma testosterone
	Gynecomastia	High plasma estradiol (from conversion of testosterone by aromatases)
	Small testicles	

Table 1: Clues to androgen abuse

On the other hand, if patient declined cessation of AAS, close supervision, monitoring and treatment of adverse effects of AAS are indicated (harm reduction approach). For instance, monitoring blood pressure, hematocrit and lipid panel, and getting a baseline echocardiogram seem appropriate. Treatment is also provided for any complication of AAS such as anti-hypertensive treatment for high blood pressure and phlebotomy if hematocrit value is close to 54% [20].

Conclusions and current needs

AAS abuse is common among men and is likely underestimated due to reluctance of abusers of disclosing such information and lack of confidence in the physician with respect to his/her knowledge in the field of AAS. Medical providers must be more aware of this problem and approach suspected abusers in a non-judgmental fashion. They should explain to their patients that AAS abuse has serious complications including increased CV events and infertility. Randomized trials are urgently needed to guide providers about optimum treatment strategies for withdrawal symptoms following cessation of androgen abuse. In addition, for those men not willing to stop AAS, close medical supervision should be available for any adverse effects, which again should be evaluated in controlled studies with long-term follow-up. The availability of such studies should enhance the ability of physicians for appropriate management of AAS abuse.

Conflict of interest

The author has no conflict of interest to declare.

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