ISSN PRINT 2319 1775 Online 2320 7876

Research paper UGC CARE Listed (Group -I) Journal Volume 11, S Iss 4, 2022

Effect of Anabolic Steroids Abuse in Gym Visitors

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ABSTRACT:

Background: The term "anabolic steroids" (also known as "anabolic-androgenic steroids" or "AAS") refers to a broad class of both artificial and naturally occurring androgens. In 1935, the endogenous androgen testosterone was isolated, described, and made available for external administration. Since then, extensive research and development have resulted in the production of a large number of testosterone derivatives, each of which has a different structural makeup and distinct features. There is a dearth of reviews that explore the causes, effectiveness, and patterns of AAS usage, despite the fact that there is a wealth of literature on the negative effects of AAS use. The objective of this review is to close this gap while also educating and updating the clinician on the underlying basic science of AAS and the prognostic implications of AAS use. Conclusion: The negative effect profile of AAS use has long been a point of debate, with opinions on how serious the problem is varied. Our study demonstrates that the negative effects of AAS are actually distributed throughout several organ systems, although further, ideally prospective-designed trials are still needed to determine their full extent. The clinician may utilize the increased risk of early death, the emergence and potential hemorrhagic rupture of hepatic adenomas, and the potential for continuing infertility for up to two years after AAS discontinuation as arguments to discourage users from abusing AAS.

Keywords: Anabolic steroids, gym visitors, steroid abuse.

INTRODUCTION:

The term "anabolic steroids" (also known as "anabolic-androgenic steroids" or "AAS") refers to a broad class of both artificial and naturally occurring androgens [1]. In 1935, the endogenous androgen testosterone was isolated, characterized, and made available for exogenous administration [2]. Since then, extensive research and development have resulted in the production of a large number of testosterone derivatives, each of which has a different structural makeup and distinct features. The Russian weightlifting team at the 1954 Olympics was one of the first teams to use AAS in the context of competitive sport [3]. A 2014 meta-analysis of 187 research estimated the lifetime prevalence of AAS usage in men to be around 6%, and a 2005 paper suggested its prevalence among male gym goers to be over 25%. These

ISSN PRINT 2319 1775 Online 2320 7876

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findings indicate that the use of AAS has become increasingly widespread among non-elite users [4, 5]. But for a variety of reasons, the typical doctor might not see a lot of clinical cases involving the use of AAS. First, the most serious side effects of AAS such as a twice as high cardiovascular mortality rate or an elevated risk of early death from all causes manifest abruptly and permanently [6]. Additionally, a study found that 56% of AAS users are not proactive in sharing their use status with their doctors. This is concerning because only 10% of people report using unsafe injection methods and almost 100% report experiencing subjective side effects [5]. Some have proposed that the main cause of this underreporting is a widespread mistrust among AAS users of the level of expertise the typical physician possesses regarding AAS [8]. Additionally, when taking a patient's history, doctors hardly ever ask specifically about AAS use, missing an opportunity to build rapport on the subject [7].

Indeed, anecdotal reports of advice given by clinicians inaccurately purporting that AAS are ineffective for muscle growth have surfaced, with some commentators arguing that such blanket statements can prematurely sever any chance of developing rapport and generating critical discussion with AAS users [8]. There is a dearth of reviews that discuss the causes, effectiveness, and patterns of AAS misuse, despite the fact that there is a wealth of literature on the negative effects of AAS use. This review aims to close this gap while also updating and reintroducing the physician to the basic science of AAS and its prognostic implications.

A review of testosterone:The production of testosterone and upkeep of a spermatogenic environment are the key responsibilities of the testes [9]. The main hypothalamus pituitary gonadal (HPG) axis orchestrates these two interrelated activities. Gonadotropin releasing hormone (GnRH), which is released pulsatilely from the hypothalamus and travels through the hypophyseal-portal system to the anterior pituitary, is the initial action of the axis [10]. GnRH stimulates the anterior pituitary, which then secretes follicle-stimulating hormone (FSH) and luteinizing hormone (LH) into the bloodstream. FSH increases spermatogenesis by acting on Sertoli cells in the seminiferous tubules. LH stimulates the production of testosterone by targeting Leydig cells near the seminiferous tubules [11].

The interaction of serum testosterone with the androgen receptor is what causes its well-known effects. When testosterone binds to the intracytosolic receptor, it is transported to the nucleus where it directly controls protein synthesis and gene transcription [13]. Any steroid, whether organic or synthetic, that binds to the androgen receptor and stimulates postreceptor functions is considered an androgen [14]. The androgen receptor modification is not the main driver of testosterone activity. Prereceptor, receptor, and postreceptor processes are constantly being produced under physiologic conditions [13]. A component of the serum testosterone is changed by aromatase into 17 -estradiol, while a different portion is changed by 5 -reductase into dihydrotestosterone (DHT) [15].

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Since aromatase is abundant in adipose tissue, obesity may lead to higher levels of 17 - estradiol as a result of enhanced testosterone conversion by aromatase [16]. An endogenously generated steroid called nandrolone (19-nortestosterone) functions as an intermediary in the aromatase reaction that turns testosterone into estradiol. Derivatives of 19-nortestosterone are one of the primary categories of AAS. The bloodstream does not contain significant amounts of 19-nortestosterone under physiological conditions [17]. In contrast to other AAS, nandrolone is also a powerful progestogen with relatively less androgenic effects in tissues like the scalp and prostate [18]. Both the androgen receptor and the progesterone receptor are agonists by it. Testosterone has got barely any anabolic.

Even while endogenous testosterone levels are in the lower range of what is considered normal for males, they are still sufficient to saturate androgen receptors under normal physiologic conditions. The question of whether supraphysiologic levels of testosterone generate further anabolic effects on muscle development and strength has been debated for decades [19]. At this time, most experts agree that androgenic anabolic steroids do fact benefit both. In one study, 61 eugonadal males were randomly divided into five groups and given varying amounts of exogenous testosterone for 20 weeks in each group. The 600 mg injection group gained an average of 7.9 kg of lean muscle mass and shed around 1 kg of body fat by the trial's end.

Surprisingly, participants were explicitly told not to perform any strength training or vigorous aerobic exercise during the research period, and this instruction was repeated every 4 weeks [20]. The anabolic effects of exogenous testosterone may be explained by processes other than androgen receptor modulation since androgen receptors are often saturated by physiologic levels of testosterone, yet supraphysiologic dosages of testosterone can improve muscle growth and body strength [21]. One method includes regulation of the glucocorticoid receptor. Animal studies have demonstrated that exogenous testosterone is beneficial at preventing atrophy secondary to glucocorticoid usage [22].

TYPES OF AAS USERS:

On the basis of an analysis of international literature and each author's personal research experiences with AAS abusers, Christiansen and colleagues proposed a classification method in 2016 . They suggested four categories: expert type, yolo type, athlete type, and wellbeing type. Expert type is the first category. They concentrated on a two-dimensional view of AAS usage, which is effectiveness and perceived danger, in their synthesis of these typologies. Christiansen and colleagues characterised "the Expert type" as adopting a scholarly approach to AAS, having their use patterns studied, and generally taking measured risks. The "YOLO type" (You Only Live Once) is in some ways the archetypal opposite because of their propensity for spontaneous behaviour and their drive for quick advancement.

The "Athlete type" is generally involved in competitive sports and is driven by success in their field. The "Wellbeing type" is focused on physical attractiveness and demonstrates

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reduced risk use behaviours [23]. In 2018, Zahnow and colleagues used cluster analysis and multinomial logistic regression on survey data from a sample of 611 AAS users throughout Wales, England, and Scotland to advance these postulated typologies [24]. They found four separate categories in their data, each of which appeared to match to one of the four typologies put forward by Christiansen. The "YOLO type," the "Wellbeing type," the "Athlete type," and the "Expert type" were specifically identified by 11%, 38%, 25%, and 25% of respondents, respectively.

PERFORMANCE-ENHANCING MEDICINES (PEDS) AND AAS USE:

In the bodybuilding world, using PEDs to reduce the adverse effects of AAS is a common use. In a 2018 study of 231 AAS users, 56% of respondents stated that they have used postcycle therapy in some capacity [25]. In order to explore the numerous websites and forum groups where users might ask for and/or give advice on minimising adverse effects caused by AAS, Karavolos et al. employed a methodical online search procedure. Up to one-third of the websites they investigated made the claim to sell AAS and/or treatments said to counteract its negative effects without a prescription, they discovered.

NEGATIVE EFFECTS OF USING AAS:

There is debate over the unfavourable effect profile of exogenous AAS delivery, with some suggesting that data interpretations may have been overdone [26]. According to a 2010 review, the frequency of somatic and psychiatric side effects was low, and the typical user received AAS with the goal of enhancing their athletic performance [27]. According to some research, cardiovascular side effects may account for up to 33-66% of premature deaths in AAS users, with the remainder primarily coming from liver failure, cancer, and suicide [28].

Cardiovascular side effects have been hypothesised to have the greatest mortality altering influence. According to a 2019 retrospective research of 545 AAS users, mortality rates were around three times higher than those of male controls who were matched (95% CI 1.3-7.0) [29]. Additionally, compared to the control group, the AAS cohort was shown to have a statistically significant higher likelihood of presenting for hospital admission. The most frequent finding in postmortem reviews of 19 instances in which autopsy had ruled out extra cardiac causes of death was left ventricular hypertrophy with concurrent fibrosis [30]. Four theories have been put up as potential explanations for cardiac death in AAS users, namely the atherosclerosis potentiation model, the thrombosis model, the coronary vasospasm model, and the direct cardiac injury model [31]. Four theories have been put up as potential explanations for cardiac death in AAS users, namely the atherosclerosis potentiation model, the thrombosis model, the coronary vasospasm model, and the direct cardiac injury model [31]. Theoretically, the exogenous AAS administration's stimulation of haematopoiesis predisposes people to an increased risk of thrombosis, although there is now only minimal case research and epidemiological evidence to support this.

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INFERTILITY:

Secondary to the lowering of intratesticular testosterone levels, which may result in azoospermia or oligozoospermia, is infertility after AAS use [32]. The 12- to 24-month mark has been given as the period for the majority of AAS users, and it is generally believed that restoration of sperm count and quality following AAS cessation would occur with the passage of time [33,34]. One 2020 cross-sectional study of 72 AAS misusers and 31 healthy controls noted no significant difference in sperm output, sperm concentration, and sperm motility between former AAS users and nonusers, with the mean recovery time for these parameters post-AAS cessation being 14 months, 10 months, and 37 months, respectively [35]. A 2021 Danish study of 545 males who tested positive for AAS found that the AAS group had a significantly decreased fertility rate compared to the healthy control group in the decade before the positive result, with a rate ratio of 0.74. Although there was no significant group difference for rates of assisted reproduction, the AAS group continued to have a 7% lower total fertility rate than the control group in the years following AAS cessation [36]. As a result, current research hints that most former AAS users may be able to resume their reproductive lives. To give more conclusive findings, however, additional study examining real-world outcomes, such as the time to become pregnant, is necessary.

HEPATIC SIDE EFFECTS:

Hepatotoxicity is a side effect of AAS usage that is frequently reported. According to one study, 8% of all cases of drug-induced liver injury were connected to AAS use [37]. It's noteworthy that only 17-alkylated AAS seems to be hepatotoxic. Increased oxidative stress brought on by higher levels of mitochondrial -oxidation has been proposed to be a contributing factor, albeit the exact mechanism underpinning this has not yet been fully understood [38]. The range of AAS-induced liver damage has a wide range of potential pathologies. In their study of 182 asymptomatic Brazilian AAS users, Schwingel and colleagues found that 38% of them had elevated liver indicators, 12% had hepatic steatosis, and there was one incidence of each of hepatocellular adenoma, focal nodular hyperplasia, hepatitis B, and hepatitis C. Additionally, there are case reports of hepatocellular carcinoma (HCC) developing as a result of the use of AAS, with the observation that 4% of hepatic adenomas develop into HCC.

PSYCHIATRIC SIDE EFFECTS:

AAS use has been linked to psychiatric measures such as the Hostility and Direction of Hostility Questionnaire (HDHQ) and the Symptoms Check List-90 (SCL-90), with the severity of misuse being inversely correlated with symptom intensity . AAS use-related aggression has traditionally been referred to as irritable "roid fury" in lay language. Hypomanic or manic disorders have also been characterised as two possible manifestations. Pope et al. compared psychiatric outcome measurements between a group that received

ISSN PRINT 2319 1775 Online 2320 7876

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exogenous testosterone and a placebo group. Young Mania Rating Scale (YMRS) scores for the AAS group were much higher, according to the study's findings, with 16% of the group being labelled as slightly or significantly hypomanic . A study that used structural MRI brain imaging on AAS users and nonusers found a negative correlation between cortical thickness and brain volume and AAS use, suggesting that AAS may predispose users to decreased cognitive skills or structural brain abnormalities. Notably, this is in light of research linking AAS usage to worse results on cognitive tests. The idea of a connection between supraphysiological testosterone levels and dementia through androgenic development of extra oxidative stress has also been proposed by certain authors [39].

Although some potential causes include direct toxicity or bile acid nephropathy secondary to AAS associated liver disease, AAS related renal damage is less researched than other side effects [40]. There is a wide range of potential kidney damage caused by AAS, from momentary changes in creatinine levels to advanced chronic kidney disease or secondary focal-segmental glomerulosclerosis.

CONCLUSION:

An extremely diverse range of manufactured and endogenous androgens, each with a distinct chemical structure and side effect profile, make up anabolic-androgenic steroids. Typological representations of the anabolic-androgenic steroid base have been made, and cross-sectionally obtained data support the concept that different user types exist within the community. However, more research is required to conceive more potential typologies, particularly from datasets beyond the United Kingdom, and this field is still in its infancy.

Exogenous testosterone is typically administered for specific time periods, and then the substance is gradually tapered off over a predetermined period of time, according to general consumption patterns found in the AAS community. Others mix various AAS or augmentative medications as part of the same regimen.

AAS is linked to a wide range of negative consequences that affect many different organ systems. Some AAS users employ self-initiated therapies like tamoxifen, anastrozole, or hCG injections in an effort to lessen adverse effects like gynecomastia, androgenic steroid-induced hypogonadism, or sexual dysfunction. The negative effect profile of AAS use has long been a point of debate, with opinions on how serious the problem is varied. Our study demonstrates that the negative effects of AAS are actually distributed throughout several organ systems, although further, ideally prospective-designed trials are still needed to determine their full extent. The clinician may utilise the increased risk of early death, the emergence and potential hemorrhagic rupture of hepatic adenomas, and the potential for continuing infertility for up to two years after AAS discontinuation as arguments to discourage users from abusing AAS.

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REFERENCES:

1. M. Roman, D. L. Roman, and V. Ostafe, "Computational assessment of pharmacokinetics and biological effects of some anabolic and androgen steroids," Pharmaceutical Research, vol. 35, no. 2, p. 41, 2018.
2. J. L. Dotson and R. T. Brown, "*e history of the development of anabolic-androgenic steroids," Pediatric Clinics of North America, vol. 54, no. 4, pp. 761–769, 2007.
3. C. Wu and J. R. Kovac, "Novel uses for the anabolic androgenic steroids nandrolone and oxandrolone in the management of male health," Current Urology Reports, vol. 17, no. 10, p. 72, 2016.
4. D. Sagoe, H. Molde, and C. S. Andreassen, "*e global epidemiology of anabolic-androgenic steroid use: a meta-analysis and meta-regression analysis," Annals of Epidemiology, vol. 24, no. 5, pp. 383–398, 2014.
5. A. B. Parkinson and N. A. Evans, "Anabolic androgenic steroids: a survey of 500 users," Medicine & Science in Sports & Exercise, vol. 38, no. 4, pp. 644–651, 2006.
6. I. *iblin, H. Garmo, and M. Garle, "Anabolic steroids and cardiovascular risk: a national population-based cohort study," Drug and Alcohol Dependence, vol. 152, pp. 87–92, 2015.
7. H. G. Pope, G. Kanayama, and M. Ionescu-Pioggia, "Anabolic steroid users' attitudes towards physicians," Addiction, vol. 99, no. 9, pp. 1189–1194, 2004.
8. S. Griffiths, S. B. Murray, and D. Mitchison, "Anabolic steroids: lots of muscle in the short-term, potentially devastating health consequences in the long-term," Drug and Alcohol Review, vol. 35, no. 4, pp. 375-376, 2016.
9. S. Basaria, "Male hypogonadism," Lancet, vol. 383, no. 9924, pp. 1250–1263, 2014.
10. K. Skorupskaite, J. T. George, and R. A. Anderson, "*e kisspeptin-GnRH pathway in human reproductive health and disease," Human Reproduction Update, vol. 20, no. 4, pp. 485–500, 2014.
11. D. Santi, P. Cr´ epieux, and E. Reiter, "Follicle-stimulating hormone (FSH) action on spermatogenesis: a focus on physiological and therapeutic roles," Journal of Clinical Medicine, vol. 9, no. 4, p. 1014, 2020.
12. M. Y. Roth, K. Lin, and J. K. Amory, "Serum LH correlates highly with intratesticular steroid levels in normal men," Journal of Andrology, vol. 31, no. 2, pp. 138–145, 2010.
13. D. J. Handelsman, "Androgen physiology, pharmacology, use and misuse," Edited by K. R. Feingold, B. Anawalt, A. Boyce, G. Chrousos, W. W. de Herder, and K. Dhatariya, Eds., MDText.com, Inc, South Dartmouth, MA,USA, 2000.

ISSN PRINT 2319 1775 Online 2320 7876

Research paper **© 2012 IJFANS. All Rights Reserved,** UGC CARE Listed (Group -I) Journal Volume 11, S Iss 4, 2022

14. C. A. Quigley, A. De Bellis, and K. B. Marschke, "Androgen receptor defects: historical, clinical, and molecular perspectives," Endocrine Reviews, vol. 16, no. 3, pp. 271–321, 1995.

15. R. S. Swerdloff, R. E. Dudley, and S. T. Page, "Dihydrotestosterone: biochemistry, physiology, and clinical implications of elevated blood levels," Endocrine Reviews, vol. 38, no. 3, pp. 220–254, 2017.

16. M. Grossmann, "Hypogonadism and male obesity: focus on unresolved questions," Clinical Endocrinology, vol. 89, no. 1, pp. 11–21, 2018.

17. D. J. Handelsman, "Chapter 138 - androgen physiology, pharmacology, and abuse," in Endocrinology: Adult and Pediatric, J. L. Jameson, L. J. De Groot, D. M. de Kretser, L. C. Giudice, A. B. Grossman, and S. Melmed, Eds., pp. 2368–2393, W.B. Saunders, Philadelphia, PA, USA, Seventh edition, 2016.

18. E. W. Bergink, P. S. Janssen, and E. W. Turpijn, "Comparison of the receptor binding properties of nandrolone and testosterone under in vitro and in vivo conditions," Journal of Steroid Biochemistry, vol. 22, no. 6, pp. 831–836, 1985.

19. S. Bhasin, W. E. Taylor, and R. Singh, "*e mechanisms of androgen effects on body composition: mesenchymal pluripotent cell as the target of androgen action," Journal of Gerontology: Series A, vol. 58, no. 12, pp. M1103–M10, 2003.

20. S. Bhasin, L. Woodhouse, and R. Casaburi, "Testosterone dose-response relationships in healthy young men," American Journal of Physiology. Endocrinology and Metabolism, vol. 281, no. 6, pp. E1172–E1181, 2001.

21. G. D. Albano, F. Amico, and G. Cocimano, "Adverse effects of anabolic-androgenic steroids: a literature review," Health Care, vol. 9, no. 1, p. 97, 2021.

22. J. M. Eason, S. L. Dodd, and S. K. Powers, "Use of anabolic steroids to attenuate the effects glucocorticoids on the rat diaphragm," Physical 6erapy, vol. 83, no. 1, pp. 29–36, 2003.

23. A. V. Christiansen, A. S. Vinther, and D. Liokaftos, "Outline of a typology of men's use of anabolic androgenic steroids in fitness and strength training environments," Drugs: Education, Prevention & Policy, vol. 24, no. 3, pp. 295–305, 2017.

24. R. Zahnow, J. McVeigh, and G. Bates, "Identifying a typology of men who use anabolic androgenic steroids (AAS)," International Journal of Drug Policy, vol. 55, pp. 105–112, 2018.

25. J. M. Armstrong, R. A. Avant, and C. M. Charchenko, "Impact of anabolic androgenic steroids on sexual function," Translational Andrology and Urology, vol. 7, no. 3, pp. 483–489, 2018

ISSN PRINT 2319 1775 Online 2320 7876

Research paper UGC CARE Listed (Group -I) Journal Volume 11, S Iss 4, 2022

26. J. R. Hoffman and N. A. Ratamess, "Medical issues associated with anabolic steroid use: are they exaggerated?" Journal of Sports Science and Medicine, vol. 5, no. 2, pp. 182–193, 2006.

27. J. van Amsterdam, A. Opperhuizen, and F. Hartgens, "Adverse health effects of anabolic-androgenic steroids," Regulatory Toxicology and Pharmacology, vol. 57, no. 1, pp. 117–123, 2010.

28. S. Achar, A. Rostamian, and S. M. Narayan, "Cardiac and metabolic effects of anabolic-androgenic steroid abuse on lipids, blood pressure, left ventricular dimensions, and rhythm," 6e American Journal of Cardiology, vol. 106, no. 6, pp. 893–901, 2010.

29. H. Horwitz, J. T. Andersen, and K. P. Dalhoff, "Health consequences of androgenic anabolic steroid use," Journal of Internal Medicine, vol. 285, no. 3, pp. 333–340, 2019.

30. P. Frati, F. P. Busard` o, and L. Cipolloni, "Anabolic Androgenic Steroid (AAS) related deaths: autoptic, histopathological and toxicological findings," Current Neuropharmacology, vol. 13, no. 1, pp. 146–159, 2015.

31. M. Torrisi, G. Pennisi, and I. Russo, "Sudden cardiac death in anabolic-androgenic steroid users: a literature review," Medicina, vol. 56, no. 11, 2020.

32. R. El Osta, T. Almont, and C. Diligent, "Anabolic steroids abuse and male infertility," Basic and Clinical Andrology, vol. 26, p. 2, 2016.

33. L. E. Crosnoe, E. Grober, and D. Ohl, "Exogenous testosterone: a preventable cause of male infertility," Translational Andrology and Urology, vol. 2, no. 2, pp. 106–113, 2013.

34. P. Y. Liu, R. S. Swerdloff, and P. D. Christenson, "Rate, extent, and modifiers of spermatogenic recovery after hormonal male contraception: an integrated analysis," Lancet, vol. 367, no. 9520, pp. 1412–1420, 2006.

35. N. Shankara-Narayana, C. Yu, and S. Savkovic, "Rate and extent of recovery from reproductive and cardiac dysfunction due to androgen abuse in men," Journal of Clinical Endocrinology & Metabolism, vol. 105, no. 6, 2020.

36. D. L. Smit, M. M. Buijs, and O. de Hon, "Disruption and recovery of testicular function during and after androgen abuse: the HAARLEM study," Human Reproduction, vol. 36, no. 4, pp. 880–890, 2021.

37. J. Windfeld-Mathiasen, K. P. Dalhoff, and J. T. Andersen, "Male fertility before and after androgen abuse," Journal of Clinical Endocrinology & Metabolism, vol. 106, no. 2, pp. 442–449, 2021.

ISSN PRINT 2319 1775 Online 2320 7876

Research paper UGC CARE Listed (Group -I) Journal Volume 11, S Iss 4, 2022

38. M. Robles-Diaz, A. Gonzalez-Jimenez, and I. Medina-Caliz, "Distinct phenotype of hepatotoxicity associated with illicit use of anabolic androgenic steroids," Alimentary Pharmacology & 6erapeutics, vol. 41, no. 1, pp. 116–125, 2015.

39. P. Bond, W. Llewellyn, and P. Van Mol, "Anabolic androgenic steroid-induced hepatotoxicity," Medical Hypotheses, vol. 93, pp. 150–153, 2016.

40. P. A. Schwingel, H. P. Cotrim, and C. R. Santos, "Recreational anabolic-androgenic steroid use associated with liver injuries among Brazilian young men," Substance Use & Misuse, vol. 50, no. 11, pp. 1490–1498, 2015.

41. L. Wang, C. Wang, and W. Li, "Multiple hepatocellular adenomas associated with long-term administration of androgenic steroids for aplastic anemia: a case report and literature review," Medicine, vol. 99, no. 28, p. e20829-e, 2020.