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Anabolic androgen steroids cardiovascular impact. Literature review

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Abstract

Introduction and Purpose: The use of anabolic steroids is widespread. They can be administered either therapeutically or by individuals looking to enhance their athletic performance. This review aims to summarize the scientific understanding regarding the link between the use of AASs and the incidence of cardiovascular complications.

Materials and methods

The literature available in PubMed and Google Scholar databases was reviewed using the keywords.

Description of the state of knowledge: A lot of studies suggest that AASs can be harmful to the cardiovascular system. It has a wide range of long-term and often irreversible consequences. AASs cause myocardial hypertrophy which results in systolic and diastolic disfunction. They disrupt lipid metabolism, act as vasoconstrictors, and promote the calcification of blood vessel walls, thereby increasing the risk of heart attack and stroke. By promoting blood clotting, they increase the incidence of pulmonary embolism, deep vein thrombosis, and cerebral venous sinus thrombosis. The effect on the occurrence of hypertension is not definitively proven. However, a very dangerous consequence of using anabolic steroids is the increased risk of life-threatening arrhythmias -especially ventricular arrhythmias- which can lead to sudden cardiac death.

Conclusion: The use of anabolic steroids increases the risk of arrhythmias, heart attacks, and thromboembolic complications, ultimately leading to a higher risk of sudden cardiac death. However, the cause-and-effect relationship is not always clearly captured, and further research is necessary to draw appropriate conclusions.

Key words: anabolic androgen steroids; cardiovascular risk; heart attack; arrhythmia; sudden cardiac death; athletes.

Materials and methods: We reviewed studies from the PubMed and Google Scholar using phrases: anabolic androgen steroids mechanism, anabolic androgen steroids cardiovascular effects, anabolic androgen steroids heart infarct, anabolic androgen steroids sudden cardiac death, anabolic androgen steroids sudden cardiac arrest, anabolic androgen steroids pulmonary embolism, anabolic androgen steroids deep venous thrombosis, anabolic androgen steroids stroke, anabolic androgen steroids arrhythmias, anabolic androgen steroids vascular calcification, anabolic androgen steroids in athletes.

Introduction:

Anabolic Androgenic Steroids (AASs) are testosterone derivatives most commonly administered by intramuscular injection or oral ingestion, with an estimated prevalence rate of 3.3% to 6.4% in males and 1.3% in females [1,2]. Many designer steroids that are neither declared on the labels nor included in the WADA (World Anti-Doping Agency) list can be found in nutritional supplement samples [3]. The pursuit of beauty standards has contributed to the rise of a body image disorder: muscle dysmorphia, where people perceive themselves as not muscular enough [4]. AASs are used to promote muscle deposition after burns, surgery, radiation therapy, and aging-related sarcopenia [18]. Furthermore they are approved for treating wasting syndrome in human immunodeficiency virus infection (HIV), hypogonadism, anemia accompanying renal and bone marrow failure, endometriosis, and cancer [5]. However, recreational sportspeople represent the largest share of AAS users [4]. The dose of AASs used by athletes depends on individual needs and the athletic requirements of the particular sport: endurance athletes (doses near or slightly below physiological replacement concentrations), sprinters (doses 1.5–2 times replacement concentrations), and traditional strength athletes (doses 10–100 times replacement concentrations) [6]. Anabolic steroids are often taken simultaneously with other drugs, such as diuretics to evade identification during anti doping controls of urine; tamoxifen to reduce the gynaecomastia effect; chorionic gonadotropin, which promotes endogenous testosterone synthesis; and growth hormone (GH), which also has an anabolic effect [7]. AASs have either anabolic or androgenic impacts-none of them has a purely anabolic function [8]. AASs in very high doses are administered in cycles called ‘stacking,’ which can last up to 48 weeks and are associated with serious risks of

cardiovascular adverse effects [9]. The consequences of AASs abuse are long-term, and even several years after discontinuation, strength athletes still have slight concentric left ventricular hypertrophy compared to AASs- free strength athletes [10]. However, Giovani Corona et al have shown that the use of anabolic steroids may benefit patients with hypogonadism by improving their metabolic profile thereby reducing cardiovascular risk [11].

State of knowledge of cardiovascular effects

Cardiac hypertrophy and myocardial function

AASs users display higher left ventricle (LV) mass index, thicker LV walls, and more concentric LV geometry than non-users [12,13,14]. Right ventricular systolic function was also impaired among AASs users compared with non-users [13]. Myocardial hypertrophy results in impaired ventricular inflow [15]. AASs abuse causes systolic and diastolic disfunction [16,17].

Acute Myocardial infarction (AMI) and coronary artery atherosclerosis

AASs abuse causes a decrease in high-density lipoprotein (HDL-C) and apolipoprotein A1 and an increase in low-density lipoprotein (LDL-C) and apolipoprotein B, which promote atherosclerosis [19]. The main pathway of thrombosis is due to erythrocytosis, thrombocytosis, and platelet hyperactivity [20]. Furthermore, AASs increase levels of thromboxane A2 and other procoagulant factors (especially fibrinogen, factors VIII and X), homocysteine, as well as the endothelial release of proteins C and S, with decreased fibrinolytic activity and prostacyclin synthesis, which induces prothrombotic action [5,9,12]. Physiological doses of androgens cause arterial vasodilation by inducing relaxation of vascular smooth muscle cells (VSMCs) or by inducing nitrogen oxide (NO) synthesis in endothelial cells (ECs) [21]. However, applying supraphysiological doses results in coronary vasoconstriction [22]. AASs can disrupt the absorption of vitamins B6 and B12, causing hyperhomocysteinemia, which increases cardiovascular risk [23,24]. Due to decreased myocardial perfusion, AASs can increase AMI risk [20,25]. In another study, G. Caleb et al. did not demonstrate that anabolic steroids increase the risk of a heart attack. However, these data should be approached with caution, as the authors themselves note that definitive conclusions cannot be drawn from their study due to the very low quality of the data [26].

Stroke, Cerebral venous sinus thrombosis (CVST), deep venous thrombosis (DVT), pulmonary embolism (PE)

In the cerebral arteries, epithelial cells produce receptors that bind steroid hormones and steroid-metabolizing enzymes (e.g., aromatase) so that brain vessels can be influenced by steroids [21]. Cerebral venous sinus thrombosis (CVST) is a very rare condition with an annual incidence of 0.22 to 1.57 per 100,000 [27]. CVST can occur in people who use AASs, but further research is needed to determine whether it is incidental or not [28,29,30]. H.S. Melsom et al. concluded that AASs users display a higher thickness of the intima media of the common carotid artery (cIMT), which is an early sign of arteriosclerosis, and also higher carotid-femoral pulse wave velocity (PWV) in comparison to non-users [31]. PWV increase is linked with arterial stiffness and atherosclerosis [32]. Cerebral artery occlusion can be correlated with intraventricular thrombi that were transported through the bloodstream [7]. Consequently, AASs abuse can cause ischemic stroke events, especially when other risk factors, such as hyperhomocysteinemia, are present [34,35,36,38]. Other adverse effects include deep venous thrombosis (DVT) and pulmonary embolism (PE), cases of which have been reported in individuals using anabolic steroids [33,28,37]. However, at therapeutic doses, AASs (such as danazol) have been used in patients with protein S deficiency to reduce the incidence of DVT [39].

Hypertension

The potential mechanism may be related to AASs' ability to increase the activity of the sympathetic nervous system, baroreflex control, and endothelial dysfunction [40]. The relationship between AASs abuse and blood pressure (BP) is controversial and requires further research. Some studies have shown a correlation between AAS use and hypertension [10,13,41,42,43], with an increase in systolic blood pressure (SBP) of 10-12 mmHg [41], but others did not obtain similar results [44,45].

Vascular calcification

Vascular calcification increases morbidity and mortality in cardiovascular diseases [46]. In vitro studies revealed that treatment with testosterone or dihydrotestosterone resulted in increased calcification of mouse VSMCs. In the same study immunohistochemical analysis showed the expression of the androgen receptor (AR) in the calcified tissue of the human

femoral artery and in calcified human valves [47]. As a result, we can conclude that long-term steroid abuse is associated with an increased risk of coronary arteriosclerosis, as measured by increased coronary calcium seen with electron beam computed tomographic imaging [48].

Arrhythmias

Testosterone shortens QT interval on ECG in both genders and may cause lower incidence of torsades de pointes in males [49]. AASs users display a higher incidence of abnormal SAECG (Signal-averaged ECG) measurements, which is a risk factor for cardiac arrhythmias [50]. Chronic AASs use has been shown to cause sympathetic dominance, which can be proarrhythmic. Arrhythmias that can occur, especially during physical activity, include atrial fibrillation (AF), supraventricular and ventricular ectopic beats, ventricular tachycardia (VT), and ventricular fibrillation (VF) [7,14,51].

Sudden cardiac death (SCD) and sudden cardiac arrest (SCA)

AASs abuse is correlated with SCD events. Most of these fatal events were reported in powerlifters and bodybuilders [52-56,57]. Athletes initially have a 2-3 times higher risk of sudden cardiac death than the general population, so supplementing with anabolic steroids further increases this risk. The risk of death can be 6 to 20 times higher in athletes who use AAS than in other athletes. Post-mortem autopsy findings can include foci of fibrosis and myocardial necrosis, concentric cardiac hypertrophy and cardiomegaly, dilated cardiomyopathy, atherosclerosis, inflammatory infiltrate, eosinophilic myocarditis, coronary stenosis, and left ventricular apoplexy [2,55,58].

Conclusions:

AASs produce toxic effects on the cardiovascular system, and it is necessary to ensure that more people know this about AAS, including medical personnel. The use of anabolic steroids increases the risk of arrhythmias, heart attacks, and thromboembolic complications, leading to a higher risk of sudden cardiac death. SCD and SCA mechanisms aren't clear. It can be caused by use of some substances that potentiate AASs, such as recombinant human growth hormone, insulin, thyroid hormones (mainly T3), and diuretics [24,50]. Further research is essential to obtain a complete view of the relationship between AASs and cardiovascular complications.

Disclosure:

Author Contributions

Conceptualization, supervision and project administration: Bartosz Balcer, Natalia Dolata
Methodology: Bartosz Balcer, Natalia Dolata
Software, validation, formal analysis, investigation, resources, writing original draft preparation: Bartosz Balcer, Natalia Dolata
Writing review editing and visualization: Bartosz Balcer, Natalia Dolata
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All authors have read and agreed with the published version of the manuscript.

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The data presented in this study is available upon request from the corresponding author.

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All authors declare that they have no conflicts of interest.

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