

The Indiscriminate Use Of Androgenic Anabolic Steroids: The Contribution of Cardiovascular Imaging

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Abstract

Anabolic androgenic steroids (AAS) are natural or synthetic compounds similar to the male hormone testosterone. The literature also describes cases of AAS abuse, such as in bodybuilding, cosmetics, recreational use, and aesthetic enhancement. It was discovered that AAS alone directly induce myocardial injury, with the main pathological finding in autopsied hearts being left ventricle (LV) hypertrophy in frequent association with myocyte hypertrophy, increased collagen deposition in the matrix, increased activity of the cardiac angiotensin, and myocardial fibrosis.

Patients who used illicit AAS present decreased LV systolic function. In athletes who self-administer AAS, LV hypertrophy and elevated sympathetic modulation were observed, as well as elevated blood pressure (BP). High doses of AAS can cause the user to have ventricular arrhythmias and sudden death. Furthermore, AAS users demonstrated greater coronary artery plaque volume than non-users. The objective of this review is to revisit the main effects of the use of AAS on cardiac changes through cardiovascular imaging as well as to establish a difference in relation to athletic heart syndrome.

Introduction

Anabolic androgenic steroids (AAS) are natural or synthetic compounds that mimic the effects of the male hormone testosterone. Alterations to their molecular structure are made to modify their bioactivity, delay absorption into the bloodstream, minimize androgenic effects, and maximize anabolic effects. While testosterone replacement therapy is the current standard treatment for pathological hypogonadism in men, the increasing and indiscriminate use of AAS for aesthetic and competitive purposes lacks support in the literature.

The misuse of anabolic steroids is also reported in bodybuilding, cosmetics, recreational use, and aesthetic enhancement.¹

Keywords

Steroids; Global Longitudinal Strain; Doppler Echocardiography; Tomography.

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Illicit AAS use has been linked to decreased left ventricular (LV) systolic function. Long-term AAS use has been associated with LV impairment, while recent use has been linked to decreased LV ejection fraction, myocardial hypertrophy, and diastolic dysfunction.^{1,2}

Independent studies have found associations between increased plasma total testosterone levels and decreased LV ejection fraction and LV myocardial hypertrophy. Cardiac hypertrophy resulting from AAS abuse is frequently associated with sudden death and arrhythmias in athletes.^{1,3}

Bowman et al. studied autopsies of athletes who used AAS. Their main findings were cardiomegaly (33%) and hypertrophy (30%). The most frequently reported histological changes were foci of fibrosis (79%) and necrosis (52%) of myocardial tissue.

The increased dimensions of the cavities, wall thickness, and LV mass are typical consequences of high-intensity physical training and are included in the physiological cardiac remodeling of athletic heart syndrome, which can be a confusing factor regarding the use of AAS.^{4,5}

The objective of this review is to assess the primary effects of AAS use on cardiac changes using cardiovascular imaging and to distinguish these effects from those of athletic heart syndrome.

Main changes of anabolic steroids in the cardiovascular system

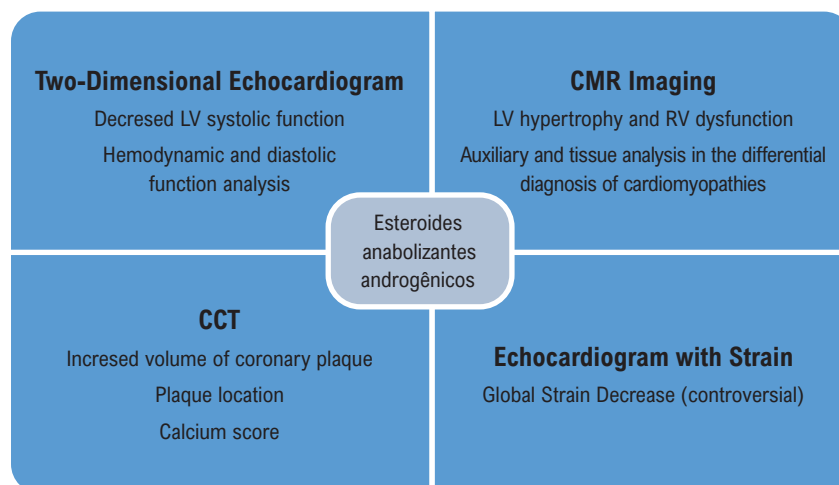
Metabolic changes

One well-documented factor related to the effects of AAS on the cardiovascular system is its action on plasma lipids. Studies show that users of these drugs had increased low density lipoprotein (LDL) and decreased high density lipoprotein (HDL).^{6,7} Their actions may be related to the increased activity of the enzyme hepatic triglyceride lipase (HTGL), responsible for regulating lipids and lipoprotein levels, stimulating the formation of atherosclerotic plaques with consequent plaque increase.^{8,9} Such changes increase the risk of coronary disease three to six times.¹⁰

Coronary artery disease (CAD)

AAS use may also be associated with acute myocardial infarction and sudden death in younger people. In a case study, a 20-year-old man using AAS experienced sudden cardiac death (SCD) accompanied by pulmonary hemorrhage.⁶ Similar findings were reported in another case involving a 31-year-old bodybuilder who had been using AAS for ten years and presented with chest pain due to an

Central Illustration: The Indiscriminate Use Of Androgenic Anabolic Steroids: The Contribution of Cardiovascular Imaging



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CCT: Coronary Tomography; CMR: Cardiac Magnetic Resonance

acute myocardial infarction, which resulted from occlusion of the right coronary artery.¹¹

Some studies have shown beneficial effects on the coronary arteries of patients using AAS, through the release of nitric oxide and the inhibition of smooth muscle contraction. However, animal studies have shown that the misuse of AAS, such as nandrolone in high doses, can cause a vasoconstriction response and lead to side effects.¹²⁻¹⁴

Arterial hypertension

Another widely discussed topic in the literature is the impact of AAS on blood pressure (BP). Studies indicate that AAS use among athletes leads to an increase in BP, which may persist even after discontinuation of the drug. For instance, one study showed that systolic BP remained approximately 6 mmHg higher at rest in AAS users compared to non-users, even after five months of cessation.¹⁵

This increased BP could be attributed to greater sodium and water retention, as the structure of AAS is similar to aldosterone, potentially leading to increased blood volume and BP.¹² Another hypothesis suggests that AAS may affect the sympathetic nervous system. In spontaneously hypertensive rats, blocking the androgen receptor was effective in reducing BP in the early stages, indicating the significant role of testosterone in the initial phase of hypertension. Additionally, changes in endothelium-dependent vasodilatory responses or alterations in baroreflex control could also contribute to increased BP.¹⁵

On the other hand, some authors have not observed an increase in BP associated with AAS use. Nottin *et al.* found no

differences in BP between bodybuilders using AAS and non-users. Similarly, a study with weight-lifters did not observe increased BP at rest or during exercise in AAS users.^{7,16} Similar findings were reported in previous studies, where rats treated with AAS and trained by swimming did not exhibit significant changes in BP. The discrepancies in the literature regarding the effects of AAS on BP may be attributed to variations in study methodologies.^{11,17}

Ventricular remodeling

Hypertrophy may be related to the increased afterload of isometric exercise.¹⁸ Possible associations between AAS and LV hypertrophy can be explained as secondary to hypertension or as a direct effect on the myocardium. Notably, studies on isolated myocytes have shown that AAS bind to androgen receptors and can directly cause hypertrophy, potentially due to tissue regulation of the renin-angiotensin system.¹⁹⁻²¹

In fact, clinical studies suggest a distinct form of LV hypertrophy in AAS users, as evidenced by echocardiographic changes in the myocardium before the onset of visible hypertrophy.²²

Arrhythmias

Animal experiments suggest that AAS abuse may lead to cardiac ischemia during peak exercise, possibly due to accelerated atherosclerosis caused by lipoprotein abnormalities over years of abuse.²³ Additionally, AAS can increase platelet aggregation and thrombus formation through various mechanisms, including increasing

platelet production of thromboxane A2 (a potent platelet aggregator), reducing prostacyclin production (a platelet aggregation inhibitor), and raising fibrinogen levels.²⁴

The relative clinical contributions of these mechanisms are uncertain. Still, their combined effects could plausibly explain instances of acute infarction or ventricular arrhythmias in young athletes without traditional cardiac risk factors.²⁵

Revisiting the concept of athletic heart syndrome

The physiological hypertrophy of athletic heart syndrome is characterized by homogeneously distributed symmetrical parietal thickening involving all cardiac cavities. The chamber thickening induced by exercise is proportional to the type of physical activity performed and its load (mainly combined power and resistance disciplines). It is reversible after temporary detraining, generally after three (3) months.²⁶

A physiological enlargement of both ventricles is usually observed (mainly with endurance athletes), along with proportional atrial dilation. Despite hypertrophy and enlargement of cardiac chambers, cardiac systolic function is not compromised in athletes, with no significant differences compared to non-athlete individuals. Likewise, LV diastolic function is normal, and an increased contribution of early filling velocity can be seen at rest with pulsed Doppler $E/A > 2$. Aortic root diameters are generally normal in athletes.⁶

The human heart feels the demand and adapts in both the short and long terms. Assessing cardiac remodeling in athletes presents several challenges, including the impact of training load — particularly in amateur athletes — and the difficulty in establishing sport- and sex-specific reference ranges. Additionally, factors such as race and the use of performance-enhancing substances, like AAS, further complicate the assessment.²⁷

The morphology of the LV in the athlete's heart is typically studied using echocardiography, revealing a distinct pattern of LV dilation and hypertrophy. Current recommendations from the European Association of Preventive Cardiology (EAPC) and the European Association of Cardiovascular Imaging (EACVI) suggest echocardiography as a secondary investigative tool to differentiate between an athlete's physiological heart adaptations and underlying cardiac conditions. However, contrary to these guidelines, echocardiography is often used as a primary screening tool in the cardiovascular assessment of both professional and amateur athletes, even when clinical and electrocardiographic assessments are normal.^{28,29}

For endurance athletes exhibiting LV and/or right ventricular (RV) dilation with mildly reduced ejection fraction at rest, stress echocardiography can be used to evaluate contractile reserve during exercise. A significant improvement in contractility during exercise indicates physiological cardiac remodeling, whereas a lack of improvement or subnormal response suggests a pathological condition (e.g., dilated cardiomyopathy, non-compacted

LV, arrhythmogenic cardiomyopathy). Likewise, exercise-induced ventricular arrhythmias also support the hypothesis of underlying heart disease.³⁰

Cardiac magnetic resonance (CMR) can more accurately assess cardiac structure and function, as well as characterize the myocardium, detecting relevant changes, including the quantification of myocardial fibrosis. CMR also enables the assessment of myocardial tissue characteristics, including fat and water content, fibrosis, and cardiomyocyte mass.^{31,32}

While the vast majority of athletes have suitable echo windows, it is important to note that echocardiography is not used as the gold standard for measuring cavity, mass, and intracavitary volumes. Instead, its routine use in suspected athletic heart syndrome is primarily due to its ability, within a coherent clinical context, for tissue characterization. Therefore, CMR is useful for assessing athletic heart syndrome, as the precision of the measurements is superior to echocardiography.

Given the complex structure of RV morphological assessment, CMR is more reproducible than echocardiography. Increases in RV mass, end-diastolic volumes, and stroke volumes relative to non-athletes have been described. The relationship between LV and RV size was maintained, leading to the conclusion that athlete's heart syndrome involves balanced remodeling of both ventricles' diameter.³²

Anabolic androgen steroids and changes in the echocardiogram

AAS use resulted in impaired LV systolic function, as assessed by left ventricular ejection fraction (LVEF) and longitudinal strain, and demonstrated in a study by Baggish *et al.* This finding was driven almost entirely by AAS users who were under the influence of drugs at the time of the study, suggesting that LV dysfunction may be dynamically related to AAS use.³³

In subsequent analyses examining the association of outcomes with duration and use of AAS: 41 (71%) of the 58 AAS users had an LVEF below the limit of 52% calculated by the Simpson method. In contrast, non-users had a largely normal LVEF.³³

In another analysis, the primary outcome variables LVEF and E' , AAS users showed significant deficits compared to non-users, therefore compromising the parameters of systolic and diastolic function in parallel.

Regarding ventricular remodeling, AAS users exhibited higher LV mass index, thicker LV walls, and more concentric LV geometry than non-users.³⁴

AAS users also showed impaired diastolic function, both in relation to non-users and also as defined by current diagnostic criteria according to the American Society of Echocardiography. Twenty-nine (50%) drug users had values below the normal E' threshold of 8.5 cm/s. Similar associations with AAS use have been found in other studies.³⁵

No association was found between the duration of AAS use and the primary outcome variables (for every additional

10 years of AAS exposure, the estimated mean change [95% CI] in LVEF was -3.3% [-8.3% to 1.6%], $p = 0.19$; and the estimated mean change in E' was 0.1 cm/s [-1.0 to 1.2 cm/s], $p = 0.90$).³⁵

When assessing the associations separately for AAS users and non-users, there was a significant association between increased LV mass index and decreased LVEF among AAS users (estimated mean change [95% CI] in LVEF for each increase of 10 g in LV mass index -1.6% [-2.4 to -0.8%], $p < 0.001$). In contrast, no significant association between LV mass index and LVEF was seen in non-users (estimated mean change in LVEF for each 10 g increase in LV mass index -0.2% [-1.5 to 1.2%], $p = 0.80$).^{35,36}

The Speckle-Tracking Echocardiography (STE) is another advanced echocardiographic strain imaging technique that provides new insights into the characterization of myocardial properties in athletes, detecting subclinical ventricular systolic function in early-stage heart disease when LVEF is still normal.³⁷

The LV global longitudinal strain (GLS) obtained by STE is the most used parameter in clinical practice, but there is controversy in the literature, whose studies show differences. Therefore, a reduction in longitudinal deformation in athletes should be considered a subclinical sign of LV contractile dysfunction and should raise the suspicion of myocardial disease, particularly in the presence of hypertrophy or equivocal LV dilation.³⁷

Data regarding the interpretation of parameters derived from VR and STE in athletes are still controversial. Chronic, strenuous physical training appears to have a detrimental effect on RV function, with reduced RV strain immediately after endurance running, followed by complete recovery. Finally, RV deformation imaging can help differentiate between physiological and pathological conditions by identifying regional wall motion abnormalities in patients with arrhythmogenic cardiomyopathy.³⁷

Myocardial work is a novel echocardiographic index of LV contractile function, being less dependent on load and adjusting parameters derived from STE for afterload. Increased afterload in various physiological and pathological conditions can result in impaired effort. This is particularly relevant for athletes whose BP and loading conditions may vary between exams and different phases of their training programs, and it may also be beneficial for monitoring AAS users.³⁸

The use of coronary tomography (CCT) in users of anabolic androgen steroids

Recent technological advances have expanded the role of CCT beyond the evaluation of coronary arteries and large vessels. LV morphological and functional assessment is performed using an ECG-controlled retrospective scanning protocol. The assessment of LV volumes, systolic volume, ejection fraction, and mass showed excellent correlation with the CMR assessment.³⁹

LV functional assessment by CCT is particularly useful in claustrophobic patients, as they are unable to perform CMR, or if there are any contraindications to CMR

(although rare among athlete patients). Otherwise, CCT cannot be recommended as a first-line imaging technique for LV functional assessment in athletes, given the greater radiation exposure required. The use of iodinated contrast media allows the assessment of the LV and myocardial fibrosis (with analysis of late iodine enhancement) by CCT, even if not routinely used in clinical practice, with good agreement with the same assessments performed by contrast-enhanced CMR.

All athletes with ambiguous anomalous coronary artery anatomy, suspected following echocardiography, should undergo CCT based on institutional preferences and knowledge. In cases of suspected coronary atherosclerotic disease, CCT is valuable for coronary artery calcium scoring and non-invasive coronary angiography. When dilation of the aortic root or ascending aorta is suspected or confirmed, a comprehensive tomographic evaluation of the aorta is recommended.

While regular aerobic exercise is known to be beneficial for the primary and secondary prevention of cardiovascular diseases, the impact of lifelong resistance exercise on the heart has only recently been studied. In a minority of susceptible veteran athletes, exercise can trigger adverse events such as SCD, often attributable to silent CAD. Several studies have demonstrated a higher-than-expected prevalence of CAD in veteran athletes, and failure to detect those at risk using routine cardiovascular screenings such as exercise testing has potentially devastating consequences. CCT, including calcium scoring, has revealed CAD in 25-53% of veteran athletes and offers prognostic benefits by determining plaque morphology and total atherosclerotic burden.⁴⁰

AAS use has been linked to increased coronary atherosclerosis, with disease severity strongly associated with the cumulative duration of AAS use over a lifetime. Taken together, our findings suggest that prolonged AAS use is associated with adverse cardiovascular phenotypes characterized by both myocardial disease and CAD.⁴¹

AAS users had significantly greater coronary plaque volume than non-users. When investigating the relationship between ATT measurements and the duration of AAS use, we observed strong associations between lifetime use time and all angiographic measures of coronary pathology (table 1). However, there was no significant change in the association between AAS use and plaque volume (estimated mean difference between users with and without drugs in classifications: -0.07 SD units [-0.56 to 0.41]; $p = 0.76$). It is noteworthy that three AAS users had experienced prior myocardial infarctions due to atherosclerotic disease, as documented by cardiac catheterization. These events occurred at ages 38 (ST-segment myocardial infarction with complete occlusion of the left anterior descending artery), 43 (Myocardial infarction without ST-segment elevation with 99% occlusion of both the right coronary and left circumflex coronary arteries), and 46 (ST-segment elevation myocardial infarction with complete occlusion of an obtuse 2nd marginal artery), after 17, 11, and 5 years of accumulated exposure to AAS over their lifetime, respectively.⁴²

Table 1 – Main changes in cardiovascular imaging methods with the use of AAS

Method	Finding
Echocardiogram	Decreased LV systolic function Hemodynamic and diastolic function analysis Reduction of cardiac mechanics (strain), (controversial)
CCT	Increased volume of coronary plaque Plaque location Calcium score
CMR Imaging	LV hypertrophy and RV dysfunction Auxiliary and tissue analysis in the differential diagnosis of cardiomyopathies

LF: Left Ventricle; RV: Right Ventricle; CCT: Coronary Tomography; CMR: Cardiac Magnetic Resonance

CMR in AAS users

CMR is the most valuable imaging method for the differential diagnosis between physiology and pathology in athletes, aiding in the discrimination of diseases where the echocardiography does not provide clarification.⁴³

CMR is the gold standard for defining myocardial morphology, assessing parietal mobility, size of cardiac chambers, and tissue definition. It provides a precise and reproducible assessment of the volume and mass of the heart chambers, as well as the global and regional contractile function. This method is the preferred choice for accurately assessing the morphology and function of the RV.¹²

CMR represents the superior method for identifying myocardial fibrosis and its distribution pattern through the assessment of late gadolinium enhancement, native T1, and extracellular volume (ECV) mapping. Furthermore, CMR can identify edema and fat in the myocardial walls.²²

In particular, the identification of myocardial fibrosis can differentiate between athletic heart syndrome and pathological LV hypertrophy (Figure 1), as fibrosis often accompanies cardiac remodeling due to pathology.⁴⁴

In hypertrophic cardiomyopathy, midwall fibrosis is typically found in areas of extreme hypertrophy, although it can also occur in non-hypertrophied segments.³⁶

Myocardial fibrosis has been observed in athletes with a higher prevalence than in healthy non-athlete populations. In healthy athletes, myocardial fibrosis involves less than 3% of the myocardium. It varies greatly in quantity, location and pattern, but is generally found in the RV or interventricular septum. The prevalence of myocardial fibrosis in individuals with athletic heart syndrome appears to increase with a longer history of resistance training. However, the prognostic significance of myocardial fibrosis in individuals with athletic heart syndrome is unknown.⁴⁵

Regarding diffuse interstitial fibrosis, studies comparing ECV in athletes and controls reported similar or lower ECV values in athletes.⁴⁵

Stress CMR, usually with exercise, can be used to identify reduced functional reserve and early-stage cardiomyopathy

when resting functional assessment is mildly abnormal. However, further studies are needed to evaluate the cost-effectiveness of stress CMR imaging in this setting.⁴⁵

A cross-sectional cohort design was used with 21 strength-trained participants who underwent heart CMR imaging and STE. Thirteen participants (30 ± 5 years) who had been taking AAS for at least two years and were currently on a “use” cycle were compared with age- and training-matched controls ($n = 8$; 29 ± 6 years) who had never taken AAS.²³

AAS users had a significantly greater absolute LV mass (220 ± 45 g) compared to non-users (163 ± 27 g; $p < 0.05$), but this difference was removed when indexed to fat-free mass. AAS users also had a reduced RV ejection fraction (AS $51 \pm 4\%$ vs. NAS $59 \pm 5\%$; $p < 0.05$) and a significantly lower myocardial tissue velocity ratio E' by LV tissue Doppler (AAS $0.99[0.54]$ vs. non-AAS users $1.78[0.46]$; $p < 0.05$). Maximum LV longitudinal strain was lower in AAS users ($-14.2 \pm 2.7\%$ vs. $-16.6 \pm 1.9\%$; $p < 0.05$). There was no evidence of focal fibrosis in any participant. AAS use was associated with significant LV hypertrophy, although in line with greater fat-free mass, reduced LV strain, diastolic function, and reduced RV ejection fraction in male bodybuilders. There was, however, no evidence of focal fibrosis in any AAS user.²³

Conclusions

The indiscriminate use of AAS among athletes has been on the rise and is linked to LV hypertrophy and dysfunction. Cardiologists must be vigilant about this public health concern and should assess its impact on users early on using cardiovascular imaging.

Author Contributions

Conception and design of the research: Bispo I, Zago IM; acquisition of data, analysis and interpretation of the data: Zago IM; writing of the manuscript and critical revision of the manuscript for intellectual content: Bispo I.

Potential Conflict of Interest

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

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There were no external funding sources for this study.

Study Association

This study is not associated with any thesis or dissertation work.

Ethics Approval and Consent to Participate

This article does not contain any studies with human participants or animals performed by any of the authors.

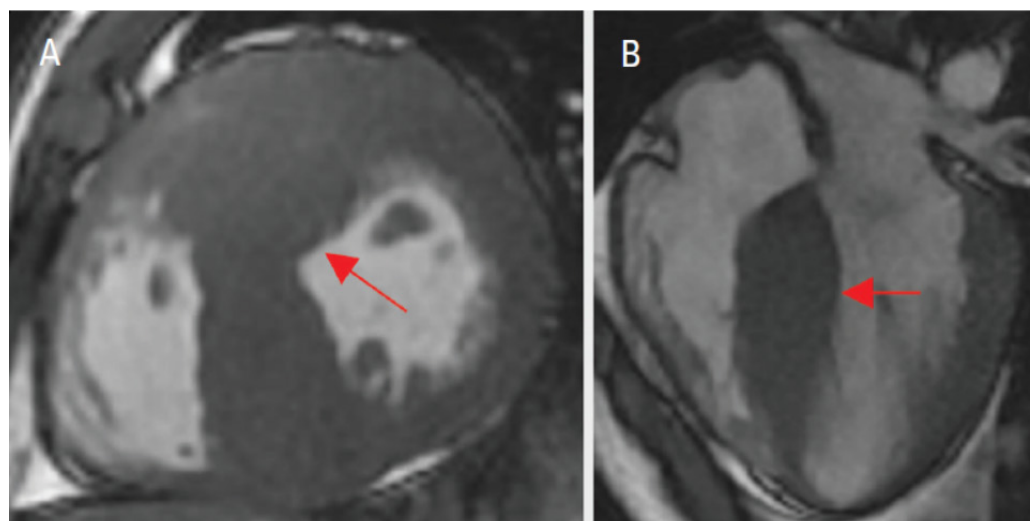


Figure 1 – CMR Imaging for the differential diagnosis of cardiomyopathy Hypertrophic x Athlete's Heart

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