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Beauty, brawn, and burden: cardiovascular consequences of aesthetic AAS use

Abstract

The non-medical use of androgenic anabolic steroids (AAS) for aesthetic and performance enhancement has surged globally, especially among young adults and athletes. Despite public perception of these substances as shortcuts to physical perfection, accumulating evidence reveals significant cardiovascular toxicity associated with their use. This opinion paper synthesizes recent findings from clinical and experimental studies, highlighting mechanisms of cardiovascular damage, including left ventricular hypertrophy, arrhythmias, dyslipidemia, thrombosis, and sudden cardiac death. We argue that cardiologists and physicians must be at the forefront of prevention, early detection, and multidisciplinary management of AAS-related cardiovascular complications.

Keywords: anabolic steroids, cardiovascular risk, left ventricular hypertrophy, sudden cardiac death; performance enhancement

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Introduction

The pursuit of enhanced physical performance and idealized body image has led to the widespread misuse of androgenic anabolic steroids (AAS) outside their approved clinical indications. Originally developed for treating hypogonadism and cachexia, AAS are now commonly used by athletes, bodybuilders, and recreational users aiming for muscle mass, strength, or aesthetic goals.[1,2] However, this non-therapeutic usage is increasingly recognized as a major public health concern due to its systemic adverse effects—especially on the cardiovascular system.[3,4]

Cardiologists are now more frequently encountering young patients with unexpected myocardial infarctions, arrhythmias, or cardiomyopathy, many of whom have histories of AAS use. This paper aims to provide a concise, evidence-based overview of the cardiovascular consequences of AAS, highlighting the urgent need for clinical awareness and preventive action.

Main Body

Prevalence and patterns of use

Surveys indicate that AAS use may affect up to 3–4 million individuals in the United States alone, with global prevalence growing rapidly, especially among gym-goers and recreational athletes.[2] In Brazil, a recent cross-sectional study found that 22% of female high-intensity functional training (HIFT) practitioners reported current or past AAS use, with aesthetics being the primary motivation. However, there is a noticeable trend toward increased use for performance enhancement, particularly among women with longer training history.[1] Users often engage in "cycling" and "stacking," combining multiple AAS with other substances, increasing the risk of adverse events. Importantly, these regimens often involve supra-physiological doses—10 to 100 times higher than endogenous testosterone levels.[4]

Structural and functional cardiac alterations

AAS induce direct myocardial changes through androgen receptor-mediated pathways, leading to both structural and functional cardiac remodeling. These alterations stem from the anabolic effects of supraphysiological androgen exposure on cardiomyocytes and vascular smooth muscle cells, promoting hypertrophic growth, collagen deposition, and altered gene expression. Clinical imaging studies consistently demonstrate that long-term AAS users exhibit concentric left ventricular hypertrophy, impaired diastolic filling patterns, and, in many cases, reduced left ventricular ejection fraction when compared to non-users.[3] These findings are particularly concerning because they are often observed in young, otherwise healthy individuals, with no prior history of cardiovascular disease or traditional risk factors.

A prospective cardiac MRI study by Baggish et al.[5] revealed elevated levels of myocardial fibrosis in chronic AAS users, characterized by delayed gadolinium enhancement—a marker of interstitial collagen deposition. Myocardial fibrosis reduces ventricular compliance and electrical stability, predisposing individuals to heart failure and arrhythmias. Pope et al.[2] further confirmed that such fibrotic changes may persist even after cessation of AAS use, indicating potentially irreversible myocardial remodeling. These pathological changes may be dose-dependent and time-dependent, reinforcing concerns about prolonged and high-dose exposure. Notably, diastolic dysfunction, often asymptomatic in early stages, may precede systolic impairment and serve as a harbinger of impending heart failure.

Moreover, animal studies and human biopsy data suggest that androgen receptor overactivation increases oxidative stress, mitochondrial dysfunction, and local inflammatory cytokine production in cardiac tissue.[3,4] These mechanisms may accelerate cardiomyocyte apoptosis and further compromise myocardial integrity. Collectively, the evidence underscores that AAS cardiotoxicity is not limited to transient physiological adaptations but represents a continuum of pathological remodeling with long-term clinical consequences, including dilated cardiomyopathy, heart failure with preserved or reduced ejection fraction, and sudden cardiac death.[2,5,6]

Arrhythmogenesis and sudden cardiac death

AAS appear to promote arrhythmogenesis through a multifactorial set of mechanisms involving both electrophysiological and structural myocardial alterations. At the cellular level, supraphysiological concentrations of androgens interfere with cardiac ion channel expression and function—particularly affecting potassium (K^+), sodium (Na^+), and calcium (Ca^{2+}) channels—which can lead to abnormal action potential propagation and repolarization

disturbances. These alterations contribute to the development of early afterdepolarizations and increased dispersion of repolarization, key substrates for ventricular arrhythmias.

In parallel, AAS enhance sympathetic nervous system activity by increasing circulating catecholamines and upregulating β-adrenergic receptors, creating a pro-arrhythmic state particularly during physical exertion or emotional stress. This autonomic imbalance, when superimposed on a structurally compromised myocardium—characterized by hypertrophy, fibrosis, or dilated chambers—further increases the risk of malignant ventricular tachyarrhythmias.

Structural remodeling also plays a critical role: myocardial fibrosis, as demonstrated in cardiac MRI studies of long-term AAS users,[5] disrupts normal electrical conduction pathways and creates reentrant circuits, facilitating the development of sustained arrhythmias. These electrophysiological abnormalities are clinically significant, as increasing case reports have described episodes of ventricular fibrillation, torsades de pointes, and sudden cardiac death (SCD) in otherwise healthy, young AAS users, frequently occurring during intense exercise or competition.

A systematic review by Perry et al.[3] highlighted that AAS use is associated with a significantly increased risk of QT interval prolongation, ventricular arrhythmias, and fatal cardiac events. Importantly, some of these effects may not be evident on routine clinical evaluation, reinforcing the need for high clinical suspicion and potentially advanced cardiac screening in known or suspected AAS users. The arrhythmogenic risk profile appears to be dose-dependent, duration-related, and possibly potentiated by concomitant use of stimulants or other substances often present in "stacking" regimens. Given the potentially fatal nature of these events, cardiologists must remain vigilant for arrhythmic complications in this population, even in the absence of overt structural heart disease.

Lipid profile and atherogenesis

Androgenic anabolic steroids (AAS) exert profound and multifaceted effects on lipid metabolism, constituting one of the most clinically significant pathways of cardiovascular risk associated with their use. AAS consistently suppress hepatic production of high-density lipoprotein (HDL) cholesterol while increasing low-density lipoprotein (LDL) cholesterol levels. This atherogenic shift stems in part from the androgen-mediated downregulation of apolipoprotein A-I synthesis, which is essential for HDL formation and reverse cholesterol transport. Concurrently, AAS stimulate hepatic triglyceride lipase activity, which accelerates HDL catabolism and further depletes protective lipoproteins. Elevated LDL levels, conversely, result from upregulated apolipoprotein B expression and impaired LDL receptor-mediated clearance.[3,4] This dyslipidemic profile directly accelerates atherosclerosis through endothelial dysfunction, increased oxidative stress, and lipid accumulation within the intima. The resulting foam cell formation and plaque progression occur even in the absence of traditional cardiovascular risk factors, highlighting the potency of AAS as independent drivers of vascular injury. In addition, AAS have been shown to increase arterial stiffness and impair flow-mediated dilation, reflecting early disruption of vascular homeostasis.[2] The long-term impact of these lipid abnormalities is evidenced by imaging studies. A notable cross-sectional analysis by Bjørnebekk et al.[6] revealed that chronic AAS users presented significantly elevated coronary artery calcium (CAC) scores compared to age- and activity-matched controls. Elevated CAC is a validated surrogate marker of subclinical atherosclerosis and a strong predictor of major adverse cardiovascular events. These findings suggest that vascular aging in AAS users is not merely accelerated, but pathologically distinct, reflecting sustained metabolic and inflammatory insults.[6] Moreover, supraphysiological androgen exposure may induce a state resembling factitious Cushing syndrome, particularly when AAS are used in conjunction with glucocorticoids or other catabolic agents common in "cutting" cycles. This iatrogenic hypercortisolism is characterized by central adiposity, insulin resistance, hypertension, and dyslipidemia—each of which is a well-established cardiovascular risk factor. The convergence of these metabolic abnormalities with AAS-induced dyslipidemia creates a synergistic environment of vascular toxicity, magnifying the likelihood of myocardial infarction, stroke, and sudden cardiac death.[3,4]

Thrombosis and endothelial dysfunction

Pro-thrombotic states have been consistently reported in association with anabolic androgenic steroid (AAS) use, mediated by multiple converging mechanisms. AAS stimulate platelet aggregation through increased thromboxane A2 production and reduced prostacyclin activity, shifting the hemostatic balance toward thrombosis. In parallel, AAS-induced erythrocytosis elevates hematocrit and blood viscosity, impairing microcirculatory flow and predisposing to vascular occlusion.[7,8] These hematological and vascular changes collectively increase the risk of thrombotic events, even in young, athletic individuals.

Endothelial dysfunction plays a central role in this process. AAS reduce nitric oxide (NO) bioavailability, increase reactive oxygen species (ROS) production, and promote inflammation within the vascular endothelium.[9] These effects impair vasodilation, increase leukocyte adhesion, and enhance the expression of adhesion molecules and pro-inflammatory cytokines—hallmarks of an activated endothelium. The cumulative consequence is a pro-thrombotic endothelial phenotype with diminished capacity to maintain vascular homeostasis.

Moreover, AAS influence the fibrinolytic system by increasing levels of plasminogen activator inhibitor-1 (PAI-1), thereby reducing clot degradation and favoring thrombus persistence.[10] This fibrinolytic suppression, in conjunction with endothelial injury and hypercoagulability, creates a dangerous milieu conducive to arterial and venous thromboembolic events. Notably, case reports and clinical series have documented myocardial infarction, stroke, and deep vein thrombosis in AAS users with no other identifiable risk factors.[5]

Given this multifactorial pathophysiology, standard cardiovascular risk assessments may underestimate actual danger in individuals using AAS. Biomarkers of endothelial dysfunction, such as asymmetric dimethylarginine (ADMA) or flow-mediated dilation (FMD) testing, may offer early warning signs of vascular impairment and help guide clinical decision-making.[2]

Reversibility and management challenges

Some cardiovascular effects of AAS may be partially reversible upon cessation, particularly functional alterations such as mild diastolic dysfunction or early endothelial impairment. However, more advanced structural cardiac changes—including left ventricular hypertrophy and myocardial fibrosis—along with vascular remodeling and atherosclerotic plaque development, tend to persist and may progress even after discontinuation of AAS use.[5] These long-term sequelae reflect the chronic impact of supraphysiological androgen exposure on myocardial and vascular tissue, resulting in irreversible damage that increases the lifetime risk of heart failure, ischemic heart disease, and arrhythmias.

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Compounding the difficulty of cardiovascular recovery is the psychological and physiological syndrome of AAS withdrawal, which often includes depressive symptoms, fatigue, irritability, anhedonia, and loss of libido. In some cases, users may experience major depressive episodes or even suicidal ideation, especially when AAS use was linked to body image issues or identity constructs tied to muscularity. These psychiatric symptoms not only reduce treatment adherence but may drive users to relapse, restarting AAS use to alleviate emotional and physical distress. Studies have shown that withdrawal from AAS can lead to hypogonadotropic hypogonadism, sometimes persisting for months, further complicating recovery and hormonal stabilization.

In this context, a multidisciplinary approach becomes essential, integrating cardiovascular care with endocrine monitoring, psychiatric evaluation, and behavioral support. Cardiologists must coordinate with endocrinologists to assess residual hormonal dysregulation and its cardiovascular implications, while psychiatrists address mood disorders and underlying dependence syndromes. In parallel, addiction specialists can offer structured interventions, including cognitive-behavioral therapy and peer support programs, to prevent relapse and encourage long-term abstinence. This integrated model is particularly crucial given that many AAS users do not perceive themselves as substance-dependent and may delay seeking help until significant clinical complications arise. Therefore, proactive engagement, empathetic dialogue, and longitudinal follow-up are key components of effective care for this high-risk population.

Conclusions

The aesthetic and performance-enhancing use of AAS poses a serious threat to cardiovascular health. Despite growing scientific evidence, public awareness and clinical recognition remain insufficient. Cardiologists and frontline physicians must maintain a high index of suspicion when evaluating young patients with unexplained cardiovascular symptoms and engage in preventive counseling for at-risk populations. Long-term strategies should include public education, screening programs, and policy-level interventions to curb the silent epidemic of AAS-related cardiac morbidity and mortality.

Conflicts of interest

The authors declare that they have no conflict of interest.

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