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EFFECTS OF ANABOLIC STEROID ABUSE ON THE DEVELOPMENT OF HEART FAILURE AND CARDIOVASCULAR INCIDENTS

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ABSTRACT

Anabolic androgenic steroids (AASs) are synthetic derivatives of testosterone primarily indicated for the treatment of androgenic deficiency states. Non-clinical use of these agents has increased in recent years, particularly among athletes and individuals seeking rapid muscle hypertrophy and enhanced physical performance. Supraphysiological, off-label dosing is associated with significant systemic adverse effects. Elevated steroid levels disrupt endocrine homeostasis by suppressing the hypothalamic-pituitary-gonadal (HPG) axis. Furthermore, AAS misuse has been linked to dyslipidemia, activation of the renin-angiotensin-aldosterone system (RAAS), vascular remodeling, oxidative stress, and left ventricular hypertrophy. These mechanisms collectively add to structural remodeling of the myocardium and blood vessels, thereby increasing the risk of cardiovascular disease (CVD). Emerging evidence also indicates a higher prevalence of cardiomyopathy, arrhythmias, and premature atherosclerosis among chronic AAS users. Recent studies have associated AAS misuse with left ventricular dilation, reduced ejection fraction, and mitral regurgitation.

This narrative review compiles current human-based research on the cardiovascular consequences of anabolic androgenic steroids misuse, explains the pathophysiology of CVD in this population, and highlights recommended clinical evaluations. A comprehensive literature search was executed using PubMed, Scopus, and Web of Science, focusing on original clinical cardiovascular imaging and translational studies published between 2000 and 2025.

KEYWORDS

Anabolic Androgenic Steroids, Cardiovascular Disease, Cardiomyopathy, Heart Failure, Steroid Misuse

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1. Introduction

Testosterone and its derivatives are frequently used in clinical medicine. The U.S. Food and Drug Administration (FDA) has approved several anabolic androgenic steroids (AASs) for specific clinical states, including testosterone and its esters for primary and secondary hypogonadism, and oxandrolone for weight gain following illness or trauma, osteoporosis-related bone pain, and anti-catabolic therapy. Shortly after their clinical introduction in the early 1950s, athletes recognized the anabolic traits of these substances to increase performance. In 1975, AASs were officially banned by the Olympic Committee. During the 1980s, illicit AAS use expanded outside professional athletes and became prevalent among recreational gym users. This period also saw a cultural shift in Western societies, emphasizing muscularity and physical dominance, as reinforced by popular media and the rapid growth of the commercial fitness industry. Consequently, young males increasingly sought rapid methods to improve their physical appearance, leading to widespread AAS use among this population. Pope et al. estimate that 2.9 to 4.0 million Americans (aged 13 to 50 years) have used supraphysiological doses of AAS for muscle mass gain or personal appearance. Notably, in recent years, use has increased among women and older adults. Globally, approximately 6% of males and 1.6% of females have used AASs.

Individuals who misuse AASs frequently use dosing methods such as “cycling” and “stacking” multiple substances to lessen side effects and enhance anabolic effects. As a result, administered doses are typically 5 to 20 times higher than therapeutic recommendations. Intramuscular injection remains the most common route of administration, although oral pills are increasingly popular. Testosterone esters (propionate, enanthate, cypionate) are the most widely used, while synthetic-based agents such as stanozolol, oxandrolone, boldenone, and nandrolone decanoate are gaining prevalence. AAS misuse is frequently accompanied by the use of sympathomimetic stimulants (ephedrine, clenbuterol, pseudoephedrine), illicit psychostimulants (cocaine, methamphetamine), and thyroid hormones, all of which further elevate cardiovascular risk and adverse health outcomes.

Recreational use of AASs emerged in the 1980s; as a result, many former users are now reaching ages at which cardiovascular events become more prevalent, stressing the need for clinical recognition and diagnosis of long-term AAS-related complications. Recent studies have demonstrated that individuals who illicitly use AASs have an increased risk of sudden cardiac death and ischemic stroke. Chronic AAS use has been associated with dyslipidemia, elevated blood pressure, atherosclerosis, left ventricular (LV) hypertrophy, systolic and diastolic LV dysfunction, and arrhythmias. This clinical presentation has been termed steroid-induced cardiomyopathy. Although the adverse cardiovascular effects of testosterone derivatives are increasingly recognized, their impact on right ventricular (RV) structure and function remains insufficiently characterized. Accordingly, this review summarizes current human-based evidence on the cardiovascular consequences of anabolic androgenic steroid misuse, with particular emphasis on mechanisms causing heart failure and cardiovascular events.

2. Methods

This narrative review summarizes current evidence regarding supraphysiological anabolic androgenic steroid use and its association with heart failure and cardiovascular disease. A comprehensive literature search was conducted across the PubMed, Scopus, and Web of Science databases, focusing on human-based studies published between 2000 and 2025. Google Scholar served as a supplementary resource.

A broad set of search terms was used, including keywords: anabolic androgenic steroids, cardiomyopathy, left ventricular hypertrophy, arrhythmia, dyslipidemia, atherosclerosis, cardiovascular disease, and athletes. Only original medical research studies were considered, including randomized controlled trials, cohort studies, case–control studies, cardiovascular imaging studies, and mechanistic human or translational investigations with clear relevance to human physiology.

Systematic reviews, meta-analyses, editorials, studies limited solely to animal models without application to human medicine, case reports, and conference abstracts were excluded. Studies were initially screened by reviewing titles and abstracts, followed by a full-text assessment of eligible articles.

Due to the diversity of the included studies and their outcomes, the findings were integrated qualitatively rather than through quantitative synthesis. Ethics committee approval was not required, as this review was based exclusively on previously published data.

3. Pathophysiology and molecular effects of anabolic androgenic steroids on the cardiovascular system

The androgen receptor (AR) is the primary mediator of the cellular effects of AASs. ARs are widely expressed throughout the human body, with high concentrations in reproductive tissues, skeletal muscle, and the central nervous system. They are also present in the myocardium, endothelium, and liver, which is critical for the development of cardiovascular disease. AR signaling promotes cardiomyocyte hypertrophy, induces endothelial dysfunction, and alters lipid metabolic pathways in the liver. Indirect effects of AASs include activation of the renin-angiotensin-aldosterone system (RAAS), increased sympathetic activity, and a prothrombotic state, all of which further elevate the risk of coronary artery disease (CAD).

3.1. Endothelial dysfunction

Physiological levels of androgens protect the vascular endothelium. ARs within endothelial cells stimulate nitric oxide (NO) synthesis, which directly regulates vascular tone. Additionally, testosterone and dihydrotestosterone inhibit voltage-gated calcium channels and stimulate calcium-dependent potassium channels, contributing to vasodilation. In contrast, supraphysiological levels of AASs have the opposite effect. They upregulate calcium currents, increase oxidative stress, leading to NO scavenging and reduced bioavailability, and elevate circulating substances such as thromboxane and angiotensin II, leading to vasoconstriction. The vasodilatory response is thus overridden by vasoconstriction. Chronic AAS abuse induces vascular smooth muscle hypertrophy, narrowing the vessel lumen. These changes can have severe clinical consequences, promoting ischemic events. AAS-induced endothelial dysfunction is an early and key step in the development of atherosclerosis.

3.2. Atherosclerosis

AAS misuse is directly associated with dyslipidemia. Multiple studies have demonstrated that synthetic testosterone intake reduces high-density lipoprotein (HDL) levels, increases low-density lipoprotein (LDL) levels, alters lipoprotein (a), and decreases apolipoprotein A (ApoA) levels. Additionally, AAS use accelerates HDL catabolism. These changes in serum lipid profiles are commonly linked to the development of atherosclerosis and, consequently, to ischemic events, coronary artery disease, peripheral arterial disease, and cerebral infarction. Baggish et al. reported that AAS abusers have higher coronary plaque volume than non-users. However, recent studies have found no correlation between AAS use and plaque accumulation in femoral and carotid arteries. Further research is needed to determine the impact of AASs on large vessels.

3.3. Hypertension

Studies on the influence of AASs on blood pressure (BP) have produced inconclusive results. Some have demonstrated a correlation between AAS use and elevated BP, while others have not found significant associations. A recent study by Junior et al. showed that AAS misuse dysregulates post-exercise hypotension; AAS abusers exhibited higher 24-hour systolic BP after exercise compared to non-users. They also had decreased plasma MR-proANP and increased aortic stiffness. These findings can also be observed in former AAS abusers. Although the precise mechanisms underlying AAS-induced hypertension remain unclear, several pathways have been proposed. AASs can directly increase sympathetic tone and modulate renin release in the kidneys. Chronic use is associated with dysregulation of the RAAS, leading to increased aldosterone secretion and expanded blood volume via sodium and water retention. This may result in increased cardiac output and, over time, elevated BP. Additionally, atherosclerosis, vascular remodeling, and increased stiffness further contribute to hypertension.

3.4. Thrombosis

AASs disrupt hemostatic balance and induce a prothrombotic state. Increased synthesis of coagulation factors, erythropoietic stimulation, platelet hyperreactivity, and inhibited fibrinolysis are key mechanisms underlying this effect. Endothelial dysfunction may cause local injury, activating platelets. Upon activation, platelets release Thromboxane A2, a pro-aggregatory and vasoconstrictive substance. It has been suggested that AASs upregulate platelet Thromboxane A2 receptors, thereby enhancing platelet aggregation and activation responses. Some studies have shown that testosterone and its derivatives induce hepatic synthesis of coagulation factors, although results remain inconsistent and further research is needed. These mechanisms collectively increase the risk of thromboembolic events, including myocardial infarction, ischemic stroke, and pulmonary embolism in AAS abusers.

3.5. Cardiomyocyte hypertrophy

One of the primary effects of anabolic androgenic steroids in skeletal and cardiac muscle is the promotion of myocyte hypertrophy. These substances bind to the androgen receptor in cardiomyocytes, activating hypertrophic gene expression and the mTOR pathway (increasing protein synthesis), leading to hypertrophy. Additionally, the literature describes the indirect promotion of hypertrophy by disturbing the RAAS. Retention of sodium and water increases BP; angiotensin (II) directly binds to the angiotensin type 1 (AT-1) receptor; and aldosterone binds to the mineralocorticoid receptor in cardiomyocytes, which initiates another hypertrophic signaling. These mechanisms promote concentric left ventricular (LV) hypertrophy, inflammation, oxidative stress, and fibrosis. It increases the risk of cardiomyopathy, myocardial infarction, and sudden cardiac arrest.

3.6. Right ventricle remodeling

While substantial evidence links AAS misuse to left ventricular (LV) dysfunction, the effects on the right ventricle (RV) remain unclear. Preliminary data suggest that RV parameters, such as systolic function and strain, are decreased in the AAS abuser population. Nieminen et al. examined two biopsy specimens of RV tissue from AAS-using weightlifters and observed myocardial fibrosis. However, the small sample size limits generalizability. Further research, including large cohort studies and meta-analyses, is needed to clarify the impact of AASs on the RV.

3.7. Cardiac fibrosis

Animal experiment have shown that supraphysiological levels of AASs can induce cardiomyocyte apoptosis via oxidative stress. This study indicates that rats treated with nandrolone decanoate exhibit significantly higher cardiac caspase-3 activity, a key enzyme in the programmed cell death pathway. Myocardial remodeling, apoptosis, inflammation, and hypertrophy induce focal fibrosis. Although myocardial biopsy is rarely performed, it has revealed focal fibrosis in AAS abusers. Advances in imaging, such as cardiac magnetic resonance (CMR), provide non-invasive methods to detect fibrous tissue. However, data on AAS use and fibrosis imaging remain inconclusive and limited. CMR without mapping may underestimate diffuse interstitial fibrosis. Chronic AAS abuse is associated with left ventricular dilation and the development of advanced heart failure. A hypertrophic-fibrosing phenotype of cardiomyopathy appears common in this population, significantly increasing the risk of premature heart failure and sudden cardiac death.

3.8. Arrhythmias

Structural remodeling and myocardial fibrosis contribute to the electrical instability of the heart. Numerous studies have associated chronic AAS misuse with an increased risk of supraventricular and ventricular arrhythmias. In a Danish cohort study by Windfeld-Mathiasen et al., arrhythmias were the most prevalent cardiovascular event among AAS users. Supraventricular arrhythmias, particularly atrial fibrillation and atrial flutter, were more common compared to matched non-user populations. Other studies have reported higher frequencies of palpitations, premature ventricular beats, and non-sustained ventricular tachycardia in individuals exposed to illicit AAS use. Cardiac hypertrophy, interstitial fibrosis, and chronic sympathetic activation create a substrate for re-entry mechanisms, thereby raising susceptibility to potentially life-threatening ventricular arrhythmias and premature sudden cardiac death.

4. Clinical implementation

4.1. Symptoms

Anabolic androgenic steroids exert systemic effects because androgen receptors are widely expressed across multiple tissues and organ systems. Suppression of gonadotropin-releasing hormone (GnRH) and pituitary gonadotropins is well documented, with primary symptoms including low libido, erectile dysfunction, and fatigue. Supraphysiological levels of AAS may also induce neuropsychiatric symptoms such as intense fear, depressive disorders, cravings, body dysmorphia, and insomnia. There are no pathognomonic cardiovascular symptoms specific to AAS misuse; clinical presentation depends on the underlying complication. Arrhythmias may manifest as palpitations, syncope, and dyspnea. Left ventricular hypertrophy, reduced ejection fraction, and heart failure may present as decreased exercise tolerance, exertional dyspnea, and cough. In young patients presenting with these symptoms, a thorough clinical history and examination are warranted.

4.2. Biomarkers

In patients who abuse AASs, several biomarkers can be assessed to evaluate cardiac strain, damage, and potential necrosis. The most commonly used markers include troponins (Troponin T and I), N-terminal pro-BNP (NT-proBNP), and B-type natriuretic peptide (BNP). Troponins are used to detect myocardial necrosis, while natriuretic peptides assess the severity of myocardial wall stretch. It is important to evaluate lipid profiles and monitor HDL and LDL levels, as dyslipidemia increases cardiovascular risk. Additionally, a complete blood count with hematocrit is essential given the frequent occurrence of AAS-induced erythrocytosis, which elevates thromboembolic risk.

4.3. Imaging tests

Transthoracic echocardiography (TTE), electrocardiography (ECG), and cardiac magnetic resonance (CMR) are valuable tools for evaluating cardiac dysfunction. Many studies have reported decreased left ventricular ejection fraction (LVEF) on TTE in AAS abusers. Additional findings include diastolic left ventricular dysfunction, impaired systolic strain, and increased chamber dimensions in selected cohorts. The HAARLEM study demonstrated increased LV mass, myocardial thickness, and left atrial volume. Electrocardiographic abnormalities in AAS users are heterogeneous and reflect primary structural remodeling, autonomic imbalance, and ischemic burden more than a specific steroid-related electrical signature. Beyond supraventricular arrhythmias such as atrial fibrillation, AAS users often exhibit prolonged QRS duration and higher R wave amplitudes in V5-V6 with lower S waves in V1-V2, indicating LV hypertrophy. CMR provides

a highly objective, non-invasive assessment of cardiac function, structure, perfusion, fibrosis, and inflammation. Its high resolution and reduced subjectivity have established CMR as the reference standard for evaluating ventricular volumes, mass, and myocardial tissue characterization. In addition to TTE findings, some CMR studies have reported mildly reduced biventricular ejection fractions in long-term AAS users. However, many CMR studies using late gadolinium enhancement (LGE) have not demonstrated overt focal fibrosis, possibly reflecting diffuse interstitial fibrosis rather than replacement scar, which may be underestimated by LGE imaging. Advanced tissue characterization techniques, such as T1 mapping and extracellular volume (ECV) assessment, may offer greater sensitivity for detecting subtle diffuse myocardial changes.

5. Treatment of AAS-induced cardiomyopathies

Patients with AAS-induced cardiomyopathies require comprehensive and individualized therapeutic strategies. The primary objectives are to reduce the risk of sudden cardiac death (SCD), reverse left ventricular (LV) dysfunction, and prevent adverse events. Discontinuation of anabolic androgenic steroid use should be prioritized, as it interrupts the cycle of harm. Reports indicate that partial reversibility is possible, particularly in the early stages following a reduction in AAS levels. For patients with heart failure (HF), therapy should follow the latest European Society of Cardiology (ESC) and/or American Heart Association (AHA) guidelines. The ESC recommends that all patients with heart failure with reduced ejection fraction (HFrEF) receive one drug from each of four classes: angiotensin-converting enzyme inhibitor (ACEI) or angiotensin receptor-neprilysin inhibitor (ARNI), beta-blocker, mineralocorticoid receptor antagonist (MRA), and SGLT2 inhibitor. These agents have demonstrated efficacy in reducing symptoms, hospitalization rates, and SCD risk. Diuretics should be prescribed for patients with symptoms of fluid retention. For heart failure with mildly reduced ejection fraction (HFmrEF), SGLT2 inhibitors and diuretics are recommended, with consideration of ACEI or ARNI, beta-blocker, and MRA to further reduce hospitalization and mortality risk. Patients with heart failure with preserved ejection fraction (HFpEF) should be treated with diuretics and SGLT2 inhibitors. Management of comorbidities such as dyslipidemia and hypertension is essential, as these factors contribute to increased SCD risk. If there is no improvement after optimal therapy, consideration should be given to left ventricular assist device (LVAD) implantation or heart transplantation. Additionally, an implantable cardioverter-defibrillator (ICD) should be considered for primary prevention in patients with LVEF ≤35% after at least three months of optimal therapy.

6. Reversibility of changes

The toxicity associated with supraphysiological levels of AASs may be reversible, although multiple factors—including drug type, dosage, duration, and genetic susceptibility—must be considered. Suppression of the hypothalamic-pituitary-gonadal axis is reversible in many cases. However, the reversibility of cardiovascular consequences remains uncertain. Several studies, including the HAARLEM study, have shown that left ventricular function, intraventricular end-diastolic septal thickness, and ventricular end-diastolic posterior wall thickness returned to baseline after eight months of AAS discontinuation. Other studies have reported improvement in LV function following pharmacological and LVAD treatment. Conversely, some studies indicate that even after prolonged abstinence, patients with reduced cardiac parameters continue to exhibit symptoms and irreversible changes. These mixed findings highlight the need for further research to determine the reversibility of cardiac effects associated with anabolic steroid use.

7. Limitations of current evidence

Current evidence on the cardiovascular consequences of anabolic androgenic steroid (AAS) misuse is constrained by several methodological limitations. Most data are derived from observational, cross-sectional, or small cohort studies, limiting causal inference and long-term risk estimation. Exposure is often self-reported, complicating accurate assessment of dose, duration, and specific compounds, especially among users employing complex “stacking” regimens. Interpretation of cardiac remodeling is further confounded by intensive resistance training, as functional adaptations of the “athlete’s heart” may overlap with steroid-induced pathology. Additionally, many studies rely on surrogate imaging markers rather than definitive clinical endpoints such as heart failure hospitalization or cardiovascular mortality. Heterogeneity in study design and limited longitudinal follow-up highlight the need for larger prospective cohorts to better define the true cardiovascular risk and long-term reversibility associated with chronic supraphysiological AAS exposure.

8. Conclusions

Anabolic androgenic steroid misuse is a significant and increasingly recognized risk factor for premature cardiovascular disease and heart failure in young and otherwise healthy populations. Chronic exposure to supraphysiological doses of AASs promotes a complex network of maladaptive mechanisms, including endothelial dysfunction, dyslipidemia, RAAS activation, sympathetic overstimulation, prothrombotic state, myocardial hypertrophy, fibrosis, and electrical instability. Collectively, these processes contribute to structural and functional cardiac remodeling, accelerating the development of cardiomyopathy, arrhythmias, atherosclerosis, and sudden cardiac death. Accumulating human-based imaging and clinical data indicate that AAS-associated cardiac remodeling is not simply a physiological adaptation to resistance training but may represent a distinct pathological phenotype, commonly characterized by concentric hypertrophy, impaired systolic and diastolic function, and diffuse interstitial fibrosis. Although partial reversibility has been observed following cessation—particularly in early stages—the long-term trajectory remains uncertain, and persistent myocardial dysfunction has been documented in some former users. Early identification of AAS misuse through detailed clinical history, laboratory testing, and multimodal cardiac imaging is essential, especially in young patients presenting with unexplained left ventricular dysfunction, arrhythmias, or thromboembolic events. Management should prioritize immediate discontinuation of AASs and the implementation of guideline-directed medical therapy when heart failure is present. Given the increasing prevalence of non-medical AAS use worldwide, heightened clinical awareness, preventive education, and well-designed longitudinal studies are urgently needed to better define causality, reversibility, and long-term cardiovascular outcomes in this population.

Conflict of interest: No conflict of interest to declare.

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