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CARDIOVASCULAR TOXICITY OF ANABOLIC-ANDROGENIC STEROIDS IN RECREATIONAL ATHLETES: MECHANISMS, CLINICAL PHENOTYPES, AND A PRACTICAL DIAGNOSTIC APPROACH

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ABSTRACT

Non-medical use of anabolic-androgenic steroids (AAS) has expanded beyond elite sports and is common among recreational strength trainees. Chronic supraphysiologic exposure is associated with adverse cardiovascular (CV) remodeling, accelerated atherosclerosis, and excess mortality. This review synthesizes evidence on AAS-associated CV toxicity and proposes a pragmatic diagnostic approach. Current data describe distinct clinical features in users: (i) often reversible cardiometabolic disturbances (dyslipidaemia, hypertension), (ii) structural remodeling, notably concentric left ventricular hypertrophy, (iii) impaired global longitudinal strain that can persist years after cessation, and (iv) premature coronary plaque burden linked to cumulative lifetime exposure. Furthermore, cardiac PET/CT studies indicate persistent coronary microvascular dysfunction, while registry cohorts confirm increased CV disease rates. Consequently, AAS use should be considered a major, under-recognized CV risk factor. We propose a phenotype-based assessment integrating non-judgmental history-taking, athlete-specific ECG interpretation, echocardiography with strain, and selective use of CMR and CCTA. Management should prioritize cessation support, harm reduction, and guideline-directed therapy tailored to the specific clinical phenotype (e.g., cardiomyopathy or coronary disease).

KEYWORDS

Anabolic-Androgenic Steroids, Cardiomyopathy, Coronary Atherosclerosis, Coronary Microvascular Dysfunction, Echocardiography, Cardiac MRI, Coronary CT Angiography, Sudden Cardiac Death, Recreational Athletes

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1. Introduction

The non-medical use of anabolic-androgenic steroids (AAS) is a global phenomenon that has increasingly shifted from elite sport to the general population and, in particular, to recreational strength training. A large meta-analysis estimated the global lifetime prevalence of AAS use at approximately 3.3% overall, with substantially higher prevalence in males than females [1]. In the United States, the best available estimates suggest millions of individuals have used AAS during their lifetime [2,34].

From a cardiovascular perspective, AAS use is clinically important for two reasons. First, users are often young or middle-aged and otherwise considered 'low risk' by traditional atherosclerotic cardiovascular disease (ASCVD) calculators, yet may present with myocardial infarction, cardiomyopathy, arrhythmias, or sudden cardiac death. Second, AAS exposure may act as a multi-pathway risk amplifier: it can worsen lipids and blood pressure, promote endothelial dysfunction and thrombosis, and trigger adverse myocardial remodeling [5-7,12-13,16-21].

Recent large register-based cohorts and contemporary imaging studies have strengthened the evidence base. In a Danish register cohort of men sanctioned for AAS use in fitness centers, AAS exposure was associated with a substantially increased risk of subsequent cardiovascular disease diagnoses [9]. Complementary cohort data demonstrate higher mortality and morbidity among sanctioned users compared with matched controls [3]. In parallel, mechanistic human studies using coronary CT angiography (CCTA) and cardiac PET/CT identify dose-dependent coronary plaque burden and persistent coronary microvascular dysfunction, even after cessation [10-11].

Research Objective. To synthesize contemporary evidence on mechanisms and clinical phenotypes of AAS-associated cardiovascular toxicity and to propose a pragmatic diagnostic approach relevant to sports cardiology and general practice.

2. Research materials and methods

This manuscript is a narrative review intended as a practical synthesis for academic and clinical use. A targeted literature search was performed in PubMed/MEDLINE for English-language publications from database inception through December 2025. Search terms were combined using Boolean logic and included: "anabolic androgenic steroid" OR "androgen abuse" OR "performance-enhancing drugs" AND "cardiomyopathy" OR "heart failure" OR "left ventricular hypertrophy" OR "coronary artery disease" OR "coronary plaque" OR "microvascular" OR "thrombosis" OR "echocardiography" OR "strain" OR "cardiac magnetic resonance" OR "coronary CT angiography" OR "PET".

We prioritized (i) cohort studies and register-based epidemiology with clinical outcomes; (ii) prospective observational studies capturing on-cycle and off-cycle changes; (iii) cross-sectional imaging studies (echocardiography/strain, CMR, CCTA, PET/CT) in recreational users; and (iv) high-quality guideline documents relevant to athlete evaluation and cardiovascular disease management [27-32]. Reference lists of key studies and reviews were screened to identify additional relevant publications [4-7,12,34-36].

Given the heterogeneity of exposure (agent type, dosing, duration, cycling patterns) and the frequent presence of polysubstance use and training-related confounders, we emphasize effect patterns consistently reproduced across independent cohorts rather than attempting pooled quantitative estimates.

2.1. Data sources and eligibility criteria

Data sources included PubMed/MEDLINE and major guideline repositories through December 2025. Eligible publications were English-language human studies or high-quality reviews addressing non-medical AAS exposure in recreational athletes or strength-trained individuals and reporting cardiovascular outcomes, imaging findings, vascular/hemostatic measures, or major clinical events. Therapeutic testosterone replacement studies were not a primary focus unless they provided context clearly distinct from supraphysiologic AAS exposure.

2.2. Search strategy and study selection

Search terms combined exposure keywords ("anabolic androgenic steroid", "androgen abuse", "performance-enhancing drugs") with phenotype and modality keywords ("cardiomyopathy", "heart failure", "left ventricular hypertrophy", "coronary artery disease", "coronary plaque", "microvascular", "thrombosis", "echocardiography", "strain", "cardiac magnetic resonance", "coronary CT angiography", "PET"). Key papers were additionally identified through citation chaining of seminal studies and screening reference lists of recent reviews and guideline documents.

2.3. Data collection and analysis / Statistical analysis

For included studies we extracted population characteristics, AAS exposure definition (current vs former use and cumulative duration when available), co-exposures when reported, and main cardiovascular endpoints. Evidence was synthesized qualitatively. No quantitative pooling was attempted because of heterogeneity in exposure characterization, study design, and outcomes.

2.3.1. Software

Standard word-processing and citation tools were used for manuscript preparation and reference management.

2.3.2. AI

AI tools were used solely to assist with language refinement and formatting under human supervision. Study selection, interpretation of evidence, and conclusions were determined by the authors. All AI-assisted edits were reviewed and revised to ensure factual accuracy and adherence to scientific writing standards.

2.3.3. Synthesis approach

We emphasize effect patterns reproduced across independent cohorts (dose–duration signals, convergent imaging findings, and outcome associations) rather than isolated case reports.

3. Research results

3.1. Epidemiology and patterns of use in recreational athletes

The epidemiology of AAS use is challenging to quantify because most exposure is illicit and often underreported. Nevertheless, multiple lines of evidence indicate that AAS use is not rare. A global meta-analysis reported a lifetime prevalence around 3.3% overall, and roughly 6% or higher among men in many settings [1]. In clinical practice, AAS users are typically males aged 20-40 years engaged in resistance training, bodybuilding, or combat sports [4-5].

Use patterns commonly involve cyclical exposure, frequent 'stacking' of multiple androgens and adjunctive compounds, and intermittent off-drug periods [4-5]. Importantly, polysubstance use (e.g., stimulants, recreational drugs) is common and may confound cardiovascular risk assessment [4-5]. These realities support a non-judgmental, harm-reduction-informed clinical approach that prioritizes engagement and monitoring rather than solely focusing on abstinence messaging [34-35].

3.2. Mechanisms of cardiovascular toxicity

1) Adverse cardiometabolic risk factor profile

AAS exposure can acutely and chronically worsen traditional ASCVD risk factors. The most consistently documented changes are profound suppression of HDL-cholesterol and increases in LDL-cholesterol, along with elevations in blood pressure [5-7,17,23]. Prospective data from the HAARLEM cohort and related work demonstrate that blood pressure, lipid parameters, and erythrocytosis may worsen during active AAS cycles and can partially normalize after cessation [12,23].

Hypertension and increased arterial stiffness have been reported in active and former AAS users [22]. These changes are clinically relevant because they increase LV afterload and may synergize with direct myocardial effects to promote hypertrophy, diastolic dysfunction, and eventual heart failure, particularly when combined with intensive resistance training [7,12-14].

2) Endothelial dysfunction and impaired vascular function

Endothelial dysfunction provides a mechanistic bridge between risk factor changes and clinical events. Early work demonstrated impaired flow-mediated, endothelium-dependent vasodilation among male bodybuilders using AAS [17]. More recent vascular studies report impaired vascular function in young AAS users, supporting persistent vascular injury signals beyond classic risk factors [18-21].

3) Prothrombotic milieu and altered fibrinolysis

Thromboembolic events in AAS users are biologically plausible given converging evidence of prothrombotic and antifibrinolytic changes. Laboratory studies show impaired fibrin clot lysis and abnormalities in coagulation/fibrinolysis in AAS users [24-26]. Erythrocytosis, which can occur during AAS exposure, may further increase viscosity and thrombosis risk, particularly in dehydrated athletes or those using concomitant agents that affect coagulation [5,23].

4) Direct myocardial remodeling, fibrosis, and dysfunction

Beyond risk factor mediation, AAS may exert direct myocardial effects through androgen receptor signaling and downstream pathways that influence myocyte hypertrophy, extracellular matrix remodeling, oxidative stress, and apoptosis [5-7,36]. Human imaging studies provide clinical correlates: AAS use is associated with increased LV mass and concentric remodeling, and with impairments in systolic function detectable by strain imaging even when LVEF is preserved [7,12-14].

Prospective echocardiographic data suggest that some structural changes are reversible after stopping AAS, yet functional abnormalities may persist in a subset of individuals [12,14]. Cardiac magnetic resonance (CMR) studies further demonstrate altered cardiac structure and function in long-term users [13], and contemporary cohorts continue to report measurable cardiac phenotype differences years after cessation [11,37].

5) Coronary atherosclerosis and microvascular dysfunction

Coronary artery pathology in AAS users likely reflects combined effects of dyslipidaemia, hypertension, endothelial injury, thrombosis propensity, and potentially direct vascular effects. CCTA studies in long-term users demonstrate higher coronary plaque burden and a relationship between cumulative exposure and severity of atherosclerosis [7,10].

Importantly, ischemic risk may not be limited to obstructive epicardial CAD. A cardiac PET/CT study found that myocardial flow reserve was more frequently impaired in both current and former AAS users than in controls, with accumulated duration of AAS exposure independently associated with reduced flow reserve among former users [11]. These findings support coronary microvascular dysfunction as a potential mechanistic contributor to early cardiomyopathy and ischemic symptoms in this population.

3.3. Clinical cardiovascular phenotypes

The clinical spectrum of AAS-associated CV toxicity is broad. Presentations range from asymptomatic risk factor abnormalities detected on screening to acute coronary syndromes, decompensated heart failure, or sudden death. Phenotypes frequently overlap and may evolve over time with ongoing exposure and/or after cessation.

For practical purposes, the following phenotype clusters can guide evaluation and risk stratification: (1) cardiometabolic and hematologic abnormalities; (2) coronary atherosclerosis and premature ischemic events; (3) coronary microvascular dysfunction; (4) cardiomyopathy/heart failure; (5) arrhythmias and sudden cardiac death; (6) venous and arterial thromboembolism.

1) Cardiometabolic and hematologic abnormalities

Marked dyslipidaemia (low HDL-C, elevated LDL-C and ApoB), elevated blood pressure, and erythrocytosis are common laboratory and clinical findings during AAS exposure [12,23]. These abnormalities can regress after discontinuation, but the degree and timeline of recovery vary across individuals and may depend on duration and intensity of exposure [12,23].

2) Coronary atherosclerosis and premature ischemic events

Premature coronary disease is increasingly recognized in AAS users. CCTA-based studies show greater coronary plaque volume in long-term users, including non-calcified plaque, and a dose-response relationship between cumulative AAS exposure and atherosclerotic burden [7,10]. In registry cohorts, sanctioned users have elevated risks of diagnosed cardiovascular disease, consistent with clinically meaningful event rates [9].

3) Coronary microvascular dysfunction

Microvascular dysfunction may represent an early, persistent mechanism linking AAS exposure to exertional symptoms and downstream myocardial injury. In a PET/CT study of recreational strength-trained men without established cardiovascular disease, both current and former AAS users exhibited a higher prevalence of impaired myocardial flow reserve compared with controls; among former users, the accumulated duration of AAS use remained independently associated with reduced flow reserve [11]. Clinically, this phenotype may manifest as exertional chest pain or dyspnoea with non-obstructive coronary arteries on CTA, or as reduced exercise tolerance disproportionate to structural findings.

4) Cardiomyopathy and heart failure

AAS-associated cardiomyopathy often combines concentric LV hypertrophy with varying degrees of systolic and diastolic dysfunction. Prospective echocardiographic data from the HAARLEM study demonstrate reversible LV hypertrophy and measurable cardiac dysfunction during an AAS cycle [12]. Cross-sectional imaging studies show that systolic dysfunction may persist in former users years after cessation [14], and CMR studies document structural and functional differences in long-term users [13].

In sports cardiology practice, reduced GLS can be an early marker of myocardial dysfunction even when LVEF is preserved [7,12-14]. When advanced disease is present, clinical heart failure management should follow contemporary guideline-directed medical therapy, while exercise recommendations require individualized risk assessment and shared decision making [28,31-32].

5) Arrhythmias and sudden cardiac death

Arrhythmia risk in AAS users is plausibly increased via multiple mechanisms: myocardial hypertrophy and fibrosis, microvascular ischemia, electrolyte disturbances, stimulant co-use, and autonomic effects. Large register-based cohorts demonstrate increased cardiovascular disease risk among sanctioned users [9], and observational reviews emphasize the association of AAS with myocardial infarction and sudden death in younger populations [7,38]. Because ECG findings in athletes require athlete-specific interpretation to avoid overdiagnosis and underdiagnosis, clinicians should apply international athlete ECG recommendations and escalate evaluation when abnormalities are present [27].

6) Venous and arterial thromboembolism

Thromboembolic disease may occur in AAS users through a combination of erythrocytosis, endothelial dysfunction, platelet activation, and impaired fibrinolysis [5,24-26]. Given the potential for concomitant risk factors (immobility, dehydration, stimulant use), evaluation should be comprehensive and management should follow standard thrombosis guidelines, with strong emphasis on identifying ongoing exposure and reversible risk factors.

3.4. A practical diagnostic approach

The diagnostic work-up should be individualized based on symptoms, duration/intensity of exposure, training history, and conventional risk factors. However, a structured approach can help clinicians avoid two common errors: (i) under-recognition of AAS exposure as a major CV risk modifier, and (ii) misclassification of pathologic findings as benign athlete's heart.

Step 0. Create conditions for disclosure

Many users anticipate stigma and may not disclose AAS exposure unless asked explicitly and non-judgmentally [4,34-35]. Practical strategies include normalizing questions ("Many strength athletes use supplements or hormones; have you ever used anabolic steroids or testosterone derivatives?"), asking about prior cycles, routes of administration, and concurrent agents, and documenting approximate cumulative exposure when possible [4-5].

Step 1. Identify high-risk presentations ("red flags")

Exertional chest pain, unexplained dyspnoea, syncope/presyncope, palpitations, or exercise intolerance in a young strength athlete.

Marked dyslipidaemia (especially very low HDL-C), hypertension, or erythrocytosis in the absence of other clear causes.

ECG abnormalities beyond expected athletic adaptation (e.g., pathologic Q waves, ST-segment depression, ventricular pre-excitation, prolonged QT, complete LBBB) [27].

Family history of premature sudden death or cardiomyopathy, which may interact with AAS exposure.

Step 2. Baseline evaluation

Baseline assessment should include: (i) focused cardiovascular history (including AAS and other substance use, training volume, and symptoms), (ii) physical examination with orthostatic vital signs, (iii) resting 12-lead ECG interpreted using international athlete criteria [27], and (iv) laboratory testing directed at common AAS-related abnormalities and differential diagnoses (lipid profile, Hb/Hct, liver enzymes, renal function, glucose/HbA1c, high-sensitivity troponin and natriuretic peptides when clinically indicated, and thyroid tests if symptom-driven) [5,12,23].

Step 3. Cardiac imaging and escalation

Transthoracic echocardiography is the first-line imaging test. It should assess LV wall thickness, cavity size, diastolic function, RV size/function, and should include speckle-tracking strain when available (GLS). Findings suggestive of pathology (disproportionate LV hypertrophy, reduced GLS or LVEF, RV dysfunction, regional wall motion abnormalities, or unexplained diastolic impairment) should prompt further evaluation. CMR is useful to characterize remodeling, quantify function, and detect fibrosis/scar (late gadolinium enhancement), which may influence arrhythmia and exercise recommendations [13,28].

For ischemic evaluation, choice of testing depends on pretest probability and the clinical question. In symptomatic users and those with long cumulative exposure, coronary artery assessment with CAC scoring and/or CCTA can identify subclinical and clinical plaque burden and may assist risk stratification [7,10]. When symptoms persist despite non-obstructive CCTA, microvascular dysfunction can be considered; PET/CT-based myocardial flow reserve measurement provides reference-standard assessment in specialized centers [11].

Table 1. Practical phenotype-based work-up for suspected AAS-associated cardiovascular toxicity

Clinical scenario / phenotype	Key questions & findings	Suggested tests (stepwise)	Notes
Asymptomatic screening	Cumulative exposure; cycling patterns; other substances; BP, lipids, Hb/Hct	History & exam; athlete-ECG [27]; labs (lipids, Hb/Hct) [12,23]; echo + GLS	Agree on follow-up interval; counsel cessation/harm reduction [34-35].
Exertional chest pain or dyspnoea	Angina pattern; triggers; stimulant co-use; BP; very low HDL-C	ECG + echo; CAC/CCTA if CAD suspected [7,10]; stress imaging; consider PET MFR if non-obstructive CCTA and symptoms persist [11]	Treat ASCVD risk factors; consider non-cardiac causes.
Unexplained LV hypertrophy or reduced GLS/LVEF	Disproportionate LVH; systolic/diastolic dysfunction; symptoms	Echo + GLS; CMR for phenotype/fibrosis [13]; BNP/troponin if indicated; Holter if palpitations/syncope	Differentiate athlete's heart vs cardiomyopathy; consider deconditioning [28].
Palpitations, syncope, or high-risk ECG	Symptoms at rest/exertion; family history; abnormal athlete ECG	ECG [27]; echo; ambulatory monitoring; exercise test; CMR if structural disease suspected [13,28]	Return-to-play and restriction decisions per sports-cardiology guidance [28].
Thromboembolic presentation	VTE symptoms; erythrocytosis; dehydration; injection-related complications	Standard VTE pathway (D-dimer/imaging); Hb/Hct; consider coagulation/fibrinolysis assessment [24-26]	Manage per thrombosis guidelines; address ongoing exposure and reversible risks.

3.5. Management considerations

Management should be individualized and should avoid inadvertently encouraging continued AAS use. At minimum, clinicians should provide clear education on cardiovascular risks, assess readiness to stop, and offer support for cessation. Harm-reduction-based communication can improve disclosure, follow-up, and opportunities to treat modifiable risk factors [34-35].

Risk factor management should follow contemporary cardiovascular prevention guidance, recognizing that AAS exposure may shift a young athlete into a higher-risk category than suggested by age alone. Interventions typically include strict blood pressure control, aggressive lipid management (including consideration of statin therapy when clinically indicated), smoking cessation, and management of sleep apnea and other comorbidities [28-30].

For cardiomyopathy or heart failure, standard guideline-directed medical therapy (GDMT) should be initiated promptly when diagnostic criteria are met, with careful attention to exercise prescription and return-to-sport decisions [28,31-32]. For coronary disease, management should follow chronic coronary disease and acute coronary syndrome frameworks, with special attention to the possibility of microvascular ischemia in younger patients with non-obstructive coronary arteries [11,28].

Arrhythmia management should follow standard pathways (risk stratification, rhythm monitoring, and electrophysiology referral when indicated). Exercise restrictions and clearance should incorporate the athlete's underlying phenotype, imaging findings (including fibrosis/scar on CMR), and shared decision making as recommended by sports-cardiology guidelines [28].

3.6. Limitations of the evidence base

Current evidence is limited by heterogeneity in AAS exposure (agents, dose, cycling), reliance on self-report, and selection bias (e.g., recruiting through gyms or clinical referrals). Resistance training itself produces adaptive remodeling, complicating differentiation between athlete's heart and pathologic hypertrophy. Polysubstance use is common and can confound associations with outcomes [4-5].

Nevertheless, the convergence of prospective on-cycle/off-cycle observations [12,23], advanced imaging studies identifying persistent myocardial and coronary abnormalities [10-11,13-14], and large register-based outcome data [3,9] supports the clinical relevance of AAS exposure as a cardiovascular risk modifier.

4. Discussion

The evidence base on AAS-associated cardiovascular toxicity has expanded from case reports to include prospective on-cycle/off-cycle observations, multimodality imaging studies, vascular and hemostatic investigations, and national registry analyses. Taken together, these data support a spectrum of harm that includes accelerated atherosclerosis, coronary microvascular dysfunction, maladaptive cardiac remodeling, arrhythmias, and thromboembolic events, sometimes manifesting in individuals who would otherwise be classified as low cardiovascular risk due to young age.

A recurring challenge is disentangling physiological training adaptation from pathological remodeling. Intensive resistance training can increase left ventricular wall thickness and mass; however, concentric hypertrophy accompanied by impaired GLS, reduced LVEF, unexplained diastolic dysfunction, or CMR evidence of scar/fibrosis should shift the diagnostic framing toward pathology and prompt closer follow-up and risk stratification [13-14,27-28].

Coronary evaluation should similarly reflect the full spectrum of ischemic mechanisms. In addition to dose–duration associations between cumulative AAS exposure and coronary plaque burden on CCTA [10], PET-based studies reporting impaired myocardial flow reserve years after cessation suggest that microvascular dysfunction may persist and contribute to symptoms even when epicardial stenoses are absent [11]. This has practical implications for the choice of diagnostic testing and for clinical counseling in symptomatic former users.

From a prevention perspective, AAS exposure can be viewed as a risk amplifier that worsens traditional risk factors (e.g., dyslipidaemia and hypertension) and may independently contribute to myocardial injury. Therefore, management should combine a nonjudgmental, engagement-focused approach to improve disclosure with aggressive guideline-directed treatment of the dominant phenotype (lipids, blood pressure, coronary disease, heart failure, arrhythmias, or thromboembolism) [28-32,34-35].

Key research gaps remain. Future studies should standardize exposure characterization, better account for polysubstance use and lifestyle confounding, include women and diverse populations, and clarify the degree and time course of reversibility after cessation. Prospective longitudinal cohorts linking imaging phenotypes to hard outcomes would be particularly valuable for informing follow-up intervals and return-to-exercise recommendations.

5. Conclusions

Non-medical AAS exposure in recreational athletes is associated with adverse cardiometabolic profiles, vascular dysfunction, prothrombotic changes, myocardial remodeling, and both macrovascular and microvascular coronary pathology. Evidence from imaging studies and register cohorts indicates that risk can persist beyond cessation and may be dose-dependent [9-14].

Clinicians evaluating strength athletes should routinely consider and inquire about AAS exposure. Applying athlete-specific ECG interpretation standards [27], echocardiography with strain, and selective use of CMR and CCTA can facilitate early detection and tailored management. A harm-reduction-informed, non-judgmental approach improves disclosure and enables prevention and treatment of cardiovascular disease in this underserved population [34-35].

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