Short Editorial

A New Frontier in Cardiovascular Prevention: Beyond Prohibition, Clinical Management of Anabolic Steroid Users

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Short Editorial related to the article: Anabolic-Androgenic Steroids and Acute Myocardial Infarction in Young Adults: A Literature Review Based on a Case Series

The traditional approach to Anabolic-Androgenic Steroid (AAS) use has been largely driven by prohibition and stigma. However, with prevalence rates reaching as high as 31.6% in specific populations in Brazil,[1] it has become imperative for the medical community to shift its paradigm: moving from a reactive response to acute events toward a strategic, vigilant, and proactive approach to AAS users. The current challenge is not merely to discourage use, but to identify subclinical cardiovascular disease at an early stage in young individuals who, despite an appearance of robust health, may harbor potentially fatal substrates for Acute Myocardial Infarction (AMI).

Subclinical Diagnosis: The Role of Advanced Echocardiography

Conventional cardiac assessment often fails to detect early damage, as Left Ventricular Ejection Fraction (LVEF) may remain within normal limits despite established myocardial injury. The literature highlights that AAS users exhibit alterations in cardiac geometry and function, including pathological hypertrophy, diastolic dysfunction, and biventricular impairment.[2,3]

In this setting, echocardiography incorporating advanced measurements such as myocardial strain has emerged as an essential diagnostic tool. Although LVEF may remain preserved during the early stages, myocardial deformation (strain) analysis enables the detection of incipient systolic dysfunction resulting from fibrosis and myofibrillar destruction caused by the direct toxic effects of androgens. Recent studies have demonstrated that both current and former AAS users may exhibit persistent biventricular cardiomyopathy,[4] reinforcing the need for continuous monitoring.

Unmasking Occult Dysfunction: Exercise Stress Echocardiography with Global Longitudinal Strain

If resting evaluation is already insufficient to detect early cardiomyopathy in AAS users, exercise stress echocardiography (performed on a treadmill or cycle ergometer) adds a critical diagnostic dimension by assessing contractile reserve and hemodynamic response under conditions that mimic the physiological demands of resistance training and competitive athletic activity. Unlike pharmacologic stress testing, exercise-based stress reproduces the real-world conditions under which these individuals frequently develop symptoms — atypical chest pain, disproportionate dyspnea, presyncope, or unexplained declines in performance — and, in this context, abnormalities concealed at rest often become evident.[5]

The integration of exercise myocardial strain imaging (global and regional longitudinal deformation acquired at peak exercise or immediately during recovery) represents a major advancement in this evaluation. In healthy hearts, a progressively increased Global Longitudinal Strain (GLS) is expected during exercise, reflecting preserved contractile reserve. In AAS users, even when resting LVEF and GLS remain within normal ranges, attenuation or reversal of the expected GLS augmentation under stress is frequently observed, along with regional heterogeneity — findings consistent with subclinical interstitial fibrosis, microvascular ischemia, and early exhaustion of myocardial reserve.

This pattern is particularly valuable for three reasons. First, it provides an early functional marker of androgen-induced cardiomyopathy, capable of identifying the transition from adaptive hypertrophy to pathological remodeling before overt clinical manifestations develop. Second, it allows for arrhythmogenic risk stratification, as regions exhibiting abnormal strain during exercise often correspond to electrically unstable substrates — a critical consideration in a population with an elevated incidence of sudden cardiac death. Third, it offers an objective and reproducible endpoint for longitudinal follow-up, enabling assessment of reversibility after AAS discontinuation and evaluation of therapeutic response to interventions such as Renin-Angiotensin-Aldosterone System (RAAS) blockade and intensive lipid-lowering therapy.

In clinical practice, stress echocardiography with GLS should be considered for symptomatic users, individuals with borderline abnormalities on resting studies (GLS at the lower limit of normal, concentric hypertrophy, or early diastolic dysfunction), and as a pre-participation screening tool for strength athletes with a current or prior history of AAS use. The combination of functional capacity data, blood pressure response to exercise, electrocardiographic findings, and regional myocardial deformation under stress

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provides a substantially more sensitive risk profile than any single modality alone.

Myocardial Work: Refining the Assessment of Steroid-Induced Cardiotoxicity

One important limitation of GLS is its dependence on afterload. In a population in which systemic hypertension and sustained elevations in systolic blood pressure during exercise are almost the norm — as is often the case among chronic AAS users — a reduction in GLS may underestimate true myocardial injury or, conversely, may reflect hemodynamic status more than underlying myocardial damage. It is precisely within this gap that myocardial work analysis derived from GLS finds its most elegant application.

This method integrates the longitudinal strain curve with an estimated left ventricular pressure curve (derived from noninvasive brachial blood pressure measurements), generating a pressure–strain loop from which four indices are derived: Global Work Index (GWI), Global Constructive Work (GCW) — the energy effectively converted into ventricular ejection — Global Wasted Work (GWW) — energy dissipated through out-of-phase shortening and lengthening — and Global Work Efficiency (GWE). By incorporating afterload into the analysis, these indices provide a functional assessment that is relatively independent of hemodynamic conditions, overcoming one of the major methodological limitations of GLS alone in hypertensive and hypertrophic patients.

Within the specific context of AAS-related cardiotoxicity, this refinement is particularly relevant. Riou and colleagues[6] compared strength athletes using AAS, athletes with Hypertrophic Cardiomyopathy (HCM), and healthy athletic controls. Although both hypertrophic groups demonstrated reduced longitudinal strain, GWE was significantly lower in both AAS users and HCM patients compared with controls (approximately 90% versus 93%). Even more noteworthy was the regional pattern identified: in AAS users, abnormalities in constructive work and efficiency were predominantly localized to the basal septal segments, whereas in HCM, impairment involved both septal and apical segments. This finding provides an additional diagnostic perspective: regional myocardial work mapping may help differentiate androgen-induced toxic hypertrophy from genetically mediated hypertrophy, a clinically critical differential diagnosis in athletes presenting with septal thickening.

From a pathophysiological standpoint, reduced efficiency and increased wasted work reflect segmental dyssynchrony and subclinical interstitial fibrosis resulting from the direct toxic effects of androgens on cardiomyocytes, including myofibrillar destruction, increased collagen synthesis, and electrical remodeling that predisposes individuals to sudden cardiac death.[7] Myocardial work, therefore, represents a marker that integrates functional, mechanical, and potentially prognostic information within a single tool, readily incorporated into resting echocardiographic protocols and, in more advanced centers, into stress echocardiographic evaluations as well.

Accordingly, incorporating myocardial work analysis into the assessment of AAS users should be viewed as a natural refinement step of strain-based evaluation: it adds pathophysiological specificity, reduces the confounding influence of afterload, and provides regional information that may guide differential diagnosis and, potentially, therapeutic monitoring. It should be emphasized, however, that myocardial work remains a promising and physiologically elegant tool, but it has not yet been fully established as a dominant clinical marker.

Myocardial work assessment through strain imaging — both at rest and during stress testing — represents a promising yet still evolving area of investigation in the characterization of the cardiac effects of AASs. Significant gaps remain regarding the establishment of population-specific reference values, the definition of prognostically validated cutoff points, the reversibility of abnormalities following cessation of AAS use, and the impact of therapeutic interventions on myocardial deformation over time. Advancing this field will require robust multicenter longitudinal studies capable of validating the clinical applicability of the method and supporting structured protocols for prevention, early diagnosis, prognostic improvement, and therapeutic monitoring in this unique patient population.

Active Detection of Atherosclerosis: Coronary CT Angiography

The notion that young AAS users are free from atherosclerosis is a dangerous myth. AAS accelerates atherosclerosis through profound disruption of lipid metabolism, increasing Low-Density Lipoprotein (LDL) cholesterol via hepatic lipase activity while simultaneously reducing High-Density Lipoprotein (HDL) cholesterol.[2] Even more concerning is the presence of perivascular inflammation, which may occur even in individuals with very low body fat percentages.[8]

Coronary CT Angiography (CCTA) should be considered as part of an active screening strategy for coronary artery disease in these patients. The CRISP-CT study demonstrated that the Fat Attenuation Index (FAI) measured on CCTA can detect coronary inflammation even before the development of obstructive plaques. Given that AAS users exhibit increased perivascular inflammation and endothelial oxidative stress,[8] CCTA provides substantially more precise risk stratification than traditional clinical risk scores, enabling early identification of patients at risk for plaque rupture or endothelial erosion.

Aggressive Treatment and Therapeutic Alliance

Once risk has been identified, management of comorbidities should be aggressive and multifactorial. Intervention cannot be limited solely to discontinuation of AAS use, which often requires support from a multidisciplinary team due to body image disturbances and anxiety-related disorders. Clinical management should include:

- Rigorous Lipid Control: Use of high-intensity statin therapy to mitigate accelerated atherosclerosis and endothelial dysfunction.

- Hypertension and RAAS Management: AAS use leads to hyperactivation of the RAAS, promoting fibrosis and hypertrophy. Pharmacologic blockade of this system is critical to preventing cardiomyopathy progression.
- Prothrombotic Surveillance: Given the hypercoagulable state associated with increased levels of coagulation factors II, IX, and XI, ongoing assessment of thrombotic risk is warranted.[9]

In summary, physicians' approach to AAS users should be grounded in the science of prevention. By leveraging technologies such as resting and exercise strain imaging, stress echocardiography with GLS, and CCTA, clinicians no longer need to wait for a myocardial infarction to occur before intervening. Instead, they can act during the silent phase of disease, when pathology is already present but still amenable to treatment.

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