

## **Letter to the Editor**

### **Cardiac Dysfunction and Recovery After Anabolic-Androgenic Steroid Abuse: Is Reversibility Possible?**

**Dear Editor,**

Observational evidence has demonstrated that chronic abuse of anabolic-androgenic steroids (AAS) can lead to significant cardiovascular damage, including left ventricular hypertrophy, systolic and diastolic dysfunction, and progression to severe heart failure [1-3]. Some authors have reported that cessation of AAS use, combined with appropriate diagnosis and pharmacological treatment of cardiomyopathy, may reverse the deleterious morphological and functional effects to normal parameters [4-8].

Given the high prevalence of AAS abuse, considered by some to be a public health issue, combined with the increased morbidity and mortality, particularly due to cardiovascular causes, accurately mapping these actual effects to enable precise interventions could have a significant impact on population health.

Thus, the objective of this letter is to provide a brief overview of observational evidence and case reports regarding findings on the potential reversibility of cardiac dysfunction resulting from the abusive use of anabolic-androgenic steroids (AAS).

For instance, Milevski SV. et al. (2022) [4] described a case involving a 46-year-old man diagnosed with severe dilated cardiomyopathy (LVEF 12%) after ten years of prolonged AAS abuse. Six months after discontinuing AAS use and initiating heart failure treatment, combined with testosterone replacement therapy to physiological levels, the patient experienced complete symptom reversal, with LVEF improving to 61%.

In another case report by Doleeb S. et al. (2019) [5], a male bodybuilder with a history of chronic AAS abuse for over three years presented with symptoms of dyspnea, fatigue, palpitations, and syncope at the time of dilated cardiomyopathy diagnosis, along with an LVEF of 12%. Pharmacological treatment for heart failure and cessation of AAS use led to symptom remission and an LVEF recovery to 54% after six months of clinical follow-up.

Gul U. et al. (2022) [6] reported a case involving a 47-year-old non-athlete woman who had used supraphysiological doses of AAS over an extended period. She presented with severe pulmonary edema and left ventricular dysfunction (LVEF 34%) at the time of cardiomyopathy diagnosis. Following AAS discontinuation and clinical pharmacological treatment for heart failure, the patient experienced regression of left ventricular hypertrophy (with no evidence of fibrosis) and an LVEF improvement to 57%, with no residual clinical symptoms.

Additionally, two observational studies [7,8] have also documented that the recovery of morphological and functional parameters (left ventricular hypertrophy and systolic and diastolic dysfunction) may be potentially reversible, reaching values considered normal after cessation of chronic AAS abuse and initiation of pharmacological treatment for heart failure over a period of 6 to 8 months. However, studies evaluating outcomes beyond the six-month to eight-month recovery period could offer a better insight into the durability of observed reversibility.

Nevertheless, the reversibility of cardiac damage caused by AAS abuse is not universally reported across studies. The systolic (reduced LVEF and longitudinal strain) and diastolic dysfunction (reduced E/A ratio) induced by AAS observed in the study by Baggish AL. et al. (2010) [1] did not resolve even after six months of discontinuation. Similarly, Abdullah R. et al. (2024) [2], evaluating former AAS users six years after cessation, and Rasmussen JJ. et al. (2018) [3], assessing individuals 30 months post-discontinuation, observed persistent left ventricular hypertrophy, reduced LVEF, and impaired global longitudinal strain, suggesting potential permanent cardiac damage.

**Keywords:** Anabolic-Androgenic Steroids, Steroid Abuse, Heart Damage, Cardiac Dysfunction

**Conclusion:** Substantial observational evidence indicates that chronic AAS abuse promotes left ventricular hypertrophy and progressive diastolic dysfunction, potentially advancing to severe systolic dysfunction and heart failure [1-3]. While some studies suggest partial or complete reversibility of functional alterations following cessation [4-8], others report persistent damage even years after discontinuation, such as sustained left ventricular hypertrophy and reduced global longitudinal strain [1-3]. These discrepancies may suggest that the extent and persistence of cardiac damage could be

related to the cumulative AAS abuse burden (dose and duration), individual factors potentially not captured in observational studies or case reports, or even the simple confounder of different methodologies (MRI, Echocardiography) applied in the cited studies.

Another factor that needs to be clarified in the future is the influence of potential confounders in these findings, which are almost always present in the context of AAS abuse [9]. These include the concurrent abuse of other legal and illegal substances, polypharmacy, and the use of low-quality drugs from the black market [9,10]. This highlights the need for future controlled studies, and for instance, close cardiological follow-up to enable early detection and appropriate therapeutic management in affected individuals.

#### **DISCLAIMER (ARTIFICIAL INTELLIGENCE):**

The authors declare that generative AI was used solely during the final stage of manuscript preparation (post-writing) and exclusively for linguistic refinement in the English language (Name: ChatGPT; Version: GPT-4; Model: OpenAI's Large Language Model; Source: OpenAI - <https://openai.com>). No original text was generated or substantively edited by the AI.

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