Letter to the Editor

Can AAS-Induced Cardiac Dysfunction Be Reversible?

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].Conversely, some authors have reported that, following an appropriate diagnosis of cardiomyopathy, cessation of AAS use, and initiation of clinical pharmacological treatment, the deleterious morphological and functional effects may be reversible to normal parameters [4-8].

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.

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.

In summary, substantial 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), as well as individual factors potentially not captured in observational studies or case reports. This underscores the importance of a close cardiological follow-up, aiming early detection of alterations, and appropriate therapeutic management in individuals exposed to these compounds.

References:

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