







Detrimental effects of performance-enhancing drugs on the heart: a case report of anabolic steroid induced cardiomyopathy

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Introduction: Synthetic anabolic androgenic steroids (AAS), compounds mimicking the action of endogenous testosterone in enhancing training performance, have been extensively studied during the last century. AAS abuse has become a major public health concern with an estimated worldwide lifetime prevalence of 1–5%¹. Long-term administration of AAS in supraphysiological doses may have detrimental effects on the cardiovascular system, presumably through direct action on cardiac myocyte androgen receptors. In severe cases, life-threatening conditions such as myocardial infarction, aortic dissection or cardiomyopathy, particularly dilated cardiomyopathy as the most common form, may occur. Hereby, we report a rare case of AAS-induced cardiomyopathy with an emphasis on the multidisciplinary approach.

Case report: 46-year-old male bodybuilder presented with exercise intolerance unrelated to maximum training load and post-workout water retention 6 weeks before the visit. History revealed previous administration of testosterone enanthate 500 mg every 8 to 12 days during the period of 4 years. After a month-long cessation, he started taking testosterone undecanoate 1000 mg in 6-week intervals. The cardiorespiratory part of the physical examination showed normal findings and blood pressure of 125/80 mmHg. The patient was of athletic build with no signs of increased hairiness and no palpable testicular mass. An electrocardiogram showed a normal electrical axis and sinus bradycardia. Laboratory assessment (**Table 1**) was followed by echocardiography which was in accordance with the diagnosis of AAS-induced cardiomyopathy (**Figure 1**). Further diagnostic assessment of osteoporosis, hepatic, renal and psychological complications was performed. Conclusion: Long-term administration of AAS with unknown pharmacokinetic and pharmacodynamic properties should be considered as a cause of newly diagnosed cardiomyopathy, especially in previously healthy individuals with an athletic background.

TABLE 1. Laboratory evaluation revealed unmeasurably high testosterone levels with a subsequent suppression of the pituitary-testicular axis.

Pituitary-testicular axis		Cardiac markers		Liver markers	
Testosterone	> 52.05 nmol/l	Troponin I	18 ng/l	AST	57 U/l
SHBG	65.54 nmol/l	hs-Troponin T	13 ng/l	ALT	71 U/l
FSH	<0.1 IU/l	NTproBNP	48 ng/l		
LH	<0.1 IU/l	CK; CK-MB	317 U/l; 7.9 µg/l		

ALT, alanine aminotransferase; AST, aspartate aminotransferase; CK, creatine kinase; FSH, follicle-stimulating hormone; hs, high sensitivity; LH, luteinizing hormone; MB, myocardial band; SHBG, sex-hormone binding globulin.

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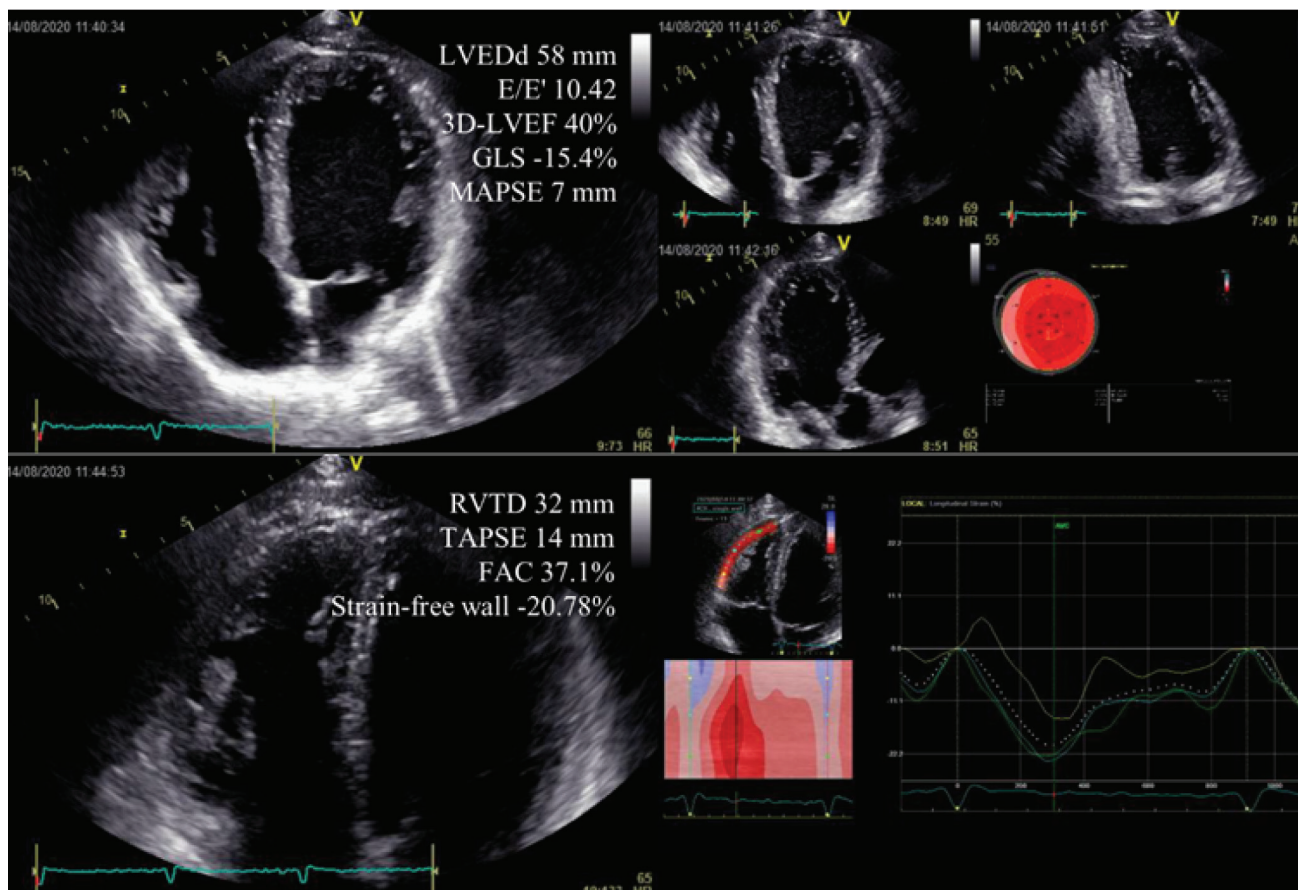


FIGURE 1. Echocardiographic assessment of anabolic steroid-induced cardiomyopathy. Representative echocardiogram images for the left ventricle (upper panel), and the right ventricle (lower panel) with corresponding values. Transthoracic echocardiography was performed using Vivid E95 Cardiac Ultrasound (GE Healthcare, Chicago, IL, USA).

3D-LVEF, three-dimensional left ventricular ejection fraction; E/E', early mitral inflow velocity to early diastolic mitral annulus velocity ratio; FAC, fractional area change; GLS, global longitudinal strain; LVEDd, left ventricular end-diastolic diameter; MAPSE, mitral annular plane systolic excursion; RVTD, right ventricular transverse diameter; TAPSE, tricuspid annular plane systolic excursion.

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