CLINICAL IMAGE

Disastrous effects of anabolic steroid abuse: a peculiar case of heart failure and toxic hepatitis in a young bodybuilder

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The use of anabolic–androgenic steroids (AAS) by professional athletes is a recurring problem in the media.[1] However, that might just be the tip of the iceberg, as most of the users are recreational athletes.[2] In addition to the desired effects on the skeletal muscle, these substances can also negatively affect various organs and systems.[3-5]

A 32-year-old male bodybuilder, without any risk factors, who has been training weightlifting for more than 5 years, reported for a routine medical examination with nonspecific symptoms. Chest X-ray revealed slight heart enlargement. Electrocardiography (ECG) as well as stress ECG test results were normal. He confessed to AAS abuse over the last 4 years; the drugs were self-administered in cycles of 10 weeks, with a 4-week interval between the cycles. The AAS most frequently used by the patient were testosterone enanthate, testosterone propionate, boldenone, stanozolol, trenbolone enanthate, and methandienone (FIGURE 1A and 1B). Echocardiography showed mild left ventricular end-diastolic diameter (LVEDD) enlargement (63 mm) and mildly reduced left ventricular

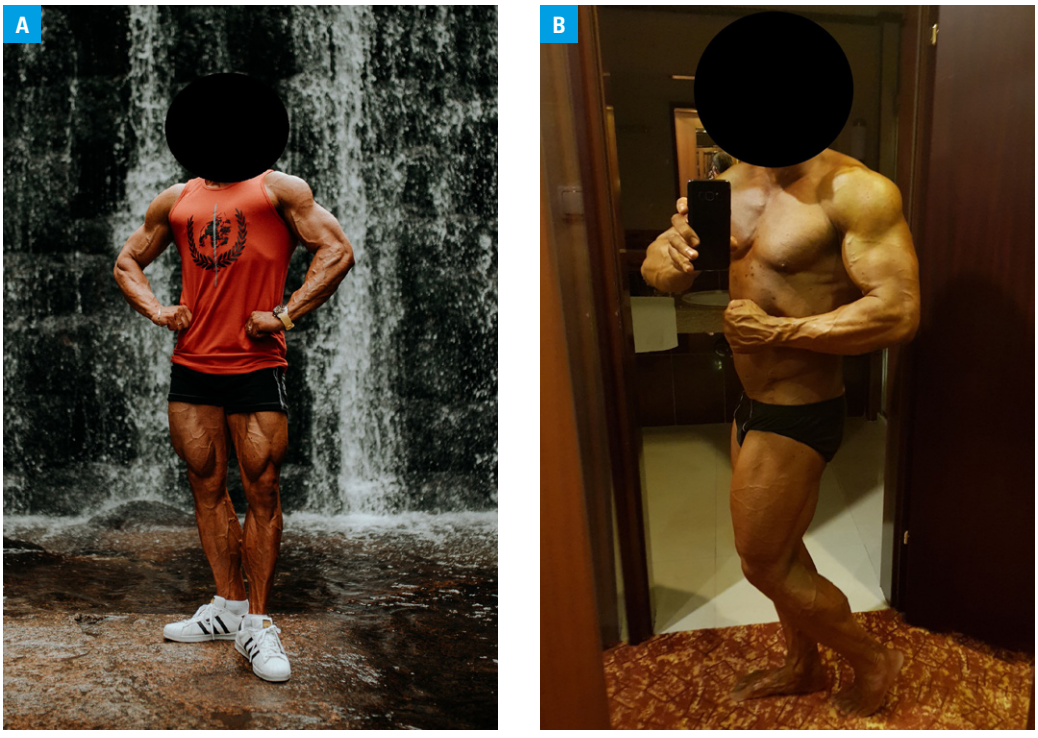

FIGURE 1 Physical presentation and imaging results of a 32-year-old bodybuilder; **A**, **B** – physical silhouette of the patient

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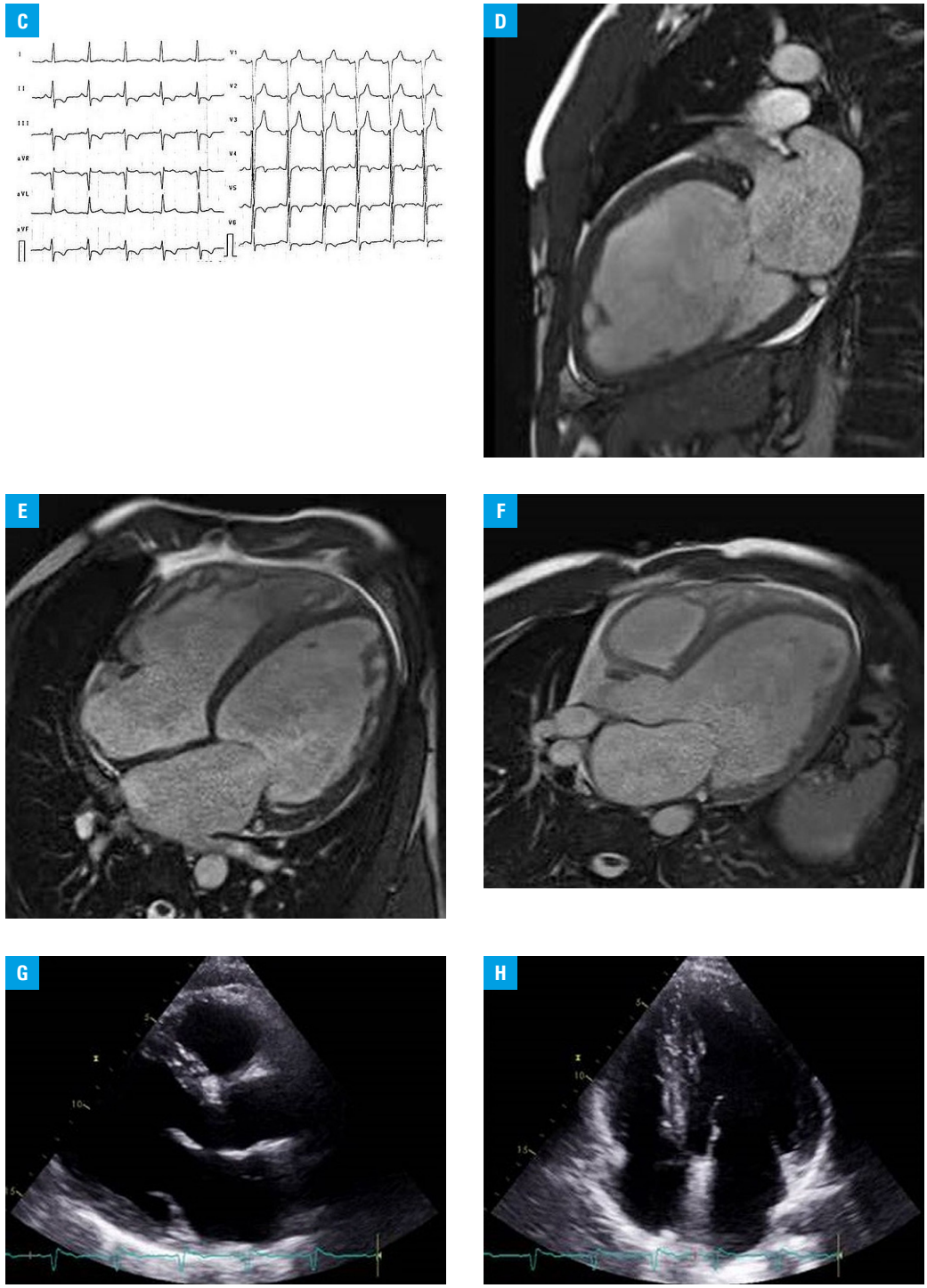

FIGURE 1 Physical presentation and imaging results of a 32-year-old bodybuilder; **C** – electrocardiogram showing left ventricular hypertrophy and inverted T waves in leads II, III, aVF, and V4–V6; **D**–**F** – cardiac magnetic resonance imaging showing dilated cardiomyopathy with left ventricular end-diastolic diameter (LVEDD) up to 90 mm and significantly reduced left ventricular ejection fraction (LVEF); **G**, **H** – follow-up echocardiography showing improvement of LVEDD (63 mm) and LVEF (40%)

ejection fraction (LVEF) of 49%. The patient was recommended to stop AAS abuse and reduce intensive weightlifting.

After 18 months he was admitted to the hospital with exertional dyspnea and symptoms of class II heart failure (HF) according to the New York Heart Association (NYHA) classification. Echocardiography revealed substantial LVEDD enlargement of 78 mm, low LVEF of 25%, and global hypokinesis. ECG presented left ventricular hypertrophy and inverted T waves in leads II, III, aVF, and V4–V6 (FIGURE 1C). Laboratory test results revealed significantly elevated levels of N-terminal pro–B-type natriuretic peptide (2288 pg/ml; reference range [RR], 0–125 pg/ml), troponin I (42.2 pg/ml; RR, 0–14 pg/ml), creatine kinase (583.8 U/l; RR, 0–190 U/l), and serum transaminases, aspartate aminotransferase (AST; 45.8 U/l; RR, 5–34 U/l) and alanine aminotransferase (ALT; 106.90 U/l; RR, 0–55 U/l). Hormone

level analysis showed elevated levels of testosterone (>15 ng/ml; RR, 2.8–8.0 ng/ml) and estradiol (273 pg/ml; RR, 11–44 pg/ml), which proved that the patient had been continuing steroid abuse. Guideline-directed medical therapy with ramipril, spironolactone, carvedilol, and furosemide was initiated, and the patient was again strongly advised to stop using anabolic steroids and reduce physical activity.

Two years later he was readmitted with symptoms of HF deterioration (NYHA class IV), pulmonary congestion, dyspnea at rest, severe edema of the legs, and jaundice. Laboratory test results revealed liver dysfunction (ALT, 211 U/l; AST, 77 U/l). Moreover, since the patient had not stopped taking AAS, hormone level analysis again showed elevated levels of testosterone (>15 ng/ml), prolactin (29.73 ng/ml; RR, 3.46–19.4 ng/ml), and estradiol (522 pg/ml). An additional diagnosis of steroid-induced toxic hepatitis was made. Coronary computed tomography angiography showed no abnormalities. Echocardiography and cardiac magnetic resonance imaging revealed dilated cardiomyopathy with LVEDD up to 90 mm and LVEF of 10% (**FIGURE 1D–1F**). The decision to insert an implantable cardioverter-defibrillator was made. The patient declared a definitive stop of AAS intake. Pharmacological therapy for HF with sacubitril/valsartan, eplerenone, carvedilol, torsemide, and dapagliflozin was initiated and heart transplantation was considered.

A 2-month follow-up showed only slight improvement of the patient's condition (LVEDD, 87 mm; LVEF, 15%). However, in the following 9 months, HF symptoms reduced to NYHA class II, the liver function stabilized, LVEDD decreased to 63 mm, and LVEF increased to 40% (**FIGURE 1G** and **1H**).

We present a rare case of AAS abuse resulting in a life-threatening clinical condition with severe HF and toxic hepatitis. However, this case shows that after AAS discontinuation and implementation of optimal medical treatment, clinical improvement is possible. This report highlights the potential catastrophic effects of AAS misuse and reinforces the warning against the use of these drugs.

ARTICLE INFORMATION

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