Acute Myocardial Infarction in a Young Bodybuilder: A Case Report and Review of the Literature

Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
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Conflict of interest: None declared

Patient:	Male, 26-year-old
Final Diagnosis:	Myocardial infarction
Symptoms:	Chest pain
Medication:	—
Clinical Procedure:	—
Specialty:	Cardiology • Endocrinology and Metabolic

Objective: Patient complains/malpractice

Background: Misuse of androgenic anabolic steroids (AAS) is a current practice associated with vigorous bodybuilding for muscular hypertrophy, especially among gym practitioners and bodybuilders, influenced by the culture of body image. In addition to liver, psychiatric, genital, urinary, dermatological, and musculoskeletal complications, AAS misuse reportedly can lead to development of cardiovascular complications, such as hypertension, dyslipidemia, cardiac hypertrophy, and early coronary disease, and potentially acute myocardial infarction (AMI) and sudden death.

Case Report: A 26-year-old male farmer who was also an amateur bodybuilder developed an extensive Killip Class I AMI in the anterior wall while using AAS. A few days before the acute event, his lipid and hormone levels were measured and found to be significantly elevated. The patient was asymptomatic after left anterior descending branch angioplasty, but he had significant electrocardiographic sequelae and ventricular dysfunction.

Conclusions: We describe the case of a young male bodybuilder using AAS who presented with AMI and was treated with primary angioplasty. Documentation of high levels of lipids and hormones 1 week before the acute event suggests some relationship between AAS and cardiovascular disease. The main effects of using these steroids on the cardiovascular system are reviewed. It is time for a new global warning about the risks of misusing AAS to obtain muscle hypertrophy. Based on current medical knowledge, these hormones should not be prescribed without a clear indication for their use.

MeSH Keywords: Anabolic Agents • Androgens • Myocardial Infarction • Somatotypes • Young Adult

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Background

Use of androgenic anabolic steroids (AAS) is widespread among amateur athletes, and especially bodybuilders, to increase muscular mass with the aim of improving their performance, but the serious health effects are often overlooked [1,2]. Many individuals try anabolic androgens because of dissatisfaction with their own body image. Factors that may contribute to the dissatisfaction include not measuring up to the "ideal standards" perpetuated in mass media, fear of being diminished or embarrassed by peers, the symbolic capital of being a musclebound person, and the perception of being able to gain muscle mass easily with the drugs [3]. Factors that compromise healthy construction of masculinity also have been mentioned as a cause for the search for a quick way to develop muscles [4]. In young adults, concomitant use of AAS has been associated with early atherosclerosis and acute myocardial infarction (AMI), among other health problems [1,5,6]. When a young patient is diagnosed with chronic or acute coronary disease, one of the principal suspects should be the misuse of AAS [7]. This paper presents a case and review of the literature.

Case Report

A 26-year-old male farmer and amateur bodybuilder was admitted to the hospital with severe pain in his left hemithorax, which had begun about 2 hours earlier. The patient had been treated for a peptic gastric disease 8 years ago and had no other history of illness. Two weeks prior, he had mild epigastric pain, which subsided after he took a proton pump inhibitor. In the 24 hours before, he had mild retrosternal and transitory pain, which worsened progressively. The pain became intermittent, of high intensity, with no radiation, and it was not related to exertion and was not relieved by a common analgesic. The patient was experiencing a period of significant professional stress, anxiety, and irritability. He had no symptoms during weight training sessions. The patient did not smoke or drink alcohol, and he denied using cocaine or stimulants and reported his cholesterol and glucose levels had been tested 1 year ago and were normal. For 6 months before developing acute coronary syndrome, he had been self-injecting t trenbolone acetate, stanozolol, and testosterone. The patient's family history included 2 cases of AMI. His father had an AMI at age 50 years and a paternal aunt had 3 AMIs after age 53 years; both had hypercholesterolemia.

Initial evaluation of the patient revealed the following: blood pressure (BP) of 122/78 mmHg, heart rate (HR) 92 beats per minute (BPM), respiration 12 inhalations per minute (IPM), weight 76 kg, height 176 cm, and body mass index (BMI) 24.5 kg/m². Physical examination was normal. Hirsutism was

Table 1. Results of laboratory tests 1 week before AMI.

Exams	Results	Reference values
Total cholesterol	249 mg/dL	<190 mg/mL
Triglyceride	60 mg/dL	<150 mg/dL
HDL cholesterol	33 mg/dL	>40 mg/dL
LDL cholesterol	204 mg/dL	<130 mg/dL
Non-HDL cholesterol	216 mg/dL	<160 mg/dL
Glucose	88 mg/dL	65–99 mg/dL
C-reactive protein	3.54 mg/L	High risk >3.0 mg/L
Follicle-stimulating hormone	0.9 mUI/mL	1.1–8 mUI/mL
Luteinizing hormone	0.4 mUI/mL	0.6–12.1 mUI/mL
Total testosterone	1953 ng/dL	241–827 ng/dL
Free testosterone	61 ng/dL	17–65 ng/dL
DHt testosterone	1296 ng/dL	16–79 ng/dL
DHEA	224 ng/mL	1.7–6.1ng/mL
Estradiol	137 ng/mL	10–40 ng/mL
Estrone	187 ng/dL	10–60 ng/dL
Cortisol	16,3 µg/dL	22.5 µcg/dL
TSH	1.09 mIU/L	0.48–5.60 µm/L
GGT	20 U/L	7–32 U/L
AST	31 U/L	<40 U/L
ALT	36 U/L	<41 U/L
Creatinine	1.16 mg/dL	0.4–1.4 mg/dL

HDL – high-density lipoprotein; LDL – low-density lipoprotein; DHt testosterone – dihydrotestosterone; DHEA – dehydroepiandrosterone; TSH – thyroid stimulating hormone; GGT – gamma glutamyl transferase; AST – aspartate aminotransferase; ALT – alanine aminotransferase.

reported by the patient but it was not visible because he shaved his body hair. There were no changes in his testicular volume.

A week before, testing of the patient's hormonal and biochemical levels had been ordered by another clinician as part of routine care. Table 1 shows the results: low levels of luteinizing and follicle-stimulating hormones. The testing also revealed dyslipidemia, with high levels of low-density lipoproteins (LDL cholesterol), low levels of high-density lipoproteins (HDL cholesterol), and elevated C-reactive protein (CRP). Levels of estrone and estradiol and dehydroepiandrosterone (DHEA) also were elevated.
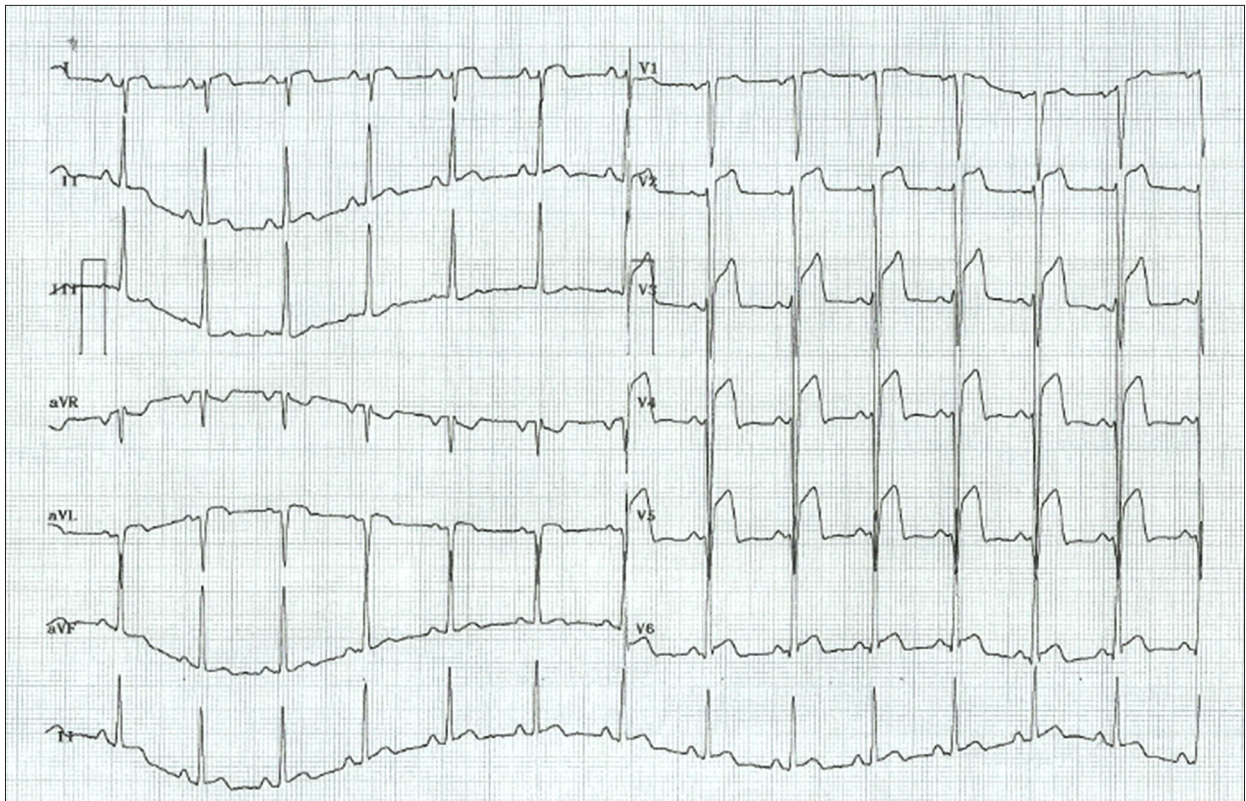
Figure 1. EKG on admission. There is a notable ST segment elevation in leads V_2 to V_6 and discrete elevation in DI and aVL derivations, confirming an extensive anterior wall AMI with ST elevation. AMI – acute myocardial infarction.

An electrocardiogram (EKG) showed ST segment elevation in leads V_2 to V_6 and in leads DI and aVL, with pathological Q waves in leads V_4, V_5, DI, and aVL (Figure 1). Markers of myocardial necrosis were elevated, as described in Table 2. Cinecoronariography and angioplasty were performed immediately, and an occlusion of the left anterior descending branch of the left coronary artery was promptly treated with angioplasty and implantation of a drug-eluting stent (Figure 2).

Doppler echocardiography (DECG) performed before the patient's discharge revealed an increase in the size of the left atrium, reduced relaxation in the left ventricle, septal and mid-apical systolic dysfunction, and increased systolic dimension, with ejection fraction of 47% (Figure 3, Table 3).

The patient was discharged after 3 days in the hospital, with no symptoms, normal vital signs, a diagnosis of Killip Class I AMI, and prescriptions for ramipril (5 mg/day), carvedilol (6.25 mg twice a day), clopidogrel (75 mg/day), acetylsalicylic acid (100 mg/day), and rosuvastatin (10 mg/day).

During an outpatient assessment 4 weeks later, the patient reported having mild palpitations and insomnia. Results of a physical examination were normal (BP 118/68 mmHg, HR 76 bpm, RF 12 IPM, weight 73.6 kg, height 176 cm, BMI 23.8 kg/m²).

Table 2. Myocardial necrosis markers on hospital admission.

Exam	First sample results	Second sample results	Reference values
Troponin	Reagent	Reagent	Non-reagent
CKMB	141 U/L	236 U/L	≤25 U/L
CK	1409 U/L	2824 U/L	26–189 U/L

CKMB – creatine kinase MB fraction; CK – creatine kinase.

His EKG showed infarction in the extensive anterior wall marked by pathological Q waves on V_1 to V_5 derivations and persistence of the ST elevation from V_2 to V_6, DI and aVL derivations (Figure 4). A new DECG showed improvement in the function and sizes of the left chambers, with no diastolic dysfunction or left atrial enlargement, with hypokinesis in the septal wall as the only finding (Table 4).

The patient's dosages of carvedilol and rosuvastatin were increased and ezetimibe was added because his HR was not yet at 60 bpm and the LDL target had not been reached. Troponin was no longer elevated and the level of B-type natriuretic peptide was normal. A drastic drop in the patient's testosterone
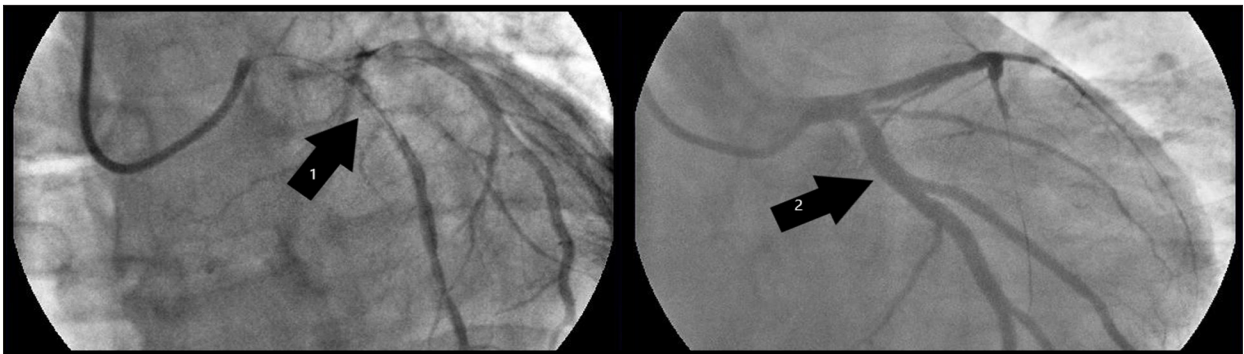
Figure 2. Emergency angioplasty of left coronary anterior descending branch. Arrow 1: Occluded artery, passed by guidewire. Arrow 2: After insertion of drug-eluting stent.
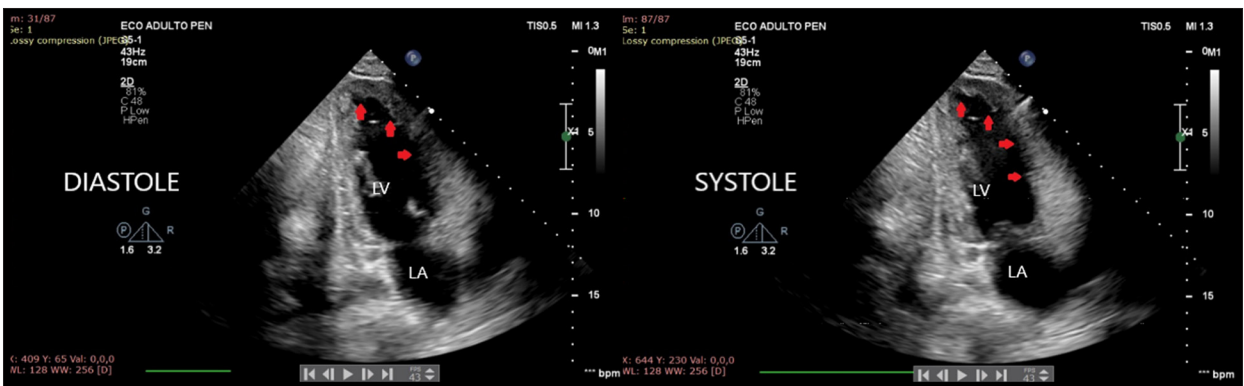

Figure 3. Pre-discharge Doppler echocardiography in a 2-chamber apical view with diastolic and systolic images. Left atrium (LA) and ventricle (LV) enlargement, anterior and apical systolic dysfunction (arrows), and concentric hypertrophy are visible.

Table 3. Pre-discharge Doppler echocardiography results.

Data	Results	Reference values
Left atrium	41 mm	19–40 mm
Aortic root	25 mm	20–37 mm
Right ventricle	17 mm	07–26 mm
Septum	12 mm	06–10 mm
Posterior wall	12 mm	06–10 mm
Left ventricle (DD)	55 mm	42–56 mm
Left ventricle (SD)	42 mm	25–40 mm
Shortening (%)	24%	≥30%
Ejection fraction	47%	≥55%
Conclusions	Hypertrophy and reduced relaxation with increased systolic dimension of the left ventricle. Mild mitral and tricuspid reflux. Enlargement of the left atrium	Normal

DD – diastolic diameter; SD – systolic diameter.

level and slight elevations in his liver enzymes also were observed (Table 5).

Four months after his AMI, the patient was asymptomatic, working at a farm, and doing regular moderate physical activity without using steroids or any drugs other than those that he had been prescribed. His physical examination was normal, but his EKG showed an extensive inactive area in the anterior wall. Treadmill stress testing showed no symptoms, ST deviation, or arrhythmia, with excellent functional capacity (14.6 metabolic equivalent).

Discussion

We describe a case of a young male bodybuilder using AAS, who presented with an AMI and was treated with primary angioplasty. The patient used AAS for muscular hypertrophy, which elevated the levels of his male hormones. The low levels of LH and FSH that were observed were due to inhibition of the hypothalamic-pituitary axis. The high levels of estrogens must have been caused by peripheral aromatization of increased androgen levels [8]. The patient had high levels of dehydroepiandrosterone, a precursor to testosterone synthesis. This finding
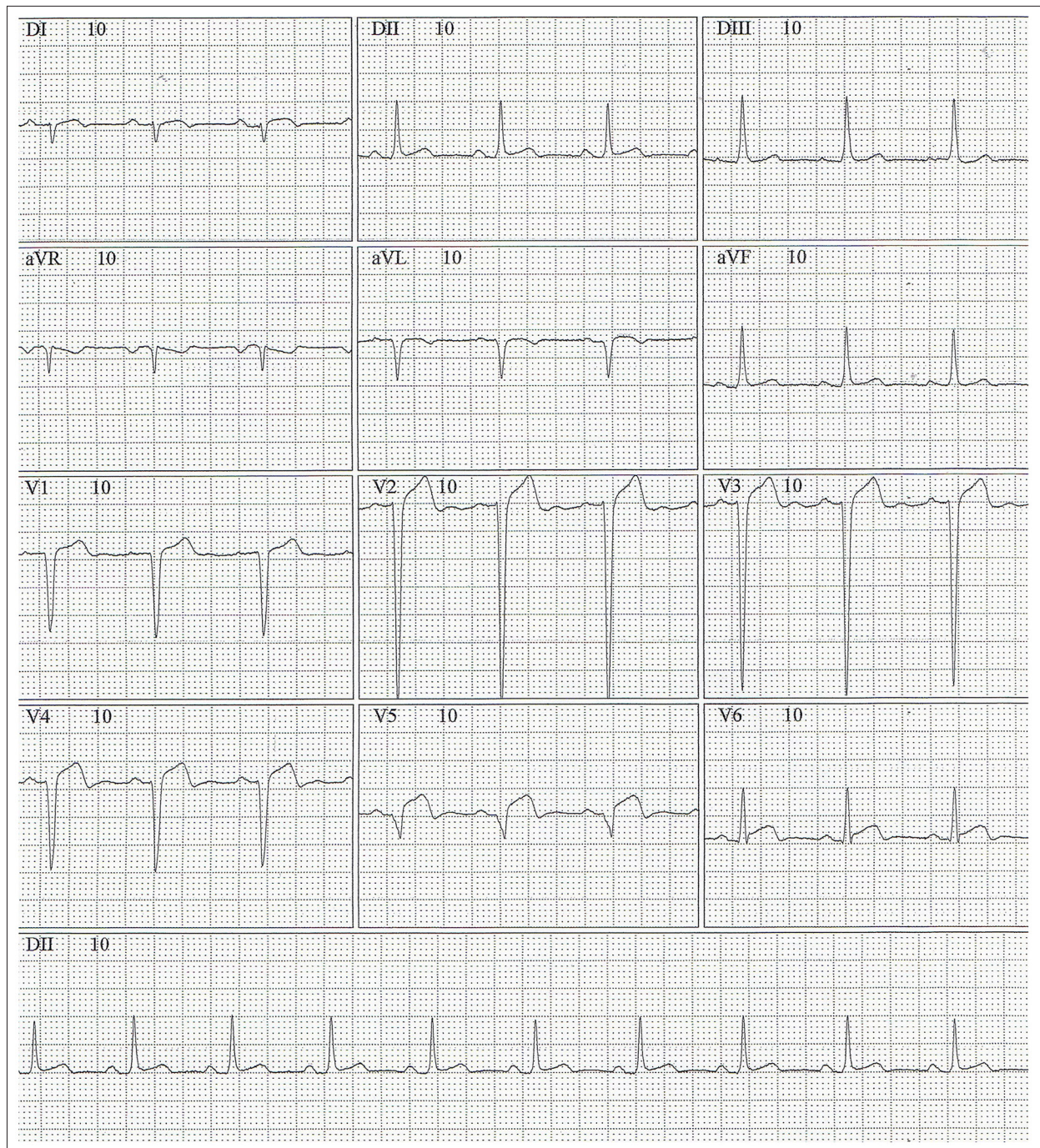
Figure 4. EKG 4 weeks after AMI, showing persistence of ST segment elevation in V_2 to V_6 derivations and pathological Q waves in leads V_2 to V_5 and DI and aVL derivations. These findings indicate large extension and left ventricle compromise.

has been associated with expression of cytochrome b5 in the zona reticularis of the adrenal glands, which can be detected in males after testosterone injection. Levels of cortisol remain normal because the zona fasciculata is not affected by this mechanism [9,10].

Replacement therapy versus AAS misuse

Use of AAS for replacement in middle-aged or older, symptomatic men with low serum testosterone levels is a well-established practice, provided they are carefully evaluated and monitored. The dosage for testosterone replacement is approximately 400 mg every 2 or 4 weeks (maximum 800 mg/month) and the

Table 4. Doppler echocardiography 4 weeks after AMI.

Data	Results	Reference values
Left atrium	38 mm	19–40 mm
Aortic root	27 mm	20–37 mm
Right ventricle	25 mm	07–26 mm
Septum	8 mm	06–10 mm
Posterior wall	8 mm	06–10 mm
Left ventricle (DD)	54 mm	42–56 mm
Left ventricle (SD)	37 mm	25–40 mm
Shortening (%)	31%	≥30 mm
Ejection fraction	59%	≥55 mm
Conclusions	Septal hypokinesis	Normal

DD – diastolic diameter; SD – systolic diameter.

Table 5. Laboratory results 4 weeks after AMI.

Exams	Results	Reference values
Troponin	Zero	Zero
BNP	61 pg/mL	<100 pg/mL
Total cholesterol	195 mg/dL	170 mg/mL
Triglyceride	68 mg/dL	<150 mg/dL
HDL cholesterol	30 mg/dL	>40 mg/dL
LDL cholesterol	155 mg/dL	<130 mg/dL
Non-HDL cholesterol	165 mg/dL	<100 mg/dL
Glucose	75 mg/dL	65–99 mg/dL
Testosterone	78 ng/dL	165–753 ng/dL
GGT	57 U/L	10–50 U/L
CK	176 mg/dL	60–174 U/L
AST	20 U/L	<40 U/L
ALT	50 U/L	<41 U/L
Blood count	Normal	Normal

BNP – natriuretic peptide; HDL – high-density lipoprotein; LDL – low-density lipoprotein; GGT – gamma glutamyl transferase; CK – creatine kinase; AST – aspartate aminotransferase; ALT – alanine aminotransferase.

goal is to achieve a level at the low end of the normal range and recovery from hypogonadism symptoms (241 ng/dL) [6]. Even this replacement use has been linked with increased cardiovascular risks. Corona et al. [6] observed that low doses can be associated with an increased risk of MI and mortality, which underscores the significant role of dose monitoring of testosterone replacement. However, other authors have shown evidence that low testosterone levels in men with congestive heart failure are linked to poor prognosis and increased mortality [11,12]. There are reports of reduced cardiovascular and reduced mortality with testosterone replacement therapy versus untreated men. Replacement therapy has been linked to less myocardial ischemia, better exercise capacity, and better glucose metabolism [12]. The patient in this report used much higher doses of AAS, starting with 600 mg per week (2400 mg/month) [13] and had a testosterone level that was more than twice normal (Table 1).

Epidemiological findings on AAS misuse

The global prevalence of AAS use has been estimated at 3.3% to 6.4% in men [2, 14] and 1.3% in women [14,15]. Most men who use AAS to gain muscular mass gain are not athletes and their principal goal is to look and feel strong [2]. Although women have less global use of anabolic steroids, a study of North American high school students showed a 5.3% use rate, similar to the incidence of depressive symptoms, use of cigarettes, and slimming pills. Authors also observed that the use was not necessarily associated with participation in sports competitions, but appears as part of a series of dangerous attitudes aiming to achieve ephemeral feeling of well-being [15]. Our patient, a 26-year-old man who was an amateur athlete interested in bodybuilding, not in sports results, belongs to the

group most likely to misuse AAS. The drugs he used – trenbolone, stanozolol, and testosterone – are the ones most frequently described in other studies in this area [16,17].

Main adverse effects of AAS misuse

The effects of AAS have been studied for decades. Studies have described endocrine, dermatological, skeletal, genitourinary, psychiatric, hepatic, metabolic, and cardiovascular effects in bodybuilders and high-level athletes [5,6,8,18–21] (Table 6). According to the literature, psychiatric symptoms are common in users of the hormones, and our patient reported stress, anxiety, and irritability before his AMI. Although the psychiatric symptoms associated with AAS are nonspecific and underestimated, they could be a clue that an individual should consider stopping use of these hormones [22,23].

Metabolic effects of AAS misuse

Changes in lipid profile were observed in this patient a few days before his AMI. The high LDL and low HDL cholesterol levels (204 mg/dL and 30 mg/dL, respectively) suggest hormonal action on lipid metabolism, which increases cardiovascular risk [5,24–27]. High levels of CRP (3.54 mg/L) have been associated with AAS misuse. The mechanism of action may by the

Table 6. Adverse effects associated with the use of anabolic steroids. (Adapted from Hoffmann JR, Ratamess NA, 2006 [5]).

Cardiovascular	Dermatological
▪ Changes in lipid profile	▪ Acne
▪ Elevated blood pressure	▪ Male baldness
▪ Reduced myocardial function	
Endocrine	**Hepatic**
▪ Gynecomastia	▪ Liver damage
▪ Sperm reduction	▪ Tumor
▪ Testicular atrophy	
▪ Impotence and infertility	
Genitourinary	**Skeletal muscle**
Men	▪ Premature epiphyseal closure
▪ Sperm reduction	▪ Tendon rupture
▪ Testicular atrophy	▪ Intramuscular abscesses
Women	
▪ Menstrual irregularity	**Psychiatric**
▪ Enlargement of clitoris	▪ Mania
▪ Masculinization	▪ Depression
Both	▪ Aggressiveness
▪ Gynecomastia	▪ Mood disorders
▪ Libido changes	

hepatic activity of these hormones or the role that CRP plays as a pro-inflammatory marker [28].

Effects of AAS misuse in cardiovascular disease

Although it is not possible to conclude that AAS caused coronary plaques in this specific case, the association has been described in the literature. Baggish et al. [28] evaluated 140 weightlifters ages 34 to 54 years with DECG and coronary tomography, with 2-year follow-up, and observed a higher incidence of left ventricular systolic dysfunction and diastolic dysfunction and greater volume of coronary plaques in AAS users than in non-users.

There are not many AAS-related AMI case reports [7,30–39]. Generally, these cases occur in men ages 25 to 35 years who are bodybuilders or fighters and have few or no traditional risk factors. In some cases, no obstructions in the coronary arteries have been found. It is possible that these patients have some characteristics more suggestive of myocarditis, which is also associated with AAS, especially in cases of diffusely myocardial dysfunction, elevated troponin, and no coronary obstruction [40]. Another explanation is the development of small-vessel disease related to misuse of AAS [41].

The main pathophysiological events associated with acute coronary syndrome in AAS users are accelerated atherosclerosis, modification in lipid profile (increased LDL and reduced HDL cholesterol), coronary vasospasm (caused by inhibition of guanylate cyclase), and coronary thrombosis due to increased platelet aggregation (increased thromboxane A2 and reduced prostacyclin) [30]. We reported a case of a young man with a familial history of coronary disease and dyslipidemia. The patient was an AAS user and presented with changes in his lipid profile and many aspects of the sexual hormonal axis 1 week before having an AMI (Table 1). We believe that he was exposed to acute cardiovascular risk when the atherosclerotic process was accelerated by a pro-coagulant, pro-inflammatory, and pro-vasospastic state.

AAS misuse and mortality

Misuse of AAS impacts mortality. In 2018, Helal et al. reported the risk of death was 6 to 20 times higher in athletes who used AAS than in other athletes [42]. Frati et al. (n=19) [41] and Montisci et al. (n=4) [43], studying autopsies of sudden death related to AAS misuse, identified a wide range of macro- and microscopic changes. They included concentric hypertrophy, hypertrophic cardiomyopathy, myocarditis, focal fibrosis, peripheral venous thrombosis, pulmonary, renal and hepatic thromboembolism, right ventricle dilatation, infarction with and without coronary occlusion, small-vessel disease, and intraventricular thrombosis. These findings indicate that, despite the known risks of death associated with use of AAS, the harms associated with its misuse are not fully understood.

Global alert for AAS misuse

Despite all the research about the risks associated with misuse of AAS, it unfortunately continues to occur. A global warning should be sent to thousands of people, especially young AAS users, who are at increased risk of AMI, sudden death, and other diseases [5,44–46]. Our case report reinforces statements made by the Brazilian Federal Medicine Council [47] and specialty societies refuting the use of AAS for aesthetic purposes or to gain benefits in sports [44–46]. Similar alerts have been issued by European public health agencies [48] and the United States government [49].

Conclusions

We described the case of a 26-year-old male bodybuilder who used AAS, presented with an AMI, and was treated with primary angioplasty. Documentation of high serum levels of lipids and hormone changes in this patient 1 week before the acute event may suggest a role for anabolic steroids in the development of AMI in young men. Based on current evidence, AAS should not be prescribed without a clear medical indication.

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