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Case Report

The Utilization of Anabolic Steroids has Led to the Emergence of Acute Coronary Syndrome in a Young Male, Ultimately Exposing an Underlying Myocardial Bridge

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Abstract

Coronary artery disease, a leading global cause of death, is commonly associated with the elderly. However, it is increasingly affecting younger individuals due to lifestyle changes, smoking, and the early onset of comorbid conditions such as diabetes and hypertension. Additionally, the misuse of Anabolic Androgenic Steroids (AAS) remains prevalent, despite their widely recognized harmful effects.

We present the case of a 20-year-old man who experienced sudden chest pain at rest, with a history of significant AAS abuse over two months.

Introduction

Anabolic steroids, which are synthetic derivatives of testosterone, were originally developed as therapeutic supplements for various medical conditions. In numerous countries, they are employed to increase muscle mass and enhance athletic performance [1]. The use of Anabolic Androgenic Steroids (AAS) has been linked to a range of cardiovascular side effects and is associated with heart toxicity [2].

Case reports commonly highlight acute myocardial infarction (MI), but a subset of individuals also experiences various other cardiovascular effects, including left ventricular hypertrophy, reduced left ventricular function, arterial thrombosis, and even instances of sudden cardiac death [3].

Here, we present the case of a 20-year-old man who presented with sudden chest pain and had a history of significant AAS abuse over two months.

Case Report

The emergency department received a 20-year-old male patient with no prior medical conditions, complaining of severe retrosternal chest pain that had persisted for five hours. Notably, he did not exhibit typical cardiovascular risk factors such as smoking, diabetes, or hypertension. It is worth noting that the patient disclosed a history of Anabolic Androgenic Steroid (AAS) abuse spanning two months.

Upon admission, his vital signs were as follows: Blood pressure measured at 130/85 mmHg, a pulse rate of 72 beats per minute, and a respiratory rate of 14 breaths per minute. An Electrocardiogram (ECG) conducted in the emergency room revealed the presence of negative T waves in the anterior region. Additionally, an elevated serum level of cardiac troponin was detected.

Figure 1: Coronary angiography showing the presence of a systolic narrowing (myocardial bridging) of the distal left anterior descending artery.

The echocardiography did not detect any regional wall motion abnormalities with preserved ejection fraction. A coronary angiography was performed, showing no significant lesions but revealing the presence of a systolic narrowing (myocardial bridging) of the distal left anterior descending artery **(Figure 1)**.

Discussion

Anabolic steroids exert a range of effects on the cardiovascular system. These effects include the development of atherosclerosis, systemic hypertension, impairment of diastolic and systolic ventricular function, and disruptions in lipid metabolism, all of which have been documented in cases of anabolic steroid abuse. Additionally, heightened platelet activity is observed in individuals using these substances, which may contribute to the occurrence of Myocardial Infarctions (MI) and potentially even strokes [3] .

Numerous instances exist of young athletes who have abused androgenic anabolic steroids, experiencing cardiovascular issues such as MI or thrombosis in various vessels, including the renal artery [4].

Another consequence of prolonged androgenic anabolic steroid abuse is left ventricular (LV) dysfunction, which can increase the risk of sudden death in affected individuals [5]. Pathological investigations in these cases have revealed thickening of small arteriole walls and intimal hyperplasia, factors that could be responsible for ischemic damage to the heart muscles and subsequent impairment of ventricular function [6].

Furthermore, the use of anabolic steroids can induce acute hyperhomocysteinemia, leading to associated thrombotic events [7].A increased level of homocysteine could be seen with lower HDL concentration, higher plasma LDL, and triglycerides.

Elevated homocysteine levels are often accompanied by lower high-density lipoprotein (HDL) concentrations, higher levels of Low-Density Lipoprotein (LDL), and triglycerides. Homocysteine is a sulfhydryl amino acid not typically found in natural human diets, and it can be toxic to endothelial cells, promoting the proliferation of smooth muscle in vessel walls and affecting the coagulation process [8, 9]. High levels of homocysteine have been associated with multivessel coronary artery disease and acute MI [10] . Anabolic steroids may interfere with the absorption of vitamins B6 and B12, potentially resulting in elevated homocysteine levels [11] . This effect might explain the elevated homocysteine levels in our patient, possibly contributing to stenosis and thrombosis.

The varied spectrum of steroid profiles and the unclear risk associated with each specific agent, along with variations in dosing, make it challenging to assess the risk for this particular patient population. Additionally, the off-label use of both animal and human compounds, coupled with the lack of regulation regarding concentration doses during production, further complicates the interpretation of the clinical risks [12].

While the administration of folate supplements may be debated, a more favorable approach involves normalizing plasma homocysteine levels through a healthy diet rich in vegetables and fresh fruits, along with moderate exercise [8]. High-dose vitamin B6 and folic acid administration after an acute MI does not reduce the risk of death of the recurrence of cardiovascular disease [13]. In fact, it may potentially harm myocardial repair and increase morbidity and mortality among patients with cardiovascular disease.

Conclusion

It is imperative for healthcare providers to remain vigilant about the potential for anabolic androgenic steroid (AAS) abuse in young athletes, particularly when significant reductions in High-Density Lipoprotein Cholesterol (HDL-C) levels are evident.

Individuals, especially athletes and those adhering to specific dietary regimens, should be under the supervision of a medical professional to closely monitor both the immediate and long-term consequences of the chemical and biological substances they may be using.

Consequently, it is crucial to launch educational campaigns aimed at enhancing public awareness regarding the severe cardiovascular complications associated with AAS abuse, with the goal of discouraging its usage.

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