

Coronary Occlusion in a Young Athlete Associated with Anabolic Androgenic Steroid: A Case Report [Version 1, 1 Approved]

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Original Submission

Received: February 07, 2017

Accepted: March 09, 2017

Published: March 14, 2017

Last Updated: May 02, 2017

Open Peer Review Status: 1 Approved

How to cite this article: Homer YanceHurtado, Thiago Andrade Macedo*, Pedro Gabriel Melo de Barros e Silva, Roger Pereira Oliveira, Leandro de Oliveira Jardim, MaurícioGonczyNunesBastos, Gustavo Iacomini Ida,ValterFurlan. Coronary Occlusion in a Young Athlete Associated with Anabolic Androgenic Steroid: A Case Report [Version 1, 1 Approved]. ClinCardiol Updates. (2017) 1: 4.1

Abstract

We describe a case of a 36 years old male athlete admitted to the emergency department with atypical chest pain, after one hour of having completed training at the gym. He reported use of anabolic steroid for the last 4 days and denied history of cardiovascular disease. The electrocardiogram was normal and high-sensitivity troponin T was high. The patient underwent coronary angiography that confirmed occlusion of the septal artery, branch of anterior descending coronary artery. The authors discuss the relationship of myocardial infarction and use of steroids in young athletes.

Keywords

Myocardial Infarction; Anabolic Steroids; Coronary Angiography

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Introduction

Anabolic androgenic steroids (AAS) are synthetic compounds similar structurally with testosterone that enhanced effects of male secondary sexual characteristics (androgenic effect) and accelerate muscle growth (anabolic effect) [1]. Its use is seeming widespread among athletes, young adolescent population, and also illicit drug users. Some of the complications associated with the consumption of these substances can be fatal, being quite associated with acute cardiovascular events. The presence of external signs such as marked muscular hypertrophy, striations, gynecomastia, testicular atrophy and cutaneous acne, can alert us to the possibility of anabolic consumption [2]. Although, in general, some patients do not present with such brands, we highlight the importance of query about AAS, mainly in young patients with no risk factors to cardiovascular disease.

Case Report

A 36 years old man athlete complained a severe and sudden anterior chest pain with radiation to left arm and neck, high intensity and duration of 20 minutes, started 1 hour after gym training. He denied history of hypertension, diabetes mellitus, smoking or family history of cardiovascular disease. The patient reported use of high doses of anabolic steroids (nandrolone decanoate and stanozolol) associated to thermogenic (caffeine) during the last 4 days. He was admitted at the emergency room after 6 hours of pain still with moderate chest pain. On the examination, the blood pressure was 112/62 mmHg, the pulse 106 beats per minute, the respiratory rate 22 breaths per minute, and the oxygen saturation 94% while he was breathing ambient air. The heart sounds were normal and clean sounds on lung fields. His electrocardiogram did not show alteration on ST-segment (figure 1) and chest X-ray was normal. Fondaparinux 2.5mg subcutaneous and orally Clopidogrel 300mg plus ASA 300mg was administered. His calculated TIMI risk score was 1 point and GRACE score 50 points, stratifying the patient as low risk.

On the laboratory tests, high-sensitivity troponin T (hs-cTnT) variation levels were 0.04 - 1.54 - 1.95 - 1.4ng/mL (normal: less than 0,01ng/mL) and the variation levels of Creatinine Kinase-MB (CKMB) was of 8.6 - 17.8 - 21.5 - 12.6ng/mL (normal: less than 7.7 ng/mL). His lipid profile: Total cholesterol: 241 mg/dL (normal: less than 200 mg/dL); Triglycerides: 120mg/dL (normal: less than 150 mg/dL); HDL: 16mg/dL (normal: greater than 40 mg/dL) and LDL: 201mg/dL (normal: less than 130 mg/dL).

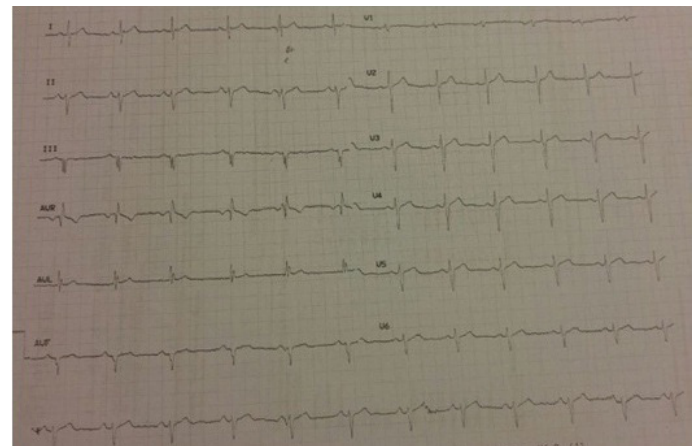


Figure 1: Electrocardiogram showing sinus rhythm and blockage of anterosuperior division, without ST-segment deviation.

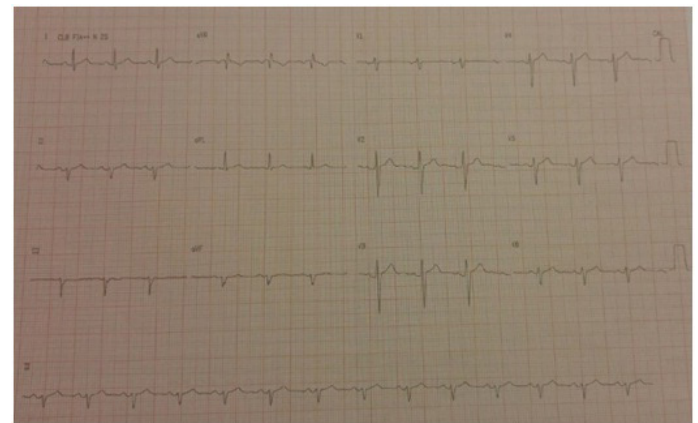


Figure 2: Electrocardiogram (6 hours later) showing sinus rhythm and blockage of anterosuperior division, without ST-segment deviation.



Figure 3: Circumflex artery (no obstructive lesions).

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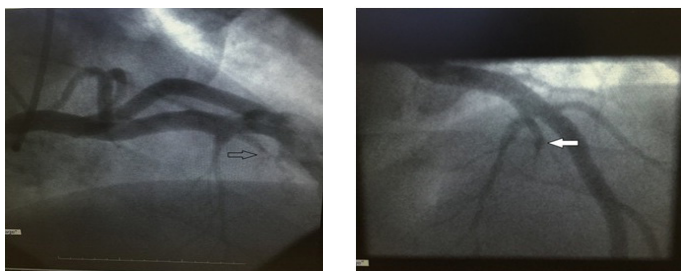


Figure 4: Arrow showing acute occlusion on large septal artery.

His two-dimensional echocardiogram showed normal left ventricular ejection fraction (74%), moderated dilatation of left atrium (45 mm) without segmental wall motion abnormalities on left ventricle. Heart valves were normal. Right ventricle showed normal function and pulmonary pressure was normal (25 mmHg). The coronariography did not demonstrate atherosclerotic plaques.

After two days of hospitalization, the patient progresses uneventfully, maintaining left ventricular function preserved. He was then chosen for clinical treatment with lifestyle modification, introduction of beta-blocker, statin and ASA and also suspension of the anabolic androgenic steroids. The patient was referred for cardiology outpatient clinic and rehabilitation.

Discussion

We presented a case of a young athlete who developed acute myocardial infarction secondary to abusive use of anabolic steroids. This case highlights the importance of quest about illicit drugs related of acute coronary syndrome (ACS) mainly in young people without risk factors for atherosclerosis. His symptoms were highly suspected of ACS (sudden anterior chest pain with irradiation to left arm and neck, high intensity and duration of 20 minutes) and came after gym training. He used abusively anabolic steroids (nandrolone decanoate and stanozolol) associated to thermogenic (caffeine) during the last 4 days. Although the electrocardiogram had not demonstrated abnormalities, Troponin presented elevation and his coronarography demonstrated occlusion of septal artery. Acute thrombosis can be induced by androgen abuse by an effect on platelet aggregation and coagulation proteins [3,4]. Moreover, steroid abuse produces mitochondrial and myofibrillar changes at a molecular level, similar to those seen in early heart failure [5]. Probably in described case it was the mechanism whereas the patient was young and had denied risk factors for atherosclerosis. Although without significant findings on ECG, biomarkers with high sensitivity such as hs-cTnT confirmed the cellular injury demonstrated by 98% of increasing from initial values. According to the most recent classification [6], the patient had a type 2 - MI caused by supply-demand imbalance probably associated with endothelial dysfunction overcharged by the presence of dyslipidemia showed by his elevated LDL. Although echocardiogram had demonstrated normal ventricular function (global and segmental wall motion) and low values of risk score (TIMI risk: 1

point and GRACE score: 50 points) was chosen stratification with coronarography due to elevated hs-cTnT. It demonstrated occlusion of large septal coronary artery. Clinical treatment was the best choice in this case because culprit artery was small, not addressed by angioplasty.

Conclusion

This case highlights the importance of investigate use of anabolic androgenic steroid. It can be related with coronary occlusion, mainly in young patients with no risk factors to cardiovascular disease.

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