

Nandrolone- induced myocardial infarction in a professional soccer player

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Introduction

Androgenic Anabolic Steroids (AAS) are often used by athletes for enhancing athletic performance but are strongly associated with detrimental cardiovascular effects including sudden cardiac death¹. Herein, we present a 24 year-old professional soccer player who suffered acute myocardial infarction after nandrolone consumption.

Case Presentation:

A 24 year-old professional soccer player presented to emergency room with squeezing retro-sternal and left sided chest pain which had started just before the game. The pain was associated with nausea, vomiting, cold sweating and dyspnea. His medical history was negative for diabetes mellitus, hypertension, or hyperlipidemia and the family history did not show any cardiovascular events. On physical examination he was in respiratory distress and had profound sweating and was obviously suffering from chest discomfort. Blood pressure was 90/55mmhg with pulse rate of

110beats /min and respiratory rate of 28/min. He was afebrile. The heart auscultation revealed S3 gallop and tachycardia. No murmur was detected. PMI(point of maximal impulse) was in proper location with normal size but the pericardium was hyperactive. He had bibasilar fine crackle in lung examination. A twelve lead electrocardiogram was obtained which showed acute ST-segment elevated anterior and inferior wall myocardial infarction (Fig. 1). The patient was treated with thrombolytic agent (streptokinase), but due to low blood pressure and ongoing chest pain coronary angiography was performed that showed diffused left anterior descending (LAD) coronary artery spasm with multiple thromboses (Fig. 2). No other intervention was done. Intravenous nitrate and antithrombotic agent (heparin) were prescribed which followed by pain relief. Transthoracic echocardiography showed ejection fraction of 45% with anterior and apical dyskinesia. Other findings were normal. Coagulation profile showed normal values including plasma homocysteine level, protein C and protein S with absent factor V Laden and prothrombin G20210A mutation. Additional coronary angiography was performed six days later which was normal (Fig. 3). He was discharged without any symptoms but with an established infarction in his heart.

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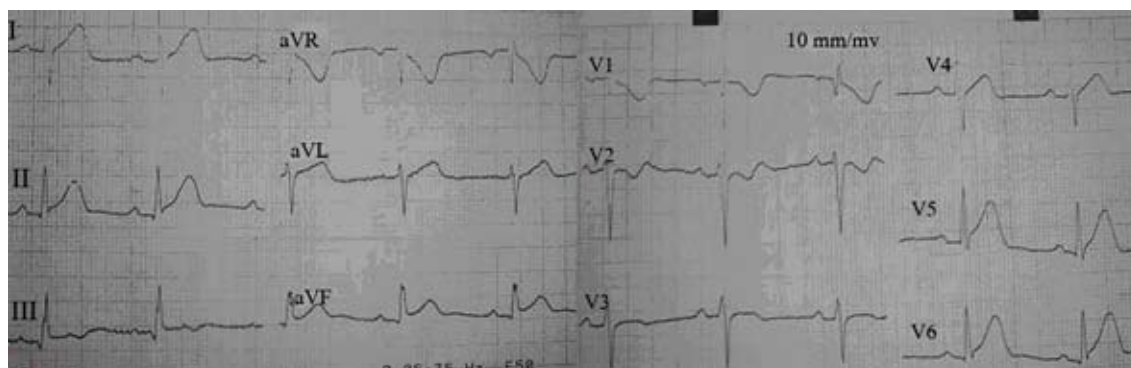


Figure 1. Shows the electrocardiogram of a 24 years old professional soccer player who has come in with compressing chest pain, sweating and dyspnea associated with nausea and vomiting. Anterior and inferior ST-segment elevation myocardial infarction is seen.

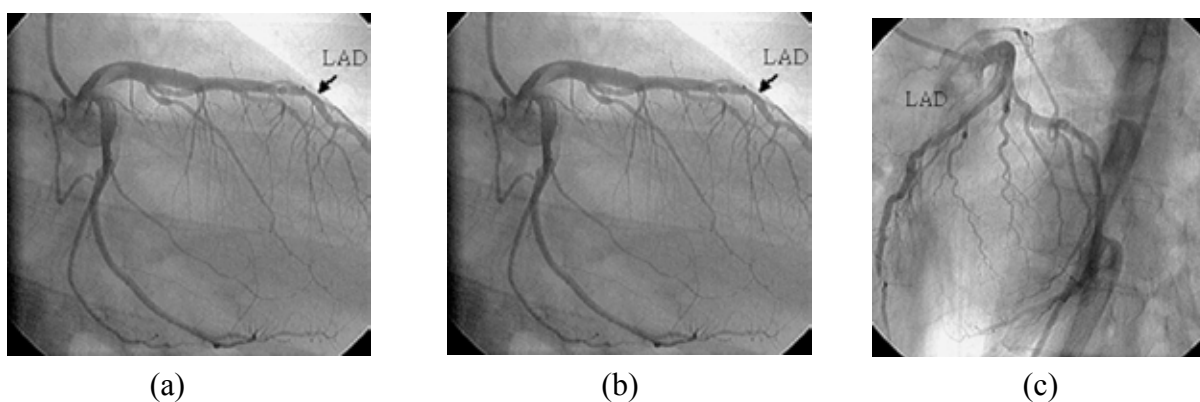


Figure 2. Shows the coronary angiography of a 24 years old professional soccer player who has come in with compressing chest pain, sweating and dyspnea associated with nausea and vomiting, (a) in right anterior oblique projection, (b) in antero-posterior cranial projection, (c) in left anterior oblique projection. Diffused Left Anterior Descending (LAD) spasm and thromboses are seen.

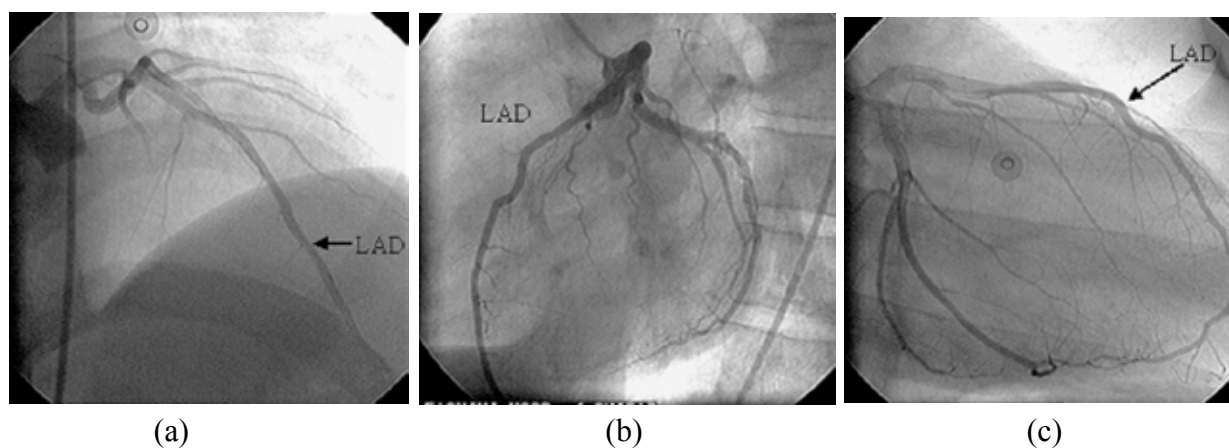


Figure 3. Shows the coronary angiography six days after acute anterior wall myocardial infarction, (a) in right anterior oblique projection, (b) in antero-posterior cranial projection, (c) in left anterior oblique projection, Normal Left Anterior Descending (LAD) is apparent.

Discussion

AAS are a class of natural and synthetic steroid hormones that promote cell growth and division, resulting in growth of several types of tissues, especially muscle and bone. Different anabolic androgenic steroids have varying combinations of androgenic and anabolic properties, and are often referred to in medical texts as AAS. Anabolism is the metabolic process that builds larger molecules from smaller ones. Anabolic steroids were first discovered in the early 1930s and have since been used for numerous medical purposes including stimulation of bone growth, appetite, puberty, and muscle growth. The most widespread use of anabolic steroids is their use for chronic wasting conditions, such as cancer and AIDS². Chemical structure of the natural anabolic hormone testosterone (17 β -hydroxy-4-androsten-3-one) is shown in Fig 4². Anabolic steroids have also been associated with numerous side effects when administered in excessive doses including elevated cholesterol (high LDL and low HDL levels), acne, elevated blood pressure, hepatotoxicity, and also cardiovascular effects². Nandrolone, or 19-nortestosterone, is an anabolic

steroid initially introduced for the treatment of anemia, osteoporosis, and breast carcinoma. it is available in several pharmaceutical formulations as a 17 β -hydroxy ester in an oily matrix or as a nandrolone salt (decanoate or sodium sulfate) in an aqueous solution. The most widely used pharmaceutical formulation is Deca-Durabolin®³. Chemical structure of nandrolone is demonstrated in Fig. 5. Non-therapeutic use of androgenic anabolic steroids is prevalent among young, competitive male and female high school athletes who hope to improve their exercise performance with these compounds. They are administered in supraphysiological doses to enhance the development of muscle mass and strength and to reduce the recovery time after strenuous training bouts. These doses are however associated with pathologic changes in numerous physiological systems. Studies using rats have shown that supraphysiological doses of anabolic steroids cause pathophysiological myocardial hypertrophy in these animals. In mice, this has been associated with inadequate vascularisation of the hypertrophied myocardium, and in isolated rat ventricular myocytes it was linked to

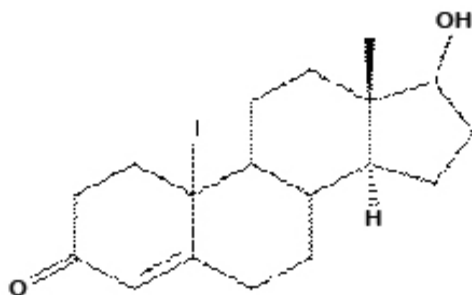


Figure 4. Illustrates the chemical structure of testosterone.

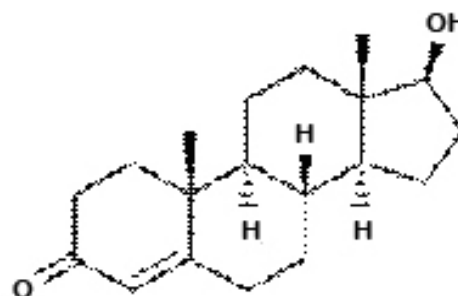


Figure 5. Illustrates the chemical structure of nandrolone.

increased apoptosis. When combined with exercise, anabolic steroid was shown to change exercise-induced physiological cardiac hypertrophy to pathophysiological cardiac hypertrophy¹. The abuse of AAS has been associated with the occurrence of serious cardiovascular disease in young athletes⁴. Chronic treatment with supraphysiological doses of nandrolone decanoate, in rats, impaired tonic cardiac autonomic regulation and provided a key mechanism for anabolic steroid-induced arrhythmia and sudden cardiac death⁵. The enhanced pumping performance of the heart by increased left ventricular diastolic filling after endurance training was attenuated by simultaneous anabolic steroid treatment which further increased the peripheral resistance⁶. However, it seemed that the anabolic steroids affect the systolic time intervals only, when their administration were combined with endurance training⁷. In addition, chronic high dose androgenic steroid administration produced a decrease in myocardial contractile reserve to beta-adrenoceptor stimulation⁸. In fatal and nonfatal myocardial infarctions patent coronary arteries were proven frequently. Besides the prothrombotic effects of AAS, an impaired endothelial function and vasospasms were discussed hypothetically as pathomechanisms. Cardiomyopathies could also occur due to AAS abuse⁹. Regular physical exercise has been associated with reduced incidence of heart diseases by decreasing plasma cholesterol, high blood pressure, obesity and glucose intolerance. Among the adaptations related to acute and chronic exercise, the improvement of heart tolerance to ischemic events represented one of the most important and clinically relevant features. Ex-

ercise adaptation was related to enhanced cardiac contractile function and reduced post-ischemia infarct size, which was dependent on the regularity and intensity of the exercise. The molecular mechanisms involved in these adaptive responses include increased expression of heat shock proteins, induction of nitric oxide synthase, protein kinase C activation as well as increased antioxidant enzyme activities. The imbalance between ROS generation and the intracellular levels of antioxidant defenses leads to oxidative stress, a condition that has been associated with apoptosis, neurodegenerative diseases and ischemia-reperfusion (I/R) injury. Exercise-induced cardio protection is impaired by supraphysiological doses of nandrolone decanoate in treadmill-exercised rats. The increased antioxidant enzyme level promoted by exercise is impaired by nandrolone decanoate treatment, which could be associated with the cardiac deleterious effects of this drug¹⁰. In addition, abuse of AAS in presence of hypercholesterolemia can enhance atherogenicity and vasospasm as well as attenuation of vasorelaxation¹¹. Acute fatal myocardial infarction was first reported in 1990. Two footballers, aged 18 and 24, sustained fatal cardiac arrests while at training sessions. Both were considered fit and healthy. Autopsy revealed features of a hypertrophic cardiomyopathy in the 18-year-old; the 24-year-old had findings of a myocarditis. In both cases, the coronary arteries were normal and there was no evidence of coronary thrombosis. Urine in both subjects contained the anabolic steroid oxymesterone. While a causal relationship was hard to prove, it was possible that the anabolic steroid contributed to an increasing cardiac

size in the first subject and augmenting his response to catecholamines and causing an arrhythmogenic event. In the second case, the inflammatory changes could have been a focus for an arrhythmia. It would appear that the abuse of anabolic steroid should be considered in any athlete presenting with an acute vascular event¹². Our patient suffered ST-segment elevated myocardial infarction. His blood tests for enhanced thrombogenicity were negative,

with positive history of nandrolone consumption for a period exceeding one year. Also the coronary angiography was in favor of intra coronary thrombosis and coronary artery spasm. In regard to absence of other predisposing factors, angiographic findings described and also considering the history of AAS consumption, we assume that AAS abuse was the underlying cause of his problem.

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