# Anabolic-androgenic steroids: a bad tenor for cardiovascular orchestra
## (Myocardial infarction with intracoronary thrombus induced by anabolic steroids)

*Anabolik androjenik steroidler: kardiyovasküler orkestra için kötü tenor*
*(Anabolik steroidlere bağlı gelişen intrakoroner trombus ve miyokard infarktüsü)*

Dear Editor,

With great interest I read the case report by Güneş et al (1), in which multiple occlusions of left anterior descending artery in a patient, a power athlete using androgenic-anabolic steroids (AAS), were demonstrated. Although they did not mention any data concerning doses and durations of AAS use (massive or small, longer or shorter) and exercise intensity (strength training is a risk factor for cardiovascular complication in athletes) before the event, I agree with the authors that AAS may facilitate development of cardiovascular accident.

Even though the compound was banned by the International Olympic Committee, athletes have been using AAS as a performance enhancer for many years (2).

Really, although the exact mechanism is not known, the AAS have been linked to cardiovascular events. Sudden cardiac death, myocardial infarction, pulmonary embolism, stroke, and other atherosclerotic/atherothrombotic events in bodybuilders taking anabolic steroids have appeared in the previously published literature (3).

With regard to detrimental effects, AAS predominantly affect regulations of cardiac, vascular and hemostatic systems. Endomyocardial biopsy specimens have revealed increased fibrous tissue and fat droplets in the myocardium of AAS abusers (4). The link between AAS and life-threatening ventricular arrhythmias has also been suggested (4). Physiologic adaptive ventricular hypertrophy does not increase ventricular repolarization in athletes (5), however, athletes using large doses of AAS have associated increased duration of the repolarization (6).

What is more, AAS also affect cardiac function and a difference in the left ventricular fraction shortening was also observed between users off and user on groups (7). This may indicate non-adaptive changes within the myocardium, and thus cause the heart to become a less effective pump. Furthermore, Pearson et al. (8) found that left ventricular diastolic functions were reduced in a group of AAS users. Earlier it was speculated that myocardium is overstimulated to irregular grow by AAS, and they may lead to cell disarray in the myocardium, like as in hypertrophic cardiomyopathy (9). Completely recovery to pretraining ventricular morphology is not obtained even though anabolic steroids are discontinued for a long time period.

On the other hand, AAS cause detrimental effects in the vascular structure and function. Several mechanisms could account for the endothelial dysfunction. Once such mechanism could take place through low high-density lipoprotein-cholesterol (HDL-C) since AAS induce a profound suppression of HDL-C (4,10-12). Chronic administration of anabolic steroids causes a reversible reduction in serum HDL-C levels, predisposing to premature atherosclerosis. Another mechanism could be that AAS have a direct effect on vascular function. The different risk factor except atherosclerotic effects for cardiovascular events is that AAS may lead to hypertension via increase in sodium chloride retention and expansion of the blood volume ( 13).

Morever, AAS have been implicated in arterial thrombosis in young athletes without known thrombotic risk factors (3,10,14). The mechanisms of AAS contribution to arterial thrombosis or premature cardiovascular disease are unclear. They also activate the hemostatic system with increased concentrations of components of clotting and homocysteinemia (14,15). Previous studies on the pathophysiological mechanisms of hyperhomocysteinemia suggest that the atherogenic propensity results from endothelial dysfunction and injury followed by platelet activation and thrombus formation (14,15).

As a conclusion, whatever mechanisms affect on cardiovascular system in a subject using AAS, it is only true that AAS are maladaptive regulators for cardiovascular morphologic and functional synchrony. In any case it seems clear that the drug history should be mentioned when face to a cardiovascular event in an athlete and AAS use should be forbidden for preventing a new event and better health.

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## Author's Reply

Dear Editor

I could't reach my notes and could not contact the patient by phone about the dosages of anabolic steroids he used. But, he was getting the anabolic steroids intermittently, especially before competitions, starting with lower doses and increasing gradually within 2 to 3 months of periods. Thanks to our colleague for contributions.

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