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CASE REPORT

Peripheral Arterial Thrombosis in Two Young Men Using Anabolic Steroids

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Introduction

Abuse of anabolic steroids (AS) is a worldwide problem. Athletes and teenagers striving for improved results or better looks risk their physical and mental health. Numerous side-effects of AS on hepatic, renal and genital function have been described. However, it is not yet fully understood how AS effects the cardiovascular system.

AS increase the serum level of low density lipoproteins (LDL) and decrease the serum level of high density lipoproteins (HDL).[1] AS affect a number of haemostatic functions, such as enhanced platelet aggregation,[2,3] increased synthesis of procoagulant factors[4] and impaired fibrinolysis.[5] Several papers regarding coronary events in athletes using AS have been published.[6,7] However, we have found only one previous report concerning peripheral arterial occlusive disease.[8]

Case Reports

Case 1

A 37-year-old male athlete and bodybuilder, who had used AS in periods during the last 10 years was admitted with sudden onset of severe pain in his left leg. Angiography showed occlusion of all major arteries of the leg. Heparin infusion was given for 3 weeks. This resulted in relief of rest pain. When he was discharged he had claudication and a walking distance of approximately 100–200 metres. This remained constant over time. Two years later he was admitted to our clinic. He had no family history of cardiovascular disease and had never smoked tobacco. The maximum walking distance measured on a treadmill was 100 metres. No pulses could be felt in his left groin. Ankle pressures were 60 mmHg on the left side and 140 mmHg on the right side compared to a brachial pressure of 135 mmHg. Routine laboratory tests including haemoglobin (145 g/l) and platelets (268×10^9/l) were normal. Angiography demonstrated a long occlusion of the left external iliac, common femoral, superficial femoral and popliteal arteries (Fig. 1). The only patent arteries visualised below the groin were the deep femoral artery and the anterior tibial artery. The arteries of the contralateral leg were normal with no sign of atherosclerotic disease.

The patient was treated with a bypass operation. An 8 mm PTFE graft from the common iliac to the deep femoral artery was used, in combination with a femorotibial bypass using an *in situ* saphenous vein graft. The distal reconstruction occluded within a week and was successfully reconstructed with a composite graft. The patient was discharged from the hospital free of symptoms after 1 week. Eighteen months later he was readmitted due to gradual return of claudication. A repeat angiogram confirmed that both reconstructions had occluded. Reoperation with insertion of a new jump-graft from the common iliac to the deep femoral artery, with further extension to the fibular and posterior tibial arteries, was performed. For the distal reconstruction a reversed saphenous vein graft from the contralateral leg was used. This reconstruction occluded after 3 weeks. No

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attempts to further reoperations were made, and the patient was discharged with claudication. One year later his situation is stable with a severe claudication and a maximum walking distance of 50 metres, but no rest pain.

Case 2

A 27-year-old male who smoked 20 cigarettes per day had chronic back-pain due to a traffic accident 8 years ago and was overconsuming Treo-Comp® (acetic salicylic acid, ASA, 500 mg, codeine 30 mg, caffeine 50 mg), approximately 8 tablets/day. Except for this, he was previously healthy and had no family history of cardiovascular disease. He was an active bodybuilder and had used oral AS for the last 13 months. The AS were consumed during three periods with increasing doses. During the last period, which lasted for about 2 months he had used methandione 50 mg/day, stanozolol 30 mg/day and oxymetholone 50 mg/day. Two weeks after finishing this period he experienced sudden onset of pain in his right calf and foot. He was referred to the county hospital, where he spent the night. He was sent home the following morning, and the pain in his leg was diagnosed as an effect of overtraining. Measurement of arterial toe-pressure arranged by his GP was found to be 20 mmHg in his right foot and 115 mmHg in the left (reference >90 mmHg). A colour-coded ultrasound examination revealed occlusion of the upper and middle thirds of all the arteries in the lower leg. The preliminary diagnosis was arterial thrombosis. Haemoglobin concentration and platelet numbers were within normal limits (145 g/l, 281 $\times$ 10^9/l). Thrombolysis was attempted with an infusion of streptokinase, without any clinical improvement. Angiography confirmed occlusion of all three arteries of the lower leg with only the distal parts of the anterior and posterior tibial arteries patent (Fig. 2). No signs of anatomical abnormalities of the popliteal artery could be demonstrated. A popliteodistal bypass was planned. However, at surgical exploration no macroscopic signs of arterial disease could be seen, and thrombectomies on both tibial arteries could easily be performed. Intraoperative angiography showed no residual thrombi or irregularities of the vessel walls. The arteriotomies were closed with vein patches. Both vessels occluded within 6 h. A reoperation with a bypass from the popliteal artery to the most distal portion of the posterior tibial artery was then performed using a reversed saphenous vein graft. Despite this reconstruction being patent he developed progressive gangrene of the distal foot leading to a fore-foot amputation.

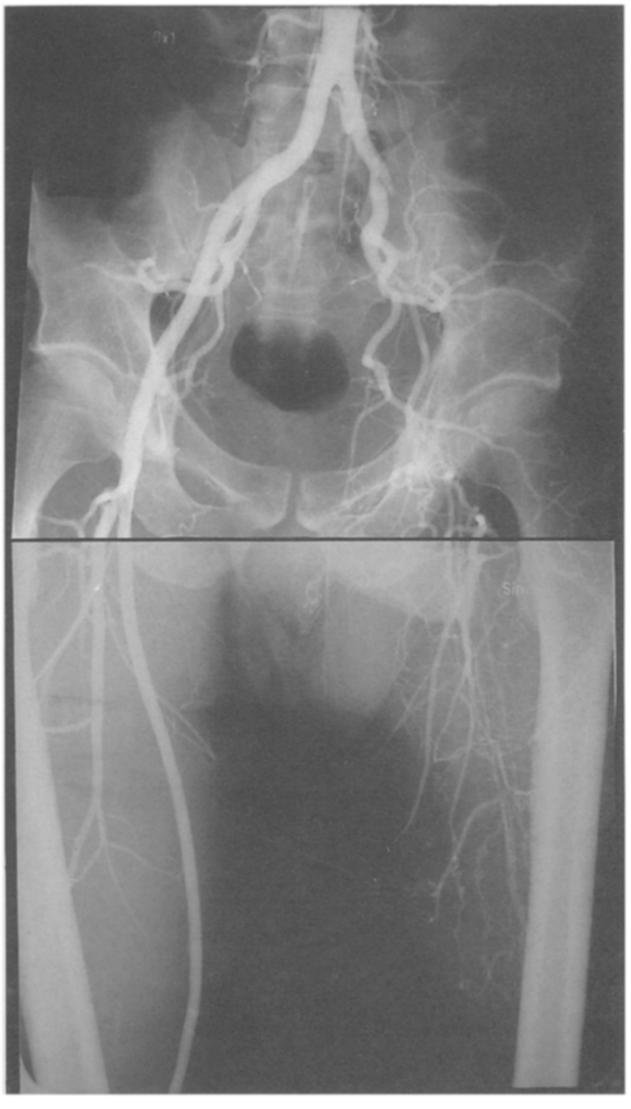

Fig. 1. Case 1. Angiography showing occlusion of the external iliac artery and the superficial femoral artery.

Laboratory studies of the coagulation profile 3 months after the operation revealed only minor abnormalities with low protein S, 28% (ref 40–100%), and a slight increase of von Willebrand's factor, 2.80 IE/ml (ref 0.40–2.00 IE/ml). All other laboratory investigations including antithrombin, protein C, activated protein C resistance, plasminogen, tissue plasminogen activator, plasminogen activator inhibitor type 1, prothrombine complex, fibrinogen and thrombin time were normal.

Discussion

From a cardiovascular point of view the side effects of

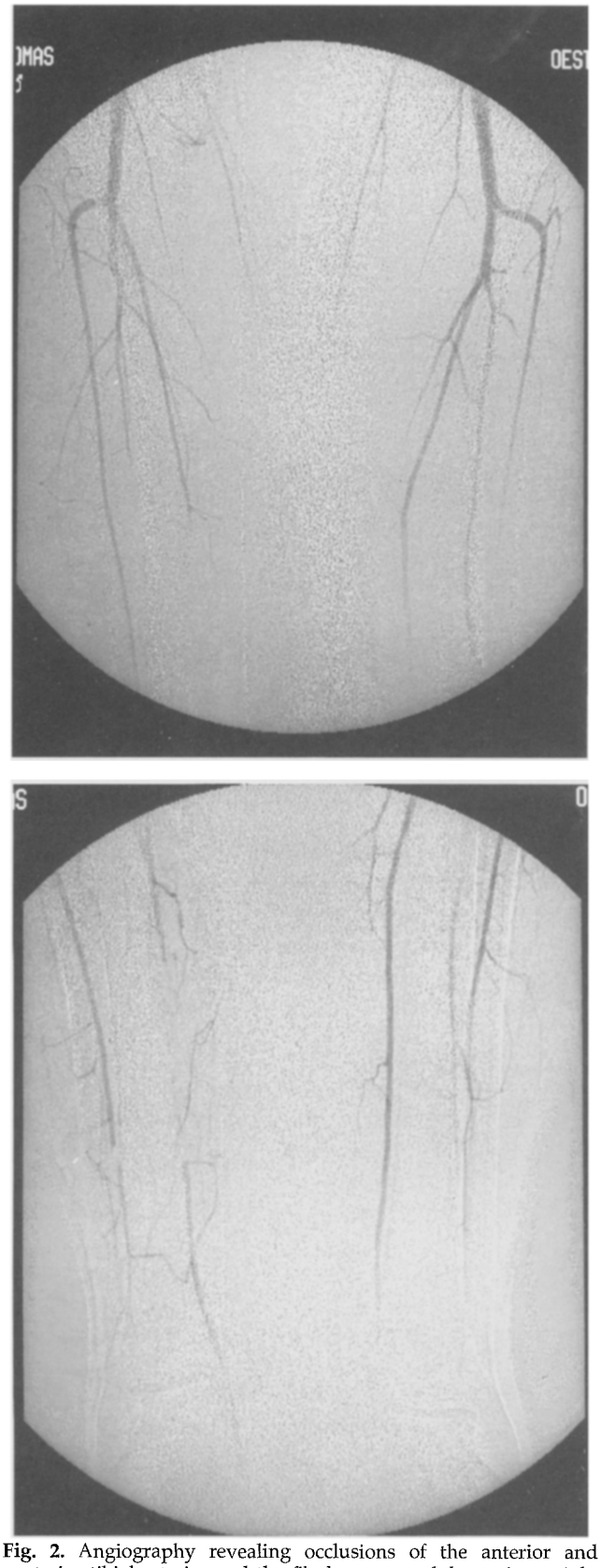

Fig. 2. Angiography revealing occlusions of the anterior and posterior tibial arteries and the fibular artery of the patients right leg.

AS related to haemostatic mechanisms and vascular wall biology are of special interest. Platelets are important in the pathogenesis of cardiovascular disease. Platelet numbers, function and aggregation are effected by androgens.[2,3] Changes in coagulation factor synthesis[4] and fibrinolytic activity[5] have also been reported. AS increase low density lipoprotein (LDL) and decrease high density lipoprotein (HDL) level[1] causing an adverse lipid profile. Increased insulin resistance and diminished glucose tolerance have been demonstrated in powerlifters ingesting AS.[9] Increased LDL and insulin resistance are associated with impaired fibrinolysis.[10,11] Recent reports describe myocardial infarction, in young and otherwise healthy persons using AS.[6,7] These studies suggest a link between the use of AS and an increased risk of cardiovascular disease.[12]

Our first patient was initially considered to be suffering from Burger's disease. The fact that he had never smoked does not support that diagnosis. His angiogram showed no signs of atherosclerosis. In the second patient, smoking was an extra risk for arterial disease. However, acute arterial occlusion at the age of 27 without any other risk factors is extremely rare. His medication (containing ASA), should also be protective against thromboembolic events. At exploration no signs of primary arterial disease, such as calcification or increase of wall thickness could be detected. Both patients had unusual anatomic sites of their arterial thrombosis.

The specific cause of arterial thrombosis in these two patients cannot be proven, but their abuse of AS is likely to have been a predisposing factor. The mechanism could be due to a hypercoagulable state, increased platelet aggregation and impaired fibrinolysis. The failure of reconstructive surgery and protracted course of ischaemia in both our patients seems to justify a conservative attitude regarding reconstruction or amputation in these cases.

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