Acute myocardial infarction and renal infarction in a bodybuilder using anabolic steroids

Anabolik steroid kullanan vücut geliştirme sporcusunda akut miyokart enfarktüsü ve renal enfarktüs

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A 41-year-old male bodybuilder was admitted with acute inferior myocardial infarction. The patient had been using oxymetholone and methenolone to increase his performance for 15 years and quitted smoking three years before. He underwent successful primary percutaneous coronary intervention (PCI) and bare metal stenting for total occlusion of the proximal right coronary artery. Angiography also showed a critical lesion in the left anterior descending (LAD) coronary artery. Five hours after primary PCI, the patient had severe right flank pain. Abdominal computed tomography showed a large renal infarction in the right kidney. Subcutaneous enoxaparin was added to dual antiplatelet treatment. Doppler renal ultrasound performed on the eighth day showed findings of reperfusion in the right kidney and normal-size kidneys. Transthoracic echocardiography demonstrated disappearance of previously detected thrombus remnant in the left ventricle and only mild hypokinesia around the apical and middle segments of the inferior and inferoseptal walls. The patient was discharged on the 10th day. Renal arteriography during elective LAD intervention 18 days after discharge showed complete revascularization, stent patency, and improved blood flow. This is the first case of renal infarction that developed in the early hours of primary PCI, despite effective anticoagulant and antiplatelet treatment. Intensive coronary artery and left ventricular thrombi may be explained by the use of anabolic steroids.

Key words: Abdominal pain/etiology; anabolic agents/adverse effects; kidney/blood supply; myocardial infarction/chemically induced; renal artery obstruction/etiology; weight lifting.

Kırk bir yaşında, vücut geliştirme sporcusu erkek hasta akut inferiyor miyokart enfarktüsü tanısıyla yatırıldı. Hasta performansını artırmak için 15 yıldır oksimetolon ve metenolon kullanmaktaydı ve sigarayı bırakalı üç yıl olmuştu. Hastaya başarılı primer perkütan koroner girişimle (PKG) proksimal sağ koroner arterdeki tam tıkanıklık için çıplak metal stent takıldı. Anjiyografide sol ön inen koroner arterde de kritik lezyon saptandı. Primer PKG'den beş saat sonra hastada şiddetli sağ böğür ağrısı gelişti. Abdominal bilgisayarlı tomografide geniş sağ renal enfarktüs saptandı. İkili antitrombosit tedavisine enoksaparin eklenen hastada sekizinci günde yapılan renal Doppler ultrasonografide sağ böbrekte reperfüzyon bulguları izlendi ve her iki böbrek de normal büyüklükte bulundu. Transtorasik ekokardiyografide sol ventrikküldeki trombüs kalıntısının kaybolduğu, inferiyor ve inferoseptal duvarların apikal ve orta segmentlerinde hafif hipokinezi olduğu görüldü. Onuncu günde taburcu edilen hastaya, taburculuğundan 18 gün sonra, sol ön inen artere yönelik elektif girişim sırasında yapılan renal arteriyografide tam revaskülarizasyon görüldü, stent açıktı ve kan akımı düzelmişti. Sunulan olgu, primer PKG sonrası erken saatlerde, güçlü antikoagülan ve antitrombosit tedaviye rağmen renal enfarktüs geliştiği bildiren ilk olgudur. Hastanın kullanmakta olduğu anabolik steroidlerin yoğun koroner ve sol ventrikküldeki trombüs yüküne neden olduğu düşünüldü.

Anahtar sözcükler: Abdominal ağrı/etyoloji; anabolik ilaç/yan etki; böbrek/kan desteği; miyokart enfarktüsü/kimyasal yolla oluşan; renal arter tıkanıklığı/etyoloji; ağırlık kaldırma.

Renal infarction is a rarely diagnosed condition and its incidence is estimated as %0.004 during baseline visits in the emergency service.[1] Thromboembolism is the leading cause and generally is of cardiac origin.[2,3] It is difficult to diagnose renal infarction due to nonspecific symptoms and findings. In addition, it is often too late for thrombolytic or interventional procedures during diagnosis.[1,2] Anticoagulation therapy

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is usually an effective approach in decreasing morbidity and mortality. Therefore, it is critical to differentiate renal infarction from more commonly seen conditions in clinical practice, such as dehydration due to diuretics, renal dysfunction and contrast nephropathy due to heart failure.

CASE REPORT

A 41-year-old male bodybuilder was admitted to the emergency service with chest pain of four-hour onset. He was diagnosed as having acute inferior myocardial infarction. He was sent to the catheterization laboratory following 300 mg aspirin and 600 mg clopidogrel administration. After 10,000 IU intravenous heparin, primary percutaneous coronary intervention (PCI) was performed for a total occlusion localized proximal to the right coronary artery with a door-balloon time of 25 minutes. A thrombus aspiration catheter (Export Aspiration Catheter, Medtronic, Minneapolis, USA) was used due to severe thrombus burden before balloon angioplasty (2.5 x 20 mm, Biotronik, Buelach, Switzerland) and a bare metal stent was implanted (3.5 x18 mm Ephesos, Nemed, Turkey). Slow TIMI III flow was obtained without any complication. Angiography of the left coronary system also showed a critical lesion in the left anterior descending (LAD) coronary artery.

Five hours after primary PCI, the patient had severe pain in the right flank while on treatment with tirofiban infusion. Physical examination showed only mild abdominal tenderness over the right lower quadrant. Blood tests and urinalysis were nonspecific, and emergency abdominal ultrasound (US) did not show a specific pathology. Abdominal computed tomography (CT) performed at the 12th hour of flank pain revealed a large renal infarction in the right kidney (Fig. 1). Simultaneous transthoracic echocardiography showed a highly mobile, round, thrombus remnant, 5 x 6 mm in size, attached to the left ventricle apex with a very thin stalk (Fig. 2). Subcutaneous enoxaparin (1 mg/kg twice daily) was added to aspirin and clopidogrel treatment. Color and power Doppler renal US performed on the eighth day showed findings of reperfusion in the right kidney and bilateral normal-size kidneys. Transthoracic echocardiography demonstrated no thrombus in the left ventricle, but there was mild hypokinesia around the apical and middle segments of the inferior and inferoseptal walls.

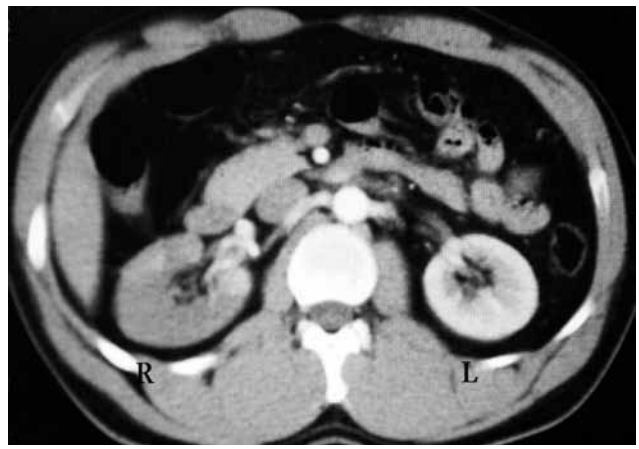

Figure 1. Computed tomography scan showing a large infarction in the right kidney as hypodense areas and normal left kidney size.

Medical history revealed that the patient had been using oxymetholone and methenolone to increase his performance for 15 years and quitted smoking three years before. Lipid parameters were as follows: total

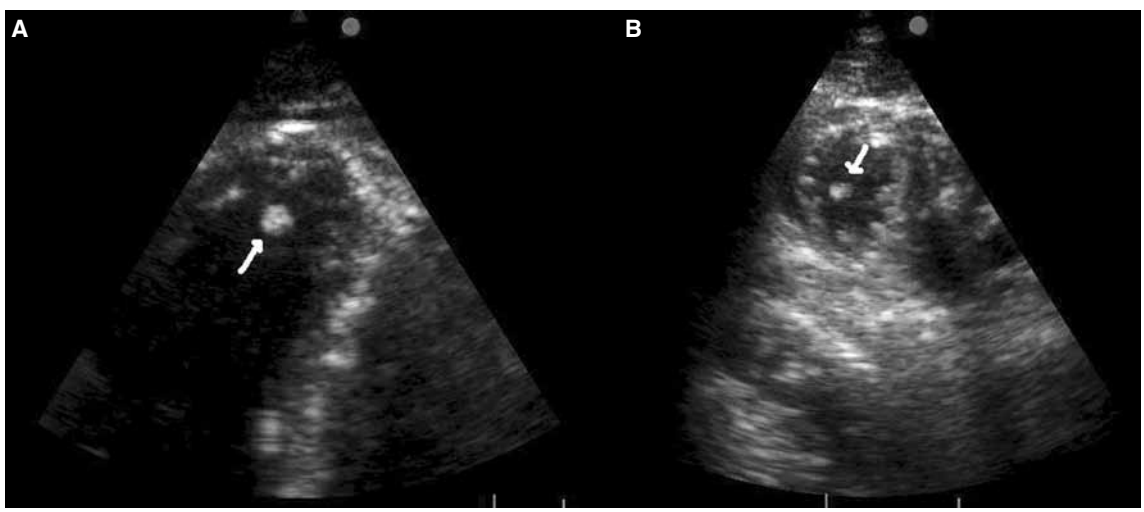

Figure 2. Transthoracic echocardiograms. **(A)** Apical four-chamber and **(B)** apical short-axis views showing thrombus remnant (arrow) attached to the left ventricular apex with a very thin stalk.

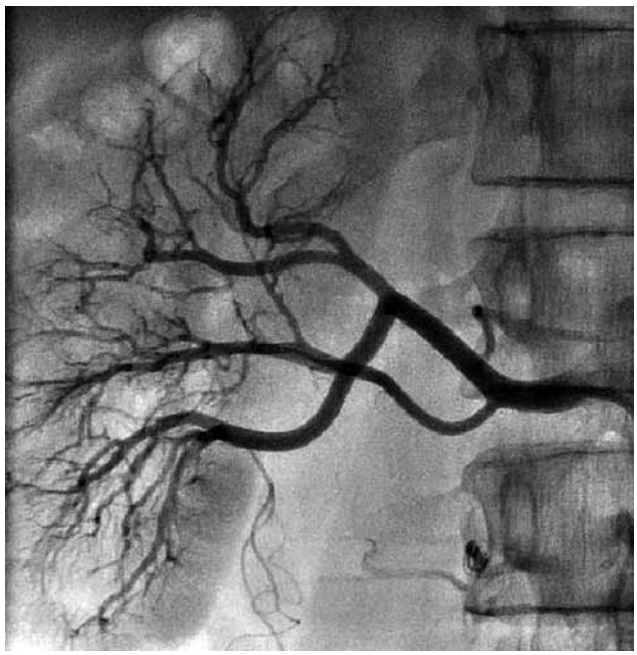

Figure 3. Right renal arteriography showing the renal artery and its main branches free from thrombus.

cholesterol 168 mg/dl, LDL cholesterol 116 mg/dl, HDL cholesterol 31 mg/dl, VLDL cholesterol 21 mg/dl, triglyceride 105 mg/dl. His fasting glucose level was normal.

The patient was discharged on the 10th day on treatment with dual antiplatelet therapy and anti-ischemic drugs. Creatinine levels were 1.53 mg/dl at baseline and 1.57 mg/dl at discharge, with a peak level of 1.78 mg/dl during hospitalization (reference range 0.7-1.2 mg/dl). Renal arteriography which was repeated during elective LAD intervention 18 days after discharge showed complete revascularization (Fig. 3), stent patency, and improved blood flow.

DISCUSSION

Acute renal infarction secondary to thromboembolism has been rarely reported in the literature. The disease is generally of cardiac origin and thromboembolism is often due to atrial fibrillation, mitral stenosis, and dilated cardiomyopathy.[2,3] Other cardiac causes include patent foramen ovale (paradoxical embolism), transient apical ballooning syndrome in the left ventricle, and sinus of Valsalva thrombosis. A review of the literature showed few cases of selective renal thromboembolism in the course of acute myocardial infarction. Of note, our patient developed renal thromboembolism despite innovative and effective treatment modalities including percutaneous revascularization and glycoprotein IIb/IIIa inhibitors.

A high level of clinical suspicion is required to diagnose renal infarction since many symptoms and findings are nonspecific.[1,2] Computed tomography should be the initial diagnostic tool; color and power Doppler US can be used to diagnose and follow the efficacy of treatment when CT is contraindicated. Renal thromboembolism should be differentiated from contrast nephropathy. Since treatment modalities for these clinical presentations are quite different, cardiologists should consider renal thromboembolism during treatment with primary PCI and effective adjuvant antiplatelet and anticoagulant therapies. We preferred to prolong the duration of anticoagulant therapy due to late diagnosis instead of intervention methods and thrombolytic therapy suggested by some authors.[2]

Although the exact mechanism is not known, several studies have shown an association between anabolic steroids and cardiovascular events.[4] In particular, atherogenic and thrombogenic effects of anabolic steroids as well as vasospastic and direct effects on myocardial damage have been emphasized. Anabolic steroids may be the main reason of or contributor to cardiovascular events. Chronic use of anabolic steroids may lead to hypertension, reduction in HDL cholesterol level, and endothelial dysfunction, which are known to play part in the development of atherosclerosis.[4,5] Increased thromboxane A2 receptor density, decreased production of prostaglandins, and increased levels of homocystein and clotting factors have been found to be the leading mechanisms for arterial thrombosis even in young healthy athletes.[6-9] In addition to quantitative changes in the levels of anticoagulant and procoagulant factors, it has been shown that there is increased platelet sensitivity to collagens.[9] Supporting this data, development of acute coronary syndromes due to severe thrombus burden induced by the use of anabolic steroids has been reported.[10] In our case, it was difficult to conclude that anabolic steroids were responsible for atherosclerosis and/or myocardial infarction, since the patient was a previous smoker. Nonetheless, anabolic steroids might be a contributing factor to severe thrombus burden in both coronary arteries and the left ventricle.

Considering thromboembolic events complicating myocardial infarction, anticoagulant therapy should be maintained for 3 to 6 months, particularly in patients with persisting mural thrombus or large akinetic myocardial areas. Some authors suggest prolonged treatment in case of previous embolic events, after large anterior myocardial infarctions with or without thrombus, and large non-anterior myocardial in-

farctions with increased wall motion abnormality or visible thrombus. We did not initiate anticoagulant therapy since, on discharge TTE, there was only mild hypokinesia in the inferior parts of the left ventricle without thrombus and we planned drug-eluting stent implantation for the LAD lesion within a few weeks. We decided to follow the patient closely while on dual antiplatelet therapy over a minimum of one year and he did not experience any problem during four months of follow-up.

In conclusion, medical history should be taken carefully with respect to the use of anabolic steroids in individuals who are interested in bodybuilding or other performance sports for the prevention and early diagnosis of cardiovascular events.

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