A Rare Cause of Acute Myocardial Infarction: Antiphospholipid Syndrome: Case Report

Akut Miyokard İnfarktüsünün Nadir Bir Nedeni: Antifosfolipid Sendrom

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ABSTRACT The antiphospholipid syndrome is an autoimmune thrombophilia characterized by the presence of plasma antibodies against phospholipids and is associated with recurrent episodes of venous and arterial thrombosis and recurrent miscarriage. Thrombosis more often affects deep venous segments of the lower limbs but arterial thrombosis may also occur and rarely, the coronary arteries can be affected. This syndrome may present as the primary condition, but may also be secondary to other diseases, particularly systemic lupus erythematosus. In this case report, the diagnosis and therapy of a 32-year old young female patient with recurrent deep venous thrombosis of the calf veins and miscarriage diagnosed as systemic lupus erythematosus and antihospholipid syndrome presenting with acute myocardial infarction due to thrombosis of the proximal left anterior descending coronary artery was presented.

Key Words: Myocardial infarction; antiphospholipid syndrome

ÖZET Antifosfolipid sendrom plazmada fosfolipidlere karşı antikorların varlığı, tekrarlayan venöz ve arteriyel trombozlar ve tekrarlayan düşüklerle karakterize otoimmün bir trombofilidir. Tromboz genellikle alt ekstremitelerin derin venlerinde görülür ancak arteriyel trombozlar da görülebilir. Nadir bazı olgularda koroner arter trombozları da bildirilmiştir. Bu sendrom primer olarak ortaya çıkabilir ya da sistemik lupus eritematozus gibi diğer bazı başka hastalıklarda sekonder olarak görülebilir. Bu olgu sunumunda sistemik lupus eritematozus ve antifosfolipid sendrom tanısı almış, tekrarlayan düşükleri ve tekrarlayan alt ekstremitede derin ven trombozu öyküsü olan 32 yaşında genç bir kadın hastada proksimal ön inen koroner arter trombozuna bağlı gelişen akut miyokard infarktüsünün tanı ve tedavisi sunulmuştur.

Anahtar Kelimeler: Miyokardiyal infarktüs; antifosfolipid sendromu

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Most coronary events in young adults are related to atherosclerosis; however, approximately 20% of them are related to nonatherosclerotic factors such as coronary abnormalities, connective tissue disorders and autoimmune disease.[1]

Antiphospholipid syndrome (APS) is a clinical entity characterized by two components; first, the presence of serum antibodies against phospholipids or phospholipid binding proteins, called antiphospholipid antibodies, of which the best known are lupus anticoagulant, anticardiolipin and anti-ß2-glycoprotein I; second, at least one of the several clinical manifestations,

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of which the most common are venous and arterial thromboses, gestational morbidity and thrombocytopenia.[2]

This syndrome may present as the primary condition, but may also be secondary to other diseases, particularly systemic lupus erythematosus (SLE). APS may be seen in 30-40% of patients with SLE. Altough the thrombosis more often affects deep venous segments of the lower limbs, arterial thrombosis may also occur. Brain vessels are the most common site of arterial thrombosis, more rarely, the coronary arteries can be affected.[3]

CASE REPORT

A 32-year-old female smoker with SLE (arthritis, autoimmune hemolytic anemia, seizures, positive serology for antinuclear antibodies) and APS (recurrent venous thrombosis and miscarriage) presented with sudden onset of severe chest pain with persistent angina and ST elevations in the anterior leads. The patient was undergoing outpatient follow-up, receiving prednisone, chloraquine and levetiracetam and also she was on warfarin therapy because of recurrent deep venous thrombosis of the calf veins. Her international normalized ratio (INR) was slightly higher than normal (INR 1.43) while admitted. She had no family history and also no history of hyperlipidemia (total cholesterol:156 mg/dl, LDL-cholesterol:75 mg/dl, HDL-cholesterol: 55 mg/dl, triglyceride: 128 mg/dl). Her vital signs were all normal and no abnormalities were noted in her cardiac, lung and chest wall examinations. She denied any history of illicit drug or oral contraceptive use. Emergent coronary angiogram showed total occlusion of the left anterior descending (LAD) coronary artery just distal to the first diagonal branch (Figure 1). The right and left circumflex coronary arteries were completely normal. Balloon angioplasty was performed leading to TIMI-I flow and revealing a huge organised thrombus (Figure 2). An aspiration catheter was advanced to the LAD and it contained only a couple of small fragments of the thrombus. After the thrombectomy, TIMI 3 coronary flow was restored and ST elevation and chest pain of the patient resolved.

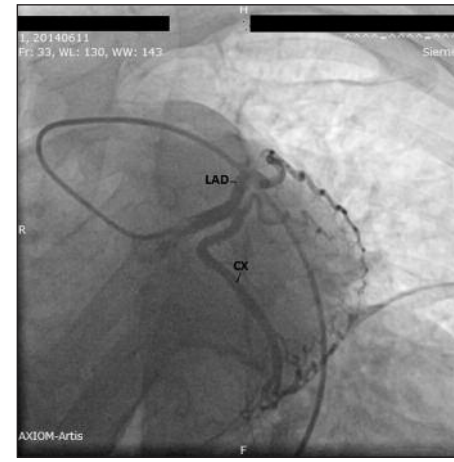

FIGURE 1: Left coronary angiogram of the patient on admission: LAD was totally occluded.

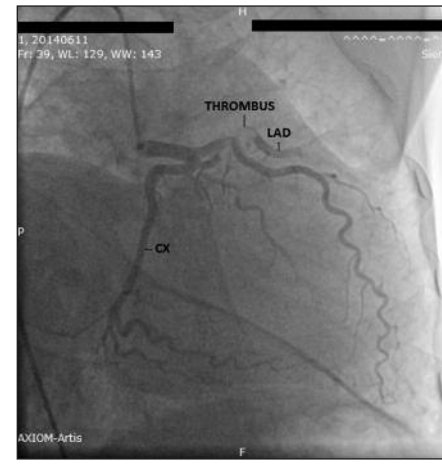

FIGURE 2: Left coronary angiogram after balloon angioplasty: a huge thrombus in the LAD.

Glycoprotein IIb/IIIa antagonist was administered during and 48 hours after the procedure along with unfractioned heparin, clopidogrel (150 mg) and acetyl salicylic acid (ASA). After 48 hours of anticogulant and antiaggregant therapy, a 3.5x12 mm bare metal stent was implanted successfully as the control coronary angiogram revealed a partly-diminished but a persistent thrombus in the LAD (Picture 3).

The echocardiogram revealed hypokinesia of the anterior wall, with a slightly depressed ejection fraction of 40% and moderate mitral regurgitation. The mitral valve was thickened with hyperechogenic vegetations (6-7 mm) on each leaflet revealing Libman-Sacks endocarditis.

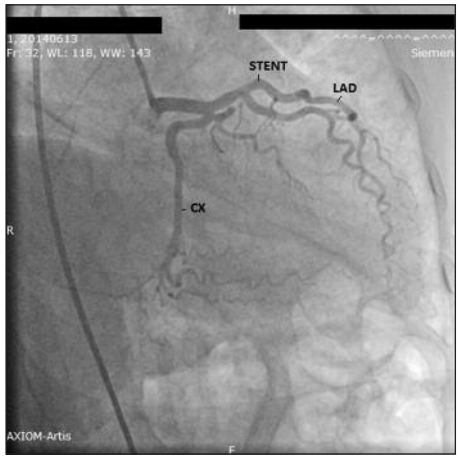

FIGURE 3: Left coronary angiogram after stent implantation to the LAD.

The patient was discharged on medical therapy with warfarin (target INR 2.5), clopidogrel (150 mg for the first week, 75 mg later on) and ASA after a long hospital stay without any ischemic event. The stent was patent in the first year control coronary angiogram.

DISCUSSION

Myocardial infarction with normal coronary arteries is an important subgroup of myocardial infarction with a frequency of at least 3-4% of all myocardial infarctions.[4] 5 to 10% of myocardial infarctions occur in adults ≤40 years of age and approximately 20% of coronary heart disease in young adults is not associated with coronary atherosclerosis. The primary causes other than coronary artery stenosis are coronary artery embolism, thrombosis, anomaly and vessel inflammation or spasm caused by a variety of mechanisms.[1]

Although SLE patients are, in most cases, young women who are considered at low risk for coronary atherosclerosis, they have a 2 to 50-fold higher risk of atherosclerotic heart disease, which is a major cause of premature mortality in this disease.[5] The mechanism responsible for the accelerated atherosclerosis process in SLE patients is multifactorial and not fully understood. There is high prevalence of traditional risk factors for atherosclerosis such as diabetes, dyslipidemia, hypertension, which may be partly secondary to adverse effects of prolonged use of corticosteroids, and intrinsic inflammatory process of the disease must play a central role in this process.[6,7] SLE patients are at a five-fold increased risk for developing coronary heart disease.[1]

In addition to these factors, there is an association of SLE with APS. There is a high prevalence of antiphospholipid antibodies in SLE patients (12-34%).[2] When APS develops in the context of SLE, prevalence of thrombosis, especially deep venous thrombosis of the lower limbs increases. Arterial thrombosis can also occur in these patients, 50% of the time in brain vessels, presenting as stroke or transient ischemic attack, in 23% of the cases, coronary arteries are involved, presenting as acute myocardial infarction (AMI) or angina, and the remaining 27% correspond to other diverse arterial beds.[2,8] The frequency of AMI was found to be 2.8% in a large study of APS patients.[8]

Although acute in situ thrombosis is the most likely mechanism of coronary occlusion causing AMI in this patient, we cannot rule out plaque rupture as the patient has atherosclerotic risk factors, like smoking and prolonged use of corticosteroids and possibility of embolism from noninfectious thrombotic vegetations.

Regarding the chronic approach of these patiens with APS, oral anticoagulation with warfarin associated with ASA use is recommended in cases of arterial thrombosis.[9] There is no evidence for the use of clopidogrel as secondary prophylaxis in these patients and its use was justified only in our patient due to coronary stent implantation.

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