

hematoma and abscess was drained and debrided. Antibiotherapy was maintained for an average of 7 days. The patient was discharged with prescription of an antiagregant therapy.

The patient was referred to our emergency service 2 months later, this time with an infected pseudoaneurysm on the left femoral bifurcation which was diagnosed and managed by surgery using the same technique. The patient was consulted with the department of psychiatry and was referred to an experienced center for treatment of substance dependence.

Discussion

In our case, the patient presented with a femoral pseudoaneurysm, due to infection caused by intravascular non-sterile injection. IV drug abuse is a significant problem of modern societies, with increasing prevalence and subsequently increasing incidence of vascular complications, including infected femoral artery pseudoaneurysms (6, 7).

The optimal management of femoral artery pseudoaneurysms in drug abusers remains a matter of debate, because these lesions are not very common and results in most published series are based on small numbers of patients. Current treatment options include (1) excision and debridement of pseudoaneurysm with ligation of the common femoral artery without revascularization, and (2) excision and debridement of the pseudoaneurysm with routine or selective revascularization (8, 9). The latter requires arterial reconstruction, which is achieved with an autogenous or synthetic graft sited either in situ or extra-anatomically, depending on the size of the arterial wall deficit and presence of contamination. The great saphenous vein has been used as autogenous graft most frequently, although some authors also report successful utilization of femoral and popliteal veins (10). In the present case, due to interrupted infection size of the aneurysm, loss of integrity and diffuse destruction of the femoral artery, aneurysmectomy and interposition with autogenous saphenous vein graft was performed.

Conclusion

In order to prevent morbidity and mortality, early diagnosis, and appropriate surgical intervention carry utmost importance in infected pseudoaneurysms. Not only should these patients with drug abuse be managed by vascular surgery, but also they should be treated for addiction. Although rare, tianeptine may cause addiction which could lead to vascular complications like pseudoaneurysms. Therefore over the counter use of tianeptine in our country is very erroneous which must be reevaluated.

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Right coronary artery spasm-a complication of cardiac pacemaker implantation

Sağ koroner arter spazmı-Kalp pili implantasyon komplikasyonu

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Introduction

Atrial or dual-chamber pacemaker implantation for symptomatic sick sinus syndrome (SSS) represents the mainstay of treatment by providing effective relief of symptoms and lowering the incidence of atrial fibrillation, thromboembolic events, heart failure, and mortality. The procedure is usually associated with low complication rates (1).

Case Report

A 65-year-old woman was admitted in hospital for recurrent syncope due to SSS. She received indication for permanent cardiac stimulation, based on symptoms and Holter monitoring results that revealed multiple episodes of sustained atrial fibrillation and atrial flutter, alternating with sinus bradycardia and sinus pauses of 2-3 seconds (2).

She was a non-smoker patient with medical history of hypertension and dyslipidemia. There was no personal or family history of coronary artery disease.

Clinical examination at hospital admission revealed irregular rhythm with heart rate of 121 beats/min, loud sharp first cardiac sound, opening snap, diastolic rumbling murmur, and systolic murmur at the cardiac apex. Blood pressure was 150/70 mm Hg. Other findings of the physical examination were not clinically significant.

The 12-lead electrocardiogram (ECG) indicated atrial flutter with variable atrioventricular (AV) block degree (2:1, 3:1), eventually converted to sinus rhythm.

Transthoracic echocardiography (TTE) documented a moderate mitral stenosis (mean gradient across the mitral valve of 6.6 mm Hg) associated with mild mitral regurgitation, mild tricuspid regurgitation, pulmonary artery pressure of 40 mm Hg, left atrial enlargement, and normal systolic left ventricular function.

A Talos DR device with two leads (Setrox S53 in right atrium (RA) and Setrox S60 in right ventricle (RV)) was implanted with no complications during procedure (Fig. 1).

One hour after the procedure, the patient developed severe chest pain with diaphoresis. Surface ECG identified significant ST segment elevation in leads II, III, aVF, and deep ST segment depression in leads I and aVL, superimposed on a paced atrial and ventricular rhythm (Fig. 2).

Blood pressure was 100/70 mm Hg associated with heart rate of 63 beats/min. TTE examination showed depressed left ventricular (LV) function (40%) with akinesia of the apical half of the anterior septum and LV apex.

The patient was immediately prepared for catheterization. Coronary angiography indicated severe (90-99%) stenosis of right coronary artery in segment II and III (Fig. 3).

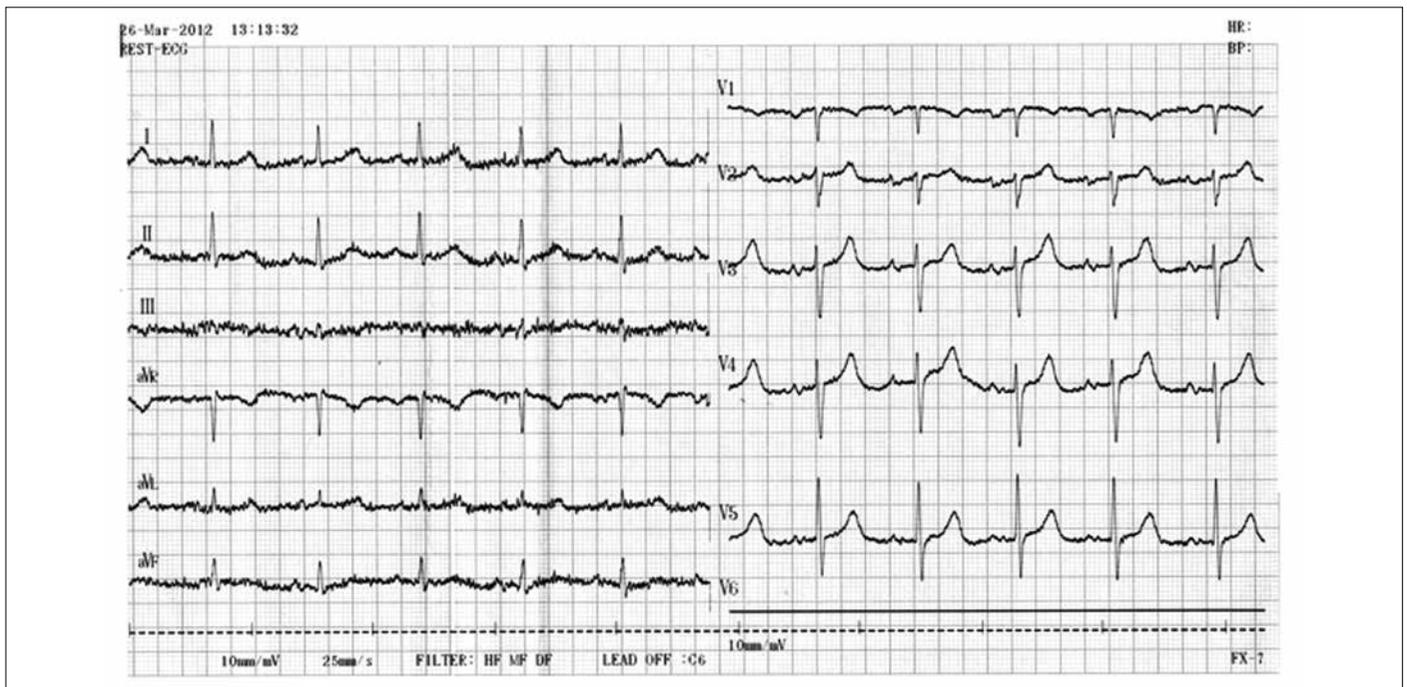


Figure 1. Electrocardiographic tracing after DDD pacemaker implantation

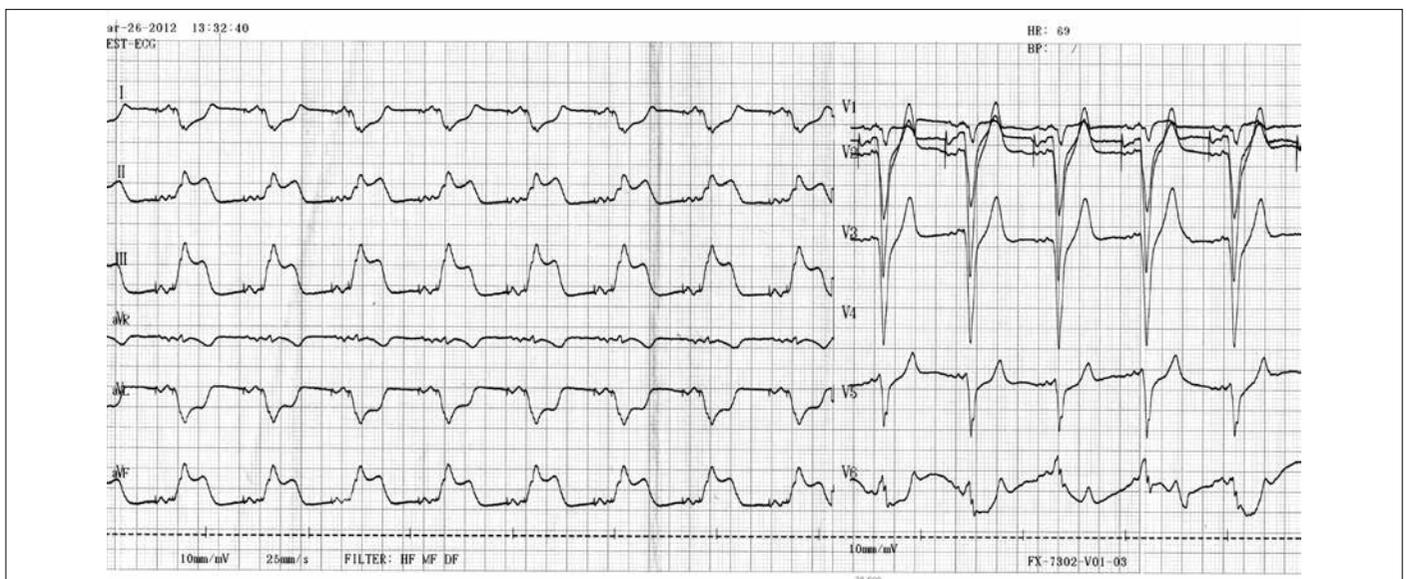


Figure 2. Electrocardiographic tracing during chest pain episode: marked ST segment elevation in leads II, III, aVF and deep ST segment depression in leads I and aVL, superimposed on a paced atrial and ventricular rhythm

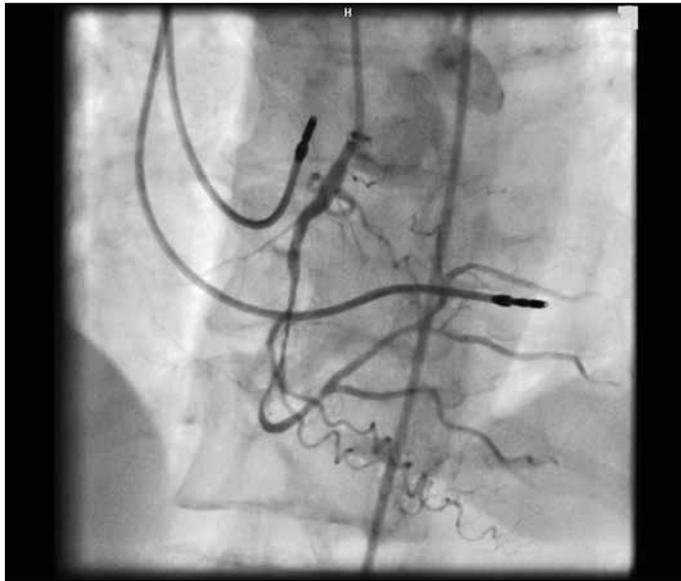


Figure 3. Coronary angiography during chest pain episode: significant and extensive narrowing of the mid-portion of the right coronary artery



Figure 4. Coronary angiography after intracoronary nitroglycerine administration: the relief of the right coronary artery spasm

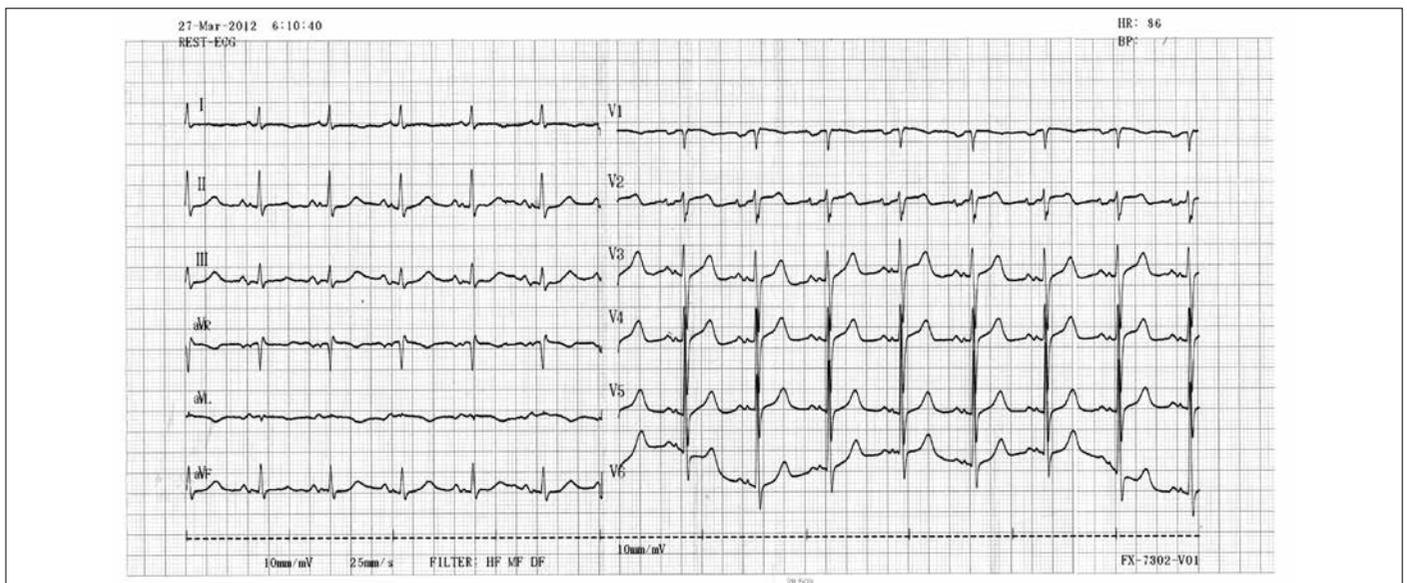


Figure 5. Follow-up electrocardiographic tracing: complete resolution of the ST-T segment changes and no residual abnormalities

After nitroglycerine injection in the right coronary artery (RCA) a normal diameter RCA achieved, with some atherosclerotic plaques but without significant stenosis. The ST segment elevation disappeared (Fig. 4).

The follow-up ECG on next day indicated no ST-T abnormalities, which suggested rapid resolution of coronary stenosis (spasm) (Fig. 5).

Cardiac enzymes remained within normal range (creatine kinase MB fraction 19 U/l- 21 U/l and creatine kinase 43.8 U/l-54U/l).

The patient was eventually discharged free of chest pain.

Discussion

Few cases of ST segment elevation and coronary spasm were reported in relation with cardiac surgery or ablation procedures. The mechanism underlying coronary spasm is not completely understood,

atheromatous or gaseous emboli, direct coronary artery manipulation, elevated catecholamine levels, hyperventilation, alkalosis, hypothermia, the release of platelet derived vasospastic factors such as thromboxane A2, autonomic nerve stimulation, proximity of the right coronary artery to the right atrial ablation lines and hypomagnesemia have all been cited as possible causes (3-6).

Sinus node dysfunction with bradycardia and syncope following RCA spasm have also been reported in the literature, some of the cases having a fatal outcome due to cardiogenic shock (7).

The exact etiology of the spasm in our case is difficult to be established. We can speculate that, right coronary artery spasm was the etiology of the syncope episodes, but the patient never complained about angina. On the other hand, an important component of the spontaneous coronary spasm is the autonomic imbalance and the spasm is likely to appear in patients with normal or with minor and nonobstructive sclerotic lesions (8). This may also

be our case if the withdrawal of the procedure related increased sympathetic tone caused coronary artery spasm followed by bradycardia and hypotension. The recently implanted device however, stimulated the heart and the patient did not developed hemodynamic deterioration.

Conclusion

Although the implantation procedure can be safely performed in a hospital with no catheterization facilities, rapid access to an available invasive catheterization laboratory was beneficial.

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