

Spontaneous coronary artery dissection: a long-term follow-up



Spontan koroner arter disseksiyonu: Uzun dönem takibi

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Introduction

Spontaneous coronary artery dissection (SCAD) is a rare cause of acute coronary syndrome with a high risk of mortality (1). Clinical presentation may vary from mild symptoms to sudden cardiac death. It is more common in women and involves coronary arteries that differ based on gender (2). Although many factors are thought to be known, its etiology is still unclear. In this case report, a patient with SCAD who remained stable during a 34-month follow-up without any intervention is presented.

Case report

A 50-year-old man was presented to emergency department with a feeling of a retrosternal pressure at rest. Pain was recurrent without radiation to the neck and arms. There was no triggered physical or emotional stress. Electrocardiography (ECG) showed negative T waves in leads V5-V6, DII-DIII-aVF (Fig. 1).

Patient's past medical history showed that there was no previous coronary artery disease. Assessment of coronary heart disease risk factors revealed that the patient had no hypertension, diabetes, family history of premature coronary artery disease (CAD) and smoking. Lipid profile was normal except a low level of high-density lipoprotein-cholesterol (HDL-C). Non-traditional risk factors such as homocysteine, lipoprotein (a), high sensitive-C reactive protein (hs-CRP), were also within normal limits. There were no symptoms or signs suggesting any inflammatory or infectious diseases.

At presentation his blood pressure was 100/65 mmHg, pulse was 78 beats/minute and he was in a sinus rhythm. The clinical examination was unremarkable. During the follow-up creatine kinase-MB increased to 62 U/L and cardiac troponin I increased to 0.97 ng/ml (upper normal limit 20 U/L; 0.04 ng/ml respectively). The patient was given metoprolol 100 mg once a day, enoxaparine 80 mg twice a day subcutaneously, nitroglycerine IV infusion once in every 18 hours, acetyl salicylic acid 300 mg once in a day and finally clopidogrel 75 mg once in day. Three hours after the initiation of the therapy, the patient was free of chest pain.

Coronary angiography on the 4th day of hospital stay revealed that a type D spontaneous dissection (Video 1. See corresponding video/movie images at www.anakarder.com) (3) extending from the mid portion to distal of the left circumflex artery's (Cx) first obtuse margin branch with TIMI-III flow beyond the dissection (Fig. 2A). A mild vasospasm was observed proximal to the obtuse marginal artery but there was no atherosclerotic plaque in the entire coronary bed. Percutaneous coronary intervention (PCI) was not planned due to long

segment dissection and absence of recurrent chest pain. Patient was discharged on the 6th day with the same doses of metoprolol, acetyl salicylic acid, and clopidogrel. Additionally 60 mg isosorbid-5-mononitrate was started. Patient was asked to be controlled with coronary angiography three months after the hospital discharge.

One month after the discharge, myocardial perfusion scintigraphy yielded that inferolateral ischemia without any fixed perfusion defect was present. Moreover, control coronary angiography (Video 2. See corresponding video/movie images at www.anakarder.com) showed that neither spontaneous recovery nor progression of the dissection existed (Fig. 2B). By contrast, proximal segment of the obtuse marginal artery was in normal size compared to that of the first angiography. As a result, according to absence of angina and extension of ischemic area beyond circumflex territory, PCI was not planned due to long segment dissection of a small size side branch.

He is still on follow-up for 34 months. Despite the persistent myocardial ischemia in inferolateral region, he has not experienced any major cardiac event or angina both at exercise and at rest.



Figure 1. Admission electrocardiogram shows negative T waves in leads V5-V6 and inferior derivations

Discussion

Spontaneous coronary artery dissection was first described by Pretty in 1931 in an autopsy study of a 42-year old woman with sudden cardiac death (4). It is a very rare cause of acute coronary syndromes. Its presentation spectrum could vary from mild exercise angina to serious myocardial infarction, cardiogenic shock or even sudden cardiac death. Nearly 70% of the SCAD cases are diagnosed in post-mortem studies caused by sudden cardiac death (2).

Spontaneous coronary artery dissection is more common in women than in men (3:1). Approximately 25% of the cases seen were middle-aged women during pregnancy or in the puerperium period. In addition, oral contraceptive use, i.e., progesterone in particular, is thought to have a role in the etiology (1, 2).

Involved coronary arteries differ according to the gender. While left anterior descending (LAD) artery and Cx artery are more commonly involved (87%) in women; right coronary artery (RCA) is the most commonly involved (67%) in men (2). However, independent from the gender, overall LAD is affected in 75% of cases and RCA is affected only in 20% of cases. Also, left Cx is affected in about 4% of cases and the rate for LMCA involvement is less than 1% of cases (5).

Although, SCAD is more commonly occurred during pregnancy, various other factors are also found to play a role in its etiology. There is a special class of SCAD, which develops on a preexisting atheromatous plaque (6). Studies with intracoronary ultrasound (IVUS) have shown that the rupture of the plaque starts bleeding between media and intima leading to the separation of these layers and eventually causing the dissection (7). However, it has been postulated that atheromatous origin of the SCAD has a better prognosis than other origins of SCAD due to well developed collaterals in these patients (6-7).

In this case, plain coronary angiography did not reveal any atherosclerotic plaque in the entire coronary artery; however, confirmation with IVUS was necessary to rule out atherosclerotic etiology (7). In order to conclude that there was no atherosclerotic plaque in certainty, IVUS should have been used. Unfortunately, at the time when the angiography was performed, IVUS was not available.

Therefore, it was not used. On the other hand, despite the unavailability of IVUS, lacking of either traditional or non-traditional risk factors for atherosclerotic disease supports that it could have non-atherosclerotic origin. Again, it is important to note that lacking of traditional or non-traditional factors does not necessarily rule out or exclude other possible sources.

Treatment options for SCAD vary from medical treatment to different coronary revascularization methods. Due to its rare occurrence, there are no studies comparing the effectiveness of the different treatment approaches.

In some cases who presented with ST segment elevation acute myocardial infarction, thrombolytic treatment was used. It is believed that the fibrinolysis of the blood clot formed in the false lumen is beneficial (8). However, due to the potential risk of worsening of the dissection by increasing the hemorrhage, fibrinolysis is usually not recommended if dissection was diagnosed visually. Vasodilating drugs including nitrates, calcium channel blockers are suggested to be used to decrease the vasospasm and prevent the further extension of the dissection (2).

Percutaneous transcatheter stent implantation is preferred in patients with a single vessel disease, which is not involving the LMCA, especially in localized and not extended dissections (9). Usually multiple overlapping stents are required to cover the dissected area.

Even though coronary bypass surgery is the therapeutic intervention of choice in dissections, its use is limited because usually the dissection extends further than that is shown on the angiogram, making it difficult to place the graft in the dissected area. Therefore, DeMaio et al (10) suggested using coronary bypass operation only in cases with left main involvement, three-vessel dissections or presence of recurrent ischemia.

In this case, thrombolytic therapy was not considered due to absence of acute ST- segment-elevation myocardial infarction at the time of admission. Coronary bypass operation was also not considered due to absence of left main coronary artery or multiple vessel dissections. As being the most beneficial therapeutic option, especially in one-vessel dissections, percutaneous coronary stent implantation was not considered for this case despite presence of ischemia limited to



Figure 2-A. Diagnostic left circumflex coronary artery selective coronary angiography. Spontaneous dissection in the 1. obtuse marginal branch is pointed with white arrow

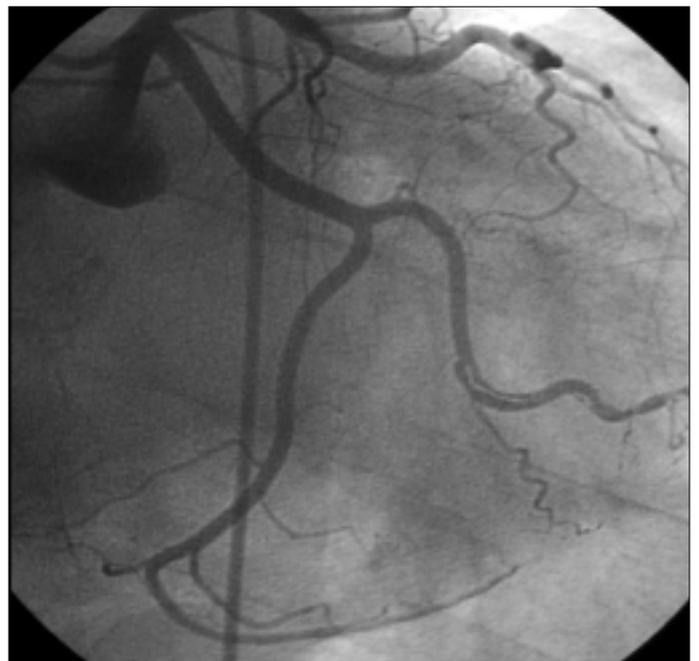


Figure 2-B. Control angiography after 3 months shows spontaneous coronary artery dissection in the same branch without any progression

dissection area in myocardial perfusion scintigraphy. Stent implantation was not thought to be appropriate because of long segment dissection of a small size (<2mm) side branch coronary artery dissection, which would require multiple overlapping stent implantations. In addition, absence of angina and lacking of ischemic area extension encouraged to do continuing follow-ups under medical therapy.

Conclusion

Spontaneous coronary dissection should be kept in mind as one of the possible causes of acute coronary syndromes even though it rarely exists. Although there is no definitive guideline for optimal treatment of SCAD, from the experiences of reported series in the literature, it is suggested that medical treatment with close follow-ups for stable patients with a preserved good left ventricular function could be beneficial.

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Pacemaker lead failure due to crush injury

Ezilme hasarına bağlı kalıcı kalp pili elektrod kusuru

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Introduction

Different techniques for pacemaker implantation such as subclavian, cephalic and transiliac vein are being used today for various circumstances (1, 2). One of the potential complications of pacemaker implantation is the lead fracture or insulation failure due to crush injury. It usually occurs after medial intrathoracic puncture of the subclavian vein and results in damaging of the pacemaker lead body by entrapment within the costoclavicular ligament and/or the subclavian muscle (3, 4). The present case report describes a patient who underwent pacemaker implantation seven years ago and developed lead failure due to crush injury detected by chest X-ray and telemetry data.

Case report

A 33-year-old woman with a history of sick sinus syndrome underwent a dual chamber pacemaker implantation (Ela DR213 Talent, Ela 4068 for atrial, and BT46D for ventricular leads) seven years ago. Because of battery depletion the pulse generator was replaced with a Guidant 1296 generator. Pacing threshold, R wave and impedance of ventricular lead during implant were 0.7 V at 0.5 msec, 7 mV and 350 ohms, respectively. Pacing threshold, R wave and impedance of atrial lead were 1.2 V at 0.5 msec, 3 mV and 650 ohms, respectively. During

follow-up telemetry data revealed intermittent sensing and pacing problem with the atrial lead. Measured impedance values were changing day by day between 540 and 1320 ohms, as shown in Table 1. Intermittent major changes in impedance values were suggestive of a lead malfunction, namely fracture that might be related to crush injury. The intracardiac electrocardiogram recordings revealed multiple artifacts and noise in the atrial channel (Fig. 1). A chest X-ray showed partial thinning and damage to the atrial lead body right at the medial puncture site of the subclavian vein (Fig. 2). All of these findings together with sensing failure probably reflected a partial fracture in the lead body, which was not detected during the implant. Since the patient had good intrinsic sinus rhythm at a rate of 55 bpm and potential complication risk during a subsequent lead revision, which was also refused by the patient, we reprogrammed the generator to VVIR mode that was well tolerated by the patient during follow-up.

Discussion

Subclavian vein puncture is commonly performed to insert the lead for permanent pacemakers and implantable defibrillators. Intrathoracic subclavian vein approach is performed in more than 65% of all endocardial leads (5). However, this medial puncture technique is potentially responsible for increased risk of lead fracture, pneumothorax and