Olgu Sunumu (Case Report)

Raynaud Fenomeninde Ani Ölüm İçin Nadir Bir Patoloji: Dirençli Koroner Vazospazm

A Rare Pathology For Sudden Death in Raynaud Phenomenon: Resistant Coronary Vasospazm

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Geliş tarihi: 25.06.2019 **Kabul tarihi:** 04.11.2019 **DOI:** 10.17517/ksutfd.582155

Özet

Vazospastik angina, genellikle geçici ST segment elevasyonu ile seyreden ve spontan ya da nitratlar ile sonlanan anjina pektoris tipidir. Bazen göğüs ağrısı ile bazen de kardiyak arrest gibi daha ciddi bir klinik yelpazede karşımıza çıkabilir. Çevresel faktörlerin hastalığın tetiklenmesi üzerindeki etkisi göz ardı edilmemelidir. Çoğu hastada semptomlar medikal tedavi ile kontrol altına alınabilir. Medikal tedavinin ana omurgası kalsiyum kanal blokerleri ve vazodilatör ilaçlardır. Biz de bu yazımızda, dirençli koroner osteal spazmı olan bir hastamızı sunmayı amaçladık.

Anahtar Kelimeler: Vazospastik angina, miyokard enfarktüsü, ventriküler fibrilasyon, raynaud fenomeni

Abstract

Vasospastic angina is a type of angina pectoris, often associated with transient ST-segment elevation, resolves usually spontaneously or with nitrates. It may be presented with chest pain, sometimes with a more serious clinical spectrum such as cardiac arrest. The impact of environmental factors on disease triggering should not be overlooked. In most patients, symptoms can be controlled with medical therapy. The main backbone of medical therapy are calcium channel blockers and vasodilator drugs. In this article, we aimed to present a patient with persistant coronary osteal spasm.

Key words: Vasospastic angina, myocardial infarction, ventricular fibrillation, raynaud phenomenon.

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INTRODUCTION

Vasospastic angina is a type of angina pectoris with transient ST segment elevation in circadian rhythm, usually resolving spontaneously or with nitrates. In these patients, symptoms associated with ischemia can often be controlled by medical therapy but malign ventricular arrhythmia induced by ischemia may develop in some patients. Here, we will present a case with ventricular fibrillation developing vasospastic angina after coronary angiography.

CASE PRESENTATION

A 41-year-old female patient was admitted to the emergency room with chest pain. In her histroy she has hypertension but not regularly taking medications, Raynaud phenomenon (diagnosed 3 years ago) and followed for 2 years without medication. An angiography had been performed about 6 months ago with a complaint of similar chest pain. A non-occlusive coronary artery disease in the mid region was found in left anterior descending artery (LAD). Electrocardiography (EKG) showed sinus rhythm at 104 / min heart rate, on precordial derivation 1-6 ischemic t wave negativity and prolonged QT **(Figure 1)**. Echocardiography revealed that there were normal ejection fraction. With high troponin value the patient was taken to the angiography laboratory after the onset of chest pain. Cardiopulmonary resuscitation was performed after sudden cardiac arrest before angiography was performed. EKG turned to sinus rhythm after defibrillation. Coronary angiography showed 99% stenosis in osteal LAD **(Figure 1)**.

Figure 1. Electrocardiographic findings

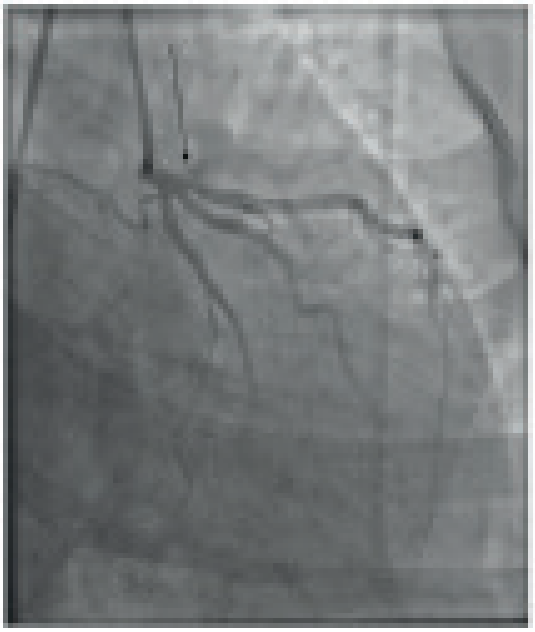

Figure 2. Coronary angiographic findings

Variant angina was considered in the patient because she had Raynaud phenomenon. Four hundred mcg nitrate was administered to the patient intracoronary way. It was seen that his spasm improved **(Figure 2)**.

The patient is medicated with 100 mg/day acetylsalicylic acid, 75 mg/day clopidogrel, 20 mg/day atorvastatin, 120 mg diltiazem two divided doses, 40 mg/day isosorbide mononitrate for 2 days. Then the VVI- intracardiac device (ICD) was implanted and patient discharged. After 4 days, the patient presented with a typical chest pain similar to first, ST segment elevation in the AVR derivation on electrocardiography **(Figure 2)**. The patient was medicated with a second generation calcium channel blocker drug, amlodipine 10 mg 1x1 and a dose titration to 10 mg 2x1 per a day. By this way, it was aimed to take advantage of both anti-anginal and anti-hypertensive effect. Isosorbide mononitrate was then increased from 40 mg once daily dose to wice a day. In the control examination, after 1 month the patient was found to have no symptoms and clinically stable and medicated with diltiazem 90 mg twice a day and amlodipine 10 mg once a day. The patient is now on follow-up for a year and during this time he did not have any angina attacks or malign arrhythmias. Diltiazem 90 mg once a day and amlodipine 10 mg nce a day were continued as medication.

DISCUSSION

Although rare in connective tissue diseases, involvement of the coronary arteries is a well known feature. Raynaud phenomenon involves especially upper extremite digital arteries but the presence of microvascular circulation disorder in the coronary arteries may cause vasospazm and angina pectoris. Raynaud phenomenon may affect visceral organs; esophageal spasm may be seen, pulmonary hypertension may develop because of pulmonary vascular bed involvement, coronary

vasospasm when coronary artery involvement and migraine when cerebral vessels affected (1). The presence of dyspnea in patients with Raynaud phenomenon may be a sign of pulmonary hypertension or coronary vasospasm (2). Approximately 24% of patients with variant angina are accompanied by the Raynaud phenomenon, which is important to emphasize the importance of vasospastic disorder in these patients (3). It is thought that the pathogenesis is mainly due to the disproportion of vasodilator and vasoconstrictor substances released from the endothelium dominated by the atherosclerotic process (4,5). The angina, which develop independently of exercise, can be triggered in the morning with sympathetic discharge peak. Management of patients with vasospastic angina is quite difficult. The results of patients treated with long-acting calcium channel blockers and nitrates are more satisfactory and these patients are more fortunate in terms of survival (6). However, symptoms persist in about 20% of patients receiving drug therapy (7). In these patients, coronary artery spasm results in severe ventricular arrhythmias and subsequent cardiac arrest. Angina attacks continued while our patient was being medicated with diltiazem and nitrate. Current guidelines do not provide a definitive treatment model for patients with persistant symptoms. Increasing the dose of both long-acting calcium channel blockers and nitrates is the essential of treatment. In addition, a road map that is generally accepted by the authorities has been identified and sudden cardiac death has been accepted as the most dangerous situation. Using implantable cardioverter defibrillator (ICD) to eliminate the fatal conditions that may occur in these patients is suggested (8-10). In conclusion, there are many reasons that may lead to persistant vasospastic angina in patients with connective tissue diseases as seen in our case. Patients with vasospasm may experience myocardial infarction, ventricular arrhythmias and sudden death. Because of the frequent occurrence of severe coronary spasm-related malignant arrhythmias, VVI-ICD should be implanted to prevent sudden death in these patients. Our case is differs from other cases in the current literature that she was using two different type of calcium channel blockers. We believe that this case may guide clinicians in the management of vasospastic angina in the future.

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