



Multi-vessel coronary vasospasm in a patient with excessive inguinal pain

Aşırı inguinal ağrısı olan bir hastada çok damar koroner vazospazm

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ABSTRACT

Coronary vasospasm is a frequent cause of chest pain. Multi-vessel coronary vasospasm increases the risk of severe ischemia, myocardial infarction, ventricular arrhythmia, and cardiogenic shock compared with single-vessel coronary vasospasm. The exact reasons for coronary artery vasospasm are uncertain. Several factors play a role in the development of coronary vasospasm. Here we present the case of a patient with multi-vessel coronary vasospasm, which occurred because of excessive inguinal pain.

Keywords: Coronary spasm, variant angina, inguinal pain, multi-vessel

ÖZ

Koroner vazospazm göğüs ağrısının sık nedenlerinden biridir. Çok damar koroner vazospazm tek koroner vazospazm ile karşılaştırıldığında ciddi iskemi, miyokard enfarktı, ventriküler aritmi ve kardiyojenik şok riskini artırmaktadır. Koroner vazospazmın gerçek nedenleri belirsizdir. Koroner vazospazm oluşumunda çeşitli faktörler rol oynamaktadır. Bu vakada aşırı inguinal ağrı nedeniyle oluşan çok damar koroner vazospazm hastası sunacağız.

Anahtar Kelimeler: Koroner spazm, variant angina, inguinal ağrı, çok damar

INTRODUCTION

Variant angina is an important cause of chest pain and is characterized by non-exertional angina pectoris with ST segment elevation on electrocardiography. Ischemic episodes are more frequently observed between mid-night and early morning hours. These can lead to myocardial infarction, ventricular arrhythmias, and sudden cardiac death. Here we present the case of a patient with variant angina pectoris with multi-vessel coronary vasospasm.

CASE PRESENTATION

A 58-year-old male patient was admitted to our clinic with the complaint of chest pain. His chest pain was like a sensation of numbness, usually beginning after mild exertion, localized to the retrosternal area, radiated to the inter-scapular region and left arm, and lasting for

1–2 minutes. The same chest pain sometimes occurred in early morning hours and during extreme emotional stress. He was a smoker and had a history of hypertension for 2 years. His medical treatment included 25 mg metoprolol and 2.5 mg ramipril per day. His physical examination revealed normal findings. His electrocardiogram was in sinus rhythm with low QRS amplitudes in extremity leads (≤ 0.5 mV) and heart rate was 93 beats/minute. Transthoracic echocardiography revealed normal left ventricular function (ejection fraction: 65%). To evaluate the coronary artery disease, a non-invasive exercise stress test was performed. At the first stage of the test, the patient complained of chest pain like a numbness sensation on the retrosternal area, radiating to the back and left arm. Because chest pain was disturbing, the exercise test stopped at 6th minute. The chest pain relieved within the 1st minute of the recovery phase and was not accompanied by ischemic electrocardiographic changes. The patient underwent coronary angiography

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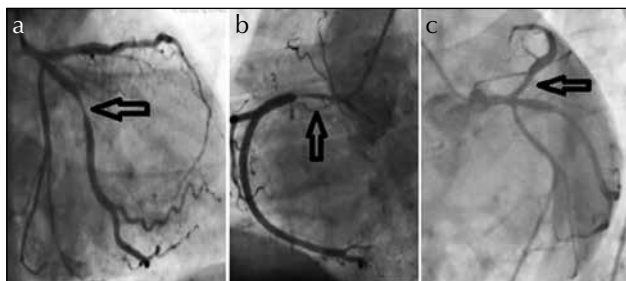


Figure 1. a-c. Arrows show critical lesion in intermediate artery (a) and right coronary artery (b) and non-critical lesion in left anterior descending artery (c)

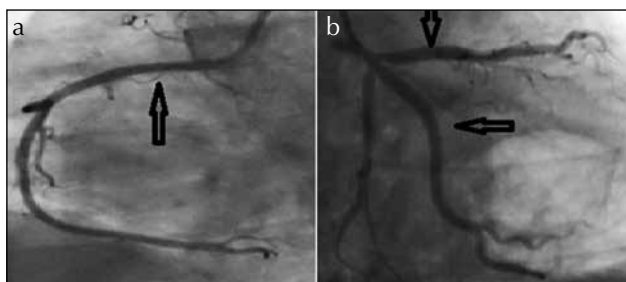


Figure 2. a, b. Six hours after diagnostic angiography, arrows show spontaneous resolution of critical and non-critical coronary lesions (a-b)

examination because of typical chest pain and cardiovascular risk factors. Our patient had lower pain threshold, so before femoral cannulation higher than normal dose of local anesthetic was administered to the right femoral region. However despite intense local analgesic administration, he complained of excessive pain. During coronary angiography, critical stenosis was detected in the mid-level of the intermediate artery (Figure 1a) and proximal portion of the right coronary artery (Figure 1b). There was a non-critical lesion in the proximal part of the left anterior descending artery (Figure 1c). Then, a percutaneous coronary intervention was planned for critical coronary lesions. A 300 mg oral loading dose of clopidogrel was administered to the patient. After 6 hours, the patient was taken to the coronary angiography laboratory again. Initially, we aimed to exclude a possible coronary spasm, and when we engaged a diagnostic catheter into the right coronary ostium, we saw that the right coronary artery was completely normal, confirming the presence of a coronary spasm (Figure 2a). Then, we displayed left coronary circulation and again observed that both the intermediate artery and left anterior descending arteries were normal (Figure 2b). We confirmed that the lesions in the left anterior descending, intermediate, and right coronary artery were all spasms. We switched the beta-blocker with a calcium channel blocker (amlodipine) and added nitroglycerin to the patient. The frequency of chest pain decreased after medical therapy.

DISCUSSION

Coronary artery vasospasm is a frequent cause of chest pain. Multi-vessel coronary artery spasm increases the risk of severe ischemia, myocardial infarction, ventricular arrhythmia, and cardiogenic shock compared with single-vessel coronary spasm (1, 2). The exact reasons for coronary artery vasospasm are uncertain. In many cases with coronary artery vasospasm, possible mechanisms were not identifiable. One of the proposed mechanisms for coronary spasm is endogen nitric oxide synthase deficiency (3). Nitrite oxide deficiency increases the susceptibility to potent vasoconstrictor mediators such as angiotensin II and endothelin I. Other precipitating factors are enhanced phospholipase C and Rho kinase activity (4, 5). They regulate the intracellular calcium level and cause smooth muscle constriction. Increased activity of these proteins was observed in patients with vasospastic angina pectoris. Vasospastic angina is also observed in patients with personality disorders those with hyperventilation, tobacco and cocaine users, those with magnesium deficiency, and those with an imbalance between sympathetic and parasympathetic systems. In addition, it has been reported that the withdrawal of medications such as calcium channel blockers and nitrates causes multi-vessel coronary spasm (6, 7). The most commonly used drugs for the treatment of vasospastic angina are nitrates and calcium canal blockers. In addition, rho kinase inhibitors, statins, and magnesium can be used. However, non-selective beta-blockers and acetylsalicylic acid should not be used in patients with coronary artery spasm due to an increased risk of vasospasm.

In the present case with multi-vessel coronary artery spasm, spontaneous resolution of coronary vasospasm was observed. We know that in some cases with multi-vessel spasm, a more serious result such as ventricular fibrillation and cardiogenic shock was observed. Our patient had a lower pain threshold; therefore, before femoral cannulation, higher than the normal dose of local anesthetic was administered to the right femoral region. However, the cannulation procedure was not completely painless. The most possible mechanism for coronary spasm in our patient may be increased sympathetic activity due to pain. Another important point was that our patient had undergone beta-blocker therapy for a long time due to coronary artery disease or hypertension. It is important to evaluate the clinical characteristics of chest pain and its main cause before beginning therapy. Because our patient had non-exertional chest pain, particularly in the morning hour, the possible diagnosis was variant angina.

CONCLUSION

Coronary vasospasm may involve more than one coronary artery and may occur because of excessive pain sensation, which demonstrates the importance of adequate analgesia before and during the procedure. All physicians performing coronary angiography should keep in mind that at least some of the stenotic images may be due to spasms, which need to be tested with intracoronary nitrate.

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