Multivessel Vasospastik Angina with ST Segment Elevation and Total AV Block

ST Segment Elevasyonu ve Total AV Blok'un Eşlik Ettiği Çoklu Damar Vasospastik Anjina

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Abstract

A 49-year-old female patient was admitted to our hospital with ST segment elevation in the inferior derivations (D2, D3, aVF) and a total atrioventricular block. The initial electrocardiography performed on the patient in the coronary intensive care unit showed no total AV block and no ST segment elevation. The patient was accepted as a transient ST segment elevation MI and a coronary angiogram was performed. The coronary angiogram showed critical lesions in the left main coronary artery (LMCA) and the right coronary artery (RCA). We thought that the patient's condition might have been caused by vasospasm and gave intracoronary nitroglycerin. Subsequently, we noticed that the vasospasm disappeared and formed TIMI-3 flow again. The patient's chest pain disappeared. The patient's diagnosis was determined to be vasospastic angina, and she was given treatment accordingly. The patient was discharged, and the chest pain did not recur at 1 year follow up.

Keywords Acute myocardial Infarction; Coronary Angiography; Coronary Artery Disease

Öz

49 yaşında bayan hasta, elektrokardiyografisinde inferior derivasyonlarda (D2-D3 ve aVF) ST segment elevasyonu ve total atrio-ventriküler blok olması nedeni ile hastanemize başvurdu. Hastanın koroner yoğun bakımda çekilen elektrokardiyografisinde ST segment elevasyonlarının gerilediği ve total atrio-ventriküler bloğun kaybolmuş olduğu görüldü. Hasta geçici ST eleve myokard enfarktüsü olarak kabul edildi ve koroner anjiyografisi yapıldı. Koroner anjiyografide sol ana koroner arter (LMCA) ve sağ koroner arterde (RCA) kritik lezyonlar görüldü. Hastanın durumunun vazospazma bağlı olabileceği düşünülerek intrakoroner nitrat yapıldı. Vazospazmın yok olduğu ve TIMI-3 akımın tekrar oluştuğu görüldü. Hastanın göğüs ağrısı ortadan kalktı. Hastaya vazospastik angina tanısı konularak tedavisi başlandı. Hasta taburcu edildi ve hastanın 1 yıl süresince yapılan takiplerinde göğüs ağrısı tekrarlamadı.

Anahtar kelimeler *Akut myokardial enfarktüs; Koroner anjiyografi; Koroner arter hastalığı*

INTRODUCTION

Coronary artery occlusion due to atherosclerotic plaque laceration or erosion is typically observed in patients who undergo coronary angiography (CAG) because of acute myocardial infarction (AMI).[1] In patients who undergo CAG because of chest pain, 20–30% are found to have normal coronary arteries.[2] This percent is higher especially in younger patients and women. In 1959, Prinzmetal et al. defined a syndrome with chest pain that is secondary to myocardial ischemia and occurs mostly during rest, that is not provoked by emotional stress or physical exercise, and occurs with transient ST segment elevation. This syndrome, referred to as variant angina pectoris, is found to be associated with AMI, ventricular tachycardia (VT), ventricular fibrillation (VF), and sudden cardiac death. The hypothesis of Prinzmetal et al. is temporary increase in coronary vasomotor tonus or vasospasm. Vasospasm causes ischemia by the excessive and temporary narrowing of the epicardial coronary artery lumen. This happens without any precipitating factor increasing myocardial oxygen consumption. The decrease in lumen diameter during vasospasm can occur in the angiographically normal or diseased coronary artery and responds to treatment with nitroglycerin. Patients with variant angina pectoris are usually young and smokers. The periods of angina generally exist between midnight and the morning hours and can sometimes occur two or three times in 30–60 minutes. The capacity of exercise is not restricted in these patients. The certain diagnosis of Prinzmetal angina is based upon positive provocative tests with angiographically normal coronary arteries in patients with chest pain and ST segment elevation. In most patients, at least one major coronary artery has proximal stenosis during the heart attack. This happens most frequently in the right coronary artery and then left anterior descending artery or together.

Our case presented at the emergency department of our hospital with severe, retrosternal chest pain that had begun one day before. They showed a total atrioventricular (AV) block with normal cardiac markers and ST segment elevation in the inferior derivations. This case, rarely seen in the literature, is reported as variant angina with multi-vessel vasospasm in the CAG that disappears by the introduction of intracoronary nitrate. Informed constent was obtained from the patient.

CASE

A 49-year-old female patient was admitted to our hospital emergency service with severe chest pain that had begun the day before and had increased 15 minutes prior. The chest pain was located retrosternal and spread around the patient's neck. Her initial electrocardiogram (ECG) had ST segment elevation in the inferior derivations (D2, D3, aVF), ST segment depression in the reciprocal derivations (D1-avL), and a total AV block (Figure 1).

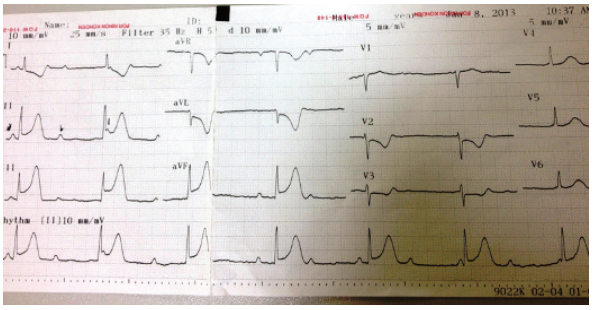

Figure 1: The initial ECG of the patient showing ST segment elevation in the inferior derivations and a total AV block

The patient was hospitalized in the coronary intensive care unit with a diagnosis of acute inferior myocardial infarction (MI) and a total AV block. The ECG done in the coronary care unit showed no total AV block and no ST segment elevation (Figure 2), and the echocardiography revealed no wall motion abnormalities. The patient was accepted as having transient ST segment elevation MI, and a coronary angiography was performed. The patient's medical history included a 40-pack/year smoking habit and coronary artery disease. She had undergone CAG due to a myocardial infarction in 2011 and again in 2012. In the CAG performed in 2011, the left main coronary artery (LMCA) had 95% stenosis in the proximal segment and a circumflex artery, and the right coronary arteries had

noncritical stenosis. A stent was implanted in the LMCA.

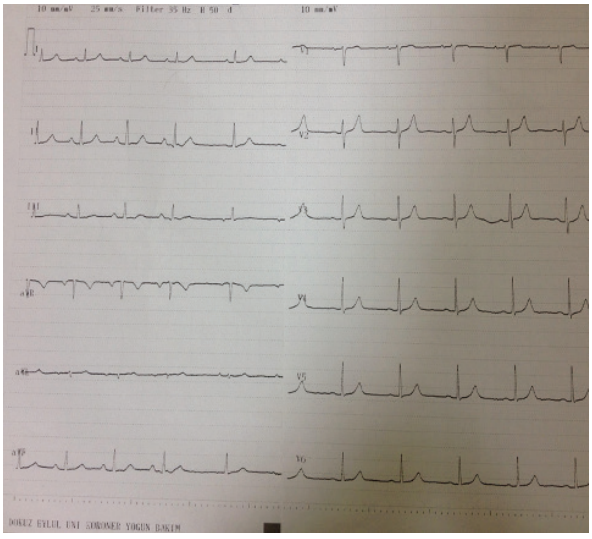

Figure 2: The intensive coronary care unit admission ECG showing no ST segment elevation and a total AV block

The following year, a stent was implanted to the RCA because of MI. She had family history of coronary artery disease. In physical examination, her blood pressure was 110/70 mmHg, and her heart rate was 82/min and rhythmic; all other findings were normal. The results of the blood tests, including a hemogram, glucose levels, and cardiac markers, were normal. A transthoracic echocardiogram revealed no wall motion or valvular abnormalities. A CAG was performed: the LMCA had 80% stenosis starting from the mid-segment of the prior stent, the LAD had only plaque, the CX had 40% stenosis in the mid region, and the RCA had 80–90% stenosis after the prior stent (Figure 3, 4). Intracoronary nitrate was applied to the LMCA and RCA lesions. After treatment, the lesions in these vessels disappeared and TIMI-3 flow occurred (Figure 5, 6). The patient was discharged with calcium channel blockers and nitrates.

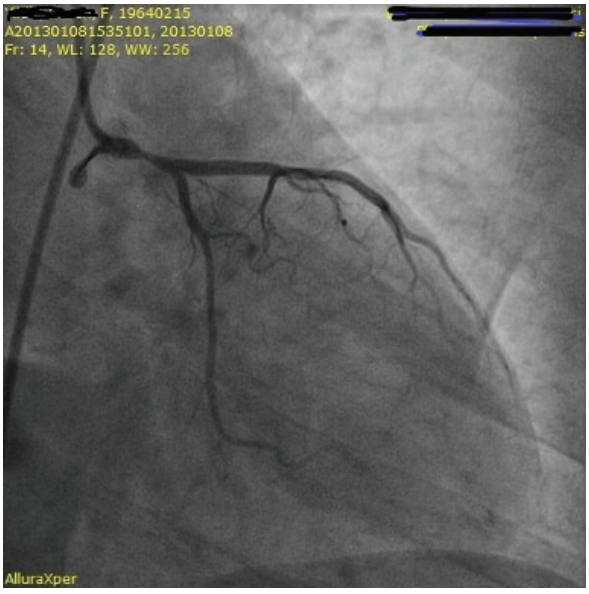

Figure 3: The coronary angiogram, shown here in the left caudal view, appears to demonstrate a significant lesion in the left main coronary artery

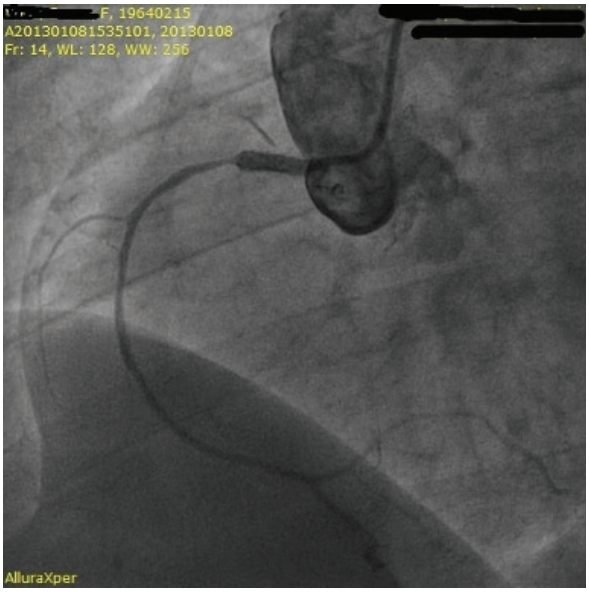

Figure 4: The coronary angiogram, shown here in the left anterior oblique view, appears to demonstrate a significant lesion in the right coronary artery

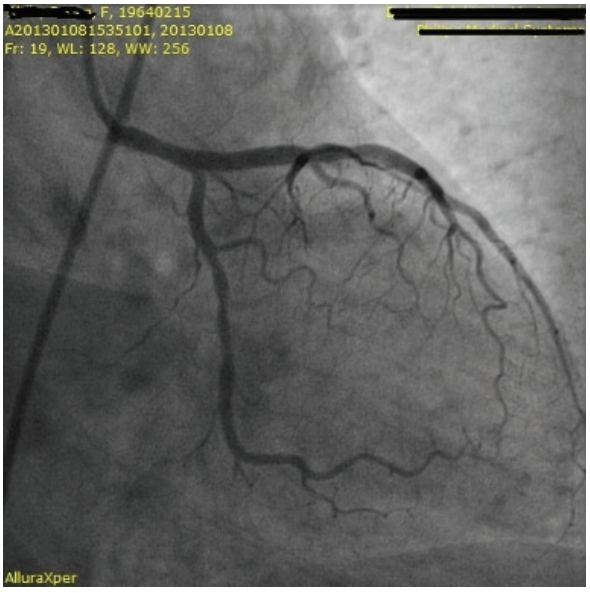

Figure 5: The coronary angiogram, shown here in the left caudal view, left main coronary artery after intracoronary nitrate application

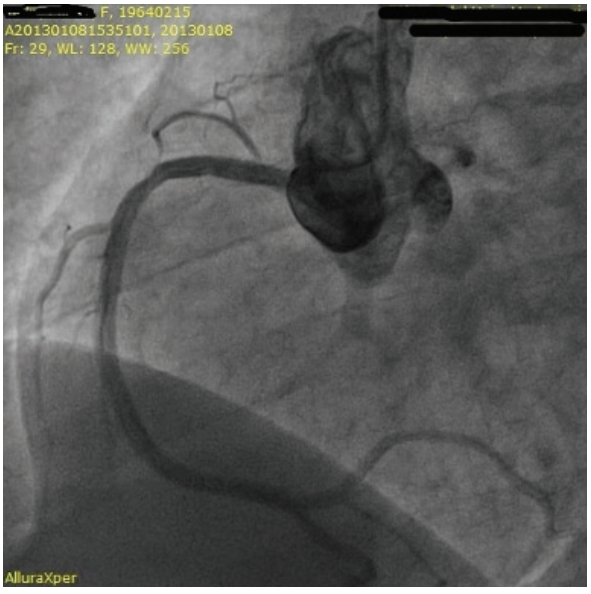

Figure 6: The coronary angiogram, shown here in the left anterior oblique view, right coronary artery after intracoronary nitrate application

DISCUSSION

Prinzmetal's angina, also called variant angina, is a syndrome characterized by spontaneous episodes of angina that are related to focal spasms of an epicardial coronary artery, which can result in severe myocardial ischemia.[3] Cenk and friends presented a case who had a serious LAD lesion disappeared after intracoronary nitrate.[4] Prinzmetal's angina also can be related to spasm of grafting artery. Jong Hyun and collegues had reported a case who experienced native coronary artery spasm and grafted artery spasm following CABG, which was successfully treated with intracoronary injection of nitroglycerine.[5] While significant coronary artery lesions are found in most patients who undergo urgent CAG because of acute coronary syndrome, in some patients the coronary arteries are found to be normal. It has been reported that the probability of normal coronary arteries is higher in young people and women.[6] Germing et al. reported that 76 of 821 patients who underwent CAG because of acute coronary syndrome had normal coronary arteries. Thirteen percent of these patients had ST segment elevation, thirty percent of them had pozitive troponin result and sixty percent of them could not been diagnosed specifically. Only a patient underwent percutan coronary intervention because of acute myocardial infarction during 1 year follow-up.

Coronary vasospasm usually cause variant (prinzmetal) angina, but also cause unstable angina pectoris, acute myocardial infarction and sudden cardiac death. In the study of Bertrand and friends.[7] Vasospasm was found most frequent in right coronary artery (% 50). Coronary vasospasm was reported 14-15 percent in the studies with patients who underwent CAG because of AMI and had normal coronary arteries.[6] Ergonovine provocation test can be applied to show up coronary vasospasm, although the sensitivity of ergonovine provocation test is very high, it's sepecificity is low, but sometimes provocation tests can make the situation worse. Mancio and collegues presented a case who have chest pain and developed ST-segment depression and transient grade 3 atrioventricular block

after injection of acetylcholine into the left anterior descending artery and ended with cardiac arrest and death.[8] The patients who have coronary vasospasm have generally good prognosis but also they have positive troponin results. The rate of non-fatal AMI is high in patients who have coronary vasospasm provocating with ergonovine.

Smoking is a major risk factor for coronary vasospasm. Smoking both increases platelet agregation and causes catecholamine release so it causes coronary vasospasm.[9] In our case; the patient is a heavy smoker. Sympathomimetic amines are vasoconstrictive agents whose usage of them can cause AMI by coronary vasospasm. Tiramine, amphetamine, ephedrine, xylometazoline, pseudoephedrine, phenylephrine and phenylpropanolamine are the main ones. In a study which involves over 160.000 patients used pseudoephedrine, AMI was dedected in six patients (1.2/100.000) and this rate was found higher according to general population (0,9/100.000), also a case who had AMI because of phenylpropanolamine was reported.[10] Our patient was not using any of them. We couldn't find any provocating factor except smoking in our patient.

Vasospastic angina can usually be controlled by vasodilators such as calcium antagonists and nitrates. Lifestyle modification, including smoking cessation and alcohol restriction is also required. Coronary stenting could be used some rare situaitions because of angina which refractory to optimal medical therapy. Our patient had not been taking any calcium channel blockers and nitrates. We gave to the patient benidipine and nitrate to avoid the recurrence of attacks.

In conclusion, we reported a case of multivessel spasm that occurred during diagnostic coronary angiography. It should not be forgotten that smoking could provocate coronary vasospasm and cause AMI so vasospastic angina should be remembered in young and smoker patients who applies with chest pain to emergency department.

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