Vasospastic angina: nocturnal chest pain, eosinophilia and delayed diagnosis

Vazospastik angina: Gece göğüs ağrısı, eosinofili ve gecikmiş tanı

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Introduction

Although the complaint of chest pain is frequently encountered in adolescents, vasospastic angina is very rarely seen in this age group (1, 2). Therefore, patients presenting with these complaints may receive incorrect treatment in clinics. The definite diagnosis of vasospastic angina, which can lead to acute myocardial infarction and sudden death, is extremely important (2).

Case Report

The 10-week history of an 18-year-old male patient revealed chest and epigastric pain, occurring particularly at night. The patient was admitted to a state hospital with the complaint of pain. He was hospitalized in the internal medicine service for three days. The electrocardiogram (ECG) was ignored although it showed ST-segment elevation in leads of II, III and aVF and on derivations of V4-6 and consequently he was treated only with an antiulcer drug (Fig. 1). Since his complaints continued during 3 days, he was transferred to our hospital for further examination and evaluation in the gastroenterology unit. In the emergency service of our hospital, ECG showed symmetrical, deep, negative T waves in leads I, II, III, aVL, aVF and V3-6 derivations and positive T waves in V1-2 and aVR derivations, considered as findings showing ischemia. Q waves were not found in all derivatives (Fig. 1). Creatine kinase-MB and troponin I were found to be respectively 63 U/L and 29.5 ng/ml. At full blood count, except a high eosinophil count (7.94%, normal range; 0.9% - 6.0%), no important findings were detected. We learned from the patient's story that he had been diagnosed with bronchial asthma and had had a childhood allergic tendency.

The patient was determined to have acute coronary syndrome and he was hospitalized in coronary care unit. Echocardiographic examination showed hypokinesia of the inferior, posterior and lateral walls. Cardiac markers which were examined again, showed a decline. Oxygen therapy, iv nitrate and heparin, oral acetylsalicylic acid and proton pomp inhibitor treatments were administered and thrombolytic treatment was not given.

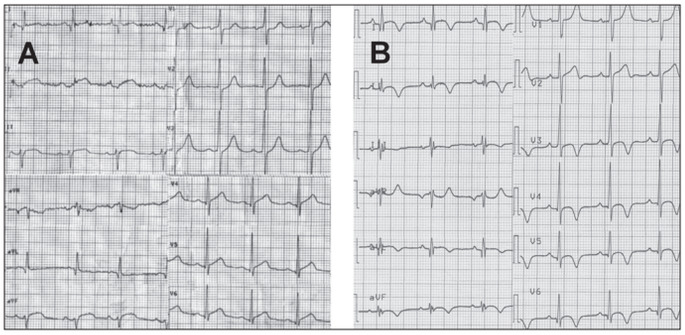

Figure 1. The electrocardiogram shows ST segment elevation in leads II, III and aVF and V4-6 derivations in the recording obtained at state hospital (A) and symmetrical, deep, negative T waves in all derivations except aVR and V1-2 in the our hospital's emergency room recordings (B)

Coronary angiography was performed on the same day. No atherosclerotic plaque, coronary slow flow or coronary dissection were found in the left anterior descending, circumflex and right coronary arteries and coronary angiography was accepted as normal. Then, calcium channel blocker (diltiazem 90 mg 2x1) was added to his therapy. Coronary provocation testing was not done because the patient was in myocardial infarction process. According to Universal Definition of Myocardial Infarction, our patient was accepted as type 2 myocardial infarction (3). The general condition of the patient improved, and he was discharged from the hospital with calcium channel blocker (diltiazem 90 mg 2x1) and low-dose acetylsalicylic acid (100 mg/day). Although the patient received appropriate treatment during monitoring, his complaints recurred two weeks later. Oral corticosteroid therapy (prednisolone 1 mg/kg/day) was added to the treatment, and all complaints of the patient improved with this drug. This therapy was continued for 2 weeks and subsequently the dosage was gradually decreased and 2 weeks later the medication was completely ceased. The left ventricular regional wall motion was determined normal during echocardiography tests performed during follow-up.

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Discussion

Variant angina is generally seen at the age of 40 years and above, and the incidence of the disease in adolescence, as in our case, has been reported very rare (1, 2, 4). At the time of chest pains ST segment elevation is determined generally and cardiac enzymes are generally normal. Chest pains generally occur at night, as in our patient (5). Again, rising cardiac enzymes and impaired regional wall motion may be observed in patients diagnosed late and with long-term complaints, as observed in our patient.

Several publications have shown a correlation between allergy and coronary spasms (6, 7). Mast cell infiltration in coronary artery spasm has been seen in the pathology of patients with variant angina (8). Beta-blockers can increase coronary artery tone and arrhythmia frequency, for this reason, calcium antagonists and nitrates are used in the initial treatment of such patients (9). The administration of corticosteroids is particularly recommended for patients exhibiting resistance against calcium antagonist treatment (10). Corticosteroid treatment has been reported as being more useful in patients with high eosinophil levels, as in our case (7).

Conclusion

The correct and early diagnosis of coronary spasm related acute coronary syndrome may prevent fatal complications. Corticosteroids may be a good choice for refractory vasospastic angina treatment, particularly when the patient has an allergic tendency.

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Diffuse left anterior descending coronary artery dissection with aortic coarctation

Aort koarktasyonu ile yaygın sol ön inen koroner arter disseksiyonu

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Introduction

Coarctation of the aorta can present in approximately 6 to 8 % of patients with congenital heart disease. It is more common in males. The vascular malformation responsible for coarctation is a defect in the media of the aorta, giving rise to a prominent posterior infolding (1). Coarctation of the aorta usually presents later in childhood as systolic hypertension. Most of these patients are asymptomatic. Some patients complain of lower extremity claudication with exercise or frequent headache. We present a case of diffuse left anterior descending coronary artery (LAD) dissection at the time of initial diagnosis of native aortic coarctation.

Case Report

A 40-year old man patient administered to the emergency department with the complaints of severe headache and palpitation lasting six hour. He had exercise dyspnea starting one week ago. He had no previous health problems, no family history of premature coronary artery disease and he did not drink alcohol and smoke. His physical examination revealed right and left arm blood pressures of both being 160/ 90 mmHg. Pulse rate was 82 beats/minute regular in upper extremity and lover extremity pulses were absent bilaterally. On cardiac auscultation there was a 2/6 systolic murmur over the 4th and 5th intercostal spaces as well as on the back interscapular space.

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