

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 lower 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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His 12-lead electrocardiogram (ECG) showed a regular sinus rhythm and ST segment elevation in leads V2- V6 and pathological Q waves (Fig. 1). Laboratory results did not show signs of typical myocardial necrosis. Serum low density lipoprotein-cholesterol, high density lipoprotein-cholesterol and triglyceride levels were found to be 142 mg/dl, 28 mg/dl, and 148 mg/dl, respectively. Doppler echocardiographic examination showed regional wall motion abnormalities (apical aneurysm) of the left ventricle and low ejection fraction (48%). Posteroanterior radiography demonstrated rib-notching. The aortic segment was not prominent as a result of tubular hypoplasia of the aortic isthmus. He underwent coronary angiography. Coronary angiography showed ectatic left main coronary artery and diffuse dissection of LAD following 1st diagonal branch (Fig. 2). On aortography, coarctation of the aorta and severe obstruction distal to the subclavian artery were detected (Fig. 3).

Chest computed tomography (Fig. 4) showed the aortic coarctation and post-stenotic dilatation clearly. Based on these results, a diagnosis

of LAD dissection induced by aortic coarctation was considered. Patient underwent both coronary artery bypass surgery and repair of the severe aortic coarctation. He has done well in his follow-up.

Discussion

Most individuals with coarctation of aorta have usually nonspecific symptoms, related either to upper extremity hypertension (headaches) or to reduced blood supply to the lower extremities (exercise-induced claudication) (2). Coronary artery disease manifests early in patients with aortic coarctation than others. The leading causes of atherosclerosis are hypertension and endothelial dysfunction. The other mechanism for coronary artery disease with coarctation of aorta is activation of sympathetic nervous system. Sympathetic activation increases myocardial contractility and increases systemic pressure that helps maintain perfusion of descending aorta and abdominal organs (3). In coronary artery disease, the addition of coarctation further increases both left ventricular afterload and myocardial oxygen demand. There are several long- term studies of survivors of coarctation (4-9). The most common cause of death in patients with coarctation of

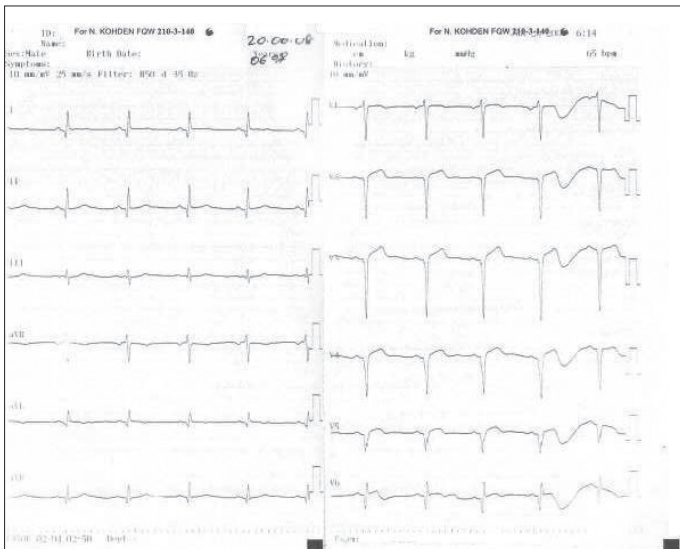


Figure 1. A 12- lead electrocardiogram shows ST segment elevation in leads V2-V6

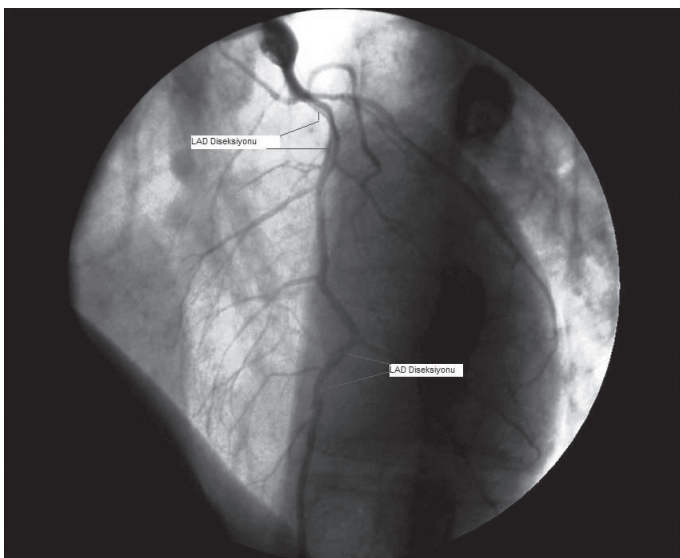


Figure 2. Coronary angiography view of diffuse left anterior descending coronary artery dissection

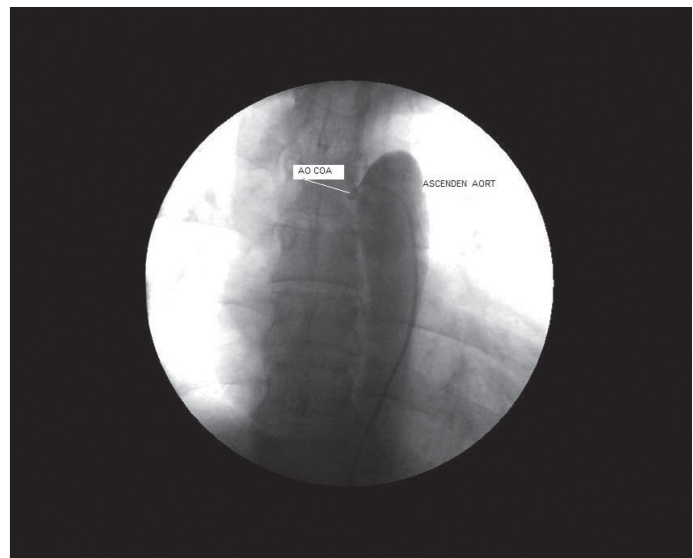


Figure 3. Aortography view of coarctation of aorta and severe obstruction distal to the subclavian artery

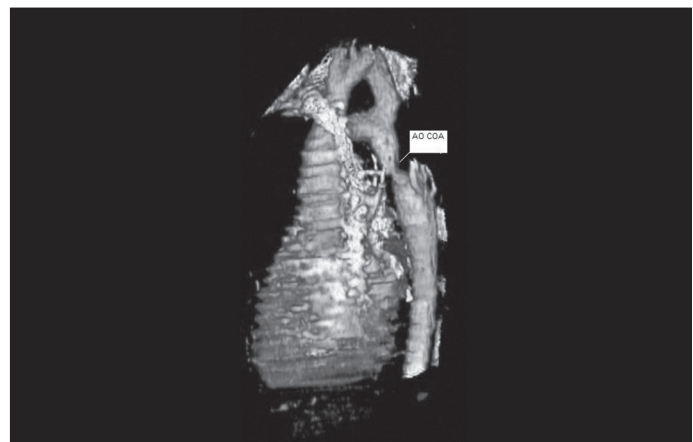


Figure 4. Computed tomography view of aortic coarctation and post-stenotic dilation

aorta includes coronary artery disease. Coronary artery disease seems to be accelerated in these patients (5). Although the etiology of this accelerated atherosclerotic heart disease is unknown, a number of factors probably play a role. Hypertension is a known risk factor for the development of coronary artery disease (6). There must be factors other than hypertension, because even considering the higher incidence of hypertension, the incidence of coronary artery disease is much higher than one would expect. Some individuals with coarctation of the aorta are known to have altered structure and function of both the coronary and other systemic arteries. Chen et al. (7) described severe atherosclerosis and calcification in internal mammary arteries of two patients with previous coarctation repair who required coronary artery bypass surgery (7). In clinical practice, the development of dissection in a patient with coronary artery disease is common immediately after balloon angioplasty because of intimal tearing. No spontaneous coronary dissection in a patient with coarctation of aorta was reported in literature.

Conclusion

Early coronary lesions should be sought in the follow-up of patients with native aortic coarctation.

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Intractable atrial flutter presented with severe bradycardia in an infant

Süt çocukluğu döneminde ciddi bradikardi ile ortaya çıkan tedaviye yanıtız atriyal flutter olgusu

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Introduction

Atrial flutter is an uncommon arrhythmia in newborns and infants. Generally, it leads to tachycardia and is not associated with congenital heart disease in neonatal period. In most cases, it is easily converted to sinus rhythm by medical or electrical cardioversion. After resolution of the arrhythmia, the long-term prognosis is excellent. However, some patients may have intractable atrial flutter despite to the treatment.

We report a 3 months of age infant who presented with severe bradycardia and diagnosed as atrial flutter without response to treatment that has not previously been reported.

Case Report

The 3 months of age infant with bilateral congenital cataract was referred to our department because of bradycardia. In physical examination, the heart rate was 40 beats per minute (bpm), respiratory rate was 60 per minute, blood pressure 85/60 mmHg and bilateral cataract formation was detected. Electrocardiography (ECG) revealed bradycardia with a ventricular rate of 57 bpm and intraventricular conduction block (QRS: 0.12 s) (Fig. 1). Atrial flutter waves were not detected on the 12-lead ECG. Mild cardiomegaly was observed on chest X-Ray (cardiothoracic ratio, 0.56). The echocardiogram revealed a

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