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Coronary artery anomalies in patients with syndrome X

Sendrom X'li hastalarda koroner arter anomalileri

To the Editor

Cardiac syndrome X (CSX) is characterized by typical angina, abnormal exercise test results, and no critical coronary stenosis (1). Coronary artery anomalies (CAAs) are a group of congenital disorders resulting in a highly variable clinical phenotype (2). There is a growing interest in the incidence of these anomalies and in their role in the manifestations of ischemic heart disease (3). To our knowledge, however, the role of structural cardiovascular abnormalities in the pathogenesis of syndrome X remains unclear. In the present study, we sought to investigate the presence and frequency of CAAs in patients with CSX.

We reviewed the records of 50 patients with CSX who had undergone exercise electrocardiogram test and coronary angiography at the Uludağ University Cardiovascular Laboratories between 2003 and 2005. The following variables were collected: time of 1-mm ST depression, onset of angina, maximum ST segment shift, number of leads (derivations) with ST depression observed at peak exercise, and exercise duration in multiples of resting O₂ consumption (METs). All patients underwent coronary angiography using the standard Judkins technique.

A total of 27 patients (54%) with CAAs were identified. The following anomalies were detected in our cohort: rudimentary right coronary artery (10 cases), left anterior descending artery (LAD) terminating before the cardiac apex (9 cases), intermediary artery (6 cases), and LAD and circumflex arteries appearing to arise from separate ostiums (2 cases). We found a trend for a lower prevalence of males in subjects with CAAs (48.1%) compared to those without (78.2%, $p = 0.06$). Cardiac syndrome X patients with CAAs showed a shorter time to 1 mm-ST depression compared to those without (6.2 ± 1.8 min versus 7.2 ± 1.8 min, $p = 0.04$). The maximum ST-segment shift at peak exercise was similar in both groups. However, a higher number of leads with ST-segment depression was evident in CSX patients with CAAs compared to those without (4.3 ± 1.0 versus 3.5 ± 0.9 respectively, $p = 0.005$). No other clinical or ergometric differences were evident.

Notwithstanding the limited sample size, our results show that more than half of CSX patients have CAAs. Since the number of leads with ST depression and time to 1-mm ST depression may have a prognostic significance in patients with ischemic heart disease (4), our data suggest that the presence of CAAs may predispose CSX patients to a worse clinical outcome compared to those without. Although subject to future confirmation, our results indicate that CAAs may play a role in the pathogenesis of CSX and related symptoms. Our study may open new avenues of investigation into the pathophysiological basis of this clinical entity.

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Effects of metoprolol and diltiazem on plasma homocysteine levels in patients with isolated coronary artery ectasia

İzole koroner arter ektazili hastalarda metoprolol ve diltiazem'in plazma homosistein düzeylerine etkisi

Coronary artery ectasia (CAE) is defined as localized or diffuse non-obstructive lesions of the epicardial coronary arteries with a luminal dilation exceeding the 1.5-fold of normal adjacent segment. Recent investigations have documented higher homocysteine levels in patients with coronary artery ectasia (CAE) (1). Beta-blocker therapy has been shown to decrease the homocysteine levels in hypertensive patients in two studies (2, 3). We aimed to investigate the effects beta-blockers and calcium channel blockers on plasma homocysteine levels in patients with CAE.

The CAE patients ($n=60$, 32 men, mean age 55 ± 11 years) were randomized into two groups, metoprolol (group 1; $n=30$, 19 men, mean age 53 ± 9 years) and diltiazem (group 2, $n=30$, 13 men, mean age 57 ± 10 years).

The baseline levels of homocysteine, folate and vitamin B12 were measured in two groups. Group 1 received metoprolol succinate 50-100 mg once daily. Group 2 received diltiazem SR 90-120 mg twice daily. The plasma homocysteine levels were measured at the baseline and at the end of 3 months in each group. After a washout period of 3 weeks the patient groups were crossed over for further 3 months. Then group 1 received diltiazem SR 90-120 mg twice daily and group 2 received metoprolol succinate 50-100 mg once daily for 3 months. The plasma homocysteine levels were measured at the end of second 3 months in each group.

In group 1, there was no significant change in homocysteine levels at the end of the 3rd month of metoprolol use when compared to baseline levels (15.6 ± 4.5 $\mu\text{mol/l}$ versus 14.5 ± 5.3 $\mu\text{mol/l}$ $p=0.14$), there was a significant decrease in homocysteine levels at the end of 3rd month in patients taking diltiazem when compared to baseline homocysteine levels (15.6 ± 4.5 $\mu\text{mol/l}$ versus 13.5 ± 4.6 $\mu\text{mol/l}$ $p=0.015$) and there was no significant difference in homocysteine levels between 3rd and 6th months (14.5 ± 5.3 $\mu\text{mol/l}$ versus 13.5 ± 4.6 $\mu\text{mol/l}$ $p=0.21$). In group 2, there was a significant decrease in homocysteine levels at the end of 3rd month of diltiazem use when

compared to baseline homocysteine levels ($12.9 \pm 5.4 \mu\text{mol/l}$ vs $11.7 \pm 3.6 \mu\text{mol/l}$ $p=0.045$), there was also a significant decrease in homocysteine levels at the end of 3rd month of metoprolol use when compared to baseline homocysteine levels ($12.9 \pm 5.4 \mu\text{mol/l}$ vs $10.4 \pm 3.1 \mu\text{mol/l}$ $p=0.011$) and there was a significant difference in homocysteine levels between between 3rd and 6th months ($11.7 \pm 3.6 \mu\text{mol/l}$ vs $10.4 \pm 3.1 \mu\text{mol/l}$ $p=0.010$).

In accordance with previous findings (2-5), we found that beta-blocker therapy significantly decreased homocysteine levels in patients with CAE. Additionally, we have showed for the first time that calcium-channel blocker therapy significantly decreased homocysteine levels in patients with CAE.

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A case with Wolf-Parkinson-White syndrome first presented with a devastating event: aborted sudden cardiac death

İlk bulgusu ani ölüm olan Wolf-Parkinson-White sendromu tanılı bir olgu

Wolf-Parkinson-White (WPW) syndrome is a cardiac conduction disorder that can present with potentially life-threatening consequences. Sudden death very rarely occurs as the initial presentation of WPW syndrome. Sudden cardiac death (SCD) is defined as a natural and unexpected death due to cardiac causes that occurs within 1 hour of onset of symptoms (1). Wolff-Parkinson-White syndrome is a cardiac conduction disorder that can present with potentially life-threatening consequences. Sudden death rarely occurs as the initial presentation of WPW syndrome (2). Unpredictable lethal complications were found in the asymptomatic WPW patients, creating uncertainty whether these patients should receive aggressive evaluation and interventional therapy.

In this case report, we presented a female children with WPW syndrome admitted to our hospital with sudden cardiac death as an initial finding of her disease.

A 13-year-old female patient admitted to our emergency department due to sudden loss of consciousness when walking in the school garden for ceremony rehearsal activity. She said to her friends that she had palpitation and chest pain just before the loss of consciousness. The headmaster immediately called out an ambulance. In about 5 minutes, ambulance arrived at the school. Ambulance paramedics reported that when they arrived, the patient was in cardiopulmonary arrest; they applied cardiopulmonary resuscitation. The patient was brought from the school to our emergency department in about 10 minutes by the ambulance. During the transportation, paramedics kept on doing cardiopulmonary resuscitation. The physical examination in the emergency department revealed that the patient was unconscious and had cardiovascular instability with clinical evidence of low cardiac output. Electrocardiography revealed ventricular fibrillation. Ventricular fibrillation was treated promptly with electrical defibrillator (1 joule/kg) and the patient returned to normal sinus rhythm. Arterial blood gas analysis revealed severe metabolic acidosis and HCO₃ replacement was performed. Intravenous fluid and dopamine infusions were started and the patient was referred to pediatric intensive care unit from emergency room. She did not have any previous history of drug usage, trauma or any infection. Before this event, she did not experience any episode of syncope, palpitation or chest pain. Family history was unremarkable. Mechanic ventilatory support was provided in intensive care unit. On physical examination at the admission to pediatric intensive care unit, the patient was unconscious with the Glasgow score of 4 (E1M3V_e). Heart rate, arterial blood pressure and body temperature were 120/min, 110/70 mmHg, 36,7 °C respectively. Pupils were isochoric; direct and indirect pupil light reflexes were positive on both eyes. There was not any sign causing suspicion of trauma. On cardiac examination, S1 and S2 were normal and there was no heart murmur. The physical examination findings of other systems were unremarkable. For brain edema, developed secondary to hypoxia, head cooling was applied. Blood ions, renal and liver function tests were all within normal limits. Computed brain tomography was normal. The electrocardiogram showed sinus rhythm with ventricular pre-excitation (short PR interval, wide QRS complex with delta wave) (Fig. 1). Prediction of accessory pathway from the resting surface electrocardiogram (ECG) according to QRS polarity was left-lateral. Because QRS polarity was positive on leads V1 and III. On the first day of hospitalization, generalized tonic-clonic convulsions were observed and antiepileptic treatment was started. Patient needed prolonged mechanical ventilatory support of 30 days due to Adult respiratory Distress Syndrome (ARDS) developed secondary to aspiration pneumonia. Due to prolonged need for respiratory support and inefficient cough and gag reflex, patient underwent open surgical tracheostomy. Patient was fed via nasoduodenal catheter then via gastrostomy. During follow up, Glasgow score improved from 4 to 8 (E4M3V1). Unfortunately, patient had severe neurological sequelae. On physical examination 40 days after admission, there was spasticity on all extremities, more prominent on upper extremities. Deep tendon reflexes were increased in all extremities. These findings were consisted with first motor neuron lesion. Baclofen, phenytoin and physical therapy program was prescribed for the bedridden patient. Cranial magnetic resonance imaging revealed multiple foci of hypointense lesion on T1-weighted images and hyperintense lesion on T2-weighted images and these findings were consisted with hypoxic ischemic encephalopathy. During follow-up, we did not observe any arrhythmia including atrial fibrillation. Electrophysiologic study (EPS) and ablation was planned.

Although SCD rarely occurs at youth, when it does, it is a devastating event for both the family and the medical community. The SCD is defined as a natural and unexpected death due to cardiac causes that occurs within 1 hour of onset of symptoms (1).

The WPW syndrome is a recognized cause of sudden death. Sudden death rarely occurs as the initial presentation of WPW syndrome (2). The exact prevalence of sudden death in WPW syndrome is unknown.