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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