



**Figure 3. First degree AV block on the ECG performed on the seventh day of hospitalization**

AV – atrioventricular, ECG - electrocardiogram

In conclusion, third degree heart block complicating hyperthyroidism is a rare situation. This situation may be a rare cause of palpitation in patients with hyperthyroidism. Therefore, before treatment, complete AV block should be suspected and evaluated with an ECG recording.

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## A case of acute rheumatic fever presenting with syncope due to complete atrioventricular block

### *Tam atriyoventriküler blok nedeni ile senkopla başvuran akut romatizmal ateş vakası*

Cases with acute rheumatic fever (ARF) are usually admitted to cardiology clinics with signs and symptoms of polyarthritis, pericarditis or heart failure. In these cases, the mostly observed electrocardiographic finding is the prolongation of PR interval (1). Rarely, second degree, third degree atrioventricular (AV) block and bundle branch blocks can also happen. In these cases with ARF; syncope, due to complete AV block is very rare and not frequently described. In the present study, we report a case with ARF admitted to our clinic with a history of syncope.

A 17 years old female patient was admitted to our emergency clinic with a history of syncope two or three hours before. She had an upper respiratory tract infection two weeks ago. She complained of bilateral

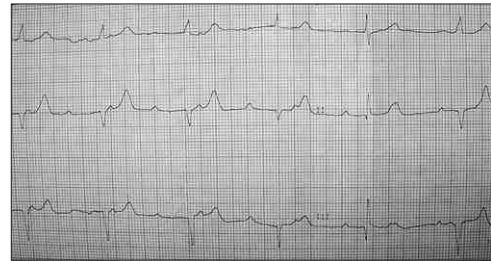
ankle pain aggravated with motion. The electrocardiogram analysis revealed complete AV block (37 beat/min) and width of QRS complex was not more than 0.1 second (Fig. 1). Cardiac auscultation revealed the systolo-diastolic murmurs at the left sternal border and apex. Both bilateral ankles were tender but no redness or swelling were noted. Initial laboratory examination revealed a white blood cell count of 13,000/ml, sedimentation rate of 85 mm/h, C-reactive protein: 132 mg/dl, Antistreptolisin O (ASO): 870 Todd units (normal<200). Other biochemical parameters were normal. Echocardiographic examination was normal except minimal mitral regurgitation.

A diagnosis of ARF was made on the basis of carditis, arthralgia, high erythrocyte count,

sedimentation rate, high ASO and a history of upper respiratory tract infection. A temporary pacemaker was implanted on the day of admittance. Penicillin G procaine 800000 twice a day and aspirin 100 mg/kg/day were ordered. Type 1 second degree AV block and then first- degree AV block (PR: 0,28 sec, rate 72 beat/min) were observed on the second and third days of the admittance respectively. On the fifth day, there was a normal sinus rhythm with a normal PR interval (PR 0.20 sec, rate 88 beat/min). Ankle pain and chest pain subsided after the first and second day of the therapy, respectively. Pericardial friction rub resolved completely on the third day. The temporary pacemaker was removed on the fourth day. She was discharged on the 12<sup>th</sup> day.

The most common manifestation of ARF is polyarthritis. A pain of pericarditis, new onset murmur, pericardial friction rub and heart failure symptoms can be observed as initial ARF symptoms. Cases with a complete AV block are rarely observed (2-4). Reasons of the conduction disturbance are not well known but are attributed, in part, to an increased vagal tone (5). It has been suggested that the site of vagal hypertonia may be in the vagal center of the medulla, but there is evidence that this excessive nerve endings of heart. Besides this, inflammation of the atrioventricular node and the His bundle may be cause of AV block.

This data showed that syncope could be the first or the most dominant clinical manifestation of ARF. Other clinical signs may be indistinct. The ARF should be remembered in young patients presenting with syncope and AV block.



**Figure 1. Complete atrioventricular block observed on the admission electrocardiography**

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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 O<sub>2</sub> 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 ( $6.2 \pm 1.8$  min versus  $7.2 \pm 1.8$  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 ( $4.3 \pm 1.0$  versus  $3.5 \pm 0.9$  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  $55 \pm 11$  years) were randomized into two groups, metoprolol (group 1;  $n=30$ , 19 men, mean age  $53 \pm 9$  years) and diltiazem (group 2,  $n=30$ , 13 men, mean age  $57 \pm 10$  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 3<sup>rd</sup> month of metoprolol use when compared to baseline levels ( $15.6 \pm 4.5$   $\mu\text{mol/l}$  versus  $14.5 \pm 5.3$   $\mu\text{mol/l}$   $p=0.14$ ), there was a significant decrease in homocysteine levels at the end of 3<sup>rd</sup> month in patients taking diltiazem when compared to baseline homocysteine levels ( $15.6 \pm 4.5$   $\mu\text{mol/l}$  versus  $13.5 \pm 4.6$   $\mu\text{mol/l}$   $p=0.015$ ) and there was no significant difference in homocysteine levels between 3<sup>rd</sup> and 6<sup>th</sup> months ( $14.5 \pm 5.3$   $\mu\text{mol/l}$  versus  $13.5 \pm 4.6$   $\mu\text{mol/l}$   $p=0.21$ ). In group 2, there was a significant decrease in