Lev's disease: insidious enemy of conduction system

İleti sisteminin sinsi düşmanı: Lev hastalığı

A 60-year-old previously hypertensive gentlemen presented with progressive dyspnea. Electrocardiography showed atrial fibrillation with left bundle branch block. Cardiac catheterization revealed normal coronary arteries, severe aortic and mitral regurgitation. Despite mild calcification of aortic valve we noted a band-like calcification arising from aortic valve, extending into and outlining the bundle of His, and probably proximal part of the left bundle (Fig. 1, 2, Video 1. See corresponding video/movie images at www.anakarder.com).

Lev's disease is often accompanied by aortic valve calcification that may invade the bundle of His, the right and/or left bundle branch. Progressive loss of myocytes lead to conduction defects including atrioventricular and bundle branch blocks.



Figure 1. Left anterior oblique projection aortography view of the mild calcification of the aortic valve and a band-like calcification extending into and outlining bundle of His



Figure 2. Aortography view of a mild calcification of the aortic valve and a band-like calcification extending into and outlining bundle of His, left anterior descending artery (LAD), right coronary artery (RCA) and aortic valve

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A frequently overlooked etiology of negative precordial T wave: solitary papillary muscle hypertrophy

Prekordiyal negatif T dalgasının sıklıkla ihmal edilen bir nedeni: İzole papiller kas hipertrofisi

Negative T wave on precordial leads is one of the most frequently encountered electrocardiographic (ECG) abnormality. Their significance depends on the electrocardiographic location, temporal evolution, and reversibility.

A 43-year-old male patient without any symptom presented with inverted T waves on ECG during a routine check-up. The ECGs showed persistent inverted T waves in lateral derivations (Fig. 1). On echocardiographic examination, left ventricular and right ventricular systolic and diastolic functions were within normal limits. Both ventricles were normal in thickness and had no wall motion abnormality. However, the posterior papillary muscle was thick in apical four-chamber view (Fig. 2). Its echogenicity has also been increased, compared to the septum and the adjacent lateral wall. The routine biochemistry, whole blood test, coronary angiography and myocardial perfusion scintigraphy were normal.

The T wave is the most variable part of the ECG. Its morphology is influenced by body position, respiration, hyperventilation, drugs, and myocardial ischemia or necrosis. Papillary muscle hypertrophy has been considered as a new echo-electrocardiographic syndrome. After review of the literature, the patient was diagnosed as solitary papillary



Figure 1. The electrocardiogram revealing negative, tented T-waves in leads V4-6. Extremity derivations mostly reveal biphasic or flat (aVL) T waves

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Figure 2. Two-dimensional echocardiographic apical 4-chamber view of the left ventricle with hypertrophic papillary muscle (dimension 25x12 mm)

muscle hypertrophy, which had been defined as diameter of at least one papillary muscle more than 1.1 cm. It is suggested that this entity is a subtype of hypertrophic cardiomyopathy.

Given the fact that the papillary muscles are frequently neglected during echocardiographic examinations, we recommend examining the papillary muscle diameter in all patients with electrocardiographic repolarization abnormalities.

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A case of Ebstein anomaly and biventricular noncompaction

Ebstein anomalisi ve biventriküler "noncompaction" olgusu

Ebstein anomaly and noncompaction affecting both ventricles are very rarely seen together in adults. A 21 years old male patient was referred to our center from a regional hospital, because of a systolic murmur determined during physical examination. We found Ebstein anomaly, moderate tricuspid regurgitation and hyper-trabeculation of both ventricular apexes with blood flow among the recesses during transthoracic echocardiographic examination (Philips I33) (Fig. 1 and Video 1. See corresponding video/movie images at www.anakarder.com). Interestingly, parasternal and apical views suggested a papillary muscle anomaly not confirmed on two-dimensional transthoracic echocardiography. We used three-dimensional transthoracic echocardiography. We used three-dimensional transthoracic echocardiography (Philips I33, X3-1 probe), which revealed a very prominent two-headed posteromedial papillary muscle, a rudimentary anterolateral papillary



Figure 1. Two-dimensional echocardiography view of blood flow between the recesses



Figure 2. Three-dimensional echocardiographic view of a prominent posteromedial papillary muscle (solid arrow) and a rudimentary anterolateral papillary muscle (dash arrow)

muscle and thick false tendon crossing the left ventricle from interventricular septum to posteromedial wall (Fig. 2, Video 2, 3. See corresponding video/movie images at www.anakarder.com). The patient was advised about possible surgical intervention in an experienced center. We also started medical therapy including warfarin.

The association of two rare cardiac disorders, Ebstein anomaly and left ventricular noncompaction, has been reported previously. However, biventricular involvement as in our case has only been reported in a family. Although we had no chance to evaluate the patient's family, our case report is also highlight the diagnostic importance of echocardiography in this very rare association without using other modalities.

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