Intramyocardial fissure

İntramiyokardiyal fissür

A 38-year-old male patient with a history of myocardial infarction was admitted to our hospital for routine follow-up control. He had a history of hypertension and chronic renal failure, followed by medical treatment with no hemodialysis (glomerular filtration rate was 25 mL/min/1.73 m^2). Transthoracic echocardiography revealed concentric left ventricular hypertrophy and intramyocardial fissure (arrows) in the posterolateral wall during systole and diastole (Fig. 1-2 and Video 1. See corresponding video/movie images at www.anakarder.com). The patient had normal global ejection fraction (65%) and no regional wall motion abnormalities. Due to the necessity of hemodialysis after gadolinium administration, cardiac magnetic resonance (CMR) imaging could not be performed because of patient discordance. Abdominal ultrasonography performed to show the possible presence of echinococcus revealed no pathologic findings. Serologic tests for hydatidosis-IHA/IFAT were also negative. We recommended continuation of the medical therapy and routine echocardiographic follow-up to the patient.

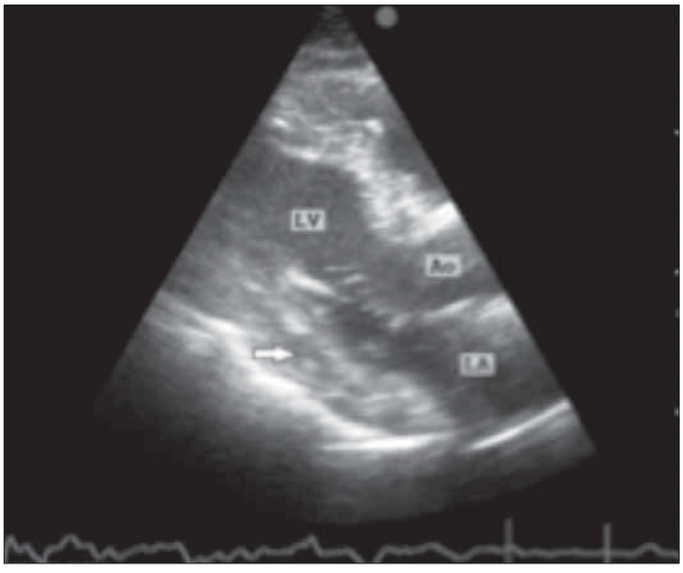

Figure 1. Parasternal long-axis echocardiographic view of intramyocardial fissure (arrow)

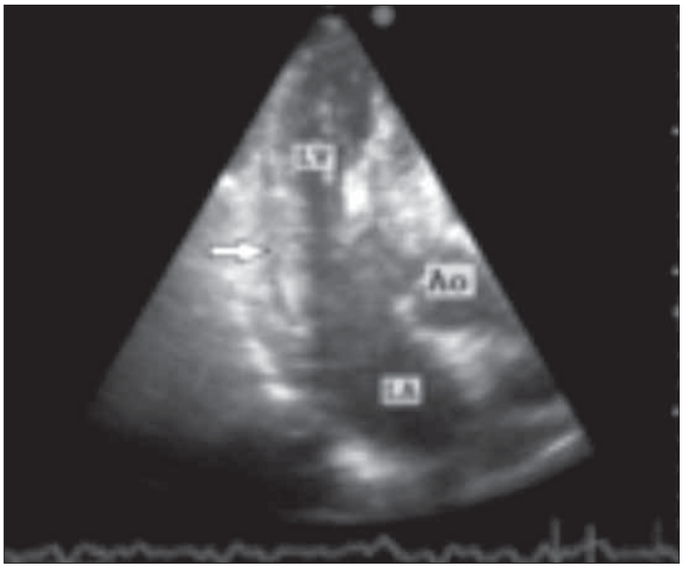

Figure 2. Video 1. Apical 5- chamber echocardiographic view of intramyocardial fissure (arrow)

We think that this fissure is a remnant of a spontaneously healed intramyocardial dissection. Thus, history of prior myocardial infarction supports our theory robustly. The intramyocardial dissection is an unusual rupture of the left or right ventricular wall, mostly secondary to myocardial infarction but can rarely be due to infection such as cardiac echinococcosis. The mechanism is a dissection among the myocardial fibers and the dissection tract is filled with blood creating a neo cavity limited by the myocardium. Diagnosis is often difficult and in most of the cases it is postmortem. It is very rare so optimal treatment strategy is not known but most of the cases were treated surgically. On the other hand, cases with spontaneous healing were also reported.

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Premature coronary artery disease in a patient with Wolfram syndrome

Wolfram sendromlu bir hastada erken yaşta görülen koroner arter hastalığı

Wolfram syndrome, also called DIDMOAD (Diabetes Insipidus, Diabetes Mellitus, Optic Atrophy, and Deafness), is a rare genetic disorder. Here we report a case of premature coronary artery disease (CAD) associated with Wolfram syndrome, which has not been reported before. The patient was a 25-year- old man who had congenital cataracts, optic atrophy; diabetes mellitus, deafness and diabetes insipidus. He had exertional chest pain for 2 months. He had no smoking history. His lipid profile and serum homocysteine levels (7.2 µmol/l) were normal. There was no family history of premature CAD. Cardiovascular examination was unremarkable. Electrocardiogram revealed T wave inversions in inferior leads. Transthoracic echocardiography revealed mild hypokinesia at mid-lateral segment of the left ventricle. Coronary angiography revealed a critical stenosis in the mid-portion of the right coronary artery (RCA) (Video 1. See corresponding video/movie images at www.anakarder.com) and non-critical plaques in the left coronary arterial system (Fig. 1). Critical stenosis in the RCA was successfully opened by a 2.75-mm X 13-mm bare metal stent (Fig. 2). The medical