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.

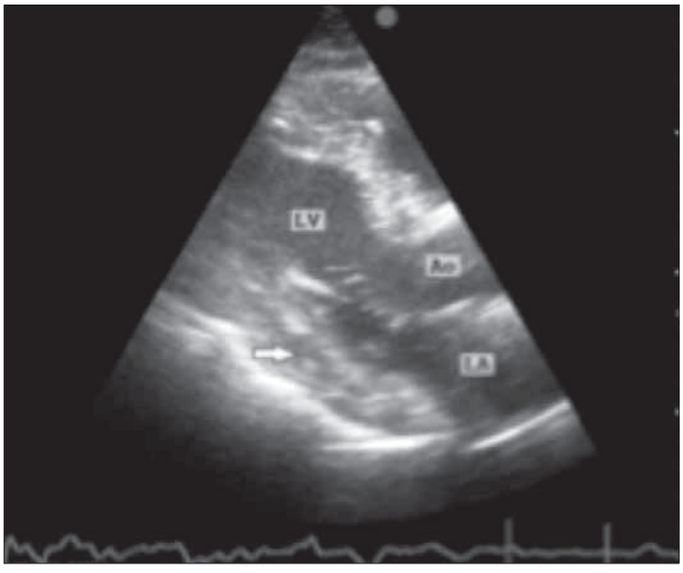

Figure 1. Parasternal long-axis echocardiographic view of intramyocardial fissure (arrow)

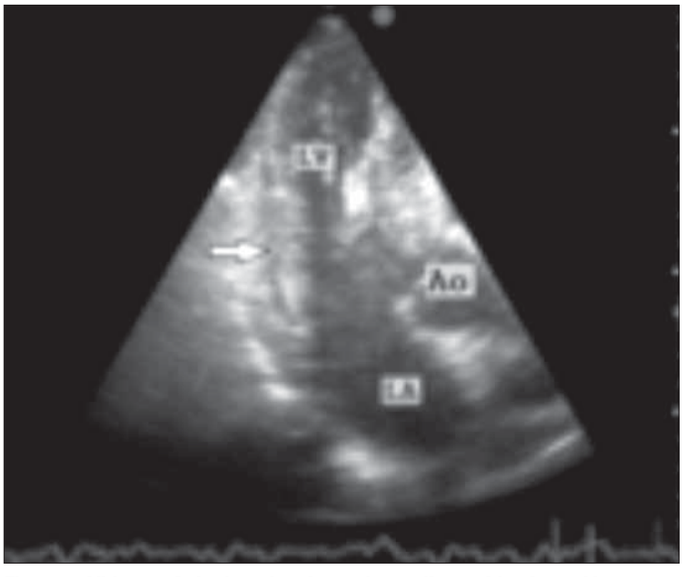

Figure 2. Video 1. Apical 5- chamber echocardiographic view of intramyocardial fissure (arrow)

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.

Yalçın Velibey, Seçkin Satılmış, Metin Çağdaş, Servet Altay, Özge Güzelburç, Dilay Satılmış[1], Kemal Yeşilçimen, Mehmet Eren
Department of Cardiology, Siyami Ersek Thoracic and Cardiovascular Surgery Training and Research Hospital, İstanbul
[1]Department of Emergency Medicine, Haydarpaşa Training and Research Hospital, İstanbul, Turkey

Address for Correspondence/Yazışma Adresi: Dr. Yalçın Velibey
Department of Cardiology, Siyami Ersek Thoracic and Cardiovascular Surgery Training and Research Hospital, İstanbul, Turkey
Phone: +90 216 444 52 57 Fax: +90 216 337 97 19
E-mail: yalchinveliyev@gmail.com, dr_yalchin_dr@yahoo.com.tr

Available Online Date / Çevrimiçi Yayın Tarihi: 18.04.2011

doi:10.5152/akd.2011.070

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

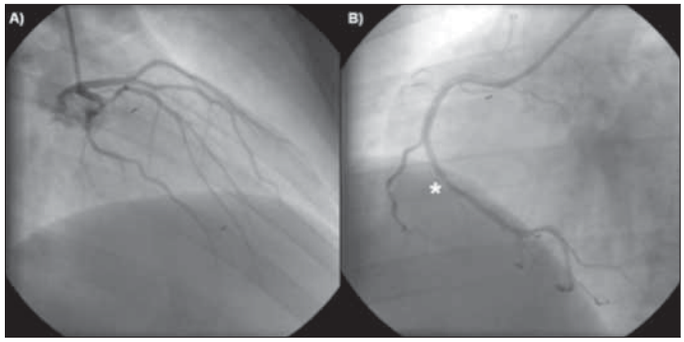

Figure 1. Coronary angiography views of non-critical lesions in the left coronary arterial system (A); and a critical stenosis of right coronary artery (asterisk), (B)

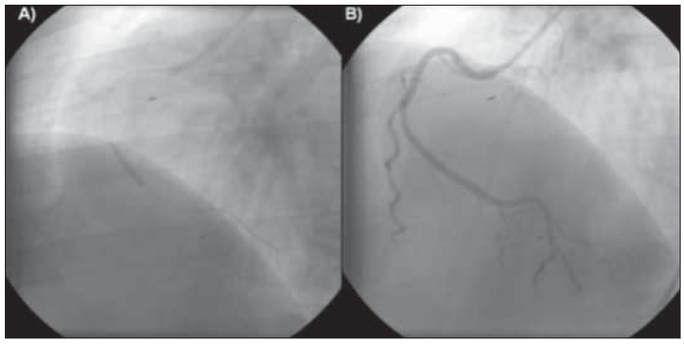

Figure 2. Critical stenosis at the mid portion of the right coronary artery was dilated by a 2.75-mm X 13-mm bare metal stent (A) with no residual stenosis (B)

therapy of the patient was optimized with clopidogrel, acetylsalicylic acid, metoprolol and ramipril. His further clinical course was uneventful; he was discharged two weeks later.

Necla Özer, Sercan Okutucu, Kadri Murat Gürses
Department of Cardiology, Faculty of Medicine, Hacettepe University, Ankara, Turkey

Address for Correspondence/Yazışma Adresi: Dr. Sercan Okutucu
Department of Cardiology, Faculty of Medicine, Hacettepe University, Ankara, Turkey
Phone: +90 312 305 17 81 Fax: +90 312 311 40 58
E-mail: sercanokutucu@yahoo.com

Available Online Date / Çevrimiçi Yayın Tarihi: 18.04.2011

doi:10.5152/akd.2011.071

A 23-year patency of a saphenous vein graft in a patient with diabetes mellitus

Diyabetik bir hastada 23 yıl açık kalan bir safen ven grefti

A 79- year-old man was admitted to our hospital with the complaint of progressive angina pectoris. Coronary artery bypass grafting (CABG) had been performed with the saphenous vein graft (SVG) to the left anterior descending artery (LAD) 23 years ago. He had type 2 diabetes mellitus for 18 years. Serum lipid parameters and electrocardiogram were normal. He was receiving clopidogrel because of aspirin-induced gastritis. Coronary angiography revealed the significant lesions in the circumflex coronary artery (CX), complete occlusions in the proximal regions of the LAD and the right coronary artery (RCA). The SVG showed an excellent patency (Video 1. See corresponding video/movie images at www.anakarder.com) Percutaneous coronary intervention was planned to the CX and the RCA, but the patient refused.

The predictors of graft patency are the diameter of the recipient vessel >2 mm (as our case, Fig. 1A-B), lower serum cholesterol, the use of aspirin after CABG. Clopidogrel is recommended in cases intolerant to aspirin after CABG.

A 30-year patency of a SVG in a 74-year-old adult and 22-year patencies of SVGs in two pediatric patients have been reported previously.

This presentation reveals the diabetic case having a 23-years patency of a SVG. This is the longest patency time in a diabetic patient with CABG in the literature. Considering that graft stenosis is more frequent in diabetic patients, this result is very remarkable.

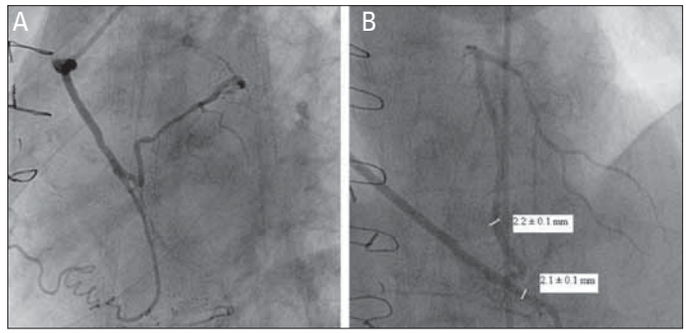

Figure 1A-B. Angiograms showing the patency of saphenous vein graft

Taner Ulus, Hande Özduman, Yüksel Çavuşoğlu
Department of Cardiology, Faculty of Medicine, Eskişehir Osmangazi University, Eskişehir, Turkey

Address for Correspondence/Yazışma Adresi: Dr. Taner Ulus
Department of Cardiology, Faculty of Medicine, Eskişehir Osmangazi University, Eskişehir, Turkey
Phone: +90 222 239 37 00 Fax: +90 222 239 90 11
E-mail: tanerulus@hotmail.com

Available Online Date / Çevrimiçi Yayın Tarihi: 18.04.2011

doi:10.5152/akd.2011.072

Coronary aneurysm and factor V Leiden mutation: A coincidence or an association?

Koroner anevrizması ve faktör V Leiden mutasyonu: Rastlantı mı yoksa ilişkili mi?

A 23-year-old male referred to our tertiary cardiology center because of chest pain, 3 ventricular fibrillation episodes in last 12 hours and troponin T elevation (1.2 µg/l). His medical history revealed recurrent deep venous thrombosis attacks on his left leg and one pulmonary embolism attack. He was a homozygous mutant on factor V Leiden