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Extreme mechanical aortic valve dehiscence: "Rocking motion" clearly assessed with echocardiography and fluoroscopy 🚳

A 19-year-old man was admitted due to sudden onset and progressively increasing shortness of breath, cough, nausea, and vomiting. The patient had undergone aortic and mitral valve replacement with mechanical prosthesis for the treatment of infective endocarditis 4 years ago.

The patient had poor general condition with a body temperature of 37.4°C, blood pressure of 120/40 mm Hg, heart rate of 120 bpm, and respiratory rate of 32/min. Cardiac auscultation showed a grade 3/6 early diastolic murmur along the left sternal border. Breath sounds were reduced, and rales could be heard



Figure 1. (a) Electrocardiogram showing sinus tachycardia and inferolateral ST segment depressions. (b) Chest X-ray showing pulmonary expansive volume load

all over the right and left lungs. There was no stigma of endocarditis. Electrocardiogram revealed sinus tachycardia and inferolateral ST segment depressions (Fig. 1a). Because the arterial blood gas analysis and chest X-ray (Fig. 1b) showed respiratory asidosis and pulmonary expansive volume load, respectively, the patient was entubated.

An emergency transthoracic echocardiogram revealed a heart rate-dependent pendular "rocking motion" of the aortic valve prosthesis, consistent with dehiscence. The aortic valve was moving to the aorta in systole, whereas it was prolapsing to the left ventricle in diastole, resulting in severe paravalvular jet of aortic insufficiency (Fig. 2, Videos 1 and 2). Transesophageal echocardiography showed no vegetations on the mitral valve, but aortic valve was difficult to assess due to acoustic shadowing and artifacts. The systolic–diastolic "rocking motion" of the prosthesis was also clearly demonstrated by fluoroscopy (Fig. 3, Videos 3 and 4).



Figure 2. Echocardiography showing the prosthetic aortic valve moving to the aorta in systole (a), whereas it was prolapsing to the left ventricle in diastole (b), resulting in severe paravalvular jet of aortic insufficiency (c) with measurement of the regurgitant jet pressure half time 115 ms (d)



Figure 3. Fluoroscopy showing "rocking motion" of the bileaflet mechanical aortic prosthesis during diastole and systole

Inotropic treatment was initiated because the patient developed shock. While awaiting an emergency surgical repair, he collapsed and could not be resuscitated successfully.

🔟 Hasan Kaya, 🔟 Bayram Arslan, ២ Mehmet Sait Coşkun, 🔟 Faruk Ertaş

Department of Cardiology, Faculty of Medicine, Dicle University; Diyarbakır-*Turkey*

Video 1. Echocardiography in the parasternal view showing the prosthetic aortic valve moving to the aorta in systole, whereas it was prolapsing to the left ventricle in diastole.

Video 2. Echocardiography in the apical four-chamber view showing the prosthetic aortic valve moving to the aorta in systole, whereas it was prolapsing to the left ventricle in diastole.

Video 3. Fluoroscopy in the 12° left anterior oblique and 27° caudal projection showing "rocking motion" of the bileaflet mechanical aortic prosthesis.

Video 4. Fluoroscopy in the 38° left anterior oblique and 32° caudal projection showing "rocking motion" of the bileaflet mechanical aortic prosthesis.

Address for Correspondence: Dr. Hasan Kaya, Dicle Üniversitesi Tıp Fakültesi, Kardiyoloji Anabilim Dalı, 21280 Diyarbakır-*Türkiye* Phone: +90 412 248 80 01-10 04 E-mail: dr_hasankaya@yahoo.com ©Copyright 2019 by Turkish Society of Cardiology - Available online at www.anatoljcardiol.com D0I:10.14744/AnatolJCardiol.2019.01336

Chest pain after a cesarean -section with a puzzling ECG

Herein, we present the case of a 31-year-old patient who had chest pain after a cesarean-section at 36 weeks of amenorrhea. As past medical history, this patient has a homozygous sickle cell disease. The patient complained of pressure in the chest 2 h after cesarean-section, radiating to the shoulders and the back. Blood pressure was 150/100 mm Hg (symmetrical on both arms) and heart rate was 98 bpm. Per-critical ECG showed an ST-segment elevation in aVR, V1–V2 with a mirror in other leads (Fig. 1). A few minutes later, the pain had disappeared and the ECG changed. Cardiac echography found a 50% left ventricular ejection fraction with homogeneous hypokinesia. There was no argument for acute pulmonary heart disease or a patent foramen ovale after contrast test. Investigations showed hemoglobin at 6 g/dl and an increase in troponin by 9ui (N<0.04ui). Cardiac-CT was performed in emergency, which showed no coronary abnormality but showed bilateral pulmonary embolism (PE) (Fig. 2).

Atypical presentations are common for PE. However, the presentation with chest pain and ST-segment elevation on ECG



Figure 1. ECG at initial management



Figure 2. Cardiac-CT

is exceptional. Two pathophysiological hypotheses have been proposed: (1) a right ventricular ischemic strain due to right ventricular dysfunction associated with low coronary output arising from a low cardiac output and (2) a paradoxical coronary embolism because of patent foramen ovale reopening due to elevated pressure in right heart cavities.

Etienne Puymirat, Discent Aidant Assistance Publique-Hôpitaux de Paris (AP-HP); Hospital European Georges Pompidou (HEGP), Department of Cardiology, University Paris-Descartes; Paris-France

Address for Correspondence: Etienne Puymirat, MD, Assistance Publique-Hôpitaux de Paris (AP-HP); Hospital European Georges Pompidou (HEGP), Department of Cardiology, Paris, France; University Paris-Descartes; 20 Rue Leblanc, Paris-*France* Phone: 33 156 09 28 51 E-mail: etienne.puymirat@aphp.fr @Copyright 2019 by Turkish Society of Cardiology - Available online at www.anatoljcardiol.com D0I:10.14744/AnatolJCardiol.2019.45787