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, 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
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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 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, Vincent 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 1 56 09 28 51
E-mail: etienne.puymirat@aphp.fr
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