Biventricular dysplasia presenting as an acute coronary syndrome

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A 45-year-old man without past medical history was referred for acute chest pain and suspected non-ST-segment elevation myocardial infarction. His cardiovascular risk factors were diabetes, obesity and current smoking. Physical examination was normal. The 12-lead ECG demonstrated ST-segment depression in leads V2 to V6 (Fig. 1A). Troponin I was elevated at 10 μg/L. Quantitative coronary angiography revealed a 65% diameter stenosis of the mid portion of the LAD coronary artery (Fig. 1B). LV angiography showed severe LV dysfunction involving the anterior wall (Fig. 1B, Video A). Because of the clinical presentation associated with the ECG and the biological and angiographic features, percutaneous coronary angioplasty of the LAD coronary artery was performed successfully with stent implantation. The following day, the patient developed sustained ventricular tachycardia that resolved spontaneously (Fig. 1C). Transthoracic echocardiography revealed severe global LV dysfunction with moderate RV dilatation. Because of the discrepancy between the moderate troponin elevation and the severe LV dysfunction, the patient underwent CMR imaging (Siemens Espree® 1.5T, Erlangen, Germany). Cine MRI acquired before contrast injection showed dilated and severely hypokinetic left and right ventricles (Fig. 1D). The RV myocardium was thin and showed segmental outward systolic wall motion particularly in the anterior wall of the RV outflow tract (Fig. 1D, Video B). T1-weighted black-blood fast spin echo images revealed the presence of intramyocardial areas of hypersignal within the RV and LV myocardium, indicative of intramyocardial fat (Fig. 1E). Late-enhanced CMR imaging acquired 10 minutes after contrast injection.
Figure 1. A. Electrocardiogram on admission showing diffuse ST-segment depression in the anterior leads. B. Coronary angiography showing left anterior descending coronary artery stenosis (arrow). Left ventricular (LV) angiography at end-diastole and end-systole with marked hypokinesis in the anterior wall (arrows). C. Sustained ventricular tachycardia. D. Steady-state free precession cine magnetic resonance imaging (cine-MRI) at end-diastole and end-systole in the four-chamber view, demonstrating dilated and severely hypokinetic left and right ventricles, along with unusual areas of hypointensity within the LV myocardium suggestive of intra-myocardial fat (arrows). Cine-MRI of right heart chambers showing outward systolic wall motion of the right ventricular (RV) outflow tract. E. Black-blood T1-weighted cardiac magnetic resonance imaging (CMRI) showing hypersignal indicative of intramyocardial fat (arrows). Corresponding areas of myocardial fibrosis on late-enhanced-CMRI acquired 10 min after gadolinium injection (0.1 mM) (arrows). Note the presence of myocardial late enhancement (fibrosis) in the RV outflow tract. RA: right atrium; RV: right ventricle; PA: pulmonary artery.

Conflicts of interest
None.

Appendix A. Supplementary data
Supplementary data associated with this article can be found, in the online version, at doi:10.1016/j.acvd.2010.03.008.