P1733

Late onset of arrhytmogenic cardiomyopathy, unmasked by myocarditis - when multi-modality imaging solves the puzzle

Luchian ML.[1]; Rimbas RC.[2]; Stoicescu CI.[3]; Marinescu AV.[3]; Nicula AI.[3]; Vinereanu D.[3]

[1]University Emergency Hospital of Bucharest, Bucharest, Romania
[2]Carol Davila University of Medicine and Pharmacy, University and Emergency Hospital Bucharest, Cardiology Department, Bucharest, Romania
[3]University of Medicine and Pharmacy Carol Davila, Bucharest, Romania

Introduction. Arrhythmogenic cardiomyopathy (AC) is a genetic disease, associated with arrhythmic sudden death in young people. Since inflammation might be also present, differential diagnosis with myocarditis is necessary. We present a challenging case, initially diagnosed with ventricular tachycardia (VT) and acute myocardial infarction (AMI), in which multimodality imaging changed the initial diagnosis.

Clinical case. A 68-year old woman with dyslipidaemia and hypertension was admitted with chest pain, negative T waves in anterior leads, epsilon wave in V1, V2 (Fig. A, B), negative troponin I (TpI), and elevated NT-proBNP (342 ng/ml). One month before, she was hospitalized for a monomorphic VT with left bundle branch block (LBBB) pattern, with hemodynamic instability, cardioverted electrically to sinus rhythm. Increased TpI suggested an AMI. She recalls an episode of flu with high fever (39°C) before admission. At current admission, transthoracic echo (TTE) revealed LVEF 52%, severe lateral wall hypokinesia, severe RV dysfunction with fractional area change 23%, and TAPSE 15 mm. Coronary angiography excluded significant lesions. Speckle tracking echo(STE) revealed patchy areas of decreased LV myocardial deformation (Fig. C), and decreased basal deformation in RV free wall(Fig. D). Cardiac magnetic resonance (CMR) revealed LVEF 59%, lateral and septal wall hypokinesia, late gadolinium enhancement (LGE) in mid-ventricular lateral wall and interventricular septum (Fig. F), with correspondent increased extracellular volume (ECV) >30%, increased native T1 time, increased T2 mapping, and small pericardial effusion (7 mm); RV was dilated (diastolic volume 107 ml/m2 and systolic volume 69 ml/m2), with severely decreased RV ejection fraction (RVEF) of 35%, hypokinesia of free wall, dyssynchronous contractions, and microaneurysms (Fig. E, F). Diagnosis was concluded for AC with delayed onset, revealed by an acute myocarditis. Betablocker, ACEI, and implantable cardioverter defibrillator (ICD) in secondary prevention were decided. Relatives were screened for the presence of AC. Patient`s daughter presented 8000 isolated monomorphic ventricular ectopic heart beats with LV origin, no echo signs, but incipient signs of AC on CMR (increased ECV on T1 mapping in 7 LV myocardial segments, without LGE).

Discussion. We presented a rare case of late arrhythmic onset of AC in a patient, in which the first aetiology of VT was considered to be ischaemic. VT with LBBB morphology can be caused by myocarditis, AMI, or AC. Similarly, epsilon wave with negative T waves in precordial leads may have multiple aetiologies. In our case, multimodality imaging approach excluded AMI and suggested an inflammatory cause for VT. CMR allowed the identification of morphologic condition AC and the trigger condition of myocarditis.

Conclusions. Our case highlights the importance of a multimodality imaging approach in diagnosing AC and myocarditis, for a correct and complete management.

Abstract P1733 Figure.