

Arrhythmogenic Right Ventricular Dysplasia

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Arrhythmogenic right ventricular dysplasia (ARVD) is a form of cardiomyopathy that usually occurs as heart failure and ventricular arrhythmias, and myocarditis is the most common finding in up to 60% of the cases. It is genetically transmitted via either the dominant or recessive autosomal mode, and is highly prevalent among young Europeans. (1, 2) Cardiac magnetic resonance imaging (CMRI) is one of the complementary tests used for diagnosing ARVD. CMRI shows presence of right ventricular high intensity signals, right ventricular outflow tract dilatation, right ventricular dilatation (greater than 42 mm during diastole), right atrial dilatation (A-P diameter > 41 mm), dyskinesias and/or aneurisms. (3, 4) The certainty diagnosis is performed by means of three criteria: with two criteria it is considered probable, whereas with only one it is uncertain. (5) The images correspond to a 57-year-old woman, carrier of Chagas disease and with a history of hypertension, who is admitted due to predominantly right-sided biventricular heart failure. The CMRI reveals significant dilatation of right chambers and severe impairment of right ventricular systolic function. Isolated deposits of fatty tissue at the level of

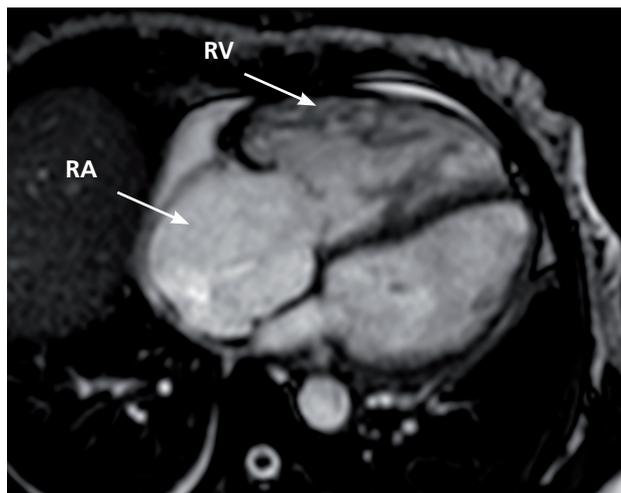


Fig. 1. Cardiac magnetic resonance imaging. White blood sequence. Cross section. Dilatation and thinning of the right chambers are observed. RA: Right atrium. RV: Right ventricle.

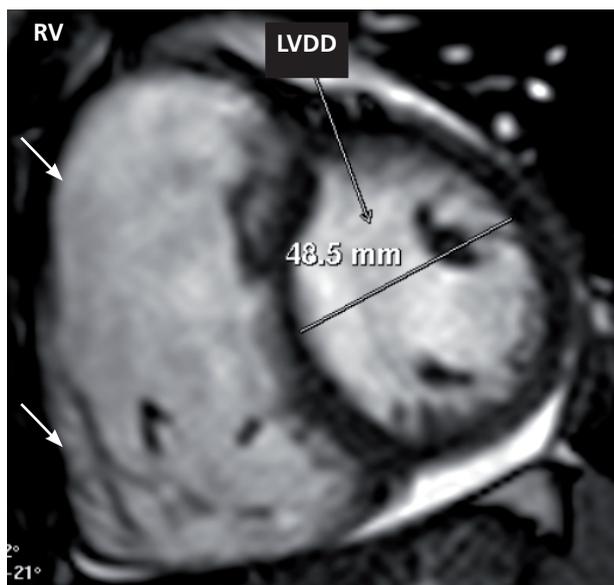


Fig. 2. Cardiac magnetic resonance imaging. White blood sequence. Sagittal section. Left ventricular wall thinning and dilatation. RV: Right ventricle. LVDD: Left ventricular diastolic diameter.

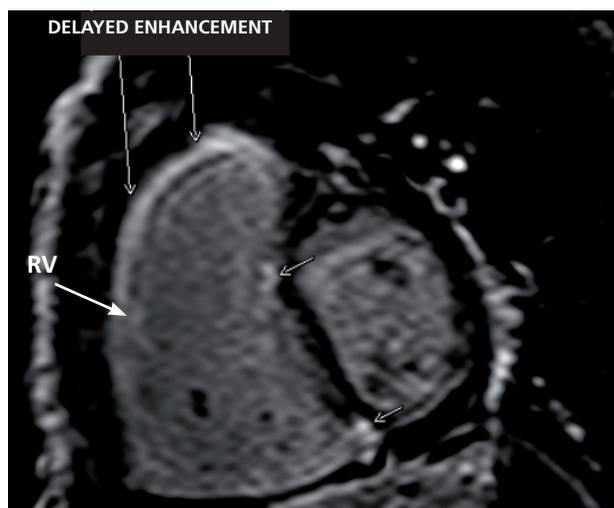


Fig. 3. Cardiac magnetic resonance imaging. Delayed enhancement sequence. Sagittal section. Delayed enhancement in patches is observed in the right ventricular wall. RV: Right ventricle.

the right ventricular free wall (Figures 1 & 2) are observed in the black blood sequence, and fibrosis-necrosis in patches are evidenced on the right ventricular walls, in the delayed enhancement sequence (Figure 3).

Conflicts of interest

None declared

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