Original Article

Utility of Myocardial Fibrosis and Fatty Infiltration Detected by Cardiac Magnetic Resonance Imaging in the Diagnosis of Arrhythmogenic Right Ventricular Dysplasia—A Single Centre Experience

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Introduction: **Cardiac magnetic resonance imaging (CMR) has evolved as a major diagnostic tool to evaluate arrhythmogenic right ventricular dysplasia (ARVD). However, there is a lack of consensus in the interpretation of findings such as fatty infiltration or myocardial fibrosis. We examined the diagnostic utility of these two features in the diagnosis of ARVD.**

Methods: **We performed fast imaging employing steady-state acquisition cine imaging, T_1-weighted black blood imaging with and without fat suppression and post-contrast delayed enhancement on a 1.5-T scanner to evaluate ventricular function and morphology, fatty infiltration and regional myocardial fibrosis in 52 subjects with suspected ARVD.**

Results: **Eight subjects met the international diagnostic criteria for ARVD. Right ventricle (RV) delayed hyperenhancement was found in 7 of 8 (88%) ARVD subjects compared to 6 of 44 (14%) subjects without ARVD ($p < 0.001$). Fatty infiltration was only identified in 1 ARVD patient, and 1 non-ARVD patient. On multiple logistic regression analysis RV enhancement remained an independent predictor for the diagnosis of ARVD ($p < 0.05$).**

Conclusion: **RV delayed enhancement is common in patients with ARVD, whereas detection of fatty infiltration of the right ventricle was rare in our patient population. The inclusion of RV fibrosis on CMR as a feature of ARVD may improve the diagnostic accuracy of this condition.**

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Keywords. Cardiac magnetic resonance imaging; ARVD; Delayed enhancement; Fatty infiltration

Introduction

Arrhythmogenic right ventricular dysplasia (ARVD) is characterised by structural and functional abnormalities of the right ventricle (RV), the development of ventricular arrhythmias and progressive heart failure in advanced cases. Histologically, diffuse or localised atrophy of the myocardium with replacement by fibrous or fatty tissue is observed.[1] The clinical presentation of the disease varies widely among patients. Symptomatic ventricular tachycardia (VT) is a common clinical presentation, leading to palpitations, chest pain, syncope or even sudden cardiac death. Congestive heart failure due to impaired right ventricular or even biventricular function represents another manifestation and is assigned to a later stage of the disease.[2] Because of the often fatal course and the potential prevention of sudden cardiac death with antiarrhythmic drug therapy or implantable defibrillators, early recognition of ARVD is of major importance.[3] Diagnosis is currently based on a set of familial, electrocardiographic, structural and histological criteria proposed by the International Task Force for Cardiomyopathies in 1994.[4]

Cardiac magnetic resonance imaging (CMR) is a promising diagnostic tool to evaluate ARVD because of its unique ability to provide tissue characterisation in addition to functional and morphological information of the RV. With further progression in knowledge and technology, CMR may replace invasive diagnostic endomyocardial biopsy (EMB) in the future. Several observational studies have reported structural findings in CMR in patients with proven or suspected ARVD.[5–16] However, the comparability of these findings is limited due to

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diverse patient selection, use of different CMR techniques and a large interobserver variability.[11,15] Overall, there is little agreement in interpretation of findings such as fatty infiltration or wall thinning. Despite the lack of consensus, these features of CMR are frequently used for assessment of ARVD, leading to numerous misdiagnoses of ARVD.[15]

In this study we analysed findings on CMR of patients referred to our centre with suspected ARVD. We performed post-contrast delayed enhancement imaging for the detection of myocardial fibrosis and T_1-weighted imaging with and without fat saturation for the detection of myocardial fat. We then examined the diagnostic utility of these two features in the diagnosis of ARVD according to the currently accepted guidelines.

Methods

Patient Selection

We performed CMR in patients referred to our centre with suspected ARVD. The suspicion of ARVD was based on a number of clinical features. These included abnormal right ventricular morphology on echocardiography, the presence of arrhythmia (including documented ventricular tachycardia with left bundle block morphology), a family history of ARVD, and unexplained syncope.

CMR Protocol

CMR was performed on a 1.5-T scanner (Signa Excite, General Electric Healthcare, Milwaukee, USA) using a dedicated cardiac coil and electrocardiographic gating. We acquired bright blood 2D FIESTA (fast imaging employing steady-state acquisition) cine imaging in three apical standard views (four-chamber, three-chamber, and two-chamber) and three short axis planes (apical, mid, and basal). Imaging parameters for bright blood cine sequences were as follows: time to repetition [TR] = 3.5, time to echo [TE] = 1.6 ms, flip angle 45°, and slice thickness = 8 mm. T_1-weighted black blood imaging with and without fat suppression was performed in three short axis planes using double inversion recovery fast spin-echo sequence. Imaging parameters for black blood sequences were TR = 2 *R–R* intervals, TE = 37 ms, and slice thickness = 8 mm. This sequence was repeated with chemical shift fat suppression manually tuned to the fat peak. Inversion recovery gradient echo sequences (MDE) were performed 10 min after intravenous administration of MRI contrast agent (0.2 mmol/kg gadolinium-DTPA, Magnevist®, Schering, Germany) in the identical long axis and short axis planes as described above. Imaging parameters for MDE sequences were TR = 6.7, TE = 3.2 ms, inversion times were individually adjusted between 175 and 225 ms to null signal intensity of normal myocardium. Ventricular function and morphology, fatty infiltration and regional myocardial fibrosis were then evaluated.

Detection of RV Fibrosis and/or Fatty Infiltration

For the detection of RV fibrosis, delayed enhancement images were evaluated in short axis and long axis planes. A threshold of signal intensity greater than two standard deviations above that of a remote region of healthy left ventricle (LV) myocardium was applied for the presence of delayed enhancement. Right ventricular trabeculations were excluded from this analysis. In order to rule out confounding effects from signal artefacts, we required an area of signal intensity corresponding to the region of interest in two orthogonal planes to confirm the presence of delayed enhancement. If there was no orthogonal plane exactly matching the region in question an extra orthogonal slice was planned directly through the area in question.

To identify fatty infiltration, T_1-weighted black blood imaging with and without fat saturation was utilised. Fatty infiltration was defined as an area of hyperintensity on T_1-weighted imaging that was suppressed out on the corresponding fat saturation image. The same sequence was also used to differentiate partial volume effects from epicardial fat and delayed enhancement on the RV free wall.

Evaluation of Ventricular Morphology

RV end-diastolic diameter was measured as the distance from interventricular septum to the free wall in the four-chamber long-axis view at the mid-ventricular level in the FIESTA sequences.[17] Discrimination of RV dilatation was made on qualitative (visual) basis. RV ejection fraction was calculated from the four-chamber long axis view by expressing the difference between the RV end-diastolic area and RV end-systolic area over the RV end-diastolic area. We have previously found this simple method for calculating RV ejection fraction to correlate well with established volumetric methods ($R = 0.80$, $p = 0.017$, Pfluger et al., unpublished data).

LV end-diastolic and end-systolic volumes were calculated using the area-length method utilising the average from the four-chamber and two-chamber long axis FIESTA views. The LV ejection fraction was then expressed as the difference between LV end-diastolic volume and LV end-systolic volume over the LV end-diastolic volume. We have also shown an excellent correlation between this biplane method and volumetric methods for the correlation of LV volume and ejection fraction (R of 0.95 and 0.96, respectively, $p < 0.001$, Pfluger et al., unpublished data).

Diagnostic Criteria for ARVD

The diagnosis of ARVD was made according to established clinical criteria of the European Society of Cardiology (ESC), adopted from McKenna et al.[4] The morphological features identified by CMR were included as diagnostic criteria for ARVD, however findings of fibrosis or fatty infiltration were not considered for the purposes of establishing the diagnosis of ARVD. Findings of patients with confirmed ARVD were compared to results of those without ARVD.

Statistics

All data are expressed as mean ± S.E. except where otherwise stated. Comparisons between groups involved proportionality the Chi Squared test or Fisher's exact test were applied. The relationship between dichotomous variables to the diagnosis of ARVD was initially examined in a

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