Journal of Cardiovascular Magnetic Resonance (2006) **8,** 361–366

ISSN: 1097-6647 print / 1532-429X online
DOI: 10.1080/10976640500527082

Taylor & Francis
Taylor & Francis Group

CARDIOMYOPATHY

Value of Repeated Cardiac Magnetic Resonance Imaging in Patients with Suspected Arrhythmogenic Right Ventricular Cardiomyopathy

David Conen,[1] Stefan Osswald,[1] Thomas A. Cron,[1] Andre Linka,[1] Jens Bremerich,[2] Dagmar I. Keller,[1] Matthias E. Pfisterer,[1] and Peter T. Buser[1]

Department of Cardiology, University Hospital, Basel, Switzerland,[1] and Department of Radiology, University Hospital, Basel, Switzerland[2]

ABSTRACT

Aim: **Diagnosis of early stages of arrhythmogenic right ventricular cardiomyopathy (ARVC) with minimal structural abnormalities is challenging. The purpose of this study was to assess the value of repeated cardiac magnetic resonance imaging (CMR) in patients referred for right ventricular arrhythmias and clinical suspicion of ARVC.** ***Methods and Results:*** **Prospective follow-up study of 18 patients (8 females) studied with CMR for suspected ARVC. Patients with implanted defibrillators (ICD) were excluded. Mean follow-up was 37 $\pm$ 16 (12–59) months. Patients were assigned to 2 categories (ARVC likely or ARVC unlikely) according to a CMR-score based on right ventricular abnormalities. Clinical follow-up revealed no disease progression in 17 patients (94%). In 1 patient, an ICD was implanted because of disease progression. Of 9 patients with initial findings suggestive of ARVC, follow-up CMR remained positive in 3 and was diagnosed as normal in 6, mainly due to the inability to confirm the presence of fatty infiltrates at follow-up (5 of 6 patients). Initially, 9 patients had a normal CMR and 8 of those remained normal during follow-up.** ***Conclusion:*** **Repeated CMR after an average follow-up of 3 years was normal in 6 of 9 patients with clinical findings consistent with early stages of ARVC at the time of baseline CMR. Thus, CMR diagnosis of early stage ARVC is difficult and should be made with caution.**

INTRODUCTION

Arrhythmogenic right ventricular cardiomyopathy (ARVC) is a myocardial disease affecting primarily the right ventricle. Histologically it is characterized by fibrofatty replacement of right ventricular myocardium. There are sporadic as well as familiar forms which are typically inherited in an autosomal dominant trait (1). ARVC has been identified as an important cause of sudden cardiac death in young subjects and athletes (2, 3). Other clinical manifestations include palpitations or syncope due to right ventricular tachyarrhythmias and electrocardiographic (ECG) changes in the right precordial leads. Structural alterations of the right ventricle are detectable by echocardiography, right ventricular angiography and cardiac magnetic resonance imaging (CMR) (4).

There is no diagnostic gold standard and early morphologic changes in the right ventricle may be very subtle. In light of the clinical difficulties of diagnosing ARVC, a score of major and minor diagnostic criteria including structural abnormalities, right ventricular fatty replacement, arrhythmias, electrocardiographic changes and family history has been proposed by the 'Task Force of the Working Group on Cardiomyopathies' (5). Despite these valuable criteria, diagnosis of early forms of ARVC remains challenging since the majority of patients with early stage disease have no or only minor symptoms. Nevertheless, early and accurate diagnosis of ARVC is crucial because of its important prognostic and therapeutic implications. Differential diagnosis includes disorders such as localized right ventricular myocarditis or idiopathic right ventricular outflow

Financial disclosure statement: None of the authors has any financial associations that might pose a conflict of interest in connection with this study.
Keywords: Arrhythmogenic Right Ventricular Cardiomyopathy, Right Ventricular Arrhythmias, Cardiac MRI, Echocardiography.
Correspondence to:
Peter T. Buser, MD, FACC, FESC
Cardiology, University Hospital
Petersgraben 4, CH-4031 Basel
Switzerland
phone: +41 61 265 52 24 fax: +41 61 265 45 98
email: pbuser@uhbs.ch

tract (RVOT) tachycardia, entities with a much better prognosis (6). While pathological findings in ARVC are usually progressive, idiopathic RVOT tachycardia is associated with no or only minimal detectable morphological changes that remain stable over time (4). Therefore, one important way to confirm or rule out the definitive diagnosis of ARVC is to evaluate the patient after a certain time-window.

Over the last decade CMR emerged as one of the most sensitive modalities to visualize the right ventricle (7) and to assess right ventricular function (8). CMR also allows to assess fatty tissue replacement and fibrosis, which are quite typical, but not specific findings for ARVC (9–13). Particularly, in patients with minimal morphologic abnormalities interpretation of these findings may be difficult, since the right ventricular free wall is very thin and often penetrated by epicardial fatty tissue in normal individuals (4, 14, 15). The prevalence of fatty tissue infiltration in the general population and the specificity of this finding in early stages of ARVC are not known. Despite these limitations, CMR emerged as the preferred diagnostic technique over the last decade compared to right ventricular angiography and biopsy (9).

Aim of the present study was to reassess by follow-up CMR studies findings detected by a first CMR study in patients, in whom the diagnosis early stage ARVC was suspected.

METHODS

Consecutive patients referred from general practitioners for arrhythmia related symptoms and/or right ventricular arrhythmias were included in this study. Institutional ethics committee approved the study protocol and all patients gave written informed consent.

Baseline evaluation included clinical examination, 12-lead ECG, 24-hour Holter ECG, echocardiography and CMR. The study population consisted of patients with right ventricular arrhythmias, in whom ARVC could not be excluded based on all information available. Only two patients were asymptomatic, both had non sustained ventricular tachycardia (NSVT) on Holter ECG. Patients with a clearly more likely diagnosis were excluded from the study. A second exclusion criterion was the implantation of a cardiac defibrillator (ICD) during follow-up in patients with increased risk for sudden death, since follow-up CMR was no longer possible. All patients were clinically evaluated after a minimal follow-up of one year. Detailed arrhythmia history was obtained, and all patients underwent clinical examination and had a 12-lead ECG, 24-hour Holter ECG, a transthoracic echocardiography and a CMR follow-up study. CMR studies were performed with a 1.5 Tesla MR scanner (Magnetom Symphony; Siemens, Erlangen, Germany) equipped with Quantum gradients (amplitude 30 mT/m; slew rate 100 T/s*m) and a 4-element (circular polarized) phased array body coil. Images were acquired in the short and long axis planes with T1-weighted turbo spin echo (TR/TE = RR-interval/7.1 ms) and TrueFisp (TR/TE = 50/1.8 ms) techniques. During the course of the study, the protocol was optimized by additional slices acquired in the axial plane. Such slices are particularly helpful for the assessment of the RV anterior free wall. Baseline and follow-up CMR studies were analyzed offline and reviewed by two physicians (one radiologist and one cardiologist), experienced in CMR diagnostics (16). Both investigators were blinded with regard to patient's history, the results of other studies as well as the baseline CMR findings. Right ventricular volume and dilation, global and regional right ventricular function, right ventricular wall thinning, moderator band thickening, localized aneurysms, right ventricular fibrosis and fatty tissue replacements were systematically assessed. According to a prespecified protocol an imaging score was calculated (Table 1). A score of one was given to each negative parameter, a score of five to each positive. A score of two was defined as probably negative, a score of three as uncertain and a score of four as probably positive. CMR-diagnosis of ARVC was made if at least two parameters were scored ≥4 (Category II). This scoring system has been shown to be predictive of arrhythmic events during follow-up (16). In a similar fashion, two-dimensional and Doppler echocardiography were analyzed by two different cardiologists experienced in the echocardiographic diagnosis of ARVC.

Table 1. CMR protocol

	Score				
Morphology					
RV					
Regional RV-wall thinning	1	2	3	4	5
Moderator band thickening	1	2	3	4	5
Aneurysm	1	2	3	4	5
RVOT					
Aneurysm	1	2	3	4	5
Tissue characterization					
RV					
Fibrosis	1	2	3	4	5
Fatty tissue	1	2	3	4	5
Function					
RV					
Regional abnormal motility	1	2	3	4	5
Global abnormal motility	1	2	3	4	5
Dilation	1	2	3	4	5

According to type and severity of abnormal right ventricular findings, results of CMR and echocardiography were classified into two diagnostic probability categories (Table 2). In category I (ARVC unlikely), there were no or only minimal right ventricular abnormalities (all scores ≤2). Patients with unequivocal findings were assigned to category II (ARVC likely, at least two parameters scored ≥4). Final assignment was made by consensus.

Continuous variables are indicated as mean ± one standard deviation. SPSS for windows (version 11.0) was used to

Table 2. Summary of the 3 categories into which patients were classified according to CMR and echo findings

Category I	ARVC unlikely: no or minimal RV abnormalities
Category II	ARVC likely (=CMR diagnosis of ARVC): typical RV alterations

RV = right ventricle.

Table 3. Baseline characteristics and results of principal examinations

Patient	Age (yrs)	Sex	Follow-up (months)	Symptoms baseline	Symptoms follow-up	ECG baseline	Arrhythmias baseline	Arrhythmias follow-up	CMR baseline (category)	CMR follow-up (category)	Echo follow-up (category)
1	36	Female	12	Palp/dizziness	Palpitations	Normal	NSVT	No	1	1	1
2	28	Male	39	Syncope	Palpitations	Normal	PVC+§	No	2	1	1
3	46	Male	14	Palp/dizziness	Asymptomatic	Negative T V1-5	No	PVC++	2	2	2
4	41	Male	33	Palpitations	Asymptomatic	Negative T V1-2	PVC+	PVC++	2	1	1
5	39	Male	33	Chest pain	Chest pain	Normal	NSVT	No	2	1	1
6	43	Male	57	Dizziness	Dizziness	Early repolarisation	SVT	PVC+	2	1	1
7	38	Female	59	Palpitations	Palpitations	PVC	PVC+	No	2	1	1
8	36	Female	55	Palpitations	Palpitations	Negative T V1-4	SVT	PVC+	2	2	2
9	41	Male	35	Asymptomatic	Asymptomatic	Normal	NSVT	PVC++	2	1	1
10	40	Female	48	Palpitations	Palpitations	PVC	PVC++	PVC+	2	2	1*
11	35	Male	58	Palpitations	Palpitations	PVC	PVC++	No	1	1	1
12	33	Female	28	Syncope	Palpitations	Incomplete RBBB	PVC+	PVC+	1	1	1
13	33	Male	38	Asymptomatic	Syncope	PVC	NSVT	NSVT	1	2	na
14	40	Female	13	Palpitations	Palpitations	Normal	No	NSVT	1	1	1
15	47	Male	35	Palpitations	Asymptomatic	Incomplete RBBB	PVC+	PVC+	1	1	1
16	24	Male	59	Syncope	Psymptomatic	Incomplete RBBB	PVC+	No	1	1	1
17	39	Female	40	Palpitations	Palpitations	Negative T V1-3	PVC++	PVC+	1	1	1
18	62	Female	58	Palp/dizziness	Palpitations	PVC	PVC+	PVC+	1	1	1

Palp/dizziness = Palpitations and dizziness; RBBB = Right Bundle Branch Block; NSVT = Non Sustained Ventricular Tachycardia; SVT = Sustained Ventricular Tachycardia; PVC = Premature Ventricular Contraction; § = +: <1000 PVC's/24 hours on Holter-ECG; ++: >1000 PVC's/24 hours on Holter-ECG; * = Restricted image quality; na = not available.

calculate Cramer's Phi correlation coefficient for the association of echocardiographic findings with findings on CMR at follow-up examination. Chi square test was used to assess statistical significance. A p-value of <0.05 was considered to indicate statistical significance.

RESULTS

The initial study population of patients referred for the assessment of right ventricular arrhythmias consisted of 26 patients. Five patients with severe ARVC and serious arrhythmic events in whom an ICD was implanted, were excluded from the present analysis (16). Four of them had an appropriate ICD shock. One patient died due to ICD dysfunction, with correct detection of ventricular fibrillation, but failure to defibrillate. According to the CMR classification, four patients with subsequent ICD implantation were in category II, and one was in category I. One patient was lost to follow-up and two patients did not give informed consent. All three had normal findings at the baseline CMR examination and neither of them had been given a diagnosis of ARVC. The remaining 18 patients (age 39 $\pm$ 18 years, eight female) were included in the present follow-up study (Table 3). Mean duration of follow-up was 37 $\pm$ 16 months (range 12–59 months). Family history was unremarkable in all patients. Only 2 patients (11%) were asymptomatic at baseline, one had atypical chest pain. Eight patients (44%) had palpitations, three (17%) had palpitations associated with dizziness, one had dizziness only, and three (17%) experienced at least one syncope. In 16 patients (89%), ventricular arrhythmias were documented at baseline (Table 3).

During follow-up, five patients (28%) remained asymptomatic, 10 (56%) felt occasional palpitations, one complained of dizziness, one had syncope and one still suffered from recurrent atypical chest pain (Table 3). There were no ECG changes during follow-up. In six patients, no arrhythmias were documented by the follow-up Holter ECG, seven (39%) had <1000 premature ventricular contractions (PVC), three (17%) had >1000 PVC's and two (11%) had NSVT.

At baseline CMR, nine patients (50%) were in category II (ARVC likely), and nine (50%) in category I (ARVC unlikely) (Table 2 and Figure 1). At follow-up CMR, in 11 patients (61%) the interpretation of CMR remained unchanged. In eight of nine patients (89%) with unlikely ARVC (category I), diagnosis was confirmed at follow-up (Fig. 1). One patient from category I at baseline received an ICD after a syncope, an increase of NSVT on Holter and new typical features of ARVC at follow-up CMR.

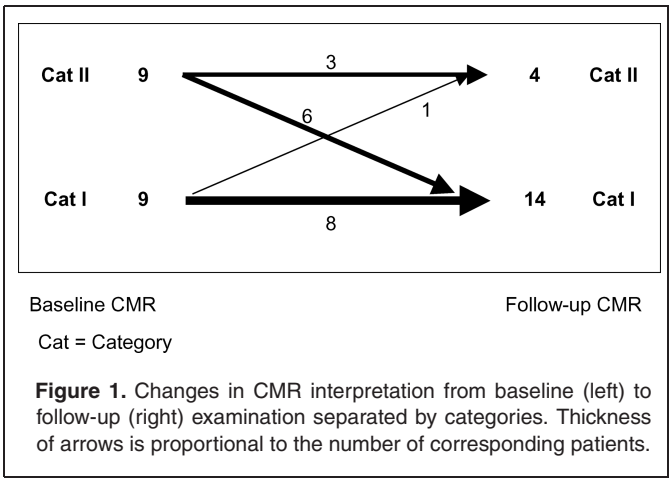

Figure 1. Changes in CMR interpretation from baseline (left) to follow-up (right) examination separated by categories. Thickness of arrows is proportional to the number of corresponding patients.

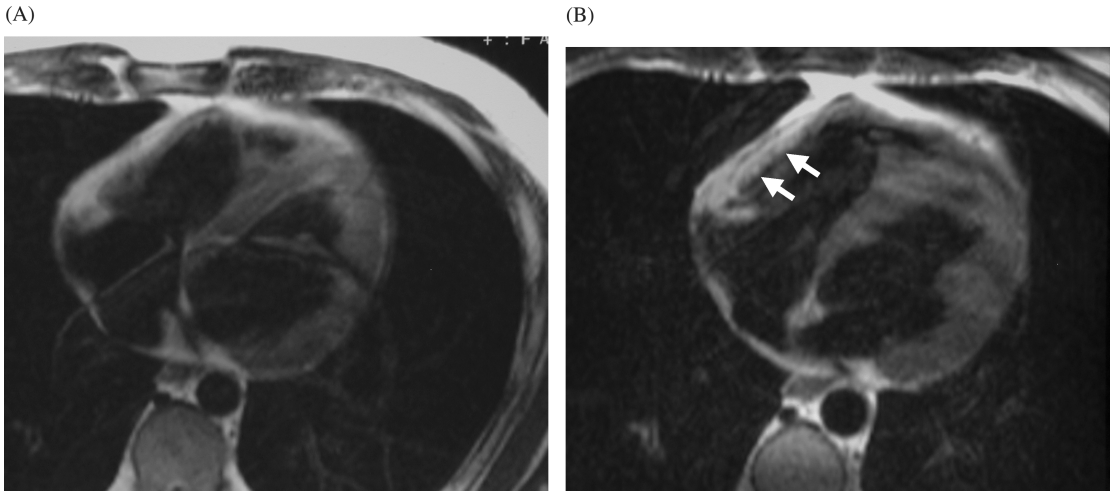

Figure 2. 33-year old male referred for suspected ARVC. T1 weighted axial TSE (A) showed normal signal in the free wall of the right ventricle (arrows) and normal dimensions of the right ventricle. This study was classified as category I (ARVC unlikely). Three years later the follow up study (B) shows progression of these fatty infiltrations, classified as category II (ARVC likely).

He was reclassified into category II (Fig. 2). In one patient from category I, an isolated aneurysm at the right ventricular apex was detected, but he remained in category I at follow-up. Three of nine patients (33%) from category II had a follow-up CMR confirming baseline findings (Fig. 3). Six patients (67%) from category II were reclassified into category I, because follow-up CMR was interpreted as normal (Fig. 4).

More detailed analysis of CMR results revealed why 67% of patients with pathologic CMR findings at baseline (category II) had normal CMR at follow-up (category I). In five of six patients (83%), interpretation of right ventricular fatty infiltrates made the main difference in the analysis of follow-up CMR. Only in one patient, interpretation of localized wall motion abnormalities and aneurysms was different and led to a reclassification from category II to category I.

Standardized echocardiographic assessment at follow-up was available for all but one patient (Table 3). Compared to CMR results, all patients with negative follow-up CMR had a negative echocardiography (one patient with RVOT wall thinning on echo, but not on CMR). Two out of three subjects in category II on CMR examination were in the same category when assessed by echocardiography. In one patient, echocardiographic findings were wall thinning, hypokinesia and aneurysm in the RVOT, whereas the same findings plus a possible fatty infiltration were attributed to the apex on CMR. The second patient had apicolateral hypokinesia and an apical aneurysm on both examinations

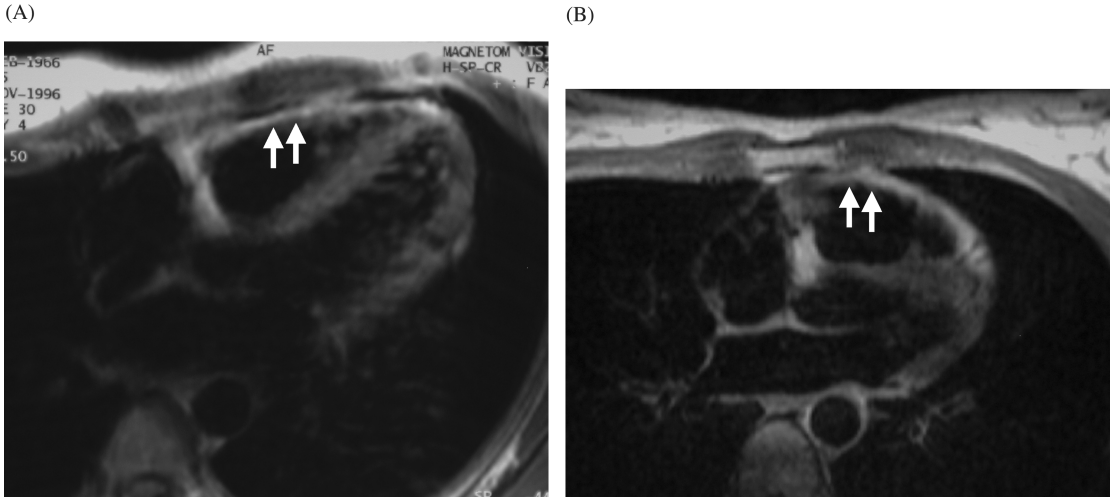

Figure 3. 36-year old female. T1 weighted TSE (A) shows transmural high signal intensity in the free wall of the right ventricle (arrows) consistent with transmural fatty infiltration in ARVC (category II). Findings on follow up scan 3 years later (B) were unchanged (category II).

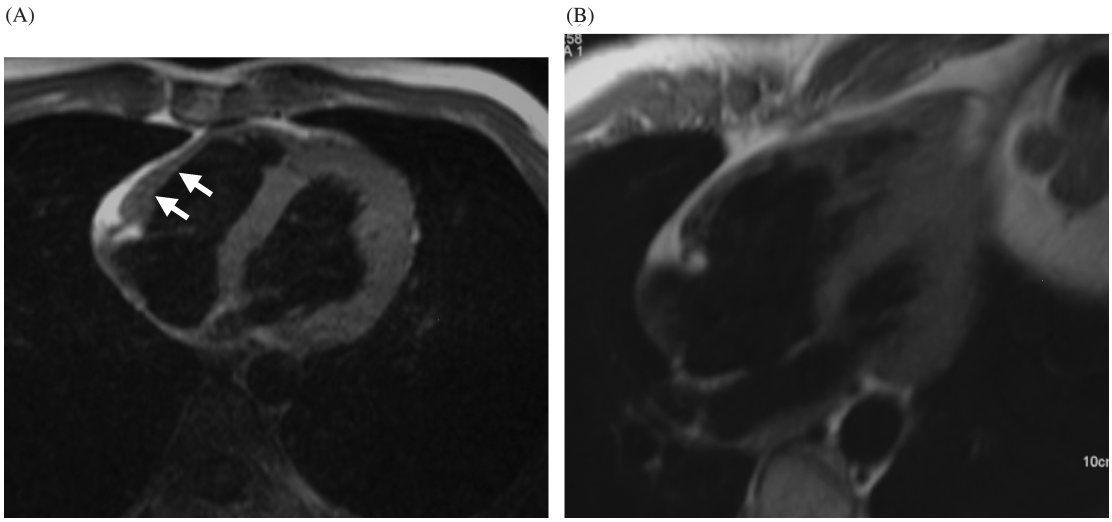

Figure 4. 41-year old male. T1 weighted axial TSE (A) shows spots with high signal intensity in the free wall of the right ventricle (arrows) suspicious of fatty infiltration (category II). Dimension and function of the right ventricle were normal. The follow up scan 3 years later (B) was normal, spots with high signal intesity in the anterior wall of the right ventricle were not present. Thus, CMR was reclassified as category I (ARVC unlikely). At reevaluation of the previous study (A) the signal disturbances were interpreted as artifacts rather than intramural fatty infiltration.

(plus apical fatty infiltration on CMR) (Table 3). The third was graded into category I, but image quality in this individual was judged insufficient for a clear diagnosis. This patient was excluded for the statistical analysis. Correlation for these two examinations was good ($r = 0.81$, $p = 0.001$).

DISCUSSION

To our knowledge, this is the first study assessing the value of follow-up CMR in patients with suspected ARVC. The main result was a striking discrepancy in the interpretation of follow-up CMRs compared to baseline. In 67% of patients who were given a CMR diagnosis of likely ARVC at baseline, these findings were not confirmed at CMR after 3 years of follow-up. On the other hand, we could confirm the diagnosis of unlikely ARVC in 89% of patients in category I at baseline. This corrobates the good negative predictive value of CMR.

Only one patient experienced clinical symptoms of ARVC progression. Possible reasons for the very low clinical event rate observed during the study may be the inclusion of patients with a low pre-test probability of ARVC on one side and the exclusion of patients with overt ARVC who qualified for an ICD, on the other.

We found a low specificity of CMR in our patients. This was mainly explained by different visualization and/or interpretation of fatty infiltrate in the right ventricular myocardium. There are several possible explanations for the different findings on follow-up CMR. Interpretation of the right ventricle is difficult and investigator-dependent (4, 14, 15). Particularly, the differentiation of pathologic fat replacement of the right ventricular myocardium as a consequence of necrosis may be difficult to be separated from harmless penetration of epicardial fat frequently seen in obese patients. This has also been recognized by other investigators, who found a high interobserver variability related to this problem (17). Likewise, the discrepancies found in our study are most likely due to different interpretation of the CMR studies. During the course of the trial, experience with this relatively new technique increased. Therefore, differentiating and interpreting RV structures have been improved, leading to improved differentiation between truly abnormal findings and normal variants. At the beginning of the study, the role of RV fatty infiltrates have probably been overinterpreted, as 5 of 6 patients were reclassified because of different interpretation of RV fatty infiltrates. Finally, the addition of axial slices to CMR protocol during the study could have contributed to the improved interpretation. In summary, the reason for the low specificity is probably a combination of lack of experience, subjectivity of diagnostic criteria, lack of gold standard and artifacts.

The clinical diagnosis of ARVC remains difficult, and therefore, is usually based on multiple clinical as well as laboratory findings. The Task Force of the Working Group on Cardiomyopathies has established criteria to facilitate clinical diagnosis of this disease (5). However, since these criteria were mainly derived from symptomatic patients with more advanced disease, the value of these quite specific but not very sensitive criteria is not known when applied to an asymptomatic low-risk population.

With regard to CMR, it has been reported that asymptomatic relatives of patients with familial ARVC have minor CMR abnormalities in up to 11%, which complicates the interpretation of minor findings (18). Furthermore, structural alterations as seen in CMR (9–13) are not very specific for ARVC. Similar changes have been described in patients with idiopathic right ventricular outflow tract tachycardia (19, 20), right ventricular PVCs (21) and even in elderly subjects with normal hearts (22). In addition, some forms of myocarditis may be very similar to ARVC (23).

However, in patients meeting the Task Force criteria, a higher incidence of right ventricular fatty infiltrations, more aneurysms and higher right ventricular volumes compared to normals have been described (24). Furthermore, prognosis in patients with ARVC and abnormal findings on CMR is worse than in those with normal CMR (16). Patients with ICD had to be excluded from the present follow-up study. All of them had an elevated CMR-score at baseline, and the event rate was high. This findings further underscore the usefulness of CMR in patients with more advanced disease.

Limitations

First, the sample size of our study was relatively small. However, in the group with positive CMR findings at baseline, the findings were striking and probably not sample size dependent. Secondly, as already mentioned, this study examined a selected low-risk population. Therefore, our results should not be applied to other groups of patients with a different pre-test probability of having the disease. Thirdly, interobserver variability was not formally addressed in the present study. All diagnoses were made by consensus finding. Fourthly, the validity of the 2 categories, that were applied in this study has not been validated prospectively. Therefore, the cut-off points may be somewhat artificial. And lastly, a general limitation of every ARVC study is the lacking diagnostic gold standard for in vivo diagnosis.

Conclusion

Our data suggest that analysis of CMR studies dealing with potential ARVC cases is difficult, probably due to a combination of interpretation errors, lack of experience and the absence of objective diagnostic criteria. A diagnosis of early stages of ARVC based on positive CMR findings alone should be made with caution. In patients with repeated normal CMR, ARVC can be safely excluded, emphasizing the high negative predictive accuracy of CMR. Finally, CMR remains a valuable tool for risk stratification in patients with suspected ARVC.

ACKNOWLEDGMENTS

We thank the Swiss Heart Foundation for financial support of the present study.

REFERENCES

1. Ahmad F. The molecular genetics of arrhythmogenic right ventricular dysplasia-cardiomyopathy. Clin Invest Med 2003 Aug;26:167–78.
2. Thiene G, Nava A, Corrado D, Rossi L, Pennelli M. Right ventricular cardiomyopathy and sud den death in young people. N Engl J Med 1988;318:129–33.
3. Shen WK, Edwards WD, Hammill SC, Bailey KR, Ballard DJ, Gersh BJ. Sudden unexpected nontraumatic death in 54 young adults: a 30-year population-based study. Am J Cardiol 1995;76:148–52.
4. Corrado D, Basso C, Thiene G. Arrhythmogenic right ventricular cardiomyopathy: Diagnosis, prognosis and treatment. Heart 2000;83:588–95.
5. Mc Kenna WJ, Tshiene G, Nava A, Fontaliran F, Blomstrom-Lundquist C, Fontaine G. Diagnosis of arrhythmogenic right ventricular dysplasia/cardiomyopathy. Br Heart J 1994;71:215–8.
6. Wichter T, Borggrefe M, Breithardt G. Arrhythmogene rechtsventrikuläre Kardiomyopathie; Aetiologie, Diagnostik und Therapie. Med Klin 1998;93:268–77.
7. Sechtem U, Neubauer S, Revel D, et al. The clinical role of magnetic resonance in cardiovascular disease. Task force report. Task force of the European Society of Cardiology, in collaboration with Association of European Paediatric Cardiologists. Eur Heart J 1998;19:19–39.
8. Pattynama PMT, Lamb HJ, Van der Velde EA, et al. Reproducibility of MRI-derived measurements of the right ventricular volumes and myocardial mass. Magn Reson Imaging 1995;13:53–63.
9. Auffermann W, Wichter T, Breithardt G, et al. Arrhythmogenic right ventricular disease: MR imaging vs angiography. Am J Roentgenol 1993;161:549–55.
10. Blake LM, Scheinmann MM, Higgins CB. MR features of arrhythmogenic right ventricular dysplasia. Am J Roentgenol 1994;162:809–12.
11. Casolo GC, Poggesi L, Boddi M, et al. ECG-gated magnetic resonance imaging in right ventricular dysplasia. Am Heart J 1987;113:1245–8.
12. Ricci C, Longo R, Pagnan L, et al. Magnetic resonance imaging in right ventricular dysplasia. Am J Cardiol 1992;70:1589–95.
13. Midiri M, Finazzo M, Brancato M, et al. Arrhythmogenic right ventricular dysplasia: MR features. Eur Radiol 1997;7:307–12.
14. Corrado D, Fontaine G, Marcus F, et al. Arrhythmogenic right ventricular dysplasia/cardiomyopathy: need for an international registry. Circulation 2000;101:e101–6.
15. Marcus FI. Update of arrhythmogenic right ventricular dysplasia. Card Electrophysiol Review 2002;6:54–6.
16. Keller DI, Osswald S, Bremerich J, et al. Arrhythmogenic right ventricular cardiomyopathy: Diagnostic and prognostic value of the Cardiac MRI in relation to arrhythmia-free survival. Int J Card Imag 2003;19:537–43.
17. Bluemke DA, Krupinski EA, Ovitt T, et al. MR imaging of arrhythmogenic right ventricular cardiomyopathy: Morphologic findings and interobserver reliability. Cardiology 2003;99:153–62.
18. Hamid MS, Norman M, Quraishi A, et al. Prospective evaluation of relatives for familial arrhythmogenic right ventricular cardiomyopathy/dysplasia reveals a need to broaden diagnostic criteria. J Am Coll Cardiol 2002;40:1445–50.
19. Carlson MD, White RD, Trohmann RG, et al. Right ventricular outflow tract ventricular tachycardia: detection of previously unrecognized anatomic abnormalities using cine magnetic resonance imaging. J Am Coll Cardiol 1994;24:720–7.
20. White RD, Trohmann RG, Flamm SD, et al. Right ventricular arrhythmia in the absence of Arrhythmogenic right ventricular dysplasia: MR images of myocardial abnormalities. Radiology 1998; 207:743–51.
21. Proclemer A, Basadonna PT, Slavich GA, et al. Cardiac magnetic resonance imaging findings in patients with right ventricular outflow tract premature contractions. Eur Heart J 1997;18:2002–10.
22. Burke AP, Farb A, Tashko G, et al. Arrhythmogenic right ventricular cardiomyopathy and fatty replacement of the right ventricular myocardium: are they different diseases? Circulation 1998;97:1571–80.
23. Chimenti C, Pieroni M, Maseri A, Frustaci A. Histologic findings in patients with clinical and instrumental diagnosis of sporadic arrhythmogenic right ventricular dysplasia. J Am Coll Cardiol 2004;43:2305–13.
24. Tandri H, Calkins H, Nasir K, et al. Magnetic resonance imaging findings in patients meeting task force criteria for arrhythmogenic right ventricular dysplasia. J Cardiovasc Electrophysiol 2003;14:476–82.