Clinical and electrophysiological differences between patients with arrhythmogenic right ventricular dysplasia and right ventricular outflow tract tachycardia

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Aims Radiofrequency catheter ablation is considered first line treatment for symptomatic patients with right ventricular outflow tract tachycardia (RVOT). The role of ablation in arrhythmogenic right ventricular dysplasia (ARVD) is more limited. As such, differentiating between the two conditions is essential.

Methods and results This study compared non-invasive findings, magnetic resonance images (MRI), invasive electrophysiological characteristics, results of ablation and long-term outcome in 50 consecutive patients with RVOT (33) or ARVD (17). Structural abnormalities were uniform in the ARVD group; in addition 18 (54\%) of the RVOT tachycardia group had MRI abnormalities. At electrophysiological study the tachycardia in the ARVD group displayed features of re-entry in over 80\%, but behaved with a triggered automatic basis in 97\% with RVOT. Ablation was complete or partial success in 12 (71\%) patients with ARVD and ventricular tachycardia (VT) recurred in eight (48\%). In the RVOT patients, ablation was a complete success in 97\% with recurrent VT in 6\%. Long-term success in the RVOT patients was 95\% in both patients with and without MRI abnormalities.

Conclusions Electrophysiological characterization can differentiate ARVD from RVOT. The finding of abnormalities on MRI does not have any bearing on arrhythmia mechanism, acute or long-term success of RFA.

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KEYWORDS
Ventricular tachycardia; Catheter ablation; Cardiomyopathy

Introduction
Right ventricular tachycardia is commonly due to arrhythmogenic right ventricular dysplasia/ cardiomyopathy (ARVD) or idiopathic right ventricular outflow tract (RVOT) tachycardia.\textsuperscript{1,2} ARVD is an inherited progressive cardiomyopathy characterized by right ventricular dysfunction due to fibrofatty replacement of myocardium, predisposing to ventricular tachycardia and death.\textsuperscript{3–7} Right ventricular outflow tract tachycardia is a benign condition, traditionally considered to be a primary electrical disease in the absence of structural heart disease.\textsuperscript{8–11} The differentiation between ARVD and RVOT tachycardia is important clinically when discussing prognosis and management options.

At the extremes of disease presentation differentiating between the two conditions is usually straightforward. However, detailed analyses using
magnetic resonance imaging (MRI),\textsuperscript{12–15} signal averaged electrocardiograms\textsuperscript{1,11} and endomyocardial biopsy\textsuperscript{1} have detected structural abnormalities in patients with RVOT tachycardia, which are similar to those seen in the early stages of ARVD. These findings have made the differentiation of ARVD and RVOT tachycardia at the time of initial diagnosis more difficult in some patients. It is unclear whether the two conditions represent separate entities or together form a continuous spectrum of disease, with RVOT tachycardia representing a concealed or early form of ARVD. The clinical and prognostic significance of the structural abnormalities detected with newer technologies is uncertain.

The aim of this study was to compare the two conditions with regard to the demographic, electrocardiographic, structural and invasive electrophysiological characteristics, in patients with ARVD or RVOT tachycardia. These findings were also analysed to identify any correlation with the acute or longer term success of radiofrequency ablation.

Methods

Patients

This study analysed details of 50 consecutive patients with right ventricular tachycardia, in the absence of coronary disease, surgical scars or left ventricular dysfunction, who were scheduled to undergo electrophysiological study and radiofrequency ablation. The right ventricular origin was determined by electrocardiographic criteria and confirmed in all cases at electrophysiological study. The patients were prospectively diagnosed as ARVD or RVOT tachycardia, according to guidelines published by the Study Group on ARVD/C of the Working Group of Myocardial and Pericardial Diseases of the European Society of Cardiology and of the Scientific Council on Cardiomyopathies of the International Society and Federation of Cardiology.\textsuperscript{16} This classification takes account of genetic, electrocardiographic depolarization and repolarization, arrhythmic, structural and histological factors. Based on this classification, the diagnosis of ARVD requires the presence of two major criteria or one major and two minor criteria or four minor criteria.

Electrocardiographic analysis

A standard 12-lead electrocardiogram was recorded during sinus rhythm free from anti-arrhythmic drug effects. The QRS/T complex in V\textsubscript{1–3} was examined for the presence of epsilon waves, right bundle branch block and T wave inversion. QRS and QT measurements were made using a Calcomp Digitizer from electrocardiograms recorded at a paper speed of 50 mm s\textsuperscript{-1}.

Signal averaged electrocardiograms were obtained using a Marquette MAC VU system. Recordings were made during sinus rhythm using standard Frank leads X, Y and Z. Two hundred and fifty beats were averaged to obtain a noise level of \textless 0.3 \mu V. The signals were amplified, averaged and filtered with a bidirectional filter at frequencies of 40–250 Hz. The duration of the total filtered QRS complex, the duration of the filtered QRS complex after the voltage decreased to \textless 40 mV, and the root mean square of the amplitude of signals in the last 40 ms of the filtered QRS complex were measured by a computer algorithm. Results were deemed abnormal if any two of the following criteria were met: (1) the filtered QRS duration was \textgeq 120 ms; (2) the duration of the filtered QRS complex after the voltage decreased to \textless 40 mV, was \textgeq 40 ms; (3) the root mean square of the voltage in the last 40 ms of the QRS complex was \textless 20 mV.

Ventricular tachycardia morphology was obtained either from a 12-lead electrocardiogram recording during sustained ventricular tachycardia or from an electrocardiogram with consistent monomorphic ventricular ectopic beats.

Structural analysis

Transthoracic echocardiography

All patients underwent transthoracic echocardiography prior to the study, which was reported by a cardiologist blinded to the clinical details. The focus on the right ventricle included measurements of overall right ventricular size and function, as well as a description of localized right ventricular aneurysms, segmental dilatation or regional wall motion abnormalities.

Magnetic resonance imaging

MRI of the heart was performed according to a standardized technique in all patients. Scans were performed either before or a minimum of 12 months after the ablation procedure. The MRI was obtained with a Magnetom Vision (Siemens, Germany) system. Using a breath-hold technique the static images were obtained as T1-weighted spin echo and T2-weighted STIR scans. Cine images were obtained using cardiac-gated sequences.
The MRI was reported by a single cardiac radiologist blinded to the clinical details. Each study was examined for abnormalities in the morphology of the right and left ventricle. Measurements of right ventricular free wall thickness were made with focal thinning defined as a wall thickness of less than 2 mm. Fatty deposition was defined as high intensity intramyocardial lesions on T1-weighted images, the presence and extent of fatty deposition was recorded. Cine studies evaluated wall motion with areas of hypokinesis, dyskinesis or aneurysm formation noted.

MRI abnormalities considered major included right ventricular dilatation, wall motion abnormalities, diffuse right ventricular wall thinning and diffuse fatty infiltration. Focal areas of right ventricular wall thinning or fatty infiltration of less than 2 cm² were defined as minor MRI abnormalities.

Electrophysiological study

All patients gave informed consent and underwent electrophysiological study and ablation according to standard protocols. Anti-arrhythmic medications and warfarin were discontinued 5 days prior to the procedure. Two quadrapolar catheters were placed into the right ventricle, at the apex and in the outflow tract.

Programmed electrical stimulation was performed from the right ventricular apex using an eight beat drive of 600 ms with up to five extra-stimuli introduced sequentially until refractory levels were reached or ventricular tachycardia was induced. Inducible monomorphic tachycardia was considered significant and recorded.

Following programmed electrical stimulation, isoprenaline was commenced and the dose titrated until the sinus rate had increased by 50% or was greater than 120 beats min⁻¹; this was repeated twice with a washout phase of 5 min. Spontaneous tachycardia and repetitive monomorphic ventricular beats were recorded (Fig. 1). Programmed electrical stimulation was repeated during the second isoprenaline infusion. In the absence of tachycardia, ventricular pacing consisting of 20 beat bursts, reducing from 400 ms to 240 ms in 20 ms intervals was performed and the reliance of induction on specific cycle lengths noted.

A minimum of 30 sites in the right ventricle were mapped in sinus rhythm to detect areas of inhomogeneous activation and slow conduction. Fragmented electrograms were defined as multi-component signals with a duration >60 ms and an amplitude of <1.5 mV.¹⁷

Ablation procedure

During sustained tachycardia, activation and entrainment mapping¹⁸ were performed to localize the tachycardia circuit. If sustained tachycardia could not be induced, the area of interest was mapped in sinus rhythm observing for fragmented electrograms and utilizing pacemapping.¹⁹ The ablation strategy involved point ablation for patients with triggered automatic behaviour. Linear ablation, targeting the diastolic pathway and exit, was attempted in patients demonstrating features of re-entry.²⁰ Ablation was performed using standard 4 mm tip catheters, temperature limited to 60 degrees. Ablation was performed for 60–120 s at each site.

Procedural success

Procedural success was defined as the absence of any inducible tachycardia at the end of the ablation procedure using both programmed stimulation and Isoprenaline. A partial success was defined as successful ablation of the clinical tachycardia but inducible non-clinical tachycardias.

Follow-up

All patients were followed-up at pre-specified intervals of 1, 6 and 12 months and then yearly. In addition, final follow-up data was collected in October 2001. At each review, patients completed standardized questionnaires regarding symptoms and underwent a standard 12-lead electrocardiogram, 48-h ambulatory electrocardiographic recordings and transthoracic echocardiography. Results of implantable cardioverter defibrillator interrogations, unscheduled outpatients appointments and hospital admissions were also collected.

Statistical analysis

Continuous variables are expressed as mean± standard deviation. Continuous variables were compared using a two tailed t-test. Discrete variables were compared using a Chi-squared test, unless the variable being analysed had fewer than five occurrences, in which case a Fisher's exact test was used. P<0.05 was considered significant.

Results

Demographic data

The study comprised 50 patients, 22 males, with a mean age of 40 (range 17–56). Seventeen of the
patients were classified as ARVD and 33 as RVOT tachycardia. The demographic, structural, electrocardiographic and clinical data is detailed in Tables 1 and 2.

There were no significant differences in symptoms between patients with ARVD and RVOT tachycardia. The mode of presentation was syncope or collapse in 42% and palpitations in the remaining 58%. Exercise potentiation of symptoms was reported in 26 (79%) of the RVOT tachycardia patients and in 10 (59%) patients with ARVD. Almost 60% of patients with ARVD had a family member with ARVD or a family history of premature sudden cardiac death. None of the RVOT tachycardia patients had a family history of tachycardia.

Electrocardiographic analysis

The routine electrocardiogram was abnormal in 52% of patients with ARVD and in 6% of patients with RVOT tachycardia. Thirty percent of the patients with ARVD displayed epsilon waves, incomplete right bundle branch block or a localized prolongation of the QRS complex in V1 or V2. T wave inversion in the right precordial leads (V1–V3) was seen in 36% of patients with ARVD and 6% of patients with RVOT tachycardia. Late potentials on the signal averaged electrocardiogram were not present in any patient with RVOT tachycardia but were present in 78% of the patients with ARVD (Figs. 2 and 3).

All patients had ventricular tachycardia or repeated monomorphic ventricular ectopic beats documented on electrocardiogram. In all cases a left bundle branch block pattern was present. All patients with RVOT tachycardia and nine (53%) of the patients with ARVD had a family history of premature sudden cardiac death. None of the RVOT tachycardia patients had a family history of tachycardia.

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Clinical characteristics of enrolled patients</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>ARVD</td>
</tr>
<tr>
<td>Number</td>
<td>17</td>
</tr>
<tr>
<td>Age</td>
<td>40±10</td>
</tr>
<tr>
<td>Male</td>
<td>11 (65%)</td>
</tr>
<tr>
<td>Syncope</td>
<td>8 (47%)</td>
</tr>
<tr>
<td>Exercise potentiation</td>
<td>10 (59%)</td>
</tr>
<tr>
<td>Family history</td>
<td>9 (53%)</td>
</tr>
<tr>
<td>ECG depolarization</td>
<td>5 (30%)</td>
</tr>
<tr>
<td>ECG repolarization</td>
<td>6 (36%)</td>
</tr>
<tr>
<td>Arrhythmias</td>
<td>17 (100%)</td>
</tr>
<tr>
<td>Structural</td>
<td></td>
</tr>
<tr>
<td>Major</td>
<td>7 (41%)</td>
</tr>
<tr>
<td>Minor</td>
<td>7 (41%)</td>
</tr>
<tr>
<td>Number of major criteria</td>
<td>1.2</td>
</tr>
<tr>
<td>Number of minor criteria</td>
<td>3.0</td>
</tr>
</tbody>
</table>

Family history, ECG depolarization and repolarization abnormalities, arrhythmias, structural and major and minor criteria are all defined according to the Study Group on Arrhythmogenic Right Ventricular Dysplasia/Cardiomyopathy of the Working Groups on Myocardial and Pericardial Disease and Arrhythmias of the European Society of Cardiology and of the Scientific Council on Cardiomyopathies of the World Heart Federation. Family history, ECG depolarization and repolarization abnormalities, arrhythmias, structural and major and minor criteria are all defined according to the Study Group on Arrhythmogenic Right Ventricular Dysplasia/Cardiomyopathy of the Working Groups on Myocardial and Pericardial Disease and Arrhythmias of the European Society of Cardiology and of the Scientific Council on Cardiomyopathies of the World Heart Federation.12

<table>
<thead>
<tr>
<th>Table 2</th>
<th>Structural abnormalities</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>ARVD</td>
</tr>
<tr>
<td>TTE–RV dilatation</td>
<td>6 (35%)</td>
</tr>
<tr>
<td>TTE–RV RWMA</td>
<td>2 (12%)</td>
</tr>
<tr>
<td>MRI–RV dilatation</td>
<td>8 (47%)</td>
</tr>
<tr>
<td>MRI–RV RWMA</td>
<td>11 (65%)</td>
</tr>
<tr>
<td>MRI–Fat &gt;2 cm&lt;sup&gt;2&lt;/sup&gt;</td>
<td>9 (53%)</td>
</tr>
<tr>
<td>MRI–Fat &lt;2 cm&lt;sup&gt;2&lt;/sup&gt;</td>
<td>7 (41%)</td>
</tr>
<tr>
<td>MRI–Thinning &gt;2 cm&lt;sup&gt;2&lt;/sup&gt;</td>
<td>10 (59%)</td>
</tr>
<tr>
<td>MRI–Thinning &lt;2 cm&lt;sup&gt;2&lt;/sup&gt;</td>
<td>7 (41%)</td>
</tr>
<tr>
<td>Major MRI findings</td>
<td>15 (88%)</td>
</tr>
<tr>
<td>Minor MRI findings</td>
<td>2 (12%)</td>
</tr>
<tr>
<td>No abnormalities</td>
<td>0</td>
</tr>
</tbody>
</table>

ARVD=arrhythmogenic right ventricular dysplasia; RVOT=right ventricular outflow tract tachycardia; TTE=transthoracic echocardiogram; RV=right ventricle; RWMA=regional wall motion abnormality; MRI=magnetic resonance imaging. Major MRI findings=right ventricular wall motion abnormality, right ventricular dilatation, fatty infiltration >2 cm<sup>2</sup> or focal thinning >2 cm<sup>2</sup>. Minor MRI findings=fatty infiltration <2 cm<sup>2</sup> or focal thinning <2 cm<sup>2</sup>.

Structural assessments

Transthoracic echocardiograms detected structural abnormalities in seven (42%) patients with ARVD. Major MRI abnormalities were present in 15 (88%) ARVD patients. All patients with echocardiographic abnormalities also had major abnormalities on MRI scans. The remaining two (12%) ARVD patients demonstrated minor abnormalities only on MRI scans.

Fig. 1 (a,b) An example of two different morphologies of ventricular tachycardia that were both seen clinically in a patient with ARVD. (c) An example of monomorphic ventricular ectopic beats which were induced with isoprenaline in a patient with RVOT tachycardia.
Structural abnormalities were detected on MRI scans in 18 (54%) patients with RVOT tachycardia but no echocardiographic abnormalities were present. The MRI abnormalities were considered major in two (6%) and minor in the remaining 16 (48%).

Right ventricular cine angiography was performed at the discretion of the cardiologist in 23 patients, 11 with ARVD and 12 with RVOT. This was reported as abnormal in seven patients with ARVD, but none with RVOT.

**Electrophysiological study and radiofrequency ablation**

Ventricular tachycardia was inducible with programmed electrical stimulation in 14 (82%) patients with ARVD, but in only one (3%) with RVOT tachycardia. All with RVOT tachycardia had ventricular tachycardia or frequent monomorphic ventricular ectopic beats during isoprenaline infusion. Ventricular tachycardia was sustained, defined as lasting longer than 30 s, in 12 (71%) of the ARVD patients and in five (15%) of the RVOT tachycardia group.

The ARVD patients had an average of 1.8 morphologies of inducible ventricular tachycardia (range 1–6), with 12 (71%) demonstrating more than one morphology (Table 3). At least one of the induced morphologies matched the clinical ventricular tachycardia in all patients. All RVOT
Tachycardia patients demonstrated only a single morphology of ventricular tachycardia or ventricular ectopic beats, which was identical to that recorded clinically. The mean cycle length of ventricular tachycardia was 360 ms (265–440 ms) in the RVOT patients and 310 ms (230–420 ms) in the ARVD patients.

Mapping of the right ventricle revealed fragmented potentials in the majority of patients with ARVD and only in a single patient with RVOT tachycardia. The sites of the fragmented potentials correlated poorly with successful ablation sites. The successful ablation sites in the RVOT patients had a mean pre-systolic activation of 28 ms (13–48 ms) and this was greater than 30 ms in 18 patients. In the ARVD group, concealed entrainment was demonstrated for 15 different ventricular tachycardias in 12 patients. Fragmented electrograms and diastolic potentials guided ablation in the remaining patients. The mean number of ablations per procedure was 10; the number of ablations in the ARVD patients (mean 18, range 7–38) was significantly greater than in the RVOT tachycardia patients (mean 6, range 1–22) (P<0.01).

**Acute success**

Acute procedural success was achieved in 32 (97%) and failure in one (3%) of the RVOT patients. Acute success was achieved in seven (41%) of the ARVD patients with a partial success in five (29%) and failure in five (29%).

There were no significant differences in demographic, electrophysiological or conduct of ablation between patients with a procedural success or failure.

**Follow-up**

The mean follow-up was 56 months (13–92 months), and all patients were followed-up for a minimum of 12 months. Recurrence of ventricular tachycardia was documented in eight (47%) of the ARVD patients and two (6%) of the RVOT tachycardia patients. Recurrence in the RVOT tachycardia patients followed a successful procedure in one patient and an unsuccessful procedure in the other. In the ARVD patients, recurrence followed a complete or partially successful procedure in three patients and in five patients with an unsuccessful. No patients died during the follow-up period.

Two of the ARVD patients had an implantable cardioverter defibrillator prior to the procedure and an additional six patients received a defibrillator following the procedure. Fifteen of the ARVD patients, including all patients who were not treated with an implantable cardioverter defibrillator, were maintained long-term on sotalol (4) or amiodarone (11). None of the patients with RVOT received an implantable cardioverter defibrillator or class 3 anti-arrhythmic agents. The RVOT patient who failed ablation was discharged on atenolol.

**Outcome in patients with MRI abnormalities**

The presence of MRI abnormalities in the patients with RVOT tachycardia did not have a significant effect on acute procedural success. The procedure was successful in 17/18 with MRI abnormalities and 15/15 without MRI abnormalities (P=ns).

Recurrent ventricular tachycardia was seen in two RVOT tachycardia patients; one patient with MRI abnormalities and an unsuccessful initial procedure, and one with asymptomatic recurrence.

### Table 3  Electrophysiological studies

<table>
<thead>
<tr>
<th></th>
<th>ARVD</th>
<th>RVOT</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Inducible with PES</td>
<td>14 (82%)</td>
<td>1 (3%)</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Inducible with isoprenaline</td>
<td>3 (18%)</td>
<td>27 (81%)</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Monomorphic VEBs only</td>
<td>1 (6%)</td>
<td>6 (18%)</td>
<td>ns</td>
</tr>
<tr>
<td>Sustained tachycardia</td>
<td>12 (71%)</td>
<td>5 (15%)</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Number of VT morphologies</td>
<td>1.8±0.8</td>
<td>1.0±0.1</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Greater than one VT morphology</td>
<td>12 (71%)</td>
<td>0</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Fragmented potentials</td>
<td>14 (82%)</td>
<td>1 (3%)</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Number of ablations</td>
<td>15±5</td>
<td>8±3</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Successful procedure</td>
<td>8 (48%)</td>
<td>32 (97%)</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Follow-up (months)</td>
<td>58±18</td>
<td>54±17</td>
<td>ns</td>
</tr>
<tr>
<td>Recurrent VT</td>
<td>8 (48%)</td>
<td>2 (6%)</td>
<td>&lt;0.01</td>
</tr>
</tbody>
</table>

ARVD=arrhythmogenic right ventricular dysplasia; RVOT=right ventricular outflow tract tachycardia; VT=ventricular tachycardia; PES=programmed electrical stimulation; VEB=ventricular ectopic beat. Sustained tachycardia >30 s; ns=not significant.
following an initially successful procedure in a patient without MRI abnormalities (Table 4). The patient with the initial failed procedure had a successful repeat procedure using an irrigated tip catheter. The patient with an asymptomatic recurrence declined a further procedure.

Discussion

This study compared 50 consecutive patients with ARVD or RVOT tachycardia, using traditional diagnostic tools, MRI and invasive electrophysiological features and correlated these with procedural success and long-term outcome following radiofrequency ablation. The major findings of the study are firstly that ARVD and RVOT tachycardia are different electrophysiological syndromes. Regardless of results from non-invasive investigations, electrophysiological findings can independently differentiate the two diagnoses on the basis of the mechanism of induction, the number of ventricular tachycardia morphologies and fragmented electrograms. And secondly, that in RVOT tachycardia the presence of minor abnormalities on MRI has no impact on electrophysiological properties, ablation success or long-term recurrence of ventricular tachycardia.

Invasive electrophysiology study

This report has demonstrated that invasive electrophysiological findings can differentiate ARVD and RVOT tachycardia. In this study ventricular tachycardia was inducible by programmed electrical stimulation in 82% of the patients with ARVD, but in only 3% of those with RVOT tachycardia. Conversely tachycardia was uniformly inducible with isoprenaline and cycle length dependent in the patients with RVOT tachycardia. Seventy percent of the patients with ARVD had more than one morphology of inducible ventricular tachycardia and the majority had significant zones of fragmented electrograms, whereas all RVOT tachycardia patients had only a single morphology of ventricular tachycardia and fragmented electrograms were rarely seen.

These electrophysiological findings suggest significant differences in underlying disease process between ARVD and RVOT patients. Arrhythmia induction with programmed electrical stimulation particularly in the presence of fragmented electrograms favours re-entry as the predominant mechanism for tachycardia in ARVD patients. Induction of ventricular tachycardia by isoprenaline with cycle length dependence is consistent with a cyclic-AMP mediated triggered activity being the predominant mechanism in RVOT tachycardia.

The more extensive disease process evidenced by zones of fragmented electrograms contributes to the different ventricular tachycardia morphologies seen in individual ARVD patients.

The findings at electrophysiological study clearly differentiate the two diagnoses on the basis of their different arrhythmic mechanisms. This allows accurate identification of patients who will be cured by ablation and remain free from recurrence or progression of tachycardia.

Structural information

With advances in technology, non-invasive imaging has become more sensitive in detecting right ventricular structural abnormalities. In this study, MRI detected twice as many structural abnormalities in the ARVD patients than a combination of transthoracic echocardiography and right ventricular cine angiography. None of the RVOT tachycardia patients had structural abnormalities on echocardiography or angiography. However, MRI detected abnormalities in 54%. Similar rates of MRI abnormalities in patients with RVOT tachycardia have previously been reported.

The extent of MRI abnormality also differed between patients with ARVD and RVOT tachycardia. Major structural abnormalities were seen on MRI in 88% of patients with ARVD but in only 6% of those with RVOT tachycardia. Minor abnormalities only were detected in the remaining 12% of ARVD patients and in 48% of patients with RVOT tachycardia.

<table>
<thead>
<tr>
<th>Table 4</th>
<th>Relationship of MRI findings in patients with right ventricular outflow tract tachycardia to acute and long-term success of radiofrequency ablation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Number</td>
</tr>
<tr>
<td>Overall</td>
<td>33</td>
</tr>
<tr>
<td>MRI abnormality</td>
<td>18 (54%)</td>
</tr>
<tr>
<td>No MRI abnormality</td>
<td>15 (46%)</td>
</tr>
</tbody>
</table>
In this report the electrophysiological characteristics did not correlate with the presence or absence of abnormalities on MRI. Mode of tachycardia induction, presence of fragmented signals and number of ablations in the RVOT tachycardia patients was similar whether MRI abnormalities were present or not. The presence of abnormalities on MRI did not have any bearing on acute or the longer term success or subsequent recurrence of ventricular tachycardia following ablation.

The finding of a degree of structural abnormality on MRI has led to speculation that RVOT tachycardia may be an early form of ARVD with the same potential for disease progression. However, studies to date have failed to show evidence of disease progression during follow-up of patients with RVOT tachycardia. This analysis supports the previous findings, with no evidence of disease progression or arrhythmia recurrence during follow-up following ablation.

Previous studies

This analysis supports the conclusions of others that ARVD and RVOT tachycardia are fundamentally different entities that can usually be distinguished clinically. Major abnormalities on standard electrocardiograms, signal-averaged electrocardiograms or transthoracic echocardiography are hallmarks of ARVD.

Contrary to previous reports, frequency of symptoms and history of syncope were similar in ARVD and RVOT tachycardia. This study enrolled only patients referred for electrophysiological evaluation and this has introduced selection bias into the sample. The patients enrolled in the study probably represent the more symptomatic end of the RVOT tachycardia spectrum. This may have blunted any expected differences in symptom frequency between the two groups.

The procedural success rates and ventricular tachycardia recurrence rates in this report are in keeping with previously published figures. Ablation is rarely curative of all arrhythmias in ARVD; the acute success rate is reported to be less than 40% with high later recurrence rates over time due to the progressive nature of the disease. The success rate of ablation in the ARVD patients in this study was 42% and the recurrence rate 48% over 58 months of follow-up. Ablation for RVOT tachycardia, on the other hand, is traditionally thought to be curative in the majority of patients and the acute success rate of 97% and long-term success rate of 94% in this report is expected.

Clinical implications

The importance of electrophysiological findings in predicting arrhythmia mechanism and long-term outcome should assist in recommending ongoing therapy following radiofrequency ablation. Even in the presence of minor structural abnormalities ablation is curative in the majority of patients with features of RVOT and a focal arrhythmia mechanism.

Recurrent ventricular tachycardia is seen in almost half of the patients with ARVD following an ablation procedure; 19% after a successful procedure and 100% following an unsuccessful procedure. The recurrence of ventricular tachycardia after a successful procedure in ARVD is significantly higher than after successful ablation of RVOT or even ventricular tachycardia associated with ischaemic heart disease. The inability of a successful procedure to reliably protect against ventricular tachycardia recurrence in ARVD patients suggests that ablation should be reserved for patients with highly symptomatic episodes and considered an adjunct to implantable cardioverter defibrillators.

This study in part attempted to address the issue of disease progression amongst RVOT tachycardia patients. Whilst no evidence of disease progression was documented, the rate of progression in ARVD is unknown and may be highly variable. The follow-up in this study may be insufficient and serial evaluations over a longer time frame would be needed to definitively answer the question of progression.

Conclusions

This analysis shows that ARVD and RVOT tachycardia can be differentiated clearly on the basis of electrophysiological characteristics. The findings suggest the two conditions behave as fundamentally different entities.

The presence of minor structural abnormalities on MRI in RVOT tachycardia patients is consistent with benign natural history, high ablation success rates and low later ventricular tachycardia recurrence.

References


