Ablation of unstable ventricular dysrhythmias guided by non contact mapping system among patients with cardiomyopathy in a growing single center experience

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Abstract We report our local experience in using the non contact mapping system in guiding catheter ablation of unstable/non sustained Ventricular tachycardia (VT) and its short and long term implications.

Patients and methods: This report includes 18 cardiomyopathic patients, 13 males, age 40.5 ± 15 yrs, who presented to our centre with VT that share in common being unstable or non sustained. The patients were subjected to radiofrequency catheter ablation guided by the non contact mapping system.

Results: Acute successful outcome was obtained in 6 out of 8 (75%) patients with scar related VTs (post myocardial infarction and arrhythmogenic right ventricular dysplasia) and 9 of 10 (90%) patients with idiopathic dilated cardiomyopathy. Long term follow up for 6–24 (16 ± 8) months showed recurrence in one case of the scar related group and in two cases of the idiopathic group, so the overall long term success rates were 62.5% vs 70% (P, NS). Regular Echocardiographic showed an improvement of 10–15% in the Ejection Fraction in successful cases of the idiopathic group (Average Post-ablation EF in the idiopathic group of 49 ± 5% vs pre-ablation EF of 41 ± 4% while 42 ± 6 pre Vs 42 ± 7% post ablation in the scar related group) (P < 0.01).

Conclusion: Non contact mapping guided RF ablation of unstable VT in patients with cardiomyopathy showed good immediate results and long term outcome both in scar related and in idiopathic cardiomyopathy patients. Successful RF ablation of non sustained ventricular dysrhythmias among patients with idiopathic Cardiomyopathy may improve LV dysfunction.

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1. Introduction

Radiofrequency (RF) catheter ablation is an effective therapy for stable monomorphic ventricular tachycardia (VT) in patients with and without structural heart disease. New mapping tools have been developed (Carto, Localisa, Realtime...
Position Management) which create maps that correlate anatomy with cardiac electrical activation to facilitate RF ablation of complex arrhythmias.\(^1,2\) However, these systems require stable tachycardia to be able to create the activation map. In case of unstable ventricular tachycardia not allowing activation mapping, systems which provide global mapping data as non-contact mapping system were reported to give good results.\(^3,4\)

With global mapping methods, substrates for short-lived and multiple arrhythmias can be defined, the data stored, and an ablation catheter navigated to the chosen site(s) enabling ablation of the target either during sinus rhythm or at the onset of a VT that would otherwise be of short duration. This has been confirmed by the experience using non-contact mapping when all target and many other VTs were successfully ablated.\(^4,5\)

In this study, we report our local experience in using the non contact mapping system in guiding RF catheter ablation of unstable ventricular tachycardias in patients with different types of cardiomyopathy and its short and long term outcomes.

2. Patients and methods

All patients with symptomatic unstable VT in patients with cardiomyopathy referred to the electrophysiology Laboratory of Saud Al-babtain Cardiac Centre (SBCC), Ad-Dammam, KSA, between December 2008 and May 2011, for the trial of RF ablation were included in the study.

2.1. Inclusion criteria

1. Significant unstable VT either due to haemodynamic instability with disturbing symptoms like syncope, presyncope that necessitate immediate termination of the VT, or frequent non sustained symptomatic VT with a significant daily arrhythmic burden \(\geq 20\%\) linked to LV systolic dysfunction.
2. Refractoriness to drug therapy or inappropriateness of medical treatment.

2.2. Exclusion criteria

I. Patients with poor prognosis due to non cardiac causes as end stage malignancy, significant renal or hepatic diseases were excluded from the study.
II. Patients with reversible ischaemic heart disease until fully investigated and any required revascularization is done.
III. Local causes that hinder implementing the procedure as LV thrombus or severe peripheral vascular disease and inability to go trans-septally if LV is the target.

- The study was approved by the local ethics committee of the Centre and patients gave informed written consents before including in the study.
- The patients were subjected to full clinical assessment, 12 lead ECG, X-ray chest, ventricular tachycardia was defined with standard electrocardiographic criteria of at least five consecutive premature ventricular contractions (PVC) at a rate \(>120\) beats/min. Echocardiographic assessment to assess LV function, LV ejection fraction (EF) and exclude left ventricular thrombus. We evaluated the chamber size of LV and right ventricle (RV), and degree of mitral regurgitation at parasternal long-axis or apical four-chamber view, respectively. All values of echocardiogram were recorded during sinus rhythm, but not at the PVC beat, nor at the post-PVC beat. All patients also Anti-arrhythmic drugs as Amiodarone, Sotalol, Beta blocker alone or in combinations had been unsuccessful for variable periods before the patients were subjected to RF ablation (Patients’ characteristics are shown in Table 1).

2.3. Electrophysiological study and mapping technique

Electrophysiological testing and RF catheter ablation were performed in the fasting state. Anti-arrhythmic drugs other than amiodarone were discontinued one week before the study.

The stimulation protocol consisted of programmed ventricular stimulation from the right ventricular apex and outflow tract (RVOT) at four different cycle lengths with up to three premature extra-stimuli. Surface ECG leads and endocardial electrograms were displayed and recorded simultaneously at a speed of 100 or 200 mm/s. Data were stored on an optical CD-Rom for further evaluation. The induced VT was initially localized to either ventricle by comparing timing of RV and LV recorded electrograms to decide the ventricle to map.

Before deployment of the multi-electrode array balloon (MEA), patients were given 10,000 IU heparin IV injection with later boluses to maintain activated clotting time at 300 to 400 s. The MEA catheter was deployed via the retrograde trans-aortic route if the LV is the target chamber except in one patient who had severe peripheral vascular disease and we had to go through the trans-septal approach. If the RV is the target chamber, the venous femoral-caval approach was used for introducing the MEA.

2.4. The mapping procedure using the Ensite system

The non-contact mapping system and the principles of its use were described in details previously.\(^3,5\) Ventricular tachycardia induced with programmed electrical stimulation was then recorded using the noncontact computerized mapping system for at least half a minute if it can be haemodynamically tolerated for this period, otherwise, recording for few seconds was considered before overstimulation of the recorded tachycardia. The virtual unipolar electrograms and isopotential maps were used for offline analysis of the recorded VTs to trace the exit, the presystolic and diastolic activation during VT, zones of slow conduction moving back from VT exit sites as described before\(^6\) (Fig. 1). Also voltage maps were used to identify scar areas by very low amplitude electrograms \(<0.5\) mV, while voltage between 0.5 and 1.5 mV was considered mixed borderline tissues and \(>1.5\) mV was coded as healthy tissues according to a previous suggested criteria.\(^7,10\)

2.5. Ablation policy

Radiofrequency ablation using an 8 mm tip IBI ablation catheter (Biotronic-Germany) with energy up to 50 W titrated to a
temperature of maximum 60 °C for 90 s per application. RF AC was administered by the use of a continuous sinusoidal unmodulated waveform of 500 kHz (Stockert GMBH ablation system). Energy was delivered between the tip electrode of the ablation catheter and a 10 × 16-cm external back plate electrode. In all patients, RF pulses were applied during sinus rhythm. Energy application was continued at each site rather than performing drag lesions and the lines were completed according to Ensite map.

In scar related VTs, when re-entry was the responsible mechanism, the linear lesions were attempted to produce lines of conduction block across the areas of diastolic activities and/or slow conduction and/or connecting two scar areas or from a scar to an anatomic boundary across an isthmus. Then the conduction block across the lines was tested by tracing the impulse propagation during pacing using the Ensite propagation map analysis and then testing the non inducibility of the VT thereafter. In post inferior infarction patients, special attention was given to the sub-mitral isthmus such that the energy delivery was continued to extend the line up to the mitral valve ring.

In cases when the nature of the tachycardia was found to be focal in origin, RF lesions were directed to the identified exit in an areal manner to isolate the identified exit. After the set of lesions is completed, the VT inducibility or spontaneous occurrence was retested.

We defined immediate success as non inducibility of the previously inducible targeted clinical VTs at the end of the procedure; acute success was defined as no clinical recurrence during the 1st 5 days post ablation, short term success within 3 months and Long term success if beyond 3 months.

2.6. Follow-up

After the ablation session, all patients had continuous ECG monitoring for at least 5 days or until ICD implantation if

<table>
<thead>
<tr>
<th>#</th>
<th>Age (ys)</th>
<th>Sex</th>
<th>Aetiology of cardiomyopathy</th>
<th>VT type</th>
<th>Site of origin</th>
<th>Pre-EF</th>
<th>Post-EF</th>
<th>Result of ablation</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>43</td>
<td>M</td>
<td>Post MI (IHD)</td>
<td>Incessant post myocardial infarction VT</td>
<td>LV BASAL INFERIOR SEPTAL</td>
<td>30%</td>
<td>35%</td>
<td>Successful ABLATION</td>
</tr>
<tr>
<td>2</td>
<td>30</td>
<td>F</td>
<td>Idiopathic LV dysfunction</td>
<td>VT arised from subpulmonary free wall RVOT Unstable VT</td>
<td>RVOT VT</td>
<td>45%</td>
<td>55%</td>
<td>Successful Ablation</td>
</tr>
<tr>
<td>3</td>
<td>28</td>
<td>F</td>
<td>ARVC/M</td>
<td>LV PVCs/NS VT</td>
<td>RV interoseptal position</td>
<td>50%</td>
<td>50%</td>
<td>FAILED</td>
</tr>
<tr>
<td>4</td>
<td>16</td>
<td>M</td>
<td>Idiopathic LV VT</td>
<td>Monomorphic PVCs, LV MID TO APICAL INFEROSEPTAL</td>
<td>45%</td>
<td>45%</td>
<td>Successful ABLATION</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>33</td>
<td>M</td>
<td>Idiopathic LV VT</td>
<td>Successful ablation, MID SEPTAL High POSTERO-SEPTAL RVOT</td>
<td>37%</td>
<td>50%</td>
<td>Successful ablation</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>29</td>
<td>F</td>
<td>Idiopathic RVOT PVCs</td>
<td>Not identified</td>
<td>-</td>
<td>30%</td>
<td>45%</td>
<td>Successful Ablation</td>
</tr>
<tr>
<td>7</td>
<td>22</td>
<td>M</td>
<td>Idiopathic RV PVCs/NS VT, VT</td>
<td>RV PVCs/NS VT, VT</td>
<td>-</td>
<td>40%</td>
<td>40%</td>
<td>Failed Ablation Of RVOT VEs</td>
</tr>
<tr>
<td>8</td>
<td>50</td>
<td>M</td>
<td>Idiopathic RVOT PVCs/NS VT</td>
<td>RVOT, (Inferoposterior septal Wall)</td>
<td>40%</td>
<td>55%</td>
<td>Successful Ablation Of RVOT VEs</td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>35</td>
<td>M</td>
<td>ARVC/M</td>
<td>RV PVCs/NS VT, VT</td>
<td>RV basal septal</td>
<td>50%</td>
<td>50%</td>
<td>Successful ABLATION PVCs</td>
</tr>
<tr>
<td>10</td>
<td>45</td>
<td>M</td>
<td>Idiopathic LVOT non sustained VT</td>
<td>LVOT NSVT</td>
<td>-</td>
<td>40%</td>
<td>45%</td>
<td>Successful ABLATION</td>
</tr>
<tr>
<td>11</td>
<td>58</td>
<td>M</td>
<td>Post MI (IHD)</td>
<td>Incessant VT</td>
<td>LV MID SEPTAL</td>
<td>25%</td>
<td>25%</td>
<td>SUCCESSFUL ABLATION</td>
</tr>
<tr>
<td>12</td>
<td>73</td>
<td>F</td>
<td>Post MI (IHD)</td>
<td>Post MI unstable VT</td>
<td>LV mid anterior septum</td>
<td>40%</td>
<td>43%</td>
<td>Successful ablation, late Recurrence SUCCESSFUL Ablation</td>
</tr>
<tr>
<td>13</td>
<td>29</td>
<td>M</td>
<td>Idiopathic RVOT NSVT</td>
<td>RVOT</td>
<td>-</td>
<td>45%</td>
<td>57%</td>
<td>Successful ablation</td>
</tr>
<tr>
<td>14</td>
<td>62</td>
<td>M</td>
<td>Post MI (IHD)</td>
<td>Haemodynamically unstable VT PVCs, NS VT, VT from the RV body</td>
<td>LV basal MID TO Ant. Septum</td>
<td>35%</td>
<td>35%</td>
<td>Successful ablation</td>
</tr>
<tr>
<td>15</td>
<td>37</td>
<td>F</td>
<td>ARVC/M, Early Recurrence Idiopathic</td>
<td>RVOT non sustained VT</td>
<td>RV basal Ant. Septum</td>
<td>55%</td>
<td>55%</td>
<td>Successful RF ablation</td>
</tr>
<tr>
<td>16</td>
<td>45</td>
<td>M</td>
<td>Idiopathic RVOT non sustained VT</td>
<td>RVOT non sustained VT</td>
<td>Antero septal RVOT just below the PV</td>
<td>45%</td>
<td>45%</td>
<td>Successful RF ablation</td>
</tr>
<tr>
<td>17</td>
<td>56</td>
<td>M</td>
<td>Idiopathic RVOT non sustained VT</td>
<td>RVOT non sustained VT</td>
<td>Low Anterior RVOT</td>
<td>45%</td>
<td>55%</td>
<td>Successful RF ablation</td>
</tr>
<tr>
<td>18</td>
<td>40</td>
<td>M</td>
<td>Post Inferior MI (IHD)</td>
<td>Unstable VT</td>
<td>LV BASAL INFERIOR SEPTAL</td>
<td>40%</td>
<td>40%</td>
<td>Successful VT ablation</td>
</tr>
</tbody>
</table>

Table 1 Criteria of individual patients serially(F: female, M: male, NS VT: non sustained ventricular tachycardia, LV: left ventricular, RVOT: right ventricular outflow tract, ARVC/M: arrhythmogenic right ventricular cardiomyopathy, IHD: ischemic heart disease, MI: myocardial infarction.)
implantation was planned. Clinical follow up and Echocardiography were performed for 1 to 2 days and at 1 and 3 months then 6 months after the ablation procedure and changes in Ventricular functions were observed.

2.7. Statistics

Continuous variables are presented as mean and SD and compared using the Student’s t test for unpaired data. Categorical data were compared by x2 analysis. Regarding success rates and EF changes in relation to the ablation method and the statistical significance of the results were analysed by Chi-square test, and the exact Fisher’s test. Values of $P < 0.05$ were considered significant.

3. Results

This report includes 18 patients, 12 males, of age 40.5 ± 15 yrs, presenting to our centre in Saud Alhabtain Cardiac Centre, KSA with ventricular dysrhythmias that share in common being unmappable by the activation mapping techniques due to unstable non sustained nature of the tachycardia or instability of the patient during the attack necessitating immediate termination. They included 5 patients with ischaemic Heart disease with a previous myocardial infarction (MI) (3 post inferior and 2 post anterior MI), 3 patients with Arrhythmogenic Right Ventricular Cardiomyopathy (ARVC) and 10 patients with idiopathic dilated cardiomyopathy in whom IHD was excluded either by myocardial perfusion imaging (MPI) or coronary angiography (CAG) and with no significant scar as assessed by MPI. The average pre-ablation Left ventricular ejection fraction (EF) was 41 ± 7%, ranging from 25% to 55%. ALL the ischaemic heart disease patients had already been revascularized – by percutaneous Coronary Intervention (PCI) in 4 patients and by Redo Coronary Artery Bypass Graft (CABG) Surgery in the fifth patient – before referral to the EP laboratory. 3 patients had previously undergone defibrillator implantation as a part of the treatment of life-threatening ventricular arrhythmias. Other 5 patients received it later after the procedure. The indications for ablation of VT were Recurrent episodes of documented haemodynamically unstable monomorphic VT in 5 patients post MI with frequent ICD shocks in three patients of them with already implanted ICDs. Rest of the patients had symptomatic palpitation and syncpe in 3 patients with ARVD, and frequent non sustained VT/PVCs in 10 patients with impaired LV function thought to be secondary to the significant burden of repetitive non sustained VT (> 20% of the 24 h Heartbeats).

3.1. Results of the mapping and ablation procedures

The overall duration of the procedure was 146.5 ± 35 min, average fluoroscopic exposure time of 27.5 ± 12 min, average RF pulses 20 ± 8 (7–40 RF pulses). By programmed stimulation, 23 VT morphologies with a CL of (230–330, 260 ± 50 ms) were induced and targeted for ablation. 12 VTs were induced in scar related cardiomyopathy patients: 7 morphologies in 5 patients with post MI VT and 5 morphologies in 3 ARVC patients. Offline investigator analysis of the isopotential maps was variable according to the complexity of the substrate and number of induced VTs reaching up to 30 min in some cases.

The VT mapping by the Ensite system showed re-entry as the responsible mechanism of VT in all scar related VT cases (8 cases). Exit sites could be identified and a diastolic pathway could be traced for a variable interval (50–160 ms) in the 12 VT morphologies induced in patients with scar related VTs (Fig. 1). Guided by the propagation map and the voltage scar maps, Lines of RF lesions in the range of 23–40 RF pulses (30 ± 8) were required according to the ablation policy mentioned above to reach the end point of non inducibility of the VT (Fig. 2).
Among the idiopathic cardiomyopathy group, 3 patients had LV origin and 7 patients found to have RV origin (5 from RVOT and 2 from RV body) and all showed a focal origin of VT and were targeted by exit isolation by RF lesions in the range 5–16 (11 ± 5) RF pulses till abolishment and noninducibility of the targeted ventricular arrhythmia. The number of RF pulses required was significantly less in patients with idiopathic forms compared to scar related VTs. The duration of the procedure and fluoroscopy exposure was significantly less also in the idiopathic group (Table 2).

Overall, acute successful outcome was obtained in 15 patients (83%) while ablation failed in three patients: One post MI case with recurrent unstable VT after a redo CABG who had an electrical storm short after his redo CABG and was uncontrollable by medical treatment. During the ablation procedure, the patient had again an electrical storm with 2 VT morphologies and their pathways were targeted by lines of RF ablations according to our ablation policy in the scar related patients. The trial of RF ablation was initially followed by 12 h of stability and non recurrence then the patient had a recurrent VT that became partially controllable by medical treatment. One week later, this patient died in a refractory VT/VF. The 2nd failed case had ARVC and the VT remained inducible despite performing a line of RF lesions across the path of the VT connecting two scars across the VT isthmus. The VT remained inducible with the changes in its pathway and morphology. The third case had frequent non sustained VT in an idiopathic cardiomyopathy patient, thought originally from the ECG and by the initial provisional crude bipolar localization to arise from the RVOT. However, the origin of the VT could not be localized on the endocardial side of the RVOT using the non contact mapping analysis and all

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**Table 2** Comparison of characteristics and outcome of patients included in the report. (VT: ventricular tachycardia, NSVT: non sustained ventricular tachycardia, PVCs: premature ventricular contractions, EF: ejection fraction, RF: radiofrequency, NS: non significant).

<table>
<thead>
<tr>
<th>Patient</th>
<th>Scar related VT group</th>
<th>Idiopathic cardiomyopathy NSVT/PVCs</th>
<th>P. value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of patients</td>
<td>8</td>
<td>10</td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>51 ± 16 yrs</td>
<td>33 ± 10 yrs</td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>6</td>
<td>7</td>
<td>NS</td>
</tr>
<tr>
<td>Pre-ablation EF</td>
<td>42 ± 7%</td>
<td>41 ± 4%</td>
<td>NS</td>
</tr>
<tr>
<td>Tried Anti-arrhythmic drugs</td>
<td>4 ± 1</td>
<td>3 ± 1</td>
<td></td>
</tr>
<tr>
<td>No of inducible VTs</td>
<td>12</td>
<td>11</td>
<td>NS</td>
</tr>
<tr>
<td>VT site of origin (LV/RV)</td>
<td>5/3</td>
<td>3/7</td>
<td>??NS</td>
</tr>
<tr>
<td>Duration of procedures</td>
<td>176.6 ± 22 min</td>
<td>85 ± 25 min</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Fluoroscopy duration</td>
<td>42.5 ± 12 min</td>
<td>25 ± 10 min</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Number of RF pulses</td>
<td>30 ± 8</td>
<td>11 ± 5</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Acute Success rate</td>
<td>75%</td>
<td>90%</td>
<td>NS</td>
</tr>
<tr>
<td>Long term success rate</td>
<td>62.5%</td>
<td>70%</td>
<td>NS</td>
</tr>
<tr>
<td>Post ablation EF</td>
<td>42 ± 6%</td>
<td>49 ± 5%</td>
<td>P &lt; 0.01</td>
</tr>
<tr>
<td>Cases with post ablation improve</td>
<td>Non/8</td>
<td>6/10</td>
<td>P &lt; 0.001</td>
</tr>
</tbody>
</table>
trials of ablation on the earliest points could not eliminate the VT.

3.2. Post ablation follow up

During long term follow up of 6–24 (16 ± 8) months, late recurrence was observed in three cases. Two cases belonged to the idiopathic cardiomyopathy patients, one case with recurrent non sustained VT from the RV body and another case with LV tachycardia with overall long term success rate in that group of 70%. The third long term recurrence occurred in one of the post MI patients i.e. belonged to the scar related group, with overall long term success rate in this group of 62.5%, however with no significant difference from the idiopathic group (Table 2).

Regular Echocardiography assessment during the follow up period showed an improvement of 10–15% in the EF in 6 successful cases all belonging to the idiopathic group with an average post ablation EF of 49 ± 5% compared to the pre-ablation EF of 41 ± 4%. The scar related group did not show a similar change (Post ablation EF of 42 ± 7% vs. pre-ablation 42 ± 6%) (P value < 0.01). (Table 2)

3.2.1. Complications

No deaths or strokes were directly related to the procedure, limited local femoral haematoma occurred in two cases.

4. Discussion

The difficulty of the ablation procedure increases when unmappable VTs are present. Many laboratories restrict ablation attempts to patients with mappable VTs. Sacher et al. document that unstable VT is present in more than two thirds of patients with structural heart disease who are undergoing ablative therapy. VT that is unmappable with a single roving catheter may be mapped with a system that simultaneously records electrograms throughout the ventricle during one or a few beats of the unstable VT, following which the VT can be terminated to allow ablation during stable sinus rhythm. This may be achieved by using a system that records electrical potentials from an electrode grid array within the cavity of the ventricle as the Ensite non contact mapping system (Endocardial Solutions, St Paul, Minnesota, USA). Sites of early endocardial activity, which are likely adjacent to re-entry circuit exits, are usually identifiable; in some cases, isthmuses and diastolic pathways have been identified. In our study, we used the non contact mapping system to record and analyse unstable ventricular dysrhythmias in cardiomyopathic patients of different pathologies. In patients whom the VT is scar related as patients after myocardial infarction or patients with ARVC, the VT mechanisms were found to be re-entrant in nature. In patients with idiopathic cardiomyopathy, non sustained ventricular dysrhythmias showed focal origin except in one case when VT exit could not be precisely determined and the nature of the VT was obscure. The voltage scar mapping could help in addition to the VT circuit analysis in guiding the RF applications done during sinus rhythm in patients with scar related VT and this resulted in acute success in about 75% of the cases. Failure in one post MI patient was associated with early mortality while over the long term follow up, one further recurrence occurred in this group.

Targeting unmappable VTs was tried in many reports using different techniques. Ellison and colleagues targeted the likely re-entry exit region in five patients with frequent unmappable VT. They did mapping during sinus rhythm and performed ablation over a region of identifiable scar that contains abnormal conduction and a presumptive VT exit guided by pace mapping. All three patients with prior myocardial infarction were free of recurrent VT during follow ups of 14–22 months. The procedure was not successful in two cases with non-ischaemic cardiomyopathy.

The voltage scar mapping and ablation of uncontrolled VTs by RF applications during sinus rhythm using electroanatomic mapping (Carto System) was described by Marchlinski et al. and they used voltage definitions of scar, borderline and normal myocardium which was later adopted in other studies and we used the same criteria in our study. They applied a more extensive series of RF ablation lines through regions of scar in 16 patients with recurrent unmappable VT (prior myocardial infarction in nine patients) guided by bipolar catheter mapping during baseline rhythm. The amount of endocardium with an abnormal electrogram amplitude was estimated using fluoroscopy in 3 patients and the magnetic mapping system (CARTO) in 13 patients. For the magnetic mapping, normal endocardium was defined by an amplitude > 1.5 mV; this measurement was based on sinus rhythm maps in 6 patients who did not have structural heart disease. Radiofrequency point lesions were extended linearly from the “dense scar” which had a voltage amplitude < 0.5 mV, to anatomic boundaries or normal endocardium to limit radiofrequency applications. During a median follow up of eight months 75% remained free of VT recurrences. One patient suffered a stroke, emphasizing the potential risk of placing extensive lesions in the left ventricle. In a post-mortem study, these voltage thresholds correlated very well with histology, since massive (> 80%) fibrosis has been found in areas with voltage < 0.5 mV, while intermediate (21%–79%) and minimal (< 20%) fibrosis have been observed in areas with voltage 0.5–1.5 mV and > 1.5 mV, respectively.

A similar methodology to ours, applied in similar patient groups was described previously by Della Bella et al. They used the non contact mapping system to guide RF ablation of unmappable VT in patients with cardiomyopathy of different aetiologies. The placement of linear lesions, by radiofrequency current, across the diastolic pathway resulted in successful ablation of 78% of targeted ventricular tachycardias in patients with scar related VTs. The rationale of the ablation design was to create a line of block extending across the diastolic pathway thus preventing sustained re-entry. These results are not far from our results in this category of patients. However, the results in ARVC and idiopathic group in our report are better than what was reported by this group where Catheter ablation was completely effective in only 1/3 of patients (33%) with arrhythmogenic right ventricular dysplasia and 1/3 (33%) of patients with idiopathic dilated cardiomyopathy in their report.

Arrhythmogenic right ventricular cardiomyopathy is associated with fibrous and fatty scar tissue in the right and often the left ventricles. When right ventricular involvement is extensive, the success of ablation is variable. Individual VTs can be ablated, but others may develop later, possibly related to progression of the disease process. Ablation is reserved as a palliative treatment for frequent episodes. Although the right
ventricle can be quite thinned, the risk of perforation during mapping does not seem to be substantially increased. In a recent report by Nair et al., long term outcome of using the Ensite non contact mapping in patients with ARVC was assessed. In 13 out of 15 patients, all the clinical and inducible VTs were ablated. In two patients, non-clinical inducible VTs could not be ablated. At 25 ± 16 months (2–52 months), all patients remained asymptomatic. Two patients had the recurrence of non-clinical VT on follow-up.12 Despite including much less number of ARVC patients in our study, however, the results reported by Nair et al. is generally similar to our results in our limited number of patients with ARVC.

It is uncertain, whether frequent non sustained VT/PVCs cause LV dilation and dysfunction even in patients with no evidence of structural heart disease and, if so, whether suppression of this non sustained dysrhythmias improves these changes. However, it has been previously reported that frequent PVC caused left ventricular (LV) dysfunction that can be reversed by suppression of PVC with anti-arrhythmic agents15,16 or radiofrequency catheter ablation (RFA).4,5 in patients with dilated cardiomyopathy. It has been reported that RFA produces clinical benefits to the patients with ventricular tachycardia17–19 and some case reports have shown that the dilated cardiomyopathy patients with PVC-depressed cardiac function and successful RFA of these arrhythmias improved LV function.70,21 In a study by Takemoto et al., A subgroup of patients with frequent (>20%) PVC demonstrated significantly enlarged LVDd reduced LVEF, increased mitral regurgitation, and deteriorated NYHA functional class as compared to the subgroup with rare (>20%) PVC. Furthermore, the ablation of these arrhythmias with acute success rate of 93% was associated with the normalization of these abnormalities without adverse effects and they suggested that Radiofrequency catheter ablation may be considered as the first choice of therapy for those patients.22 Our patients with dilated cardiomyopathy with no structural heart disease and with frequent PVCs/NSVT > 20% per day, had similar success rates of ablation and a significant improvement of functional state and LV function after a successful ablation. So, our findings also support the assumption that frequent PVCs/NSVT may be a possible cause of LV dysfunction and/ or heart failure and successful suppression of these dysrhythmias may improve LV dysfunction among the patients with idiopathic cardiomyopathy.

5. Conclusion

In our growing experience, non contact mapping guided RF ablation of unstable VT in patients with ventricular dysfunction showed good immediate results and long term outcome. This may be as effective in scar related VTs as in idiopathic cardiomyopathy types. Successful RF ablation of frequent repetitive non sustained ventricular dysrhythmias among patients with idiopathic Cardiomyopathy may improve LV dysfunction.

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References


