Express Publication

Mapping and Ablation of Polymorphic Ventricular Tachycardia After Myocardial Infarction

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OBJECTIVES The goal of this study was to describe the mapping and ablation of polymorphic ventricular tachycardia (VT) after myocardial infarction (MI).

BACKGROUND METHODS The initiating mechanisms of polymorphic VT after MI have not been reported.

METHODS The study comprised five consecutive patients (four males; age 61 ± 7 years) with frequent episodes of polymorphic VT after anterior MI (left ventricular ejection fraction 32 ± 7%) despite revascularization and antiarrhythmic drugs were studied. All patients demonstrated frequent ventricular premature beats (PBs) initiating polymorphic VT. Pace mapping and activation mapping were used to identify the earliest site of PB activity. The presence of a Purkinje potential preceding PB defined its origin from the Purkinje network. Electroanatomic voltage mapping was performed to delineate the extent of MI.

RESULTS The PBs were observed in all cases to arise from the Purkinje arborization in the MI border zone. These PBs were right bundle-branch block in all five patients, with morphologic variations in the limb leads in four; one also had a left bundle-branch block morphology. The coupling interval of the PB to the preceding QRS complex demonstrated significant variations (320 to 600 ms). During PB, the Purkinje potential at the same site preceded the QRS complex by 20 to 160 ms and was associated with different morphologies. Repetitive Purkinje activity was documented during polymorphic VT. Splitting of Purkinje activity and Purkinje to muscle conduction block were also observed. Ablation at these sites eliminated all PBs. At 16 ± 5 months follow-up using defibrillator memory interrogation, no patient has had recurrence of arrhythmia.

CONCLUSIONS The Purkinje arborization along the border-zone of scar has an important role in the mechanism of polymorphic VT in patients after MI. Ablation of the local Purkinje network allows suppression of polymorphic VT. (J Am Coll Cardiol 2004;44:1700–6) © 2004 by the American College of Cardiology Foundation

Monomorphic ventricular tachycardia (VT) in the presence of structural heart disease is largely attributed to anatomically bound macro-re-entry involving regions of myocardial scarring or the bundle branches. Improved understanding of these mechanisms has led to the ability to map and identify critical isthmuses that create the substrate necessary for these arrhythmias, thus allowing their ablation (1–5). In contrast, the mechanisms underlying the initiation and maintenance of polymorphic VT are poorly understood. Experimental studies have suggested the possibility that this arrhythmia may be maintained by migrating scroll waves, intramural re-entry, and Purkinje network re-entry (6–9).

Emerging evidence in patients with ventricular fibrillation (VF) in a variety of clinical scenarios implicates an important role for triggers originating from the distal Purkinje arborization in the initiation of this malignant arrhythmia (10–14). This study describes the mapping and ablation of polymorphic VT in relation to the three-dimensional ventricular anatomy after myocardial infarction (MI).

METHODS

Study population. The study comprised five consecutive patients (four males; age 61 ± 7 years [mean ± SD], range 50 to 67 years) with frequent episodes of polymorphic VT who underwent mapping and ablation. These patients were selected for ablation on the basis of repeated arrhythmia despite drug therapy (including beta-blockers and amiodarone), complete revascularization, and correction of any electrolyte abnormality. All patients were observed to have frequent premature beats (PBs) that occurred in isolation or initiated arrhythmia (Fig. 1) during hospitalization immediately after the arrhythmic storms. Medical therapy for arrhythmia in these patients included amiodarone, sotalol, beta-blockers, and mexiletine.

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Arrhythmias occurred in these patients after large anterior MI with residual left ventricular ejection fractions of 32 ± 7% (range 22% to 40%). In three patients, polymorphic VT occurred early after MI (four, four, and eight days, respectively). Two of these required >30 external defibrillations because of induction/degeneration into VF, such that ablation was performed during the initial hospitalization. In the third patient, although the arrhythmic episodes abated during the initial hospitalization, further recurrent episodes one month later necessitated ablation. The latter two patients presented with recurrent polymorphic VT 150 and 170 days after MI (Table 1).

**Electrophysiologic study.** Written, informed consent was obtained from all patients. In the two patients mapped during the week of MI, mapping was performed in the sedated ventilated state. The remaining patients were studied in the fasted state with sedation using midazolam and nalbuphine. An intravenous dose of 0.5 mg/kg of heparin was administered during mapping in the left ventricle. One or two multielectrode catheters were introduced percutaneously through the femoral vessels including an 8-mm tip quadripolar roving ablation catheter (Navi-Star, Biosense Webster Inc., California). The latter was introduced into the left ventricle by retrograde aortic catheterization.

Surface electrocardiograms and bipolar endocardial electrograms were continuously monitored and stored on a computer-based digital amplifier/recorder system with optical disk storage for off-line analysis (Bard Electrophysiology [Massachusetts] or EP MedSystems Work-Mate [New Jersey]). Intracardiac electrograms were filtered from 30 to 500 Hz, and measured with computer-assisted calipers at a sweep speed of 100 mm/s.

**Electroanatomic mapping.** In four patients, left ventricular electroanatomic maps were created during sinus rhythm (Fig. 2), whereas in one patient with frequent PBs, mapping of the PB was performed using the CARTO mapping system (Biosense-Webster) (Fig. 3). The system records the 12-lead electrocardiograms and bipolar electrograms filtered at 30 to 400 Hz from the mapping catheter and the reference electrogram. Endocardial contact during point acquisition was facilitated by fluoroscopy and the catheter icon on the CARTO system. Points were acquired if the stability criteria in space (≤6 mm) and local activation time (≤5 ms) were met. The border-zone of the MI was defined as previously described, as the region demonstrating bipolar voltage amplitudes of between 0.5 and 1.5 mV (4). In addition to voltage mapping, we tagged points on the map that demonstrated Purkinje potentials during sinus rhythm (Figs. 2 and 3).

**Mapping and ablation.** The origin of PBs in these patients was localized by: 1) pace mapping techniques to localize concordance with PB morphology; and 2) mapping of the earliest endocardial site activation relative to the QRS complex during PB. In one patient with frequent monomorphic PBs, an activation map of the PB was created to localize its origin. The following definitions were utilized as previously described to identify the origin of PB (10): 1) An

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**Figure 1.** Examples of frequent isolated premature beats followed by the initiation of polymorphic ventricular tachycardia.
initial sharp potential (<10 ms in duration) preceding the larger, slower ventricular electrogram during sinus beats, was considered to represent a Purkinje potential. Such a potential preceding the local electrogram at the site of earliest activation during PB indicated the Purkinje origin of the PB. 2) In the absence of such a potential at the site of earliest activation, the PB was considered to be of myocardial origin.

Ablation was performed using radiofrequency energy with a target temperature of 55°C to 70°C and a maximum power of 70 W, with a duration, to abolish PB and consolidating applications to minimize recurrence. In addition, in patients with inducible monomorphic VT (n = 3), activation and entrainment mapping of this VT was performed to identify the critical isthmus and further ablation performed at these sites. After ablation, patients were monitored for three to five days and then followed using defibrillator memory interrogation.

**RESULTS**

Polymorphic VT was observed in all patients during their hospitalization. The cycle length of arrhythmia varied from 600 to 200 ms (mean 332 ± 73 ms) and also demonstrated significant intra-individual variation between episodes of polymorphic VT.

**PBs and polymorphic VT.** All patients were observed to have frequent ventricular PBs in the immediate aftermath of recurrent episodes of polymorphic VT. Through in-hospital monitoring, these PBs were observed to trigger episodes of polymorphic VT (Fig. 1). These beats were right bundle-branch block morphology in all five patients, with morphologic variations in the limb leads in four patients. One patient additionally had a left bundle-branch block morphology PB. The coupling interval of the PBs to the preceding QRS complex demonstrated significant intra- and inter-individual variation (320 to 600 ms), with 40 to 160 ms variation within a given individual. The QRS duration of the PBs was similar to that observed during sinus rhythm; the difference from sinus rhythm being <40 ms in four patients and 60 to 170 ms in the last patient (Table 1). In four patients, a long-short sequence of activation without abnormal prolongation of the QT interval was observed to initiate polymorphic VT.

**Mapping and localization of PBs.** Pace mapping was used as a guide to identify the region of interest to perform more detailed activation mapping of PBs. The PBs observed during mapping were all localized to the left ventricle in the region of the border-zone of the MI as defined by electro-anatomic mapping, occurring within 1 cm of dense scar (Fig. 2). In these patients with post-anterior MI, we observed the following localization of PB morphology to the border-zone of the scar: inferior-axis PBs were located along the anteroseptal region; intermediate-axis PBs were localized to the anterior-lateral region; and superior-axis PBs were localized to the apico-septal region.

In all cases, at the earliest site of activation, PBs were preceded by Purkinje potentials indicating their origin from this structure (Figs. 3 and 4). During PB, the Purkinje potential preceded the QRS complex by 20 to 160 ms. At the same site, this varying conduction was associated with different PB morphologies previously documented on an electrocardiogram. Repetitive Purkinje activity, suggestive of successive beats being maintained by the Purkinje network, was observed to precede each QRS complex during the initiation of VT (Fig. 5). In addition, in two patients the Purkinje potential appeared to split during repetitive runs of PBs, and in a further two patients, conduction block between the Purkinje potential and the ventricular muscle was observed (Fig. 6).

**Radiofrequency ablation.** Radiofrequency energy was delivered for 19 ± 9 min (range 9 to 32 min) at sites demonstrating the earliest PB activation (mean fluoroscopy duration was 49 ± 25 min). In all cases, these were at sites where Purkinje potentials were observed to precede ventricular activation (Table 1). During applications, bursts of arrhythmia were observed before the eventual elimination of all PBs.

**Table 1. Baseline Characteristics and Results**

<table>
<thead>
<tr>
<th>Pt. No.</th>
<th>PVT After MI (days)</th>
<th>No. of VPB Morph.</th>
<th>VPB Morph.</th>
<th>Coupling Interval of VPB (ms)</th>
<th>PP to QRS Duration (ms)</th>
<th>Ablation Site (PP)</th>
<th>RF Time (min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>8</td>
<td>3</td>
<td>RBBB sup, inf and int axis</td>
<td>360–500</td>
<td>20–30</td>
<td>190–220</td>
<td>Left free-wall and septum</td>
</tr>
<tr>
<td>2</td>
<td>170</td>
<td>1</td>
<td>RBBB sup axis</td>
<td>400–440</td>
<td>20</td>
<td>Ant-Sept and low posterior walls</td>
<td>23</td>
</tr>
<tr>
<td>3</td>
<td>4</td>
<td>4</td>
<td>RBBB sup, inf and int axis, LBBB</td>
<td>400–500</td>
<td>20–40</td>
<td>160–190</td>
<td>Septum</td>
</tr>
<tr>
<td>4</td>
<td>150</td>
<td>3</td>
<td>RBBB sup, inf and int axis</td>
<td>500–600</td>
<td>20</td>
<td>60–80, 116</td>
<td>Ant-Sept</td>
</tr>
<tr>
<td>5</td>
<td>4</td>
<td>2</td>
<td>RBBB sup and inf axis</td>
<td>320–480</td>
<td>30–40</td>
<td>40</td>
<td>Ant-Sept</td>
</tr>
</tbody>
</table>

Ant-Sept = anteroseptal; inf = inferior; int = intermediate; LBBB = left bundle-branch block; MI = myocardial infarction; Morph = morphology; PP = Purkinje potential; Pt. = patient; PVT = polymorphic ventricular tachycardia; RBBB = right bundle-branch block; RF = radiofrequency application; sup = superior; VPB = ventricular premature beat.
Figure 2. Electroanatomic bipolar voltage map of Patient #4 performed during sinus rhythm. The voltage map delineates the region of scar border-zone. Note that the premature beats originating from the Purkinje network are in this border-zone, where successful ablation was performed (brown tags). Purple tags represent sites recording Purkinje potentials in sinus rhythm, and white tags represent fractionated complex electrograms.

Figure 3. Electroanatomic bipolar voltage and activation map in Patient #3 performed during premature beats. The activation map demonstrates the centrifugal activation from the border-zone of the scar. The voltage map (right panel) delineates the region of scar border-zone. Note that the premature beats originating from the Purkinje network are in this border-zone, where successful ablation was performed (brown tags). Purple tags represent sites recording Purkinje potentials, and white tags represent fractionated complex electrograms.
The two patients in whom the procedure was performed as a life-saving maneuver during the first week after MI had no arrhythmia requiring defibrillation after the procedure. However, they experienced some short, self-terminating runs of PBs in the few days after the procedure. The other three patients had occasional isolated PBs during in-hospital monitoring after the ablation procedure. All patients had defibrillators implanted; therefore, the follow-up was based on device memory of arrhythmia occurrence. None of these patients have had any defibrillator therapies after ablation during a follow-up of 16 ± 5 months (range 10 to 24 months). All patients have been continued on beta-blockers, angiotensin-converting enzyme inhibitors, and amiodarone or sotalol.

**DISCUSSION**

This study presents new information on the mechanisms of polymorphic VT after MI. Polymorphic VT in these patients was triggered and possibly maintained by activity originating from the distal Purkinje arborization localized to the border-zone of the MI and could be successfully abolished by radiofrequency ablation.

The damaged border-zone tissue of the scar resulting from MI has been demonstrated to play a crucial role in forming the substrate that sustains macro-re-entry and monomorphic VT (1–5). In such VT, studies based on activation and entrainment mapping allowed determination of the critical isthmus of slowed conduction that maintained VT and thus facilitated ablation (1,2). These isthmuses of surviving tissue within the border-zone of the scar can also be localized by electroanatomic mapping, with transection of these regions by ablation resulting in termination and elimination of arrhythmia (4,5). In contrast, the mechanisms underlying polymorphic VT have remained unknown.

El Sherif et al. (15) have previously implicated the role of the Purkinje network in an experimental model of anthropleurine-induced torsade de pointes. Berenfeld and Jalife (7), using a three-dimensional model of polymorphic VT, have suggested an important role for Purkinje to muscle interactions in the initial stages of polymorphic VT. In addition, they observed that when the Purkinje network was removed from this model, polymorphic VT could no longer become sustained, suggesting its potential role in the substrate maintaining arrhythmia. However, Janse et al. (16) have reported contradictory data suggesting the persistence of polymorphic VT in the absence of the Purkinje network. It has been suggested that a core of non-stationary vortex-like re-entrant activity may result in the constantly changing QRS morphology observed in polymorphic VT (9). As such, it seems unlikely that the ablation performed in the current cohort would be of sufficient extent to prevent such a migrating cascade of activation.

Interestingly, the Purkinje tissue cells have been demonstrated to be able to survive transmural infarction in experimental models, leading to speculation that their proximity to the endocardium allows exposure to cavitary blood (17). These surviving Purkinje fibers crossing the border-zone of the MI demonstrate heightened automaticity, triggered activity, and supernormal excitability, which, when coupled with prolongation of the action potential duration in this region, may result in the necessary milieu for polymorphic VT (6,7,17–19).

Emerging evidence in patients with VF in several clinical conditions has identified that the Purkinje arborization is a dominant source of triggers initiating VF (10–14). These studies have shown that ablation of these triggers was able to eliminate further arrhythmia. The observations in the current series of patients with polymorphic VT are remarkably similar, with all PBs in these patients originating from the Purkinje network located in the border-zone of the MI, regardless of the duration after the initial MI. In addition, we observed Purkinje potentials preceding runs of non-sustained polymorphic VT, repetitive activation of the Purkinje system during
polymorphic VT, and persistent Purkinje activity despite the absence of propagation to the ventricular myocardium. While these observations could support the notion of either automaticity or re-entry, they implicate the Purkinje arborization in the scar border-zone in the initiation and possibly in the maintenance of polymorphic VT in patients after MI.

**CONCLUSIONS**

The Purkinje arborization within the border-zone of scar has an important role in the mechanism of polymorphic VT in patients after MI. Ablation of this network allows suppression of clinical arrhythmia. Although they need to be confirmed in a larger cohort with longer follow-up, these
results have major implications as they provide new insights on the mechanisms of polymorphic ventricular arrhythmias.

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