Recurrent Polymorphic Ventricular Tachycardia
Treated by Ablation of Purkinje Arborization within an Infarct Border-Zone

A 70-year-old patient with 3-vessel coronary artery disease and a left ventricular aneurysm underwent coronary artery bypass grafting, together with a surgical anterior ventricular endocardial restoration (SAVER) procedure. Four days later, he suddenly developed recurrent sustained and nonsustained polymorphic ventricular tachycardia, preceded by monomorphic ventricular premature contractions, and did not respond to any antiarrhythmic drug, including lidocaine, esmolol, or amiodarone. Repeated electrical cardioversion procedures were performed (28 in total). Mapping was performed to target the earliest site of activation in the left ventricle during the ventricular premature contractions, a site where the premature beats were preceded by Purkinje potentials. That site was located along a scar border-zone. Ablation at that site resulted in the disappearance of the monomorphic ventricular premature contractions and in the complete suppression of the electrical storm. These findings appear to indicate that the area in which the Purkinje potentials were recorded along the scar border-zone played an important role in the mechanism of the polymorphic ventricular tachycardia after myocardial infarction. (Tex Heart Inst J 2011;38(3):291-4)

Ventricular premature contractions (VPCs), originating from the Purkinje system, have been shown to be responsible for the initiation of ventricular fibrillation (VF) in patients without structural heart disease.1-3 A similar mechanism has been shown to be associated with electrical storms after myocardial infarction4 and during the course of ischemic cardiomyopathy.5 The initial studies indicated that targeting Purkinje potentials during ablation of the infarct border-zone could successfully suppress the electrical storm.6,7 Although those results are preliminary, they have major implications because they provide new insight into the mechanisms underlying polymorphic ventricular tachyarrhythmias in patients with ischemic cardiomyopathy.

We report the case of a patient with unstable angina pectoris who had experienced a myocardial infarction and consequent left ventricular (LV) aneurysm 12 years before this hospital admission. The patient underwent coronary artery bypass grafting (CABG) combined with the surgical anterior ventricular endocardial restoration (SAVER) procedure.9,10 After the surgery, recurrent polymorphic ventricular tachycardia (VT) and ventricular fibrillation (VF) developed. Successful radiofrequency (RF) ablation was performed at the myocardial infarction border-zone, with the mapping guided by the Purkinje potentials.

Case Report

A 70-year-old man presented with chest pain in January 2006. Twelve years before his admission, he had experienced an anterior-wall myocardial infarction complicated by an LV aneurysm and severe LV dysfunction. Serial measurements of cardiac enzymes at 6-hour intervals were within the normal range. Coronary angiography revealed total occlusions of the middle left anterior descending and middle left circumflex coronary arteries, together with diffuse 60% to 80% narrowing of the right coronary artery, results that were similar to the coronary angiographic findings of 12 years earlier. The echocardiographic finding revealed akinesia of the LV apex, mid-septum, and mid-inferior wall, with severe LV dysfunction (ejection fraction, 0.20) and an aneurysmal change in the LV apex. Due to sustained chest pain and dyspnea, the patient underwent emergency CABG of all 3 coronary arteries (left internal mammary ar-
tery graft for the diagonal branch, saphenous vein graft for the distal right coronary artery, and radial artery graft for the obtuse marginal branch) and endoventricular patch plasty using an ovoid Vascutek® Gelseal™ 34-mm straight graft (Terumo Cardiovascular Systems Corporation; Ann Arbor, Mich) for the LV aneurysm repair (the SAVER procedure). While in the intensive care unit on postoperative day 4, the patient suddenly developed polymorphic VT, despite the absence of any preoperative VT or syncope. Recurrent polymorphic nonsustained and sustained VT, triggered by the same monomorphic VPCs with a short coupling interval (240–280 ms), appeared incessantly and degenerated into the polymorphic sustained VT (Fig. 1). The VT could not be suppressed by the intravenous administration of lidocaine (2–3.5 mg/min), esmolol (50 µg/kg/min), or amiodarone (25–37.5 mg/hr). Frequent electrical cardioversions (3–6 times a day) were required when the sustained VT or VF appeared. The electrical storm was suppressed temporarily using a lidocaine infusion (3 mg/min), but gradual tapering of the lidocaine infusion resulted in the return of the electrical storm, which required frequent cardioversions. On postoperative day 14, there were no significant changes in the blood levels of the cardiac enzymes, in the electrocardiogram, or in the echocardiogram. During the electrical storm, the serum electrolytes (including Mg++, K+, and Ca++) were maintained at the upper level of normal by means of intermittent intravenous replacement.

The patient’s clinical course from admission is shown in Figure 2. On postoperative day 17, the patient underwent RF catheter ablation at the infarct border-zone with use of a CARTO™ RMT Electroanatomical Mapping System (Biosense Webster, a Johnson & Johnson company; Diamond Bar, Calif) to create LV electroanatomical maps. Pacemapping was also used to target the identical premature beats that preceded the polymorphic VT. In addition to the voltage mapping (Fig. 3), activation mapping was performed during the VPCs, and the earliest ventricular activation was recorded by

![Fig. 1](image1)

Panels A–D show monomorphic ventricular premature contractions (*) initiating ventricular triplets and nonsustained or sustained polymorphic ventricular tachycardia.

![Fig. 2](image2)

Clinical course of the patient.

CABG = coronary artery bypass grafting; EC = electrical cardioversion; IV = intravenous; LCx = left circumflex coronary artery; NSVT = nonsustained ventricular tachycardia; OP = operation day; PLAD = proximal left anterior descending coronary artery; RFCA = radiofrequency catheter ablation; SAVER = surgical anterior ventricular endocardial restoration; SVT = sustained ventricular tachycardia; VF = ventricular fibrillation.

<table>
<thead>
<tr>
<th>Esmolol 50 µg/kg/min</th>
<th>Lidocaine IV 3.0–1.5 mg/min</th>
<th>1.5–2.5 mg/min</th>
</tr>
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<tbody>
<tr>
<td>Amiodarone IV 25–37.5 mg/hr</td>
<td>P.O. 400 mg b.i.d.</td>
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| Na⁺ | 134 | 135 | 138 | 133 | 135 | 133 | 132 | 128 | 128 | 129 | 128 | 130 | 128 | 127 | 130 | 131 |
|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|
| K⁺  | 4.1 | 4.1 | 4.2 | 4.4 | 4.9 | 5.1 | 4.9 | 5.2 | 6.1 | 5.7 | 5.7 | 5.7 | 5.2 | 5.2 | 5.8 | 5.5 | 4.5 | 4.8 |
the ablation catheter (Fig. 4). Seven applications of RF ablation were delivered at this site, but the polymorphic VT continued. While repositioning the ablation catheter near the previous ablation site, we recognized and subsequently recorded low-amplitude, high-frequency Purkinje potentials preceding the V wave, as denser mapping took place. This target point located at the infarct border-zone was compatible with the activation mapping. The infarct border-zone was defined as an area where the bipolar voltage amplitudes were between 0.5 and 1.5 mV. The Purkinje potentials always preceded the VPCs by approximately 30 to 40 ms (Fig. 5). Multiple RF energy applications were made with a target temperature of 55 to 70 °C and a maximum power of 50 W at the target area.

During the energy applications, intermittent bursts of ventricular premature beats occurred, but these were subsequently eliminated by the ablation. After 6 ablations, the episodes of VPC occurred rarely in comparison with those observed before ablation; and there were no episodes of VT, either spontaneous or induced by pacing, with or without isoproterenol. Thirteen RF applications, in total, were delivered near the infarct border zone.

On post-ablation day 3, rare VPCs were recorded, and there was 1 episode of nonsustained VT (duration, 5 sec), with no symptoms. After that, no evidence of VT or VF was observed, except for a rare episode of VPC. A follow-up echocardiogram revealed an improved LV ejection fraction (from 0.20 to 0.42). Seven days after the ablation treatment, the patient was discharged from the hospital with no antiarrhythmic medications. For the following 18 months, he was monitored in the outpatient clinic and showed no symptoms, despite the absence of antiarrhythmic prophylaxis. A recent LV ejection fraction was 0.45.

**Discussion**

It has been reported that 1% to 3% of patients undergoing open-heart surgery, including CABG, experience unexpected de novo sustained ventricular tachyarrhythm-
mias (sustained VT, VF, or both), particularly during the first 7 postoperative days.\(^\text{3,11,12}\) Recently, monomorphic VT in the presence of ischemic heart disease has been attributed mainly to anatomically bound macro-reentry that involves myocardial scar lesions or bundle branches.\(^\text{11,12}\) In contrast, the mechanisms underlying the initiation and maintenance of polymorphic VT are poorly understood.

The Purkinje system and VPCs are reported to be responsible for the initiation of VF in patients with and without structural heart disease.\(^\text{3}\) On the basis of those findings, we successfully treated an electrical storm using RF catheter ablation. Our patient experienced recurrent and medically refractory electrical storms, which were initiated chiefly by monomorphic VPCs, with short coupling intervals from the 4th postoperative day. We used a CARTO mapping system to define the infarct border-zone, and along this zone we identified low-amplitude, high-frequency Purkinje potentials preceding the VPCs. Radiofrequency energy was delivered at the sites where the Purkinje potentials preceded the earliest VPC activation in the infarct border-zone. In comparison with the time before ablation, there were, after ablation, only rare episodes of VPC and no evidence of VT. Those results indicate that the electrical storms might have been triggered by the monomorphic premature ventricular beats, which in turn appear to have been triggered by the Purkinje activity at the scar border-zone in patients with previous myocardial infarction. In this manner, the Purkinje system within the scar border-zone appears to play an important role in the initiation and maintenance of polymorphic VT.

The present report has several obvious limitations as evidence in support of a broad conclusion. First is that it reports but a single case. Although the size of the patch-plasty lesion (including the suture line) was smaller than the infarct size, the SÄVER surgery itself might have been a causative factor in the induction of VT storm. Consequently, more case studies are needed to say that Purkinje arborization within the infarct border-zone plays an important role in the mechanism of polymorphic VT after myocardial infarction. Second is that the follow-up period was short, and the arrhythmia might have subsided in the natural course of the electrical storm, rather than due to ablation. However, the frequent VPCs that preceded the polymorphic VTs were completely suppressed immediately after the RF catheter ablation, and no VF storms recurred thereafter. Although not conclusive, the results strongly suggest that the electrical storms were precipitated by the monomorphic VPCs and that the catheter ablation at that focus eliminated the trigger of the VT episodes. More conclusive findings await studies that involve more patients and longer follow-up.

References


