Focal Origin of Ventricular Fibrillation in a Patient with Ischemic Cardiomyopathy

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A 65-year-old patient with history of ischemic cardiomyopathy admitted to the hospital for chest pain and subsequently experienced incessant ventricular fibrillation (VF), requiring repeated defibrillation. Coronary angiogram was unchanged, compared to a study a year before, and acute ischemia was not considered to be the etiology of the VF. A particular premature ventricular contraction morphology was noted on telemetry prior to each episode of VF. The patient subsequently underwent successful radiofrequency ablation of a focus in the left ventricular free wall. Careful examination of initiating foci of VF or polymorphic ventricular tachycardia, with radiofrequency ablation in appropriate cases, could be potentially life-saving.

Key words: incessant ventricular fibrillation ■ radiofrequency ablation ■ ischemic cardiomyopathy

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INTRODUCTION

Three-hundred-fifty-thousand patients die each year of sudden cardiac death alone in the United States.[1] Ventricular fibrillation (VF) and sustained ventricular tachycardia (VT) are the major causes of sudden cardiac death.[1] Patients who survive an episode of sustained VT or VF have a recurrence rate of 30% in the next year, which increases to 50% in two years.[1] We report a patient with ischemic cardiomyopathy who had focal origin of incessant VF causing hemodynamic instability, which resolved after ablating the originating focus.

CASE REPORT

A 65-year-old male patient with a history of coronary artery disease (bypass grafting 10 years ago), diabetes mellitus, hyperlipidemia, and peripheral vascular disease experienced persistent substernal gripping chest pain and was evaluated in the local emergency room. He was found to be pale and diaphoretic, with runs of nonsustained VT. He was started on intravenous nitroglycerin, heparin, and eptifibatide, and transferred to our facility for suspected acute coronary syndrome. His cardiac enzymes showed a creatine kinase (CK) of 171 u/l (39–195 u/l), CK MB fraction of 3.2 u/l (0.0–5.0 u/l), and troponin T of 0.01 ng/ml (0.0–0.1 ng/ml). His serum creatinine was 2.1 mg/dl (0.5–1.4 mg/dl), sodium 135 meq/l (135–145 meq/l), potassium 4.1 meq/l (3.5–5.0 meq/l), magnesium 2.3 mg/dl (1.7–2.5 mg/dl), calcium 9.0 mg/dl (8.5–10.2 mg/dl), and albumin 3.3 g/dl (3.5–5.0 g/dl). Echocardiogram showed mildly dilated left atrium, dilated left ventricle with posterior hypokinesis, and an ejection fraction of 40%. Over the next day, the patient had numerous premature ventricular contractions (PVCs), followed by multiple runs of VF and polymorphic VT. He was started on intravenous infusion of membrane active antiarrhythmic agents, including lidocaine, procainamide, amiodarone, bretylium, and esmolol, administered as single-agent and in combination. During periods of frank cardiac arrest, he had to be revived with external electrical cardioversion along with intravenous epinephrine, atropine, and mag-

nesium sulfate as needed. He was intubated and transferred to the intensive care unit. He continued to have electrical storm requiring multiple defibrillations. At this point, the patient underwent catheterization, which revealed occluded left anterior descending artery (LAD), left circumflex artery, and right coronary arteries (RCA) at the origins. The grafts to LAD and obtuse marginal were patent, and there was diffuse disease beyond the graft insertion sites. The saphenous venous graft to RCA was occluded, and the distal RCA filled adequately from circumflex and LAD collaterals. This was unchanged compared to his previous catheterization one year ago. Acute ischemia was not felt to be the etiology for the patient's recurrent VF. He continued to have recurrent VF during the catheterization and was subsequently transferred to the electrophysiology lab.

Review of his telemetry monitor strips revealed identical-looking PVC before each episode of VT/VF (Figure 1). Electrophysiology procedure was performed using standard techniques, with placement of a catheter in the high right atrium, His bundle, and right ventricular apex. There were multiple spontaneous runs of VF during the procedure. A decision was made to ablate a suspected trigger focus indicated by the right-bundle, superior-axis ventricular PVC (Figure 2) using electromagnetic mapping with Biosense CARTO, mapping, and navigation catheter Navistar (Biosense, Diamond Bay, CA). The origin of this ventricular ectopy was mapped to the antero-lateral apical free wall of the left ventricle. Linear ablation performed in a

Figure 1. Telemetry strips showing initiation of ventricular fibrillation/ventricular tachycardia by premature ventricular contraction (PVC). The same morphology of PVC initiated ventricular fibrillation every time.

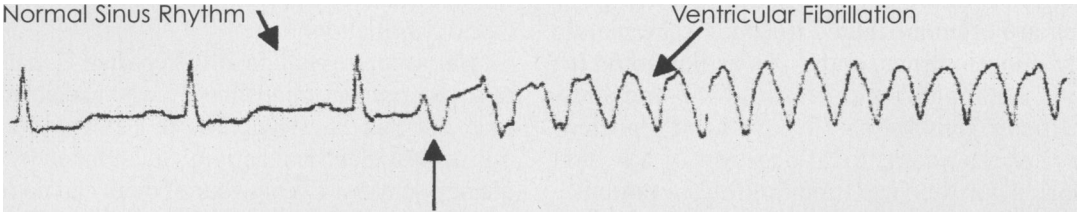
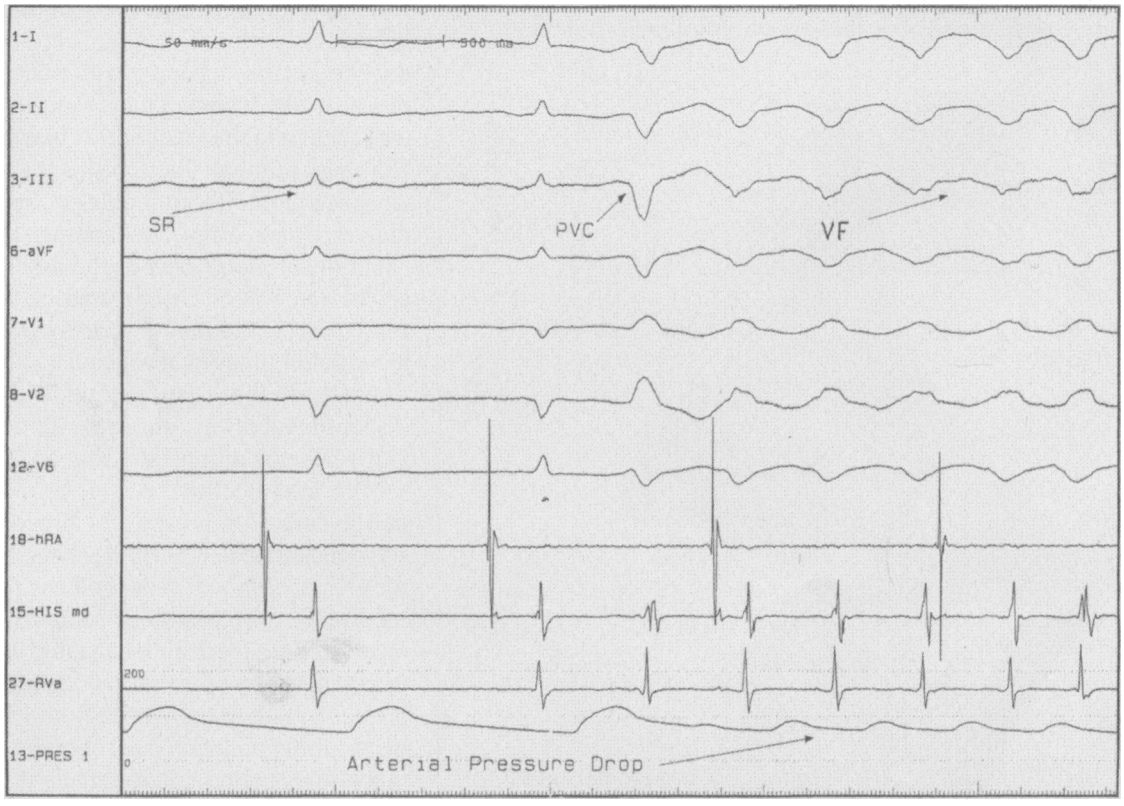


Figure 2. Tracing from the electrophysiology study showing initiation of ventricular fibrillation by characteristic premature ventricular contraction noticed in surface electrocardiographic leads (I, II, III, aVF, V1, V2, V6), intracardiac leads (hRA represents high right atrium, HIS represents His bundle, RVa represents right ventricle), and the arterial pressure line (PRES) showing the blood pressure drop following the arrhythmia.



cross-like fashion at this site was successful in eliminating the focus of origin (Figure 3).

The patient, prior to leaving the electrophysiology lab, was successfully weaned off all antiarrhythmic agents and did not have any recurrence of VT/VF for 24 hours. He was subsequently extubated the next day. Two days later, a dual-chamber pacemaker defibrillator was implanted, and the patient was discharged home. Implantable cardioverter-defibrillator (ICD) interrogation to-date showed only nonsustained VT. The patient has been off all membrane active antiarrhythmic agents since discharge and remains functional without any residual neurological injury for two years.

DISCUSSION

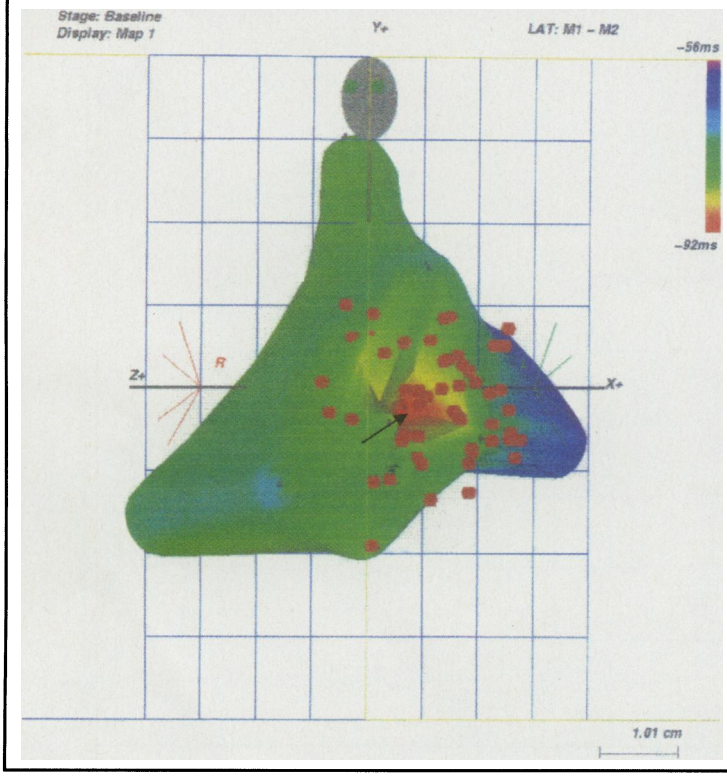
Our patient is unique in that he has ischemic cardiomyopathy with a focal origin for his VF. Polymorphic VT/VF and electrical storm occurring in this setting are often attributed to acute ischemia or electrolyte abnormalities, and this was not noted in our patient. Although focal origin of VT and successful catheter ablation of VT have been reported for more than 15 years, focal ablation of VF has been reported for the first time in 2002 in patients with idiopathic VF.[6] Our case preceded these reports and is the first one describing transcatheter ablation for VF in ischemic cardiomyopathy.

Transcatheter ablation has been tried with success in patients with VT in the absence of underlying structural heart disease and those with idiopathic right ventricular outflow tract VT or idiopathic left-sided VT and bundle-branch re-entrant VT.[2,3] Treatment of polymorphic VT/VF in ischemic cardiomyopathy usually requires revascularization or intravenous amiodarone. Our patient had unchanged coronary circulation on coronary angiogram and normal cardiac enzymes and electrolytes during his hospitalization. The use of multiple antiarrhythmic agents, including intravenous amiodarone, was not effective. The results we achieved lend credence to the fact that patients presenting with apparent disorganized ventricular arrhythmia—even in ischemic cardiomyopathy—may indeed have a focal or localized region of origin. A single scroll wave can give rise to extremely irregular electrocardiogram characteristic of fibrillation.[4,5]

Haissaguerre et al. in 2002 reported 27 patients with recurrent primary idiopathic VF who had ablation therapy in six centers. Triggers were localized by mapping the earliest electrical activity and ablated by radiofrequency delivery. Twenty-four of them had no recurrence of VF after the ablation.[6] Similar patients as described by Haissaguerre et al. and other authors[6-9] should have a close scrutiny of the initiating focus of their VF or polymorphic VT.

Catheter ablation for VF was described in patients with structurally normal hearts and in patients with nonischemic cardiomyopathy. We describe a patient with ischemic cardiomyopathy who had patent grafts with no evidence of acute ischemia at presentation. His chest pain was felt to be due to ventricular arrhythmia. Radiofrequency application delivered in a criss-cross manner to a one-square-centimeter area of the anterolateral apical free wall of the left ventricle was successful in terminating this arrhythmia. Following radiofrequency ablation and observation for an hour on the table, we then discontinued all his antiarrhythmic drugs. After 48 hours of freedom from polymorphic VT/VF and electrical storm, he received a defibrillator for subsequent prevention of sudden cardiac death. He has been followed for greater than two years and has not had a recurrence of sustained arrhythmia or received therapy from his defibrillator. The incessant nature of the presenting arrhythmia precluded him initially even from ICD treatment. Ability to attain control of the arrhythmia with catheter ablation made it possible for him to receive an ICD.

Figure 3. Electrophysiological mapping of the originating focus of arrhythmia. Approximately 1 cm^2 area of the left ventricle free wall (bright red spot in the center) with some of the tags of radiofrequency ablation (multiple small red dots). Ablation at the site eliminated the ventricular fibrillation.



Disorganized atrial and ventricular arrhythmias are now being recognized to generally have a focal mechanism of initiation.[10] Current ability to treat and cure patients with atrial fibrillation by directing catheter ablation to focal sites of origin in the pulmonary veins indicates and supports the concept that the mechanism of initiation of atrial fibrillation is different from the ability for fibrillatory conduction. In conclusion, focal initiation of polymorphic VT/VF has to be considered in certain instances, even in patients with ischemic cardiomyopathy. The ability to intervene as electrophysiologists in similar circumstances may help patients much like the one we treated.

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REFERENCES

1. Talwar K, Naik N, Juneja R. Are Drugs and Catheter Ablation Effective for Treating Ventricular Arrhythmias in Populations that Cannot Afford Implantable Cardioverter Defibrillators? *Curr Cardiol Rep.* 2001;3:459-466.

2. Vergara I, Wharton JM. Ventricular Tachycardia and Fibrillation in Normal Hearts. *Curr Opin Cardiol.* 1998;13:9-19.

3. Stevenson WG, Sweeney MO. Pharmacologic and Nonpharmacologic Treatment of Ventricular Arrythmias in Heart Failure. *Curr Opin Cardiol.* 1997;12:242-250.

4. Gray RA, Jalife J, Panfilov AV, et al. Mechanisms of Cardiac Fibrillation. *Science.* 1995;270:1222-1225.

5. Jalife J, Gray R. Drifting Vortices of Electrical Waves Underlie Ventricular Fibrillation in the Rabbit Heart. *Acta Physiol Scand.* 1996;157:123-131.

6. Haissaguerre M, Shoda M, Jais P, et al. Mapping and Ablation of Idiopathic Ventricular Fibrillation. *Circulation.* 2002;106:962-967.

7. Saliba W, Abul Karim A, Tchou P, et al. Ventricular Fibrillation: Ablation of a Trigger? *J Cardiovasc Electrophysiol.* 2002;13:1296-1299.

8. Takasuki S, Mitamura H, Ogawa S. Catheter Ablation of a Monofocal Premature Complex Triggering Idiopathic Ventricular Fibrillation. *Heart.* 2001;86:E3.

9. Kusano F, Yamamoto M, Emori T, et al. Successful Catheter Ablation in a Patient with Polymorphic Ventricular Tachycardia. *J Cardiovasc Electrophysiol.* 2000;11:682-685.

10. Reek S, Klein HU, Ideker RE. Can Catheter Ablation in Cardiac Arrest Survivors Prevent Ventricular Fibrillation Recurrence? *PACE.* 1997;20:1840-1859. ■

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