CASE REPORT

Successful catheter ablation of focal ventricular fibrillation originating from the right ventricle

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Abstract Sudden cardiac death from ventricular fibrillation (VF) typically occurs in patients with structural heart disease, but in 5 to 10 percent VF is "idiopathic," occurring in normal hearts. Recently, there has been the description and growing recognition of patients with VF that has a focal origin, the common sites being in the right ventricular outflow tract (RVOT) and sites in the left ventricle. A focus within the right ventricle outside the RVOT is rare. We present a case of a woman with VF storm that was localized to the inferobasal right ventricle and was successfully treated with radiofrequency ablation.

Keywords Idiopathic ventricular fibrillation · Radiofrequency ablation

1 Introduction

Sudden cardiac death from ventricular fibrillation (VF) typically occurs in patients with structural heart disease, but in 5 to 10 percent VF is "idiopathic," occurring in normal hearts [1]. The category of idiopathic VF includes "ion channelopathies," such as the congenital long QT syndromes, the Brugada syndrome, etc [2]. Over the past 6 to 8 years, there has also been the description and growing recognition of patients with VF that has a focal origin. The majority of these seem to originate from the right ventricular outflow tract (RVOT) [3–5]. Other foci include

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sites in the right ventricle (RV) outside the RVOT and sites in the left ventricle (LV). Bansch et al. described VF storms in four patients with recent myocardial infarction, where the arrhythmias were thought to originate from foci in the LV His-Purkinje system [6]. Despite the presence of several reports, focal VF remains a very rare arrhythmia. We present a case of a woman with VF storm that was localized to the inferobasal right ventricle (RV) and was successfully treated with radiofrequency ablation (RFA).

2 Case

A 40-year-old woman with no prior cardiac history suffered an outside-of-hospital VF arrest. She survived the event after a prolonged resuscitation. An echocardiogram and cardiac magnetic resonance imaging study showed normal left ventricular function and did not reveal any structural abnormalities in either ventricle. On hospital day two, more than 65 episodes of VF were noted despite treatment with amiodarone, lidocaine, beta blockers, calcium channel blockers, and procainamide. On hospital day five, she was transferred to our institution for further management and for possible ablation. Examination of the 12-lead electrocardiogram (ECG) revealed frequent unifocal premature ventricular complexes (PVC) with left-bundle branch morphology and left superior axis (Fig. 1). Ventricular preexcitation was electrocardiographically absent in the normally conducted beats and the QTc interval was normal, excluding Wolff-Parkinson-White syndrome and congenital long QT syndrome, respectively. The absence of epsilon waves, lack of T wave inversions in leads V1 through V3, and a structurally normal RV excluded arrhythmogenic right ventricular dysplasia. Observation of the ECGs and

Fig. 1 A 12-lead ECG showing sinus rhythm with frequent PVCs. The PVCs have a left-bundle branch block morphology with left superior axis
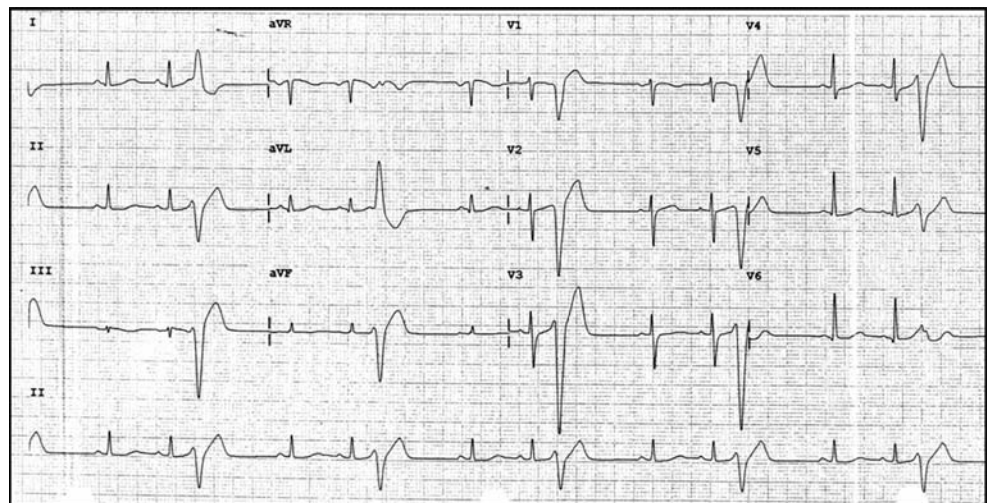
rhythms strips (Fig. 2) revealed that the initial QRS complex of each VF episode was identical in morphology to the unifocal PVCs. None of the ECGs or telemetry rhythm strips demonstrated Brugada pattern ST segment elevation making Brugada syndrome an unlikely etiology of this patient's VF episodes, and while a formal sodium channel blocker challenge was not performed, procainamide was administered as an antiarrhythmic drug without any ST changes. There were non-specific ST-T wave changes seen, especially in the post-extrasystolic beats (Fig. 2). No occlusive epicardial disease was seen with coronary angiography. Due to the refractory nature of her arrhythmias, a decision was made to proceed with catheter ablation. Electrophysiology study (EPS) revealed easily inducible polymorphic ventricular tachycardia (PMVT) and

VF in a highly reproducible manner with application of single and double extrastimuli. Frequent PVCs were not seen during EPS, limiting our ability to perform an activation sequence map. Pace mapping from the inferobasal wall of the RV revealed 11/12 lead ECG match. Additionally, catheter contact with the myocardium at this location reproducibly induced VF. Several applications of RFA (temperature cut off of 65°C and maximum power of 50 W with a 4 mm tip ablation catheter) were applied to the inferobasal myocardium of the RV (Fig. 3). On numerous occasions, RFA to this zone induced VF. A second zone (mid-inferior wall and 1 cm towards the septum from first zone) also showed moderate irritability with non-sustained PMVT and applications of RF current were also performed there. Although pharmacologic (isoproter-

Fig. 2 Rhythm strips demonstrating frequent ectopy with single PVCs, a couplet, and a triplet. The bottom strip shows unifocal PVCs followed by VF. The initial QRS complex of the VF is identical in morphology with the preceding PVC suggesting a focal zone/area of arrhythmicity
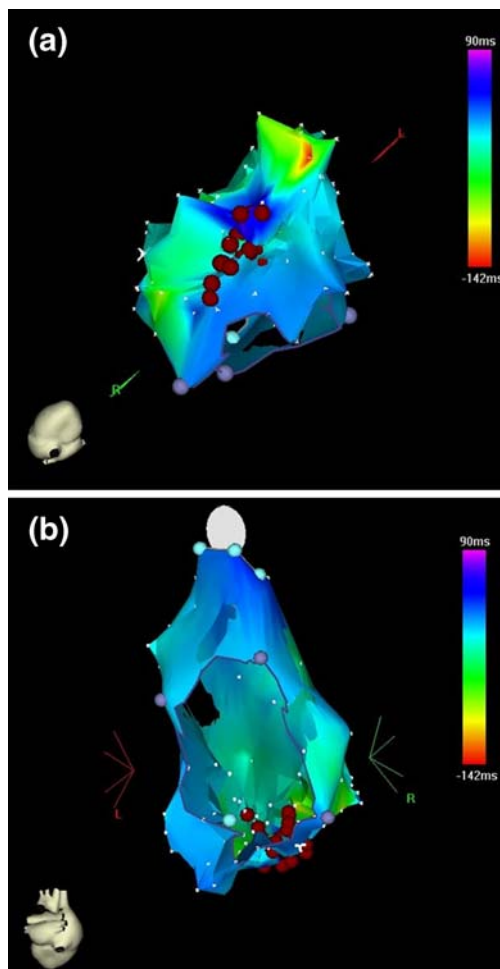
Fig. 3 An anatomic map of the RV. Panel A is an inferior view of the RV map. Panel B is a posterior view through the tricuspid annulus. The arrhythmogenic focus was isolated in the inferobasal myocardium of the RV. Pacing from this area produced QRS complexes that were very similar in morphology to the PVCs associated with VF. RFA was performed in this region

enol) and aggressive programmed stimulation still induced VF after RFA, spontaneous arrhythmias (PVCs or VF) were no longer seen. VF did not recur during the remainder of her hospitalization. Her hospital course was complicated by anoxic encephalopathy and ventilator dependence. Initial neurologic evaluation showed low likelihood of meaningful neurologic recovery. A wearable LifeVest defibrillator (ZOLL Lifecor Corp., Pittsburgh, PA) was given to the patient as a secondary preventative measure. It has been more than a year and she has since made a remarkable recovery without recurrence of VF. She has been offered the protection of an implantable defibrillator, but she has deferred the procedure due to personal reasons. In addition, a blood sample is being sent for genetic testing to investigate further if her condition is due to an ion channel defect.

3 Discussion

Idiopathic VF, especially those arising from a single focus, is an uncommon entity and RFA procedures to treat it are even more uncommon. This patient presented with VF storm requiring numerous defibrillating shocks despite aggressive antiarrhythmic drug therapy, including administration of simultaneous agents. The QTc interval was not prolonged excluding congenital long QT syndrome. Sacher et al. reported that only 25% of women with symptomatic Brugada syndrome have spontaneous type I pattern on 12-lead ECG [7]. The remaining 75% had ST elevation unmasked with provocative testing with flecainide or ajmaline. Our patient did not have spontaneous type I pattern nor did she have ST elevation with administration of procainamide, making the diagnosis of the Brugada syndrome unlikely. Recently, Haissaguerre et al. reported an increased prevalence of early repolarization in the inferolateral leads in patients with idiopathic ventricular fibrillation [8]. In their case subjects, early repolarization pattern was accentuated before the onset of arrhythmia. Early repolarization pattern was not seen by 12-lead ECGs or during EPS in our patient. Each VF episode was initiated with a QRS complex that was identical in morphology to previously seen unifocal PVCs, leading us to suspect focal VF. The QRS morphology of the "initiating beat" was suggestive of origin from the base of the RV. Mapping of this area revealed "highly irritable"/arrhythmogenic myocardium with VF occurring with even gentle catheter contact and application of RF energy, providing additional evidence for a focal source of her arrhythmia. After catheter ablation of the sites, catheter contact and application of RF energy no longer induced ventricular fibrillation. In addition, no ectopy or arrhythmias were seen during the remainder of her hospitalization. Saliba et al. previously reported a case of VF arising from the inferolateral border of the RV midway between the apex and base [9]. To the best of our knowledge, this is only the second report of idiopathic VF arising from the RV myocardium away from the RVOT.

Previous authors have reported successful RFA of idiopathic VF by mapping the ventricular ectopic beats that were seen to initiate the rhythm. Noda et al. reported 16 patients who had idiopathic VF arising from the RVOT, with all patients successfully treated with RFA without recurrence (mean follow-up of 54 months) [5]. Haissaguerre et al. reported 27 patients who had idiopathic VF, 23 of which had VF arising from the Purkinje conducting system. They reported an 89% success rate for RFA (mean follow-up of 24 months) [4]. These studies support the rationale of pursuing RFA in treating idiopathic VF that is refractory to medical therapy.

Accurate anatomic isolation of the arrhythmogenic focus is critically important in treating VF by RFA. Recordings

from multipolar catheters or a noncontact mapping catheter can generate an activation sequence map of the unifocal ectopics [10]. In our patient, the absence of ventricular extrasystoles during EPS prevented us from performing an activation sequence map. Alternatively, pace mapping can be utilized to localize the arrhythmogenic focus which was the technique used in this case. Analysis of the PVCs seen in prior ECG tracings was very helpful in localizing the arrhythmogenic site in our patient.

The mechanism of VF in acute myocardial ischemia is thought to be reentry, due to a combination of heterogeneity of repolarization and slow conduction. The mechanism of VF arising from the RV in the Brugada syndrome is thought be due to phase 2 reentry, originally described by Krishnan and Antzelevitch [11]. Haissaguerre et al. described successful catheter ablation in three patients with the Brugada syndrome [12]. In two of the three patients, catheter ablation was successfully performed in the RVOT. The observation that the Brugada syndrome is amenable to catheter ablation of a small region of the heart suggests that the arrhythmogenic focus is a microreentrant site in the right ventricle. We are unable to say as to what the underlying arrhythmogenic mechanism was in our patient (i.e. microreentry, abnormal automaticity, or triggered activity).

References

1. Consensus Statement of the Joint Steering Committees of the Unexplained Cardiac Arrest Registry of Europe and of the Idiopathic Ventricular Fibrillation Registry of the United States. (1997). Survivors of out-of-hospital cardiac arrest with apparently normal heart. Need for definition and standardized clinical evaluation. *Circulation*, *95*, 265–272.
2. Boussy, T., Paparella, G., de Asmundis, C., Sarkozy, A., Chierchia, G. B., Brugada, J., et al. (2008). Genetic basis of ventricular arrhythmias. *Cardiology Clinics*, *26*, 335–353.
3. Takatsuki, S., Mitamura, H., & Ogawa, S. (2001). Catheter ablation of a monofocal premature ventricular complex triggering idiopathic ventricular fibrillation. *Heart (British Cardiac Society)*, *86*, E3.
4. Haissaguerre, M., Shoda, M., Jais, P., Nogami, A., Shah, D. C., Kautzner, J., et al. (2002). Mapping and ablation of idiopathic ventricular fibrillation. *Circulation*, *106*, 962–967.
5. Noda, T., Shimizu, W., Taguchi, A., Aiba, T., Satomi, K., Suyama, K., et al. (2005). Malignant entity of idiopathic ventricular fibrillation and polymorphic ventricular tachycardia initiated by premature extrasystoles originating from the right ventricular outflow tract. *Journal of the American College of Cardiology*, *46*, 1288–1294.
6. Bansch, D., Oyang, F., Antz, M., Arentz, T., Weber, R., Val-Mejias, J. E., et al. (2003). Successful catheter ablation of electrical storm after myocardial infarction. *Circulation*, *108*, 3011–3016.
7. Sacher, F., Meregalli, P., Veltmann, C., Field, M. E., Solnon, A., Bru, P., et al. (2008). Are women with severely symptomatic Brugada syndrome different from men? *Journal of Cardiovascular Electrophysiology*, *19*, 1181–1185.
8. Haissaguerre, M., Derval, N., Sacher, F., Jesel, L., Deisenhofer, I., de Roy, L., et al. (2008). Sudden cardiac arrest associated with early repolarization. *The New England Journal of Medicine*, *358*, 2016–2023.
9. Saliba, W., Abul Karim, A., Tchou, P., & Natale, A. (2002). Ventricular fibrillation: ablation of a trigger? *Journal of Cardiovascular Electrophysiology*, *13*, 1296–1299.
10. Betts, T. R., Yue, A., Roberts, P. R., & Morgan, J. M. (2004). Radiofrequency ablation of idiopathic ventricular fibrillation guided by noncontact mapping. *Journal of Cardiovascular Electrophysiology*, *15*, 957–959.
11. Krishnan, S. C., & Antzelevitch, C. (1993). Flecainide-induced arrhythmia in canine ventricular epicardium. Phase 2 reentry? *Circulation*, *87*, 562–572.
12. Haissaguerre, M., Extramiana, F., Hocini, M., Cauchemez, B., Jais, P., Cabrera, J. A., et al. (2003). Mapping and ablation of ventricular fibrillation associated with long-QT and Brugada syndromes. *Circulation*, *108*, 925–928.