The Egyptian Heart Journal (2015) 67, 349–352

HOSTED BY

ELSEVIER

Egyptian Society of Cardiology

The Egyptian Heart Journal

www.elsevier.com/locate/ehj
www.sciencedirect.com

SHORT COMMUNICATION

Catheter ablation of idiopathic ventricular fibrillation using the CARTO 3 mapping system

Radu Rosu [a], Lucian Muresan [a,*], Gabriel Cismaru [a], Mihai Puiu [a], Marius Andronache [b], Gabriel Gusetu [a], Csongor Grosz [c], Lucia Bica [a], Olivia Sacaturean [a], Adina Rosu [a], Dana Pop [a], Dumitru Zdrenghea [a]

[a] *Rehabilitation Hospital, Cardiology Service, 400347 Cluj-Napoca, Cluj, Romania*
[b] *Institut Lorrain du Coeur et des Vaisseaux «Louis Mathieu», Cardiology Service, Electrophysiology Department, 54000 Nancy, France*
[c] *Clinics of the district of Neumarkt, Medical Clinic I, Nürnberger Str 12, 92318 Neumarkt, Germany*

Received 16 January 2015; accepted 6 March 2015
Available online 21 March 2015

KEYWORDS

Electrophysiology;
Ventricular fibrillation;
Catheter ablation

Abstract Ventricular fibrillation in the absence of structural heart disease represents an important mechanism of sudden cardiac death. It is initiated by triggers originating in the distal Purkinje fibers, arising from either the right or the left ventricle. Catheter ablation of these triggers has the potential of terminating the arrhythmia and preventing recurrence.

We present the case of an electrical storm in a 39-year old female patient with no cardiac past medical history, with recurrent episodes of idiopathic ventricular fibrillation, who was referred to our hospital for repeated episodes of syncope. The 12-lead ECG showed the presence of frequent ventricular premature beats (VPB), having a left-bundle branch block morphology and superior axis, with an "R on T" phenomenon, initiating non-sustained episodes of ventricular fibrillation. Using a three-dimensional, non-fluoroscopic mapping system (CARTO 3, Biosense Webster), the origin of the ventricular premature beat responsible for the initiation of VFib was identified and successfully ablated. Catheter ablation of idiopathic ventricular fibrillation using a 3-dimensional mapping system is a feasible therapeutic option for patients with this type of arrhythmia.

1. Introduction

Ventricular fibrillation in the absence of structural heart disease represents an important mechanism of sudden cardiac death. It is initiated by triggers originating in the distal Purkinje fibers, arising from either the right or the left ventricle. Catheter ablation of these triggers has the potential of terminating the arrhythmia and preventing future recurrences.[1]

* Corresponding author at: Rehabilitation Hospital, Cardiology Service, no. 46 – 50 Viilor Street, 400347 Cluj-Napoca, Cluj, Romania. Tel.: +40 (0)264 207 021x227; fax: +40 (0)2 64 45 33 53.
E-mail address: lmure_san@yahoo.com (L. Muresan).

Peer review under responsibility of Egyptian Society of Cardiology.

http://dx.doi.org/10.1016/j.ehj.2015.03.001

2. Material and methods

A 39-year old female patient with no cardiac past medical history was referred to our hospital for repeated episodes of syncope, which first occurred the day before admittance. The 12-lead ECG showed the presence of frequent ventricular premature beats (VPB), having a left-bundle branch block morphology and superior axis, with an "R on T" phenomenon, initiating non-sustained episodes of ventricular fibrillation (VFib) (Fig. 1) and intermittent right bundle branch block (Fig. 1, second panel – first and fourth beat).

Her family history was unremarkable for cases of sudden cardiac death. Her routine laboratory tests were normal, including serum electrolytes, troponins and inflammatory markers. The transthoracic echocardiogram showed no structural heart disease, with a preserved left ventricular ejection fraction and no kinetic disorder, a non-dilated right ventricle and the absence of pericardial fluid. Coronary angiography was urgently performed, showing no coronary lesions.

She subsequently developed sustained and incessant episodes of VFib, each requiring external defibrillation. Antiarrhythmic medication, including magnesium sulfate, metoprolol, lidocaine, amiodarone, isoproterenol and midazolam proved ineffective in controlling the arrhythmia, with only iv propafenone temporarily suppressing the arrhythmic events. After more than 200 external electric shocks, the patient was referred to the electrophysiology laboratory for catheter ablation of the VPB that initiated VFib.

A 3.5 mm electrode with an irrigated tip Navistar catheter was inserted inside the right ventricle via the right femoral vein and a fast anatomical map (FAM) was created using the CARTO 3 electro-anatomical mapping system. An activation mapping of the right ventricle was then performed, targeting the VPB initiating the episodes of VFib. After several failed attempts (Fig. 2, red spots), targeting local electrograms during the VPB preceding the QRS onset by up to 15 ms, a Purkinje potential was found in a zone situated in the middle third of the inferior wall of the right ventricle. The local electrogram recorded at this level showed the Purkinje potential preceding the VPB (Fig. 2), which was situated after the QRS complex in sinus rhythm. The local activation time was −67 ms, calculated from the Purkinje potential to the peak of the QRS complex in V3. (Fig. 2, second vertical panel, orange stripe), preceding the surface QRS onset by 26 ms.

The endpoint of the procedure was elimination of the local Purkinje potential and non-inducibility of the ventricular fibrillation (spontaneous and during programmed ventricular stimulation).

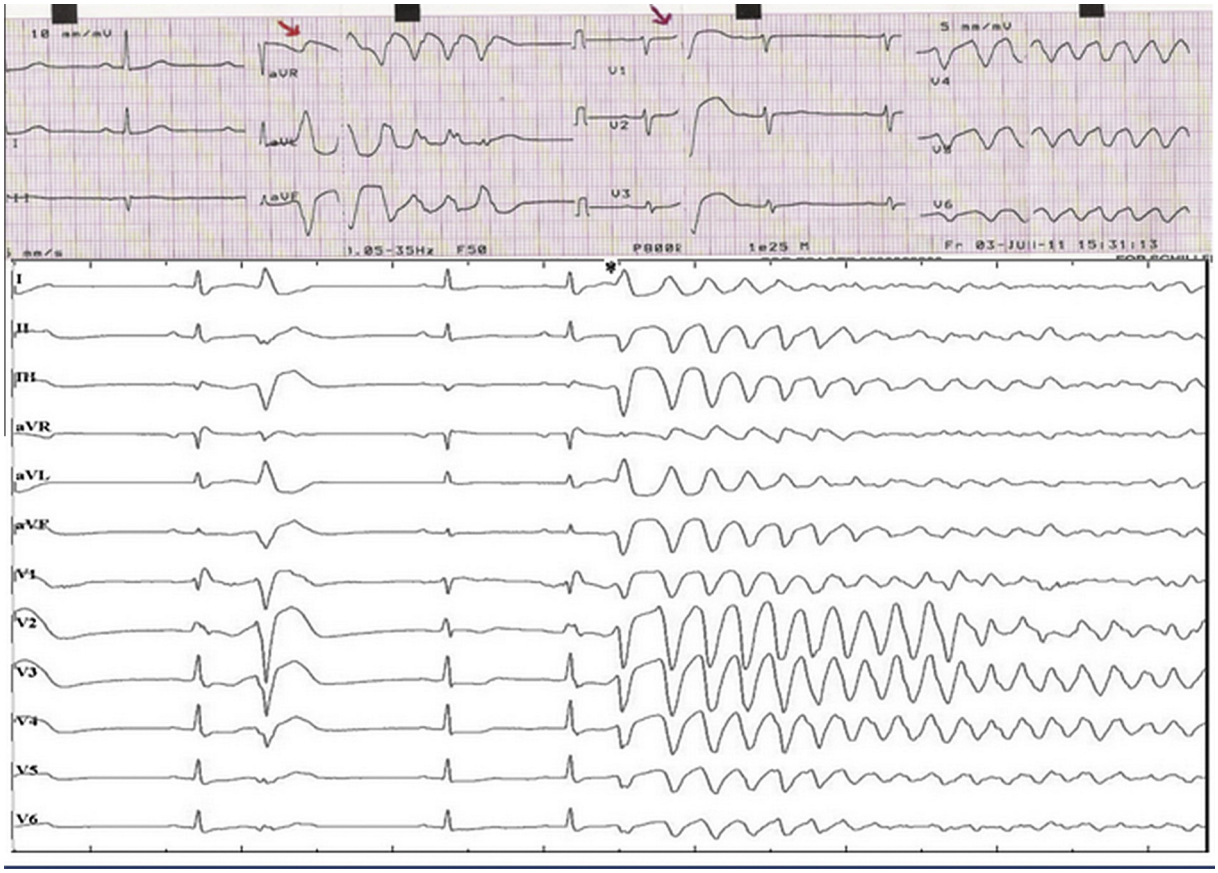

Figure 1 Top panel: twelve lead ECG (sequential recording of leads at 25 mm/s) showing a ventricular premature beat (red arrows) having a left-bundle branch block morphology and superior axis, with an "R on T" phenomenon, responsible for the initiation of a non-sustained episode of ventricular fibrillation (last part of tracing). Bottom panel: a ventricular premature beat (star) with the same morphology (see V1, V2, V3, aVL, aVF), initiating a sustained episode of ventricular fibrillation (recording speed at 25 mm/s).

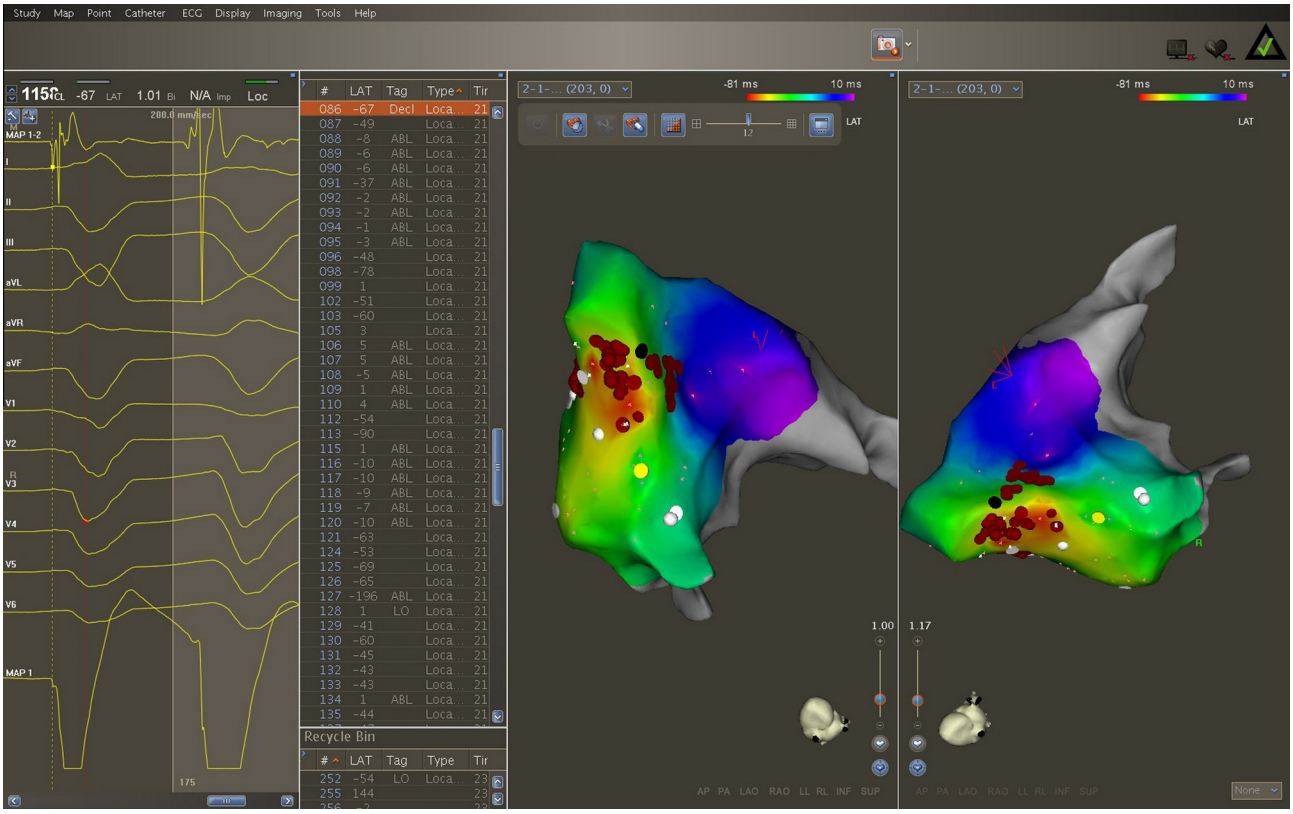

Figure 2 Activation map of the right ventricle using the CARTO 3 system. First panel: surface ECG leads and intracavitary leads, with the ablation catheter (Map 1–2) situated at the level of the Purkinje fiber responsible for the initiation of ventricular fibrillation. Note the local Purkinje potential preceding the VPB (Map 1–2). Third and fourth panel: activation map of the right ventricle performed during the frequent ventricular premature beat triggering ventricular fibrillation, showing the earliest local activation (red area) at the level of the middle third of the inferior wall. Red dots = radiofrequency applications.

3. Results

Radiofrequency (RF) applications at this site with a target temperature of 45 °C and a power of 30 W successfully suppressed the VPBs and no episode of VFib occurred over a waiting period of 30 min.

The patient had three more episodes of VFib during the next 12 h, this time being initiated by 2 other morphologies of VPBs: one with a LBBB morphology and superior axis, suggesting an origin on the inferior wall of the right ventricle and the other one with a RBBB and superior axis, suggesting an origin in the left ventricle (not shown). All three episodes were successfully converted to sinus rhythm by electric shocks. No other episode of VFib occurred and the patient was discharged from the hospital 5 days later, after an ICD was implanted. Three years after the procedure, the interrogation of the ICD showed no arrhythmia recurrence.

4. Discussion

Since the first publication on the mapping and ablation of idiopathic VFib by Haissaguerre et al.[1], several case reports and articles comprising short series of patients were published describing the role of a VPB acting as a trigger from the distal Purkinje system.[2] It is now known that the Purkinje fibers are responsible, in some cases, for the initiation of malignant arrhythmias. Predisposing factors such as ischemia, discharge of catecholamines, electrolyte imbalance, or certain drug exposure create the necessary conditions for reentry, increased automaticity or supernormal excitability. Catheter ablation has the potential of eliminating these triggers and thus controlling episodes of electrical storms.

This case describes the presence of an electrical storm in a patient with no structural heart disease, in whom no predisposing factor could be identified. The over 200 episodes of VFib required the administration of an unusually high number of electric shocks. Antiarrhythmic medication proved ineffective in suppressing the VPB initiating VFib, with the exception of iv propafenone, which only had a short-lasting effect. Radiofrequency ablation was the therapeutic option of choice, since the VFib episodes were mostly initiated by the same VPB and the elimination of this trigger resulted in control of the arrhythmia.

Haissaguerre et al.[1] described several morphologies of the VPB originating in the Purkinje system initiating idiopathic VFib. In our case, there is a remarkable resemblance between

the morphology of the VPB originating from the left ventricle and the one described by Saba in a case of idiopathic VFib in a 10-Year-Old Boy.[3]

5. Conclusion

The use of a 3-dimensional mapping system for guiding the ablation procedure has the advantage of providing clear anatomical and activation maps, which can lead a better understanding of the underlying process. This case presents a catheter ablation procedure of idiopathic ventricular fibrillation using the CARTO 3 mapping system, providing such images, hoping to contribute to a better understanding of the initiation mechanism of VFib by a VPB originating from the Purkinje fibers.

Conflict of interest

The authors declare that there are no conflict of interests.

Appendix A. Supplementary material

Supplementary data associated with this article can be found, in the online version, at http://dx.doi.org/10.1016/j.ehj.2015.03.001.

References

1. Haissaguerre M, Shoda M, Jais P, Nogami A, Shah DC, Kautzner J, et al. Mapping and ablation of idiopathic ventricular fibrillation. *Circulation* 2002;**106**:962–7.
2. Kohsaka S, Razavi M, Massumi A. Idiopathic ventricular fibrillation successfully terminated by radiofrequency ablation of the distal purkinje fibers. *PACE* 2007;**30**:701–4.
3. Saba MM, Salim M, Hood RE, Dickfeld TM, Shorofsky SR. Idiopathic ventricular fibrillation in a 10-year-old boy: technical aspects of radiofrequency ablation and utility of antiarrhythmic therapy. *Pacing Clin Electrophysiol* 2011;**34**:e85–9.