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REVIEW

Catheter Ablation of Recurrent Ventricular Fibrillation: A Literature Review and Case Examples

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Ventricular fibrillation (VF) electrical storm is a serious and life-threatening event, and is often triggered by premature ventricular complexes (PVCs). Catheter ablation of these PVC triggers have been described in a variety of clinical situations, including post-myocardial infarction (MI), patients with structurally normal heart, as well as in patients with Brugada Syndrome and Long QT Syndrome. We provide a literature review on this topic, using case examples for illustration.

Keywords Ventricular fibrillation • Ablation • Premature ventricular complexes • Electrical storm

Introduction

Ventricular fibrillation (VF) electrical storm is a rare but life-threatening event. It is often ''triggered'' by premature ventricular complexes (PVCs) falling in the ''vulnerable'' period, and has been reported to occur in a spectrum of clinical scenarios, including post-myocardial infarction patients [1,2], as well as in patients with a structurally normal heart [3,4] and in patients with Brugada Syndrome and Long QT Syndrome [5]. Although anti-arrhythmic therapies have been recommended for treatment of this disorder, the mortality from VF storm remains high. More recently, elimination of these PVC triggers using radiofrequency (RF) ablation has been reported to be successful in treating VF storm [2–6]. Here, we provide a literature review on this topic, using case examples involving patients with different underlying cardiac substrates successfully treated with catheter ablation for their recurrent VF for illustrative purposes (summarised in Table 1).

Case 1

A 40-year-old female presented with recurrent syncope. There was no family history of premature sudden death or arrhythmias. Although ambulatory electrocardiogram (ECG) monitoring showed infrequent PVCs (<20 over 24 hours), there were occasional episodes of non-sustained polymorphic ventricular tachycardia (VT). The initiating PVCs had a morphology suggestive of origin near the left posterior fascicle and they were closely coupled to the preceding QRS complex (coupling interval ~320 ms; Figure 1A). Structural heart disease was excluded by echocardiogram and magnetic resonance imaging (MRI). Coronary artery disease was also excluded with a Computer Tomography Coronary Angiography (CTCA). Flecainide testing, adrenaline testing and exercise stress testing using standard protocols yielded normal results. An initial electrophysiology (EP) study did not identify an alternative cause of syncope. The working diagnosis was idiopathic polymorphic ventricular tachycardia and an implantable cardioverter defibrillator (ICD) was inserted. She was also commenced on quinidine extended release (10 mg/kg/day).

She presented with an episode of VF three months later. A repeat EP study was performed. Spontaneous PVCs arising from near the left posterior fascicle matched the morphology of the clinical PVC and were observed to initiate and maintain polymorphic VT (Figure 1B and 1C). Ablation was

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Table 1 Summary of cases of recurrent ventricular fibrillation.

Case No.	Age at time of ablation (years)	Underlying heart disease	Site(s) of ablation
1	40	None	Left posterior fascicle
2	24	None	RVOT
3	64	Early post MI	Mid LV septum (at border zone of LV septal scar)
4	64	Remote post MI	Posteroseptal, anterobasal LV

LV = left ventricle; MI = myocardial infarction; RVOT = right ventricular outflow tract.

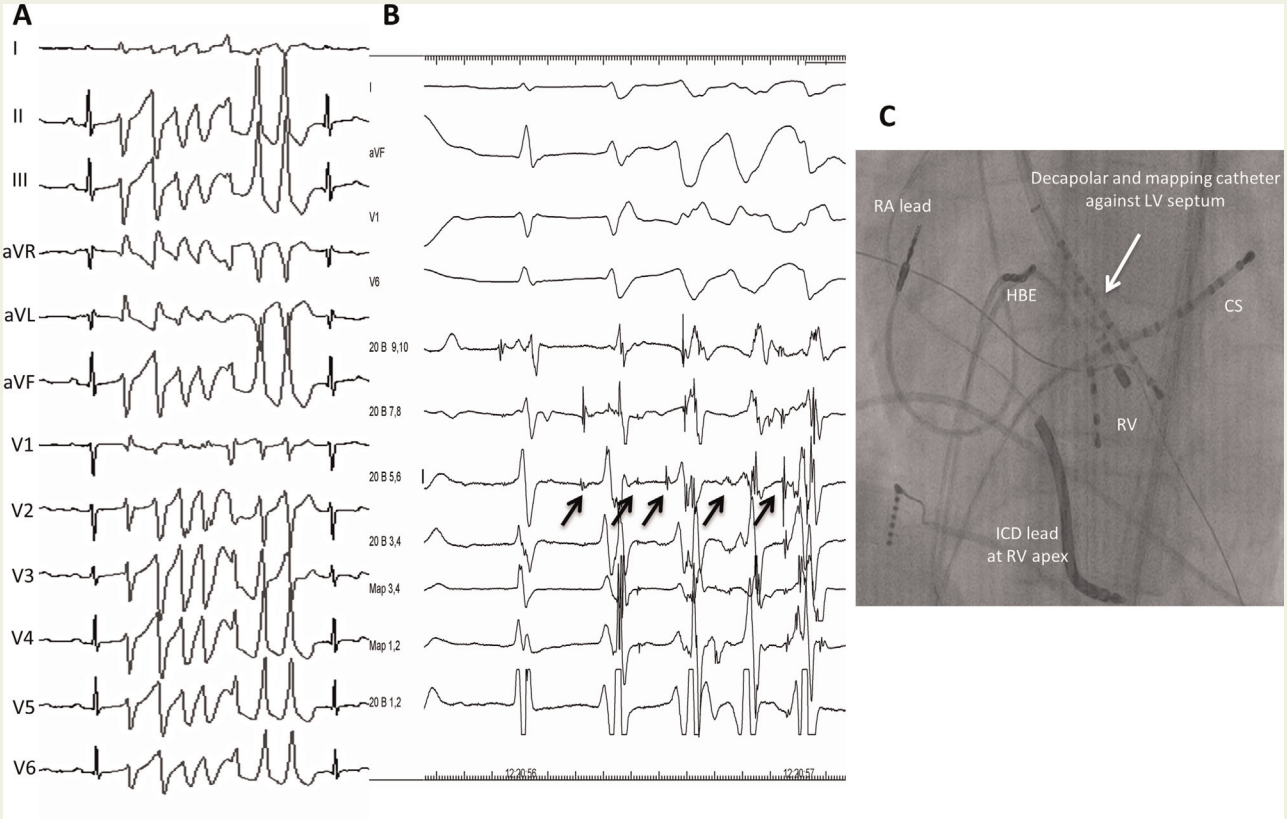

Figure 1 (A) Example of polymorphic VT on 12-lead ECG; (B) Polymorphic VT initiated by fascicular PVC in posterior septum of left ventricle (LV); Fascicular potentials preceding the initial PVC and during VT (arrows) (C) Left anterior oblique fluoroscopic view showing catheter positions (RA = right atrial, HBE = his bundle catheter, RV = right ventricular catheter, CS = coronary sinus).

performed in the posterior septum. The patient has remained arrhythmia-free without anti-arrhythmic drug therapy over a follow-up period of 17 months.

Case 2

A 24-year-old female presented following an out-of-hospital cardiac arrest. There was no history of syncope or family history of premature cardiac death. Structural heart disease was excluded by echocardiogram and MRI. Flecainide testing, adrenaline testing and exercise stress testing were normal. An ECG showed sinus rhythm with monomorphic PVCs and runs of non-sustained VT (Figure 2A). She had further monitored episodes of VF (Figure 2B). The QRS morphology of the PVCs suggested a right ventricular outflow tract (RVOT) origin.

She underwent an EP study. Activation mapping of the clinical PVC revealed earliest activation in the superior free wall of the RVOT (30 ms pre-QRS; Figure 2C). Purkinje