Successful epicardial ablation of electrical storms due to recurrent ventricular fibrillation triggered by premature ventricular contractions

Roland Richard Tilz, MD, Tina Lin, MD, Hisaki Makimoto, MD, PhD, Feifan Ouyang, MD

From the Asklepios Klinik, St. Georg, Hamburg, Germany.

Introduction

Ventricular fibrillation (VF) triggered by premature ventricular contractions (PVCs) is most frequently documented originating from the His-Purkinje system and right ventricular outflow tract (RVOT).[1,2] VF triggered by PVCs originating from elsewhere is rarely reported. We report a case of VF storm triggered by epicardial monomorphic PVCs, successfully treated with radiofrequency (RF) ablation.

Case report

A 29-year-old man was admitted to our institution for the management of a VF electrical storm. His index event occurred 11 months before this presentation, secondary to aborted sudden cardiac death requiring prolonged resuscitation. Investigations at this time included echocardiography and coronary angiography, which essentially demonstrated normal results. The result of an ajmaline test (sodium channel blocker) was negative for the assessment of channelopathies, and a myocardial biopsy did not reveal hypertrophic cardiomyopathy. Subsequently, a single-chamber implantable cardioverter-defibrillator (ICD) was implanted, and amiodarone and a β-blocker were prescribed as concomitant medical therapy. For a period of 5 months before presentation while on long-term therapeutic amiodarone therapy, the patient experienced daily nocturnal ventricular tachycardia (VT)/VF episodes documented on ICD interrogation, with 17 appropriate ICD shocks spanning this period of time and a treatment cluster of 7 episodes within the week before admission (Figure 1). Twelve-lead surface electrocardiogram (ECG) indicated a left ventricle (LV) basolateral focus (inferior axis, positive chest leads), with pseudo-delta waves suggesting an epicardial origin (Figure 2). Twenty-four-hour Holter monitoring revealed frequent nocturnal monomorphic PVCs (348) triggering each VF episode (Figure 3).

The first electrophysiological (EP) procedure was performed under sedation with midazolam, sufentanyl, and propofol. A decapolar catheter was placed in the coronary sinus and a quadrapolar catheter in the right ventricular (RV) apex via the right femoral vein. No spontaneous PVCs were seen throughout the procedure, and no PVCs, VT, or VF were inducible with programmed and burst electrical stimulation from both the RV and the LV, sedated and awake, and with and without isoproterenol infusion and intravenous β-blocker. The procedure was therefore rebooked, and all antiarrhythmic medications were ceased.

Three weeks later, the patient represented with VF storm to the emergency department and was treated with intravenous amiodarone. During a repeat EP study, again no spontaneous or induced PVCs/VT/VF was seen. Using a retrograde approach, an irrigated-tip ablation catheter was advanced into the LV via the right femoral artery and a long SL1 sheath (St Jude Medical, St. Paul, MN). Endocardial 3-dimensional (3D) electroanatomic mapping (CARTO, Biosense Webster, Diamond Bar, CA) was subsequently performed and showed no proof of substrate on bipolar voltage mapping. Using the Holter-documented clinical VT/VF, an endocardial pace map was then attempted; however, no matched pace map was reproducible. In addition, bipolar voltage mapping and pace mapping were performed in the coronary sinus, also with no evidence of substrate. Therefore, the ablation procedure was again rebooked.

While on the ward, VF triggered by monomorphic PVCs were recorded using radiotranslucent ECG patches. Using the same ECG patches, a further EP study with 3D electroanatomic mapping via combined endocardial/epicardial

KEYWORDS Ventricular arrhythmia; Epicardial ablation; Premature ventricular complex

ABBREVIATIONS **3D** = 3-dimensional; **ECG** = electrocardiogram/electrocardiographic; **EP** = electrophysiological; **ICD** = implantable cardioverter-defibrillator; **LV** = left ventricular/ventricle; **PF** = Purkinje fiber; **PVC** = premature ventricular contraction; **RF** = radiofrequency; **RV** = right ventricular/ventricle; **RVOT** = right ventricular outflow tract; **VF** = ventricular fibrillation; **VT** = ventricular tachycardia (Heart Rhythm 2014;11:146–149)

Dr Tilz received travel grants, research grants, and speakers' honoraria from Biosense Webster, and St Jude Medical, as well as research grants from Hansen Medical. Dr Lin received a fellowship grant from St Jude Medical.
Address reprint requests and correspondence: Dr Roland Tilz, Department of Cardiology, Asklepios Klinik, St. Georg, Lohmuehlenstr 5, 20099 Hamburg, Germany. E-mail address: tilz6@hotmail.com.

1547-5271/$-see front matter http://dx.doi.org/10.1016/j.hrthm.2013.10.013

Ereignis	Datum/Uhrzeit	Typ	Therapie	Dauer hh:mm:ss
V-19	09 Okt 2012 06:04	VF	41J	00:00:44
V-18	09 Okt 2012 04:40	VF	41J, 41J	00:00:58
V-17	09 Okt 2012 02:43	VF	41J	00:00:45
V-16	09 Okt 2012 01:56	VF	41J	00:00:44
V-15	07 Okt 2012 03:47	VF	41J, 41J, 41Jx1	00:01:13
V-14	07 Okt 2012 02:04	VF	41J	00:00:44
V-13	03 Okt 2012 06:13	VF	41J	00:00:44
V-12	24 Sep 2012 03:41	VF	41J	00:00:48
V-11	22 Sep 2012 04:21	VF	41J	00:00:44

Seite 1 von 2

Ereignis	Datum/Uhrzeit	Typ	Therapie	Dauer hh:mm:ss
V-10	29 Jul 2012 05:51	VF	41J	00:00:44
V-9	28 Jul 2012 23:22	VF	41J	00:00:45
V-8	28 Jul 2012 03:08	VF	41J	00:00:44
V-7	17 Jul 2012 08:11	VF	41J	00:00:44
V-6	21 Jun 2012 04:31	VF	41J	00:00:44
V-5	21 Jun 2012 03:53	VF	41J	00:00:45
V-4	21 Jun 2012 02:52	NonSustV	Nicht anhaltend	00:00:11
V-3	21 Jun 2012 02:24	NonSustV	Nicht anhaltend	00:00:05
V-2	21 Jun 2012 01:29	NonSustV	Nicht anhaltend	00:00:14
V-1	16 Nov 2011 11:53	VF	Induziert: 17J	00:00:37

Figure 1 Implantable cardioverter-defibrillator interrogation showing a treatment cluster during sleep within a period of 1 week.

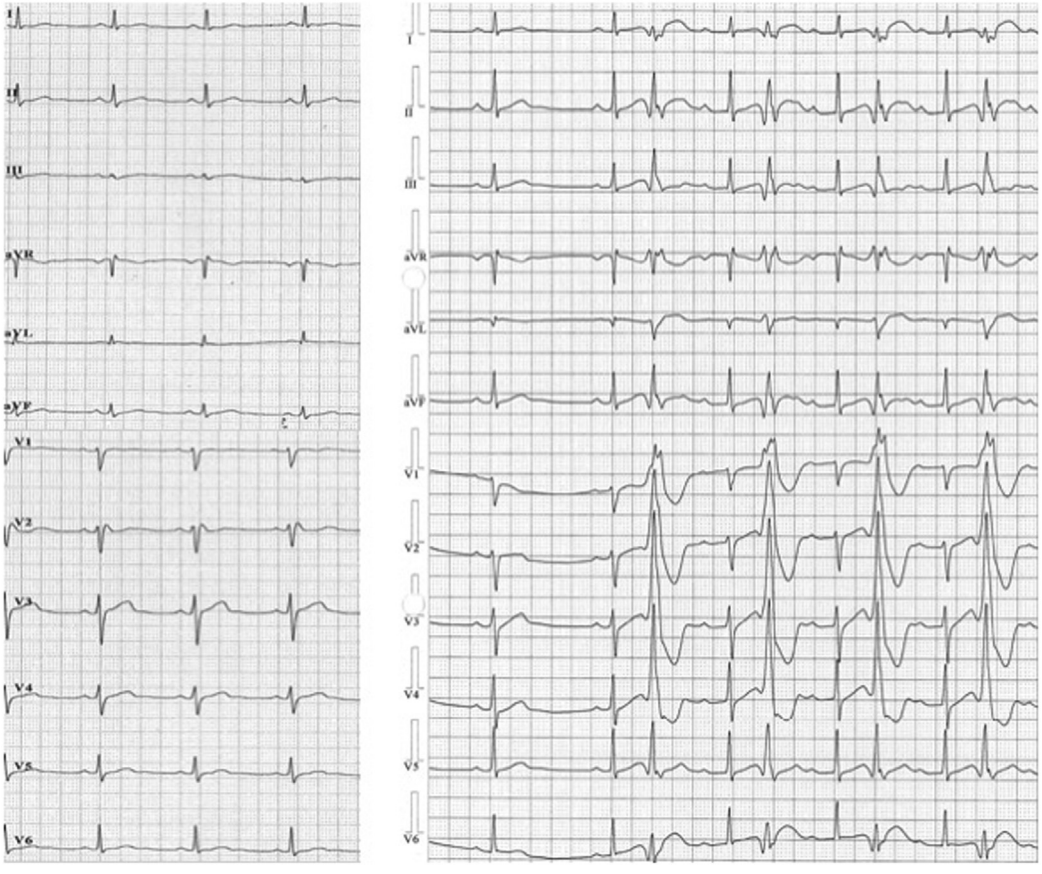

Figure 2 Twelve-lead electrocardiogram of sinus rhythm and with monomorphic ventricular ectopics.