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Review

Catheter ablation for ventricular tachyarrhythmia in patients with channelopathies

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ABSTRACT

Drug treatment and/or implantable cardioverter defibrillator (ICD) implantation are the most widely accepted first-line therapies for channelopathic patients who have recurrent syncope, sustained ventricular tachycardia (VT), or documented ventricular fibrillation (VF), or are survivors of cardiac arrest. In recent years, there have been significant advances in mapping techniques and ablation technology, coupled with better understanding of the mechanisms of ventricular tachyarrhythmia in channelopathies. Catheter ablation has provided important insights into the role of the Purkinje network and the right ventricular outflow tract in the initiation and perpetuation of VT/VF, and has evolved as a promising treatment modality for ventricular tachyarrhythmia even in channelopathies. When patients are exposed to a high risk of sudden cardiac death or deterioration of their quality of life due to episodes of tachycardia and frequent ICD discharges, catheter ablation may be an effective treatment option to reduce the risk of sudden cardiac death and decrease the frequency of cardiac events. In this review, we summarize the current understanding of catheter ablation for VT/VF in patients with channelopathies including Brugada syndrome, idiopathic VF, long QT syndrome, and catecholaminergic polymorphic VT.

Contents

1. Introduction

A channelopathy (also termed a primary electrical disorder) is defined as an inherited syndrome caused by mutations in genes encoding for ion channels, their subunits, or their associated proteins [1]. Drug therapy and implantable cardioverter defibrillator (ICD) implantation are generally used as first-line therapies for the treatment and prevention of sudden cardiac death (SCD) in channelopathy patients [1]. However, recent advances in mapping techniques and ablation technology allow us to perform ablation therapy more safely and effectively for the treatment of ventricular tachycardia (VT) and ventricular fibrillation (VF), even in channelopathies. A recent consensus report recommended that catheter ablation of VT or a triggering focus of VF should be considered as a class IIa indication in patients with VT/VF storm when adequate operator experience is available, although, presumably,

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this recommendation also includes VT/VF in patients with structural heart disease [1]. According to the European Heart Rhythm Association Survey, catheter ablation for tachyarrhythmia is currently undertaken in 5–10% of recurrent cases with channelopathies such as Brugada syndrome (BrS) and long QT syndrome (LQTS) [2]. However, the long-term effectiveness of catheter ablation for VT/VF remains to be precisely elucidated as no randomized data on the effect of catheter ablation on arrhythmic events has been collected. In this article, we summarize the clinical reports on catheter ablation for the treatment of VT/VF in channelopathic patients in Table 1 and describe the current understanding of this field.

2. Brugada syndrome

BrS is characterized by coved-type or saddleback-type ST-segment elevation in the right precordial leads of the standard electrocardiogram (ECG) or high intercostal ECG. It is associated with an increased risk of SCD due to VF [3]. Approximately 15–30% of BrS cases are attributed to mutations in *SCN5A*, and a further 10–20% of BrS cases are attributed to mutations in other genes [4,5]. ICD implantation is recommended in patients with a diagnosis of BrS who are survivors of a cardiac arrest and/or have documented VF or spontaneous sustained VT with or without syncope. Isoproterenol and quinidine are also useful for the treatment of electrical storm in BrS patients. Experimental studies have shown that heterogeneous loss of the action potential dome occurs at the right ventricular (RV) epicardial sites, resulting in a marked dispersion of repolarization which underlies the development of local re-excitation via a mechanism termed phase 2 reentry in BrS [6]. Phase 2 reentrant ventricular extrasystole can trigger polymorphic VT/VF. Therefore, the elimination of trigger ventricular premature contractions (VPCs) might suppress VT/VF.

Haïssaguerre et al. reported the electrophysiological properties and effects of catheter ablation in three symptomatic patients with BrS [7], with one patient exhibiting a familial *SCN5A* deletion mutation (2850delT). Monomorphic VPCs originating from the RV outflow tract (RVOT) were observed in all patients, with monomorphic VPCs with left bundle-branch block (LBBB) and superior axis in one patient. RVOT triggers were eliminated by radiofrequency (RF) energy applications at the earliest site (25 and 40 ms before QRS onset), and VF inducibility was modified after ablation in two patients. In the third patient, RF energy application could ablate the VPCs originating from the anterior RV Purkinje network, thus rendered the VF non-inducible. During a mean follow-up period of 7 ± 6 months, there was no evidence of recurrence of syncope, VF, or SCD in any of the patients.

Nademanee et al. reported nine symptomatic patients with BrS who experienced VF and underwent electrophysiological study and catheter ablation [8]. The patients exhibited abnormal epicardial electrograms characterized by fragmented electrograms with a relatively low voltage (< 1 mV), prolonged duration, and fractionated late potentials exclusively localized over the anterior aspect of the RVOT epicardium. Catheter ablation over these abnormal areas at the epicardial sites of the anterior aspect of the RVOT rendered the VT/VF non-inducible in seven of nine patients (78%) and normalized the Brugada ECG pattern in eight patients (89%). After a mean follow-up of 20 ± 6 months, eight of nine patients (89%) had no recurrence of VF episodes, and there were no shocks from the ICD. Amiodarone was resumed at 100 mg daily in only one patient with VF recurrence after ablation, and there were no VT/VF recurrences up to 33 months after the ablation. Thus, RVOT was suggested to be an important target for catheter ablation, as an originating site of trigger VPCs and as an arrhythmogenic substrate of VF in BrS [9–11].

3. Idiopathic ventricular fibrillation

Idiopathic ventricular fibrillation (IVF) is generally diagnosed by exclusion of apparent structural heart disease, identifiable genetic syndromes, and any other potential causes of VF [12]. Thus, IVF may not strictly be categorized as a channelopathy. The gold standard treatment for either primary or secondary prevention of SCD is the insertion of an ICD. Recent progress in understanding the mechanism of IVF strongly suggests that the Purkinje network [13–17] and the RVOT [15,18] play a pivotal role in both the initiation and perpetuation of VF.

PVCs originating from the RVOT occasionally trigger VF although these are generally considered to be benign. Noda et al reported on 16 patients who showed spontaneous VF and/or polymorphic VTs initiated by VPCs arising from the RVOT [17]. The optimal ablation site was determined by the earliest local activation site during the spontaneous target VPC and/or by pace mapping. Eventually, catheter ablation with a mean of 9 ± 4 RF energy applications was successful in 13 of 16 patients and partially successful in three patients. During programmed ventricular stimulation after ablation, nonsustained polymorphic VT was induced in two patients and VF in one patient who underwent ICD implantation. During a mean-follow-up period of 54 ± 39 months, there were no episodes of syncope, VF, or SCD (four patients received a β-blocker).

The short-coupled variant of torsades de pointes (TdP) is defined as a syndrome in which VF is exhibited secondary to a short-coupled VPC (with coupling interval < 300 ms) without obvious heart disease or QT prolongation [19]. The VPCs triggering VF may arise from the Purkinje network rather than RVOT or the working myocardium. Haïssaguerre et al summarized a cohort of 27 patients diagnosed as having IVF (without structural heart disease, QT prolongation, or a Brugada-like ECG) who underwent catheter ablation [15]. In this study, VPCs originated from the Purkinje networks in 23 patients (LV septum in 10, anterior RV in nine, both in four), and from the RVOT in four patients, and the former had a shorter coupling interval initiating VF or polymorphic VT than the latter (280 ± 26 vs 355 ± 30 ms). The interval from the Purkinje potential to the following myocardial activation varied from 10 to 150 ms during premature beat but was 11 ± 5 ms during sinus rhythm. After ablation for VPCs, 24 patients (89%) without drug therapy had no VF recurrence during a 24 ± 28 months follow-up period.

The long-term prognosis of patients with IVF after catheter ablation was reported in a multicenter study [20]. VPCs originated from the right ($n=16$), the left ($n=14$), or both ($n=3$) Purkinje systems, and from the myocardium ($n=5$) (including the RVOT [$n=4$]). After ablation, seven (18%) of the 38 patients (21 men, age 42 ± 13 years) experienced VF recurrence during a median postprocedural follow-up period of 63 months. Five of these seven patients underwent repeat ablation and had no subsequent recurrence of VF or documented VPCs for 28 months. The number of significant events (confirmed VF or aborted SCD) was reduced from 4 (interquartile range 3–9) before ablation to 0 (interquartile range 0–4) after ablation ($p < 0.01$). Taken together, short-coupled VPCs triggering VF originate predominantly from the Purkinje system and the RVOT, and catheter ablation for the triggers is feasible and is associated with long-term freedom from VF recurrence in patients with IVF.

Early repolarization (ER) is characterized by elevation of the QRS-ST junction (J point) and QRS notching or slurring (J wave) in multiple leads, especially the inferior and/or left precordial leads. Although this finding has been considered to be a benign ECG manifestation, Haïssaguerre et al reported that the ER pattern was found in 31% (64/206) of IVF patients compared to 5% (21/412) of matched control subjects [21]. An ER pattern in the inferior or inferolateral leads has been

Table 1
Summary of case reports of catheter ablation for ventricular tachyarrhythmias in patients with channelopathies.

Diagnosis	Authors	*n*	Age/Sex	Symptoms	Genes	VPC morphology/origin	Electrophysiology	Ablation	Outcome	Ref
BrS	Haïssaguerre et al.	3	39 ± 7, 2 males, 1 female	Syncope, PVT, VF	*SCN5A* mutation (2850delT) in 1	#1 RVOT in 2 pts #2 LBBB superior axis/anterior; RV Purkinje network in 1 pt.	#1 CI: 340 ± 20, 408 ± 15 ms #2 CI: 278 ± 29 ms	Successful ABL for VPCs	No rec (7 ± 6 mo)	#7
	Darmon et al.	1	18, male	Frequent VF, PVT	Not described	Posterior RVOT	-	Successful ABL for VPCs and AT	No rec (6 mo)	#9
	Nakagawa et al.	1	41, male	VF storm	Not described	LBBB inferior axis/RVOT	CI: 390–450 ms	Successful ABL for VPCs	No VF rec (29 mo)	#10
	Nademanee et al.	9	38, all males	All had out-of-hospital cardiac arrests.	Not performed	Not described	Low voltage (0.94 ± 0.79 mV), prolonged duration (132 ± 48 ms), and fractionated late potentials (96 ± 47 ms beyond QRS complex) in the anterior RVOT epicardium.	Successful ABL for substrate in the anterior of RVOT epicardium.	Only 1 of 9 pts had VF rec (20 ± 9 mo)	#8
	Sunsaneewitayakul et al.	10	36.5, all male	4 had frequent VF or storm; 6 had syncope or VF episodes.	Not performed	Posterolateral RV in 1 pt. No VPCs in 9 pts.	Late activation zone (where electrical activity was recorded by isopotential map within J point to J point + 60 ms) was observed in all patients.	Successful ABL for substrate at late activation zone of RVOT. 1 pt. had CRBBB during procedure.	Modified Brugada ECG in 3 of 4 pts (75%) and no VF storm in 4 (100%) (12–30 mo).	#11
IVF	Aizawa et al.	1	13, male	Convulsion, VF	Not described	#1 RBBB/posterolateral LV #2 LBBB superior axis/RV	Fractionated activities were recorded at posterolateral LV, J wave (+).	ABL for VPCs	VF rec 1.5 mo after 1st ABL, but no rec 3 mo after 2nd ABL.	#23
	Ashida et al.	1	18, female	Syncope, TdP	Not described	LBBB inferior axis/RVOT	QT/QTc 0.39/0.3 ms. CI: 380 ms, J wave (-)	Successful ABL for VPC	No TdP and syncope (3 yr)	#13
	Takatsuki et al.	1	62, male	Syncope, VF	Not described	RVOT	CI: 320–330 ms, J wave (-)	Successful ABL for VPC	no VF rec (20 mo)	#14
	Haïssaguerre et al.	27	41 ± 14, 13 males, 14 females	VF (23 during daily activity and 4 during sleep); LQT and Brugada ECG were excluded.	12 pts had no mutations in *SCN5A* and *KCNH2*	RBBB 10, LBBB 13, both 4. #1 Purkinje system in 23 (LV septum in 10, anterior RV in 9, both in 4) #2 RVOT in 4.	CI: 20–160 ms (75 ± 42) #1 The interval from the Purkinje potential to the following myocardial activation: 10–150 ms (38 ± 28 ms) during premature beats, 11 ± 5 ms during sinus rhythm.	Successful ABL for VPCs	24 pts (89%) had no VF rec without drug (24 ± 28 mo). 3 pts had late rec.	#15
	Betts et al.	1	32, male	Syncope, VF storm	Not described	LBBB/RVOT free wall	CI: 260–300 ms	Successful ABL for VPC	No VF rec (11 mo)	#16
	Nogami et al.	1	54, male	Syncope, VF	No mutation in SCN5A	#1 RBBB inferior axis/LV septum #2 RBBB northwest axis/LV	#1 CI: 280 ms #2 CI: 260 ms diastolic Purkinje potential and presystolic Purkinje potential were recorded from LV during PVT.	Successful ABL for VPC but isolated VPC was inducible.	No VF and syncope rec (4 yr) without drugs.	#18
	Noda et al.	16	39 ± 10, 7 males, 9 females	Syncope, VF, polymorphic VT (VF in 5 pts)	Not described	LBBB 16/RVOT septum 13, free wall 3	CI: 409 ± 62 ms, CL: 245 ± 28 ms, polymorphic changes of the QRS complex during rapid pacing in 2 pts.	Successful ABL for VPCs in 13, partially successful in 3.	No syncope, VF, SCDs (54 ± 39 months) (4 patients received a β-blocker).	#17
	Latcu DG et al	1	57, male	Aborted SCD, recurrent VF	Not described	RBBB/inferoseptal LV near posterior hemibranch (small number of VPCs)	J wave in inferior leads, no scar.	Successful ABL, J wave disappeared.	No VF rec and no occurrence of J wave (2 months).	#24
LQTS	Haïssaguerre et al	4	37 ± 8, 2 males, 2 females	Syncope, PVT, VF	No mutation in KCNQ1, KCNH2, and SCN5A	#1 LBBB inferior axis /RVOT in 1 pt. #2 RBBB superior axis/ Purkinje in LV in 1 pt. #3 Polymorphic, repetitive (bidirectional) with a positive morphology in V1/ Purkinje in LV in 2 pts.	#1, #2 CI: 503 ± 29 ms. #3 PVT cycle length: 280–420 ms lasting 3 to 45 beats, Purkinje potential proceeded VPCs/repetitive beats.	Successful ABL for VPCs	No VF rec (24 ± 20 mo), 1 pt had a rec of VPCs.	#7
	Srivathsan et al	1	39, female	VF	Not described	Purkinje in midposterior septal LV	CI: 340 ms Purkinje potential preceded VPCs by 30 ms.	Successful ABL for VPCs	No rec (6 mo)	#28

	Sanchez-Munoz et al	1	56, female	PVT, VF	No mutations in *KCNQ1, KCNH2, SCN5A, KCNE1, KCNE2,* and *KCNJ2*	LBBB inferior axis/posteroseptal RVOT	CI: 360 ± 30 ms	Successful ABL for VPC	No VF rec (14 mo) #29
	Cheng et al	1	19, male	Syncope, TdP	*SCN5A* variants (c.3578G > A, R1193Q)	Anterior lateral free wall of RV	-	Successful ABL for VPCs	No rec (29 mo) #30
	Yap et al	1	22, female	Out-of-hospital cardiac arrest	*KCNH2* mutation (c.2771G > A, p. Gly924Glu)	LBBB superior axis/inferoseptal RV	CI: 260 ms	Successful ABL for VPC	VF rec 5 mo after ABL #31
CPVT	Kaneshiro et al	1	38, female	Syncope, VF	*RyR2* mutation (c.1259G > A, p. R420Q)	#1: RBBB superior axis/inferoseptal LV. #2: RBBB inferior axis/LCC (=induced in the TET)	#1 Purkinje potential preceded VPCs by 18 ms. #2 discrete pre-potential preceded VPC #2 by 65 ms.	Successful ABL for VPCs	No VF rec (6 mo) with bisoprolol. #37

BrS, Brugada syndrome; IVF, idiopathic ventricular fibrillation; LQTS, long QT syndrome; CPVT, catecholaminergic polymorphic ventricular tachycardia; Pt, patient; VPCs, ventricular premature contractions; PVT, polymorphic ventricular tachycardia; TdP, torsade des pointes; RBBB, right bundle-branch block; LBBB, left bundle-branch block; RV, right ventricle; LV, left ventricle; RVOT, right ventricular outflow tract; LCC, left coronary cusp; TET, treadmill exercise test; CI, coupling interval; ABL, ablation; rec, recurrence; mo, months; yr, years; SCDs, sudden cardiac deaths.

reported to be associated with an increased risk for life-threatening arrhythmias, termed ER syndrome. ER has also been reported to coexist with other arrhythmogenic disorders such as BrS, short QT syndrome, and arrhythmogenic right ventricular cardiomyopathy; therefore, ER may be viewed as one of many arrhythmogenic factors that is rarely solely responsible for clinical events [22]. Aizawa et al. first reported a patient with ER-like QRS notching and VF without any apparent cause, which were suppressed by catheter ablation for the VPCs triggering the VF [23]. Recently, a case was reported of a patient with recurrent VF storm and inferoposterior ER in whom catheter ablation guided by pace mapping of the triggering VPCs successfully abolished the clinical event and caused the ER to disappear [24].

4. Long QT syndrome

LQTS is characterized by prolonged ventricular repolarization and susceptibility to syncope and SCD through VT (TdP), which can deteriorate into VF [25]. A clinical diagnosis is made from a combination of clinical history, family history, and 12-lead ECG, which typically reveals a heart rate-corrected QT interval (QT interval divided by the square root of the RR interval in seconds = QTc) of greater than 0.46 s in women and 0.45 s in men [25].

LQTS is most commonly inherited in an autosomal dominant fashion and has been associated with mutations in 15 genes. Among these, more than 75% of the mutations in congenital LQTS were of the *KCNQ1* (LQT1), *KCNH2* (LQT2), or *SCN5A* (LQT3) genes [26]. β-blocker therapy is associated with a 50% reduction in risk of cardiac events, and mexiletine is effective in LQT3 patients [27]. ICD implantation is recommended for patients with resuscitated cardiac arrest/VF or recurrent syncope who are on β-blockers.

Several studies have suggested that early afterdepolarizations arising from the Purkinje network and/or myocardial fibers are the primary triggering beats in TdP [7,28–31]. For example, Haïssaguerre et al. reported four symptomatic LQTS patients diagnosed on the basis of a corrected QT interval of > 460 ms who underwent catheter ablation [7]. In one patient, VPCs originating from the RVOT were ablated by RF energy applications. In three patients, VPCs originated from the left Purkinje system and were eliminated by ablation at multiple sites. During a mean follow-up period of 24 ± 20 months, there was no recurrence of syncope, VF, or SCD in any patient, although one patient with LQTS was maintained on a β-blocker, and another had a late recurrence of VPCs. The authors concluded that the triggers from the Purkinje network or the RVOT play a crucial role in initiating TdP or VF in LQTS, and these can be eliminated by focal ablation. Other groups also reported that VPCs originating from the RV free wall and RV inferoseptal wall could trigger VT/VF and could be eliminated by focal ablation [30,31].

5. Catecholaminergic polymorphic ventricular tachycardia

Catecholaminergic polymorphic VT (CPVT) is an inherited arrhythmogenic disorder characterized by polymorphic VT induced by physical or emotional stress with no detectable morphological abnormalities in the heart [32]. To date, five genes, cardiac ryanodine type 2 receptor (*RYR2*), calsequestrin 2 (*CASQ2*), *KCNJ2* (which encodes Kir2.1), calmodulin 1 (*CALM1*), and triadin (*TRDN*), have been identified as being involved in CPVT [33]. Typical arrhythmias include bidirectional VT and polymorphic VT that can degenerate into VF and thus SCD. β-blockers and additional administration of flecainide or verapamil have been reported to be effective for the prevention of VT/VF [34]. An ICD is considered the definitive therapy for the prevention of SCD, although failure of the ICD to prevent SCD has been reported in

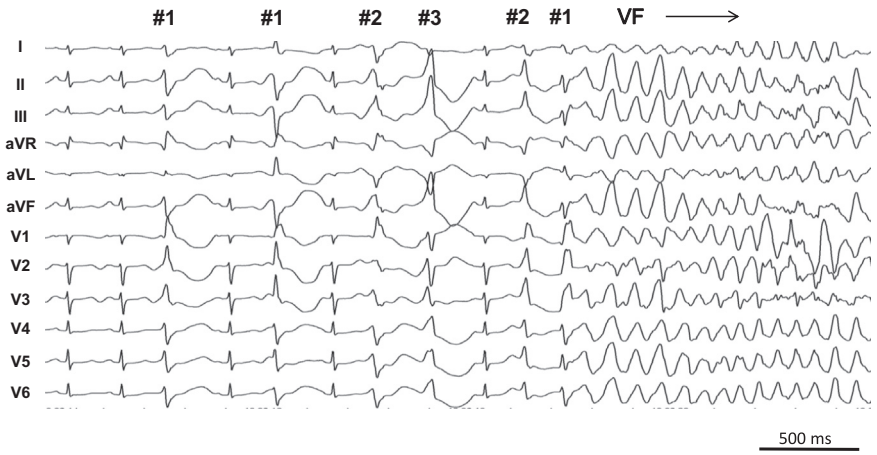

Fig. 1. Twelve-lead ECG recording during epinephrine stress test. Continuous intravenous infusion of epinephrine was started at a rate of 0.025 μg/kg per min, and the QT interval did not change. At a rate of 0.1 μg/kg per min, VPC #1 (right bundle branch block configuration and superior axis), VPC #2 (right bundle branch block configuration and inferior axis; the same as that induced in treadmill exercise testing), and VPC #3 (left bundle branch block configuration and inferior axis) were induced. Subsequently, VPC #1 following VPC #2 suddenly induced ventricular fibrillation, which was successfully terminated with electric shock. (Cited from [37]. With permission from Lippincott Williams & Wilkins.).

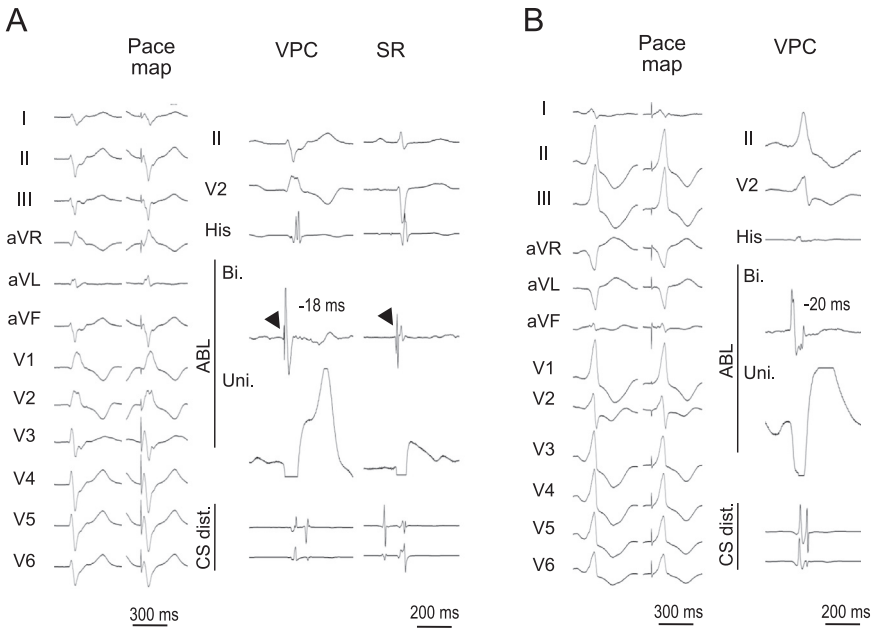

Fig. 2. Electrophysiological study and catheter ablation for catecholaminergic polymorphic ventricular tachycardia. (A) Activation mapping and pace mapping for VPC #1. A Purkinje potential was recorded from the left ventricular inferoseptum and preceded the QRS onset by 18 ms (arrowheads). The unipolar electrogram recorded from the distal electrode showed a QS pattern. Perfect pace mapping was obtained at this site. (B) Activation mapping and pace mapping for VPC #2. A local bipolar electrogram recorded from the LCC showed a discrete prepotential that preceded the QRS onset by 65 ms and was associated with a QS pattern in the unipolar electrogram. Perfect pace mapping was obtained at this site. ABL, ablation catheter; Bi., bipolar; CS, coronary sinus; His, His bundle; RVa, right ventricular apex; LCC, left coronary cusp; SR, sinus rhythm; Uni., unipolar; VPC, ventricular premature contraction. (Cited from Ref. [37]. With permission from Lippincott Williams & Wilkins.).

several cases because the ICD shock delivery might lead to catecholamine release, resulting in an electric storm [35].

Cerrone et al. reported that the mechanism of CPVT was due to delayed afterdepolarization-induced triggered activity in a focal Purkinje network in a knock-in (RyR2) mouse [36]. In addition, chemical ablation of the RV endocardial cavity with Lugol's solution, which selectively destroys the Purkinje network, could convert bidirectional VT into monomorphic VT in a CPVT model of anesthetized mice. Therefore, the Purkinje network is considered to be a critical contributor to arrhythmogenic triggers in CPVT and may be a promising therapeutic target for catheter ablation.

Our group presented the first case report of the successful catheter ablation of bidirectional VPCs triggering VF in patients with CPVT [37]. This case was of a 38-year-old woman who had often experienced syncope since childhood. The patient's daughter also had similar episodes of syncope and developed VF during treadmill exercise testing, which was successfully defibrillated with an electric shock. Witnessing this situation, the patient also lost consciousness, with documented VF which was converted to sinus rhythm by cardiopulmonary resuscitation. There were no significant abnormalities in her resting 12-lead ECG, echocardiography, or coronary angiography. Genetic analysis revealed that she and her daughter had same missense mutation (c.1259 G > A, p.R420Q) in *RyR2*, and both were diagnosed as having CPVT.

During the patient's epinephrine stress test (Fig. 1), multifocal VPCs (VPC #1, RBBB, and superior axis; VPC #2, RBBB, and inferior axis, the same VPC configuration as that induced during the treadmill exercise testing; and VPC #3, LBBB, and inferior axis) appeared, and VPC #1 following VPC #2 subsequently induced VF. We performed catheter ablation targeting the catecholamine-

induced VPCs (Fig. 2). VPC #1 was recorded at the left ventricular inferoseptal area near the posteromedial papillary muscle, where a presystolic Purkinje potential preceded VPC #1 by 18 ms. RF energy application to this site accelerated the VPCs, and additional applications around the target site subsequently eliminated VPC #1. After the procedure, VPC #2 continued to occur, and a local bipolar electrogram recorded on the left coronary cusp showed a discrete prepotential that preceded the onset of VPC #2 by 65 ms. RF energy application to the left coronary cusp abolished VPC #2. After ablation at both sites, neither the VPCs nor VF was inducible, even with an infusion of epinephrine at up to 1.2 g/kg per min. The patient underwent ICD implantation and was discharged from the hospital on bisoprolol. During a 16-month follow-up after ablation, no episodes of syncope or ICD therapy occurred. Thus, catheter ablation of the bidirectional VPCs triggering VF may become an adjunctive therapeutic option for CPVT.

In addition to ventricular tachyarrhythmia, patients with CPVT are likely to be complicated with supraventricular tachyarrhythmia such as atrial fibrillation and supraventricular tachycardia (SVT). Typically, the onset of supraventricular tachyarrhythmia is observed at lower heart rates than ventricular arrhythmia in patients with CPVT. Supraventricular tachyarrhythmia may be a precursor to VT and may be a cause of frequent and inappropriate shocks from an ICD associated with SVT with a rapid ventricular response. Therefore, catheter ablation targeting SVT may be effective in reducing the number of cardiac events in patients with CPVT.

6. Conclusions

VT/VF in patients with channelopathies can be suppressed by elimination of the triggered VPCs originating from the Purkinje network and/or around the RVOT, and/or by substrate modification of possible re-entry circuits. Further studies are required to evaluate the precise mechanisms and long-term prognosis of channelopathies.

Conflict of Interest

All authors declare no conflict of interest related to this study.

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