Endo/epicardial ablation of ventricular arrhythmias with contact force-sensing catheters in arrhythmogenic right ventricular dysplasia/cardiomyopathy

Dursun Aras, Fırat Özcan, Serkan Çay, Özcan Özeke, Meryem Kara, Serkan Topaloğlu

Department of Cardiology, Türkiye Yüksek İhtisas Training and Research Hospital; Ankara-*Turkey*

Abstract

Objective: To control ventricular arrhythmia in arrhythmogenic right ventricular dysplasia/cardiomyopathy (ARVD/C), ablation may be required both from the endocardial and epicardial side. In this study, we analyzed the results of contact force–sensing (CFS) catheters in the endo/epicardial ablation of ventricular arrhythmias in ARVD/C.

Methods: We included 17 patients with ARVD/C, 5 of whom had premature ventricular contractions (PVC), and the rest of them were admitted with a ventricular tachycardia (VT) storm, between September 2014 and October 2016. We divided patients into two groups: the PVC and VT groups. Irrigated CFS catheters (Smart Touch, Biosense Webster, Inc.) were utilized in all procedures.

Results: In the PVC group, the mean ratio of PVC during the 24-hour Holter monitoring was 31.8±7.6%. The mean contact force during mapping and ablation in the right ventricle was 13±1.2 and 12.8±1.9 grams, respectively. The mean follow-up duration was 15±3.1 months for the PVC group. The left ventricular ejection fraction improved in all patients (52.8±10%). All patients in the VT group underwent endo/epicardial ablation, except one. The mean contact force during the endocardium and epicardium mapping was 12.5±1.2 and 12.5±4.6 grams, respectively. The mean contact force during ablation for the endocardium and epicardium was 12.1±1.4 and 12.8±1.9 grams, respectively. All clinical and non-clinical VTs were ablated successfully, except in 2 patients who still had non-clinical VTs. The mean follow-up was 15.5±4.5 months. None of the VT patients experienced electrical storm or death. Two patients had single shock, and 1 patient had two shocks during the follow-up.

Conclusion: Endo/epicardial ablation of ventricular arrhythmias with CFS catheters in ARVD/C seems to be promising. *(Anatol J Cardiol 2019; 21: 187-95)*

Keywords: ventricular tachycardia, arrhythmogenic right ventricular dysplasia/cardiomyopathy, ablation

Introduction

Arrhythmogenic right ventricular dysplasia/cardiomyopathy (ARVD/C) is a genetic myocardial disease, characterized with progressive replacement of right ventricular myocytes with the fibrous and adipose tissue (1). The disease process progresses from epicardium to endocardium and leads to the formation of islets of myocytes among the fibrous and adipose tissue, forming a substrate for reentrant ventricular tachycardias (VT) (2). The ARVD/C can present with sudden cardiac death and recurrent ventricular arrhythmias that may be difficult to control with antiarrhythmic drugs (AADs). Endocardial ablation results are not satisfactory (3, 4). Adjuvant epicardial ablation to endocardial ablation provides a better ventricular arrhythmia control (5-7). Previous studies were carried out with ablation catheters without the contact force–sensing (CFS) feature.

In this study, we report our institutional experience on the ablation of ventricular arrhythmias in ARVD/C with a CFS ablation catheter.

Methods

Patients

The study population was identified from the Türkiye Yüksek İhtisas Training and Research Hospital registry. We included 5 patients with premature ventricular contractions (PVC) and 12 patients with electrical storm, who underwent ablation between September 2014 and October 2016. All patients fulfilled diagnos-

Address for correspondence: Dr. Fırat Özcan, Türkiye Yüksek İhtisas Eğitim ve Araştırma Hastanesi, Kardiyoloji Kliniği, 06590 Ankara-*Türkiye*
Phone: +90 532 785 40 00 E-mail: drfozcan@gmail.com

Accepted Date: 28.12.2018 **Available Online Date:** 05.03.2019

DOI:10.14744/AnatolJCardiol.2018.58534

Anatol J Cardiol 2019; 21: 187-95
DOI:10.14744/AnatolJCardiol.2018.58534

tic Task Force Criteria for ARVD/C. We divided patients into two groups: the PVC and VT groups. Our inclusion criteria for PVC ablation were decreased left ventricular ejection fraction (EF) or being symptomatic despite the medical treatment. The inclusion criterion for patients with VT was recurrent VT that could not be controlled with AAD. All patients gave their written informed consent, and the Ethics Committee of our hospital approved the study.

Electrophysiological study

AADs were abandoned ≥5 half-lives prior to procedure, but amiodarone was discontinued 3 days before. AADs were only resumed in case of ongoing recurrent unstable arrhythmias. Conscious sedation was used unless patient underwent epicardial mapping. Patients who underwent epicardial mapping had general anesthesia. The 6F quadripolar catheter with a 5-mm interelectrode distance was positioned at the RV apex. In patients, a deflectable 3.5 mm open irrigated-tip catheter with a CFS ablation catheter (Thermocool Smart Touch, Biosense Webster, Diamond Bar, CA, USA) was used for mapping and ablation. A programmed ventricular stimulation protocol was performed for the right ventricle and the right ventricular outflow tract for the induction of VT with triple extra stimulus with two driving cycle lengths.

Endocardial voltage mapping

A meticulous electroanatomic map of the endocardial RV surface was formed with a fill threshold of 20 mm. The bipolar signals were filtered at 30–500 Hz (CARTO-3 system, Biosense Webster, Inc.) and were displayed at the 100 mm/s speed. The signal amplitude of bipolar electrograms were manually revised for confirmation after being automatically gauged during mapping. Accordingly, a 3D bipolar voltage map as color gradients was formed to illustrate the electrogram signals. The tricuspid annulus was defined by both the fluoroscopic catheter position and electrogram recordings exhibiting, an approximately equal amplitude of atrial and ventricular bipolar signals. Valvular structures were labeled by location tags on a 3D map. Multiple electrograms were recorded to verify valvular sites. Steep hallows on the endocardial shell outline with sudden reductions in the signal slew rate and were deleted from the voltage maps. The referent bipolar signal amplitude value for defining a normal endocardial site was >1.5 mV. Values below 0.5 mV were defined as dense scars. During endocardial mapping, the referent value of unipolar voltage at the RV free wall to predict abnormal voltage area at confluent epicardial surface was <5.5 mV.

Epicardial mapping

Epicardial puncture was performed via inferior approach (8) to avoid RV and liver injury. The puncture was performed on the left of the xiphoid process with a superficial entry angle while compressing the epigastric region to reduce the possibility of an intra-abdominal organ injury. Epicardial mapping and ablation were performed via a 8F deflectable sheath. Epicardium voltage mapping was carried out with a 20 mm fill threshold. Contralateral of RV endocardial shell was identified as the RV epicardium. Bipolar voltage criteria to define a low-voltage zone at the epicardium were below 1 mV, and values below 0.5 mV were applied for the determination of dense scar regions. To differentiate an abnormal voltage zone from the coronary vessel and epicardial fat, low voltage zones with late potentials and broad multicomponent or split signals were considered. Zones having a bipolar voltage greater than 1.0 mV and showing an abnormal, multicomponent, split, or late signal were also tagged and, if adjacent to confluent areas of low-voltage sites, targeted for ablation during substrate-based ablation (Fig. 1 and 2) (9).

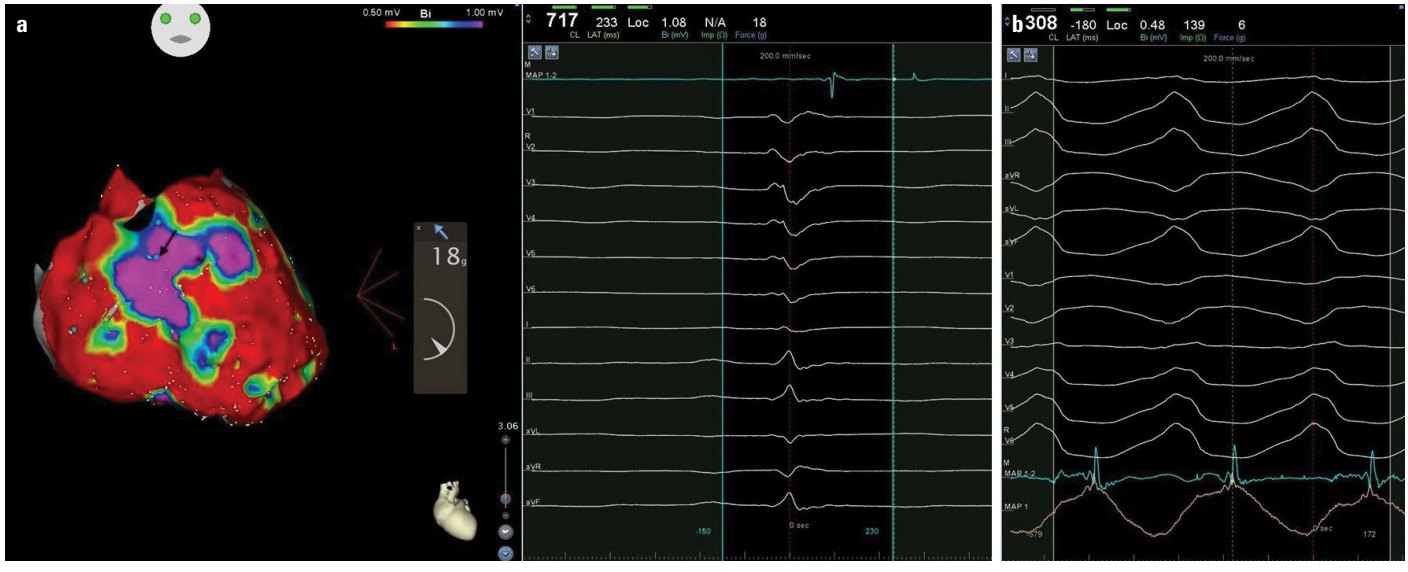

Figure 1. (a) Epicardial bipolar 3D mapping late potential recorded at the border zone (catheter image shown). (b) Ventricular tachycardia at the epicardial surface. Diastolic potential recorded at the scar border zone (black arrow) where the ablation terminated ventricular tachycardia

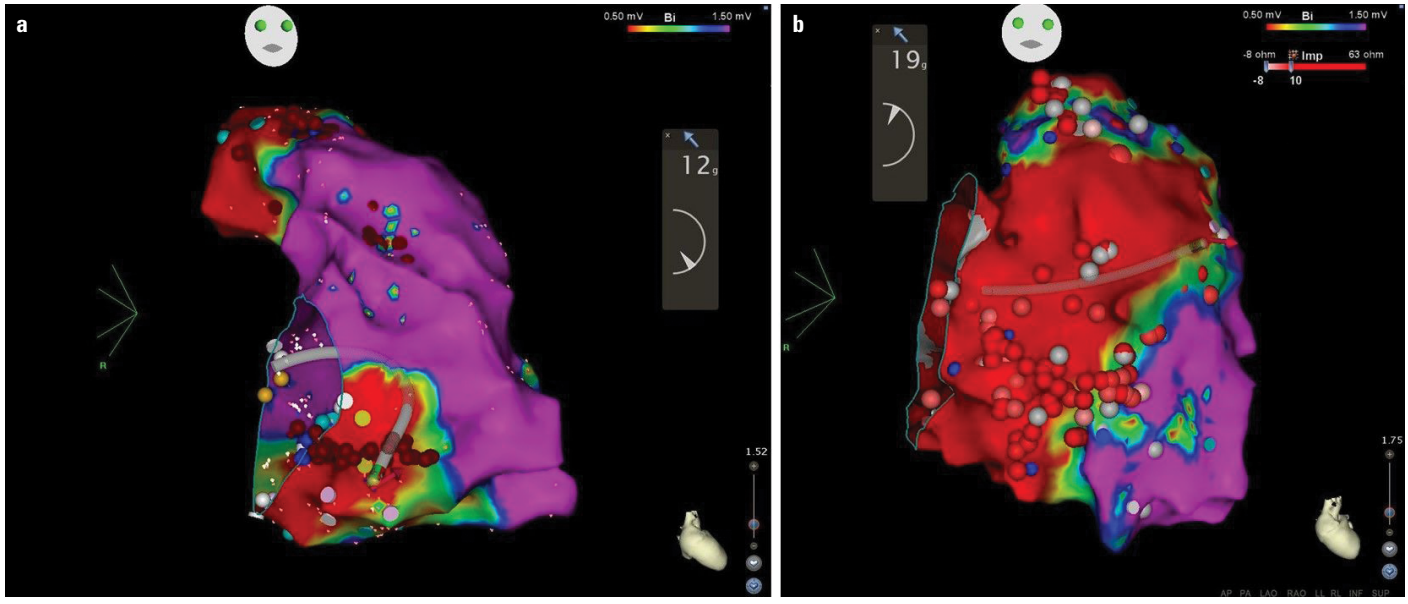

Figure 2. (a) Endocardial mapping showing inferior subtricuspid and the right ventricle outflow tract bipolar low-voltage areas. (b) Endocardial mapping showing subtricuspid and anterior right ventricle low bipolar voltage areas

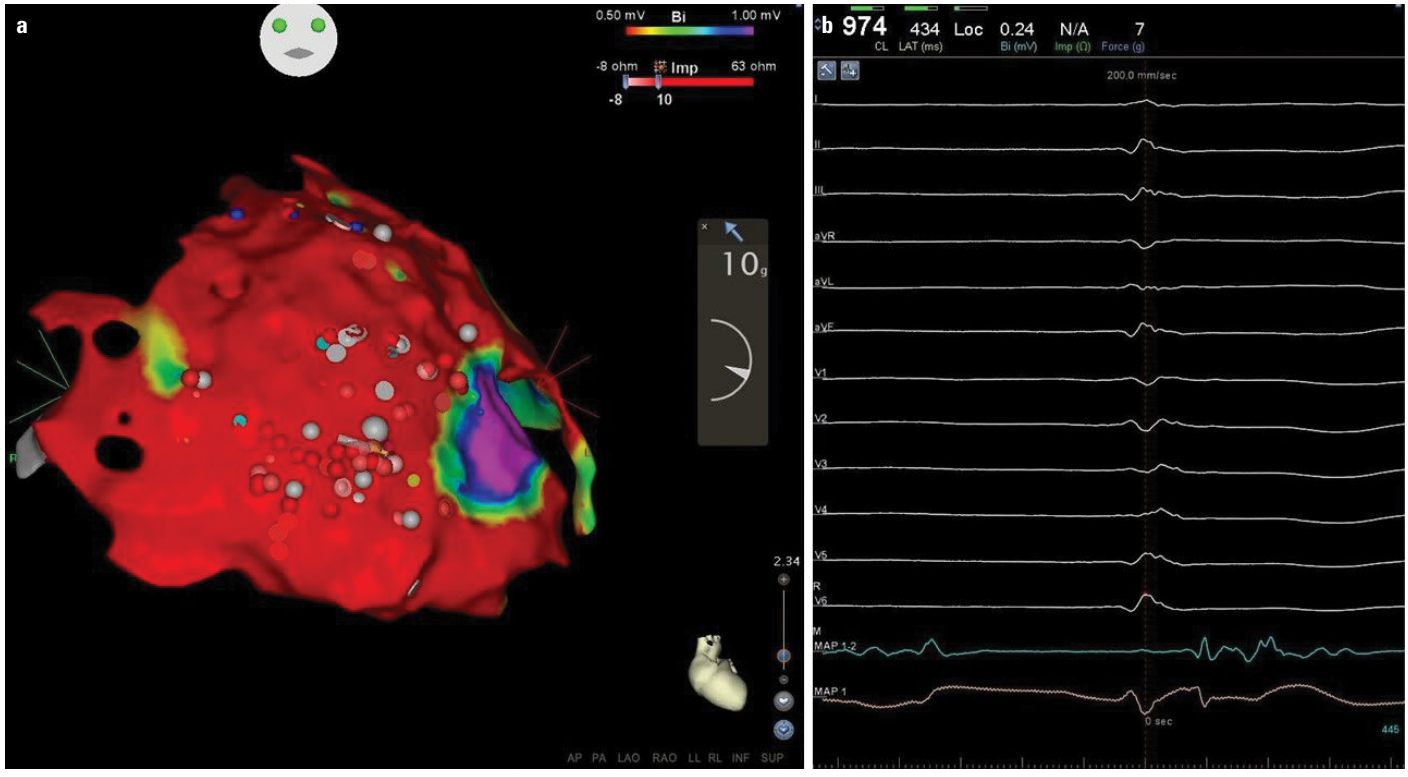

Figure 3. (a) Epicardial electroanatomic mapping. (b) Diastolic continuous electrogram recorded at the epicardial surface

Ablation

The 12-lead ECG morphology of all spontaneous VTs and PVCs was collected. If the ECG of VT was not present, then the intracardiac near-field and far-field electrograms of the implantable cardioverter defibrillator (ICD) were evaluated and compared with the inducible VT(s) during the procedure. Activation and entrainment mapping were performed to localize clinical and non-clinical VTs in patients without hemodynamic deterioration during VT. Critic isthmuses defined entrainment with concealed fusion with a post-pacing interval of 30 msec, and the stimulus to QRS ranged between 30% and 70% of the tachycardia cycle length. Substrate-based ablation was performed after the VT ablation. Areas with low voltage and late potentials, diastolic potentials, broad fragmented signals, and split potential were paced, and if the stimulus QRS was above 30 msec, these sites were defined as slow conduction channels, and ablation

Anatol J Cardiol 2019; 21: 187-95
DOI:10.14744/AnatolJCardiol.2018.58534

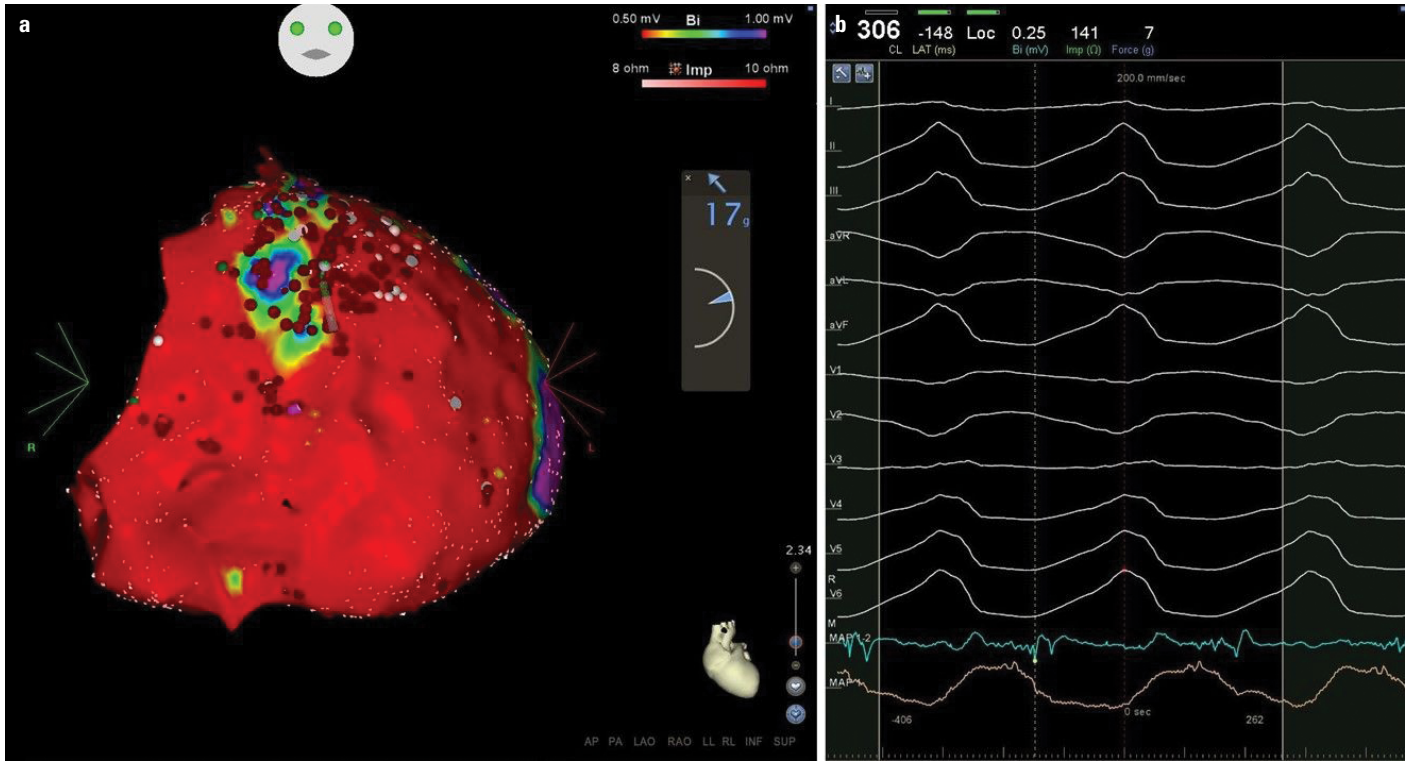

Figure 4. (a) Electroanatomic mapping of the epicardium. (b) Ventricular tachycardia. Diastolic continuous signal recorded at the scar border zone where a successful ablation was performed

was applied. VTs with hemodynamic compromise were mapped in the sinus rhythm, and pace mapping was used to define the site of VT (Fig. 3 and 4). Areas with a long stimulus QRS reproducing the best QRS match were ablated. Substrate-based ablation was carried out in all patients after clinical and non-clinical VT ablation. Patients with PVC underwent the ablation of all PVC morphologies, and a substrate-based ablation was performed after the ablation of PVCs. The best pace mapping with the earliest signal was sought to ablate for PVC.

Linear lesions were placed from the best pace match with a long-stimulus QRS region or through channels and transected the abnormal myocardium to valve annulus extending to normal myocardium. Also, linear lesions from the scar/abnormal voltage zone to scar/abnormal voltage zone, scar/abnormal voltage zone to valve annulus, and the encirclement of abnormal voltage site were performed depending to the anatomic location and the zone size. Ablation of abnormal electrograms continued until the signal modification was achieved. The abnormal electrograms within a 3 cm radius from the site of origin were specifically targeted. Extensive substrate ablation was performed especially at the epicardium. A coronary angiography and high output pacing were performed before ablation to avoid the coronary artery and phrenic nerve injury. Ablation was avoided within the 1 cm proximity to the coronary artery. A more extensive ablation was performed if multiple VT morphologies were present. If the VT inducibility persisted, above-mentioned strategies were repeated. The radiofrequency energy application was 40 watts for the endocardial and 35 watts for the epicardial surface, and a 42° maximum impedance drop was 12–15 Ohms. A contact force of 9 grams was applied to achieve an effective ablation both at the epicardium and endocardium. Radiofrequency energy duration was set to 60 seconds but increased to 180 seconds at the zones with transient VT suppression. Continuous intraarterial pressure monitoring was performed, and at the end of the epicardial ablation, the intrapericardial fluid amount was monitored with transthoracic echocardiography. A total of 125 mg of methylprednisolone was applied in the intrapericardial space. After the epicardial ablation, a pigtail catheter was left in the pericardial space for 24 hours.

Endpoints and follow-up

The endpoint of the study was freedom from any ventricular arrhythmias (PVC, VT, ventricular fibrillation). After the procedure, the VT induction was performed with two drive-cycle lengths up to three extra stimuli. The non-inducibility of any VT is defined as a complete procedural success, and induction of only non-clinical VT is defined as partial success. Patients who underwent VT ablation were given amiodarone and beta blockers for 6 months after the procedure. Patients who underwent VT ablation were monitored with device interrogation every 6 months for detection of ventricular arrhythmias. Patients who underwent PVC ablation were monitored with 24-hour Holter 1, 6, and 12 months from the procedure date. After a year, they were interviewed by telephone.

Statistical analysis

All continuous variables are expressed as the mean±standard deviation. A descriptive statistics analysis was also performed (IBM SPSS Statistics for Windows, Version 21.0. Armonk, NY, USA).

Results

Patients

The study population comprised 17 patients diagnosed with ARVD/C according to the Task Force Criteria. The study population was divided into two groups: the PVC and VT groups. Baseline characteristics are demonstrated for the PVC (5 patients) and VT (12 patients) groups in Tables 1 and 4.

The mean age of patients in the PVC group was 34.6±12.8 (3 females, 2 males), and all had LV systolic function impairment, except 1 patient (the mean EF, 40.4±12.1%). The mean PVC ratio in 24-hour Holter monitoring was 31.8±7.6% and with more than one PVC morphology (3.4±0.9). One patient who was implanted for primary prophylaxis had ICD for low ejection fraction in the PVC group.

The mean age of patients in the VT group was 36.6±6.3 (7 males, 5 females), and all had a normal LV systolic function, except 1 patient (the mean EF, 55.5±8.6%). All VT patients were implanted ICD previously (mean duration, 44.8±14.1 months) and on two ADD. Two patients in the VT group were implanted ICD for secondary prophylaxis. All VT patients were admitted with electrical storm (the mean number of shocks within 24 hours, 6.6±1.6). Two patients had a prior endocardial ablation history at other centers within 1 year.

Procedure

Epicardial ablation was not performed in the PVC group. Procedural characteristics of the PVC group are demonstrated in Table 2. The mean endocardial point count was 295.6±67.3 in the PVC group. The mean areas of bipolar low voltage and the scar were 17.4±8.4 and 10.2±5.4 cm^2, respectively. The mean low unipolar voltage area was larger than low bipolar voltage (21.5±9.5 cm^2). The mean contact force in the right ventricle during mapping and ablation was 12.6±1.1 (Table 3) and 12.8±1.9, respectively. All patients had a subtricuspid scar, except one. The mean procedure time and fluoroscopy time were 128.8±13.3 and 2±0.7 minutes, respectively. The total mean RF time was 20.2±3.25 minutes. The sites of origin for PVC's were subtricupid, lateral free RV, and anterior RVOT. All PVC morphologies could not be eradicated in 2 patients from the PVC group. Other patients had a successful ablation of all PVC morphologies.

All patients in the VT group underwent endo-epicardial ablation, but there was 1 patient who refused. The mean TCL of clinical VT and the median number of VT were 303±38.1 msec and 3, respectively (Table 5). The mean endocardial and epicardial points were 454±156.3 and 650.4±131.5, respectively (Table 5 and 6). The low-bipolar-voltage area was found less frequently in the endocardium than in epicardium (112.1±44.7 cm^2 versus

Table 1. Characteristics of patients who underwent premature ventricular contraction ablation

	Sex	Age	ICD	EF (%)	PVC (%)	Drug	Syncope	PVC mor.
Patient 1	M	27	-	45	25	BB	-	4
Patient 2	F	47	-	52	26	BB	-	3
Patient 3	M	22	-	40	30	BB	-	2
Patient 4	F	27	+	20	44	BB	-	4
Patient 5	F	50	-	45	34	BB	-	4

ICD - implantable cardioverter defibrillator; EF - ejection fraction; PVC - premature ventricular contraction percentage for 24 hours; PVC mor. - the number of premature ventricular contraction morphologies; BB - beta blocker

Table 2. Procedure characteristics of patients who underwent premature ventricular contraction ablation

	Endo. point	Bipolar low vol. (cm^2)	Bipolar scar loc.	Bipolar scar (cm^2)	Uni. <5.5 mv	Uni. <5.5 (cm^2)
Patient 1	312	20.3	Ant RV/subtric	10.1	Ant RVOT/ant RV/ subtric.	25.4
Patient 2	215	10.7	Ant RV	4.7	Ant RV/lateral free RV wall	8.7
Patient 3	243	11.3	Subtric	11.3	Subtric/ant RV	18.2
Patient 4	384	30.9	Subtric/RVOT	18.8	Subtric/ant RV/RVOT	34.6
Patient 5	324	14.2	Subtric	6.4	Subtric/lateral RV free wall	20.8

Endo. point - endocardial points taken by a 3D mapping system; Bipolar low vol. - measurement of endocardial areas with the voltage below 1.5 mv; Bipolar scar loc. - location of areas with voltage lower than 0.5 mv; RVOT - right ventricular outflow tract; ant, anterior; RV - right ventricle; Subtric - subtricuspid; Uni. <5.5 - location of endocardial areas with unipolar voltage lower than 5.5 mv; Uni. <5.5 (cm^2) - measurement of areas with unipolar voltage lower than 5.5 mV

Anatol J Cardiol 2019; 21: 187-95
DOI:10.14744/AnatolJCardiol.2018.58534

Table 3. Contact force and radiofrequency time and follow-up characteristics of patients who underwent premature ventricular contraction ablation

	Mean CF	RF time	Fl. time	Proc. time	Fol. up	PVC	Fol. up EF
Patient 1	14	18.4	2	110	19	-	55
Patient 2	13	20.3	1	124	13	2%	63
Patient 3	11	19.5	2	129	17	13%	48
Patient 4	12	25.7	3	146	11	15%	38
Patient 5	13	17.3	2	135	15	3%	60

Mean CF - contact force during mapping in grams; RF time - radiofrequency time in minutes; Fl. time - fluoroscopy time in minutes; Proc. time - procedure time in minutes; Fol. up - follow-up; PVC - percentage of premature ventricular contraction at the end of the follow-up observed with 24-hour Holter monitoring; Fol. up EF - follow-up ejection fraction as percentage

Table 4. Characteristics of patients with electrical storm who underwent ventricular tachycardia ablation

	Age	Sex	ICD	Shock	Syncope	Arrest	EF	Drugs	Abl. history
Patient 6	44	M	48	4	-	-	54	Ami-BB	-
Patient 7	45	M	24	8	-	-	58	Ami- BB	-
Patient 8	39	F	60	6	-	-	63	Ami-BB	+
Patient 9	25	M	54	5	-	-	60	Ami-BB	-
Patient 10	41	M	72	7	-	-	35	Ami-BB	+
Patient 11	34	F	38	9	+	-	53	Sot-mex	-
Patient 12	38	M	29	5	-	-	59	Ami-BB	-
Patient 13	29	F	56	6	-	-	67	Sot-mex	-
Patient 14	38	M	36	8	-	-	45	Sot-mex	+
Patient 15	42	M	42	9	+	-	53	Ami-BB	-
Patient 16	29	F	31	6	-	-	62	Ami-BB	+
Patient 17	36	F	48	7	-	-	58	Sot-mex	-

ICD - duration of implantable cardioverter defibrillator in months; Shock - number of shock episodes within 24 hours; EF - left ventricular ejection fraction; Drugs - administered drugs to control ventricular tachycardia prior to ablation; Ami - amiodarone; BB - beta blocker; Sot - sotalol; Mex - mexiletine; Abl. history - previous presence of ablation

Table 5. Procedure characteristics of patients who underwent ventricular tachycardia ablation for electrical storm

	cVT CL	n-cVT	n-CVT-CL	cVT circuit	n-cVT circuit	End/Epi. point	End low vol (cm^2)	End. Scar Loc.	End. scar (cm^2)
Patient 6	220	-	-	Epicardium	-	270/423	65	Subtric	45
Patient 7	320	2	367-340	Epicardium	Endo-epi	426/656	53	Subtric	24
Patient 8	324	3	340-410	Epicardium	Endo-epi	725/907	170	Ant RV/subtric	144
Patient 9	340	2	390-510	Epicardium	Endo-epi	685/770	168	Ant RV/subtric	106
Patient 10	270	1	380	Endocard.	Endo	436	180	Free wall RV/Ant RV	125
Patient 11	310	4	260-350	Endocard.	Endo-epi	543/612	145	Free wall RV/subtric	98
Patient 12	360	2	290-320	Epicardium	Endo-epi	489/546	120	Ant RVOT/subtric	65
Patient 13	280	1	310	Endocard.	Epicardium	602/710	92	Ant RV/subtric	72
Patient 14	310	2	350-380	Epicardium	Endocard	310/ 563	78	Inf RV/subtric	34
Patient 15	340	2	280-310	Epicardium	Endo-epi	350/ 624	66	Free wall RV/Ant RV	28
Patient 16	280	3	240-270	Epicardium	Endo-epi	286/ 761	120	Free wall RV/subtric	67
Patient 17	290	1	300	Endocard	Epicardium	326/ 583	95	Inf RV/subtric/Ant RV	48

CVT CL - clinical ventricular tachycardia cycle length; n-CVT - the number of non-clinical ventricular tachycardia episodes; n-CVT-CL - the range of the cycle length of non-clinical ventricular tachycardia; c-VT circuit - the location of clinic VT circuit; endocard - endocardium; n-cVT circuit - the location of non-clinic VT circuit; endo - endocardium; epi - epicardium; Endo. point - endocardial points taken by a 3D mapping system; Epi. point - epicardial points taken by a 3D mapping system; Endo low vol (cm^2) - area of the endocardial region with a voltage below 1.5 mv; Endo. scar loc - endocardial location of areas with a voltage lower than 0.5 mv; RVOT - right ventricular outflow tract; ant - anterior; RV - right ventricle; subtric - subtricuspid; End. scar (cm^2) - the area of the endocardial region with a voltage below 0.5 mV

Table 6. Procedure characteristics of patients who underwent ventricular tachycardia ablation

	Epi. low vol (cm^2)	Epi. scar loc	Epi. scar (cm^2)	En-RF	Ep-RF	Fl. time	Proc. time	Success CVT	Success n-CVT
Patient 6	220	RVOT/ant RV/subtric	180	21	35	6	228	+	N/A
Patient 7	445	Nearly whole epicardium	380	26	37	5	270	+	+
Patient 8	310	Nearly whole epicardium	286	26	42	7	258	+	+
Patient 9	156	Ant RV/ant RVOT/subt.	100	28	38	8	210	+	+
Patient 10	N/A	N/A	N/A	34	N/A	2	157	+	+
Patient 11	185	Ant RV/free wall RV/subt.	169	20	31	7	196	+	+
Patient 12	224	Ant RVOT/ant RV/subt.	156	19	29	9	213	+	-
Patient 13	157	RV Ant/subtric.	115	25	34	5	245	+	+
Patient 14	462	Nearly whole epicardium	430	23	31	6	286	+	-
Patient 15	485	Nearly whole epicardium	420	26	38	7	247	+	+
Patient 16	200	Ant RV/free wall RV/subt	143	29	33	6	273	+	+
Patient 17	246	Inf RV/subtric/Ant RV	168	21	29	5	259	+	+

Epi. Low vol - area of epicardial regions with a voltage below 1 mV; Epi. scar loc - location of area with a voltage below 0.5 mV; RVOT - right ventricular outflow tract; Ant - anterior; RV - right ventricle; subtric. - subtricuspid; N/A - not applicable; Epi. scar (cm^2) - the measurement of area with a voltage lower than 0.5 mV; RF time - endocardial radiofrequency time in minutes; Ep-RF time - epicardial radiofrequency time in minutes; Fl. time - fluoroscopy time in minutes; Proc. time - procedure time in minutes; Success CVT - success clinic ventricular tachycardia; Success n-CVT - success non-clinical ventricular tachycardia

Table 7. Mean contact force and follow-up in patients who underwent ventricular tachycardia ablation

	End. CF	Epi. CF	Delay	ATP	Shock	Death	Follow-up
Patient 6	13.3	19.4	16	-	-	-	18
Patient 7	14.1	13.4	22	-	-	-	19
Patient 8	12.5	11.7	18	-	-	-	9
Patient 9	14.3	16.8	15	-	-	-	10
Patient 10	12.2	n/a	-	-	2	-	23
Patient 11	12 .9	13.7	16	-	-	-	21
Patient 12	13.1	10.9	19	1	1	-	16
Patient 13	14.2	11.6	17	-	-	-	15
Patient 14	13.5	15.5	21	-	-	-	17
Patient 15	13.1	13.8	23	-	1	-	12
Patient 16	11.7	16.8	19	-	-	-	10
Patient 17	10.2	14.9	17	-	-	-	16

End. CF - the mean endocardial contact force during mapping on the endocardial surface in grams; Epi. CF - the mean epicardial contact force on the epicardial surface in grams; Delay - the mean activation delay among endocardial and epicardial opposite points in milliseconds; ATP - the number of anti-tachycardia pacing during the follow-up; Shock - the number of shock episodes during the follow-up; Follow-up - in months

280.9±125.3 cm^2). Similar to this finding, the scar area was larger in the epicardium than endocardium (231.5±124.4 cm^2 versus 71.3±39.1 cm^2). The mean contact force at the epicardium and endocardium was 14.4±2.6 and 12.9±1.1 grams, respectively (Table 7). The mean contact force at the epicardium and endocardium during ablation was 12.1±1.4 and 12.8±1.9 grams, respectively. A total RF time was greater at the epicardium (24.8±4.3 versus 34.2±4.1 minutes). The total duration of procedure and fluoroscopy was 222.1±36.4 minutes and 6±1.7 minutes, respectively. The mean activation delay at the opposite points for the epicardium and endocardium; the infundibulum, the lateral free RV wall, the anterior wall, the inferior wall, the apex, and the inferolateral tricuspid annulus were compared. Epicardial activation was found to occur later than in the endocardium (Table 7).

All clinical and non-clinical VTs were ablated successfully, except in 2 patients who had 1 non-clinical VT still inducible at the end of the procedure. Eight of 12 patients had clinical VT circuits were at epicardium. One patient did not have non-clinical VT. However, all other patients had nonclinical VTs at the endocardium or epicardium. Ten of 12 patients had a low voltage and the scar at the endocardial subtricuspid region. Nearly whole epicardium of 4 patients was covered with the scar. A low bipolar voltage and the scar distribution in the VT and PVC groups were at the RV anterior wall, lateral free RV wall, RVOT, subtricuspid

Anatol J Cardiol 2019; 21: 187-95
DOI:10.14744/AnatolJCardiol.2018.58534

region, and inferior RV. After the procedures, no complications occurred.

Follow-up findings

The mean follow-up duration was 15±3.1 months for the PVC group (Table 3). All PVC morphologies could not be eradicated in 2 patients, but a total percentage of PVC was reduced significantly in these 2 patients from the PVC group. The percentage of PVC of these patients at 24-hour Holter monitoring remained reduced during the follow-up. Other patients had a successful ablation of all PVC morphologies. During the follow-up, the percentage of PVC was found to be less than 5%. All patients' LV systolic function improved during the follow-up (mean EF, 52.8±10%).

The mean follow-up duration was 15.5±4.5 months for the VT group (Table 7). None of the VT patients experienced electrical storm during the follow-up. There were no deaths, but 1 patient was admitted for decompensated with heart failure. Three patients suffered shock during the follow-up. One of them had undergone endocardial ablation only and experienced shock twice in 10 months. One of the other 2 patients had had partial success, and the other had a complete procedural success.

Discussion

In this study, ablation of ventricular arrhythmias with CFS ablation catheters in the ARVD/C patients refractory to medical treatment was found to be promising. The long-term follow-up success rates are similar to previous studies performed with non-contact force catheters.

In the ARVD/C patients, the disease process usually begins in the subepicardium and progresses to the subendocardium (1). This leads to a more extensive involvement of the VT substrate and circuits on the epicardium compared to the endocardium (10, 11). Studies investigating favorable outcomes for the VT control found that the endocardial and epicardial ablation provides a better VT suppression (6, 7). The VT free survival with endo/epicardial ablation is 71% in the long term without requiring AAD (5-7). On the contrary, the absence of epicardial approach lacks similar success (3, 4). Due to better outcomes, we prefer the endo/epicardial approach in our patients with ARVD/C.

The contact force is essential in identifying the ventricular arrhythmia substrate (12). Some markers such as impedance, tactile feedback, and electrogram amplitude are considered as indirect measures of tissue contact. However, those markers are poor indicators of actual contact (13). Poor contact can cause misidentification of normal or abnormal tissues and mistakenly recognition of electrograms as lower than in the amplitude or missing abnormal electrograms that demonstrate critical sites of the VT circuit. Contact resuming in systole and diastole was associated with noticing abnormal electrograms with higher frequency to define the scar tissue (13). Best cutoff values of the optimal force for the right ventricle, left ventricle, and epicardium are 9, 8, and 8 g, respectively (12).

Contact force is one of the determinants of the optimal lesion formation during ablation. Impedance, and electrode temperature are poor predictors of the electrode–tissue contact force and lesion size (14). The lesion volume surges as the contact increases with the same power and duration (15). A low power and greater contact can form a lesion deeper than high power and lower contact (14). The power and duration of ablation can be adjusted to optimize lesion with imparted contact force.

A high contact force out of optimal range declines in efficacy and surges in complications (16). Monitoring contact force during the procedure can prevent potential complications such as perforation and steam pop. Additionally, during the epicardium mapping and ablation, the catheter tends to point outward to the lungs (16). Knowing the force vector orientation data can prevent inadvertent lung injury and facilitate an efficient epicardial lesion ablation.

In atrial fibrillation ablation, the CF sensing is linked with better outcomes in terms of ablation duration, fluoroscopy, and procedure duration, and a better lesion formation with superior clinical outcomes (17, 18). However, much fewer studies investigated CF sensing for scar-related ventricular arrhythmias. The mapping and ablation of scar-related ventricular arrhythmias with CFS catheters might have better outcomes by better defining substrate and optimal ablation lesions.

In this study, we performed endo-epicardial ablation for VT and endocardial ablation for PVC in ARVD/C patients. Irrigated CFS catheters were used for ablation. During and after the procedure and follow-up, no complications occurred. In the VT group, epicardial electroanatomic substrate was covering a larger area than the endocardial side. We found a delayed activation on the epicardium considering contralateral endocardial sides. Clinical VT circuits were mostly located at the epicardial surface. However, non-clinical VTs were found at both the endocardial and epicardial sides. Three patients had VT recurrence. One of them had only endocardial ablation and refused epicardial ablation. If adjuvant epicardial ablation had been added to the treatment, the outcome may have been better. The other 2 patients underwent endo-epicardial ablation, 1 with partial success, and the other with complete success. None of the patients experienced a VT storm during the follow-up.

In the PVC group, the endocardial voltage mapping demonstrated larger low unipolar regions than low bipolar regions, which indicates the presence of more low-voltage area at the epicardial side. All PVC morphologies were ablated in 3 of 5 patients. In the remaining 2 patients, all morphologies could not be ablated. During the follow-up, the PVC burden was observed to decrease significantly for these 2 patients. The left ventricular systolic function improved in all patients.

Study limitations

Our study was small and non-randomized and has inherent limitations due to these factors. The follow-up duration is not adequate considering the progressive nature of ARVD/C that can

result in new arrhythmic events or recurrence due to change in the VT substrate with time. Epicardial adjuvant ablation may increase our success rate for PVC patients, but we preferred to follow the clinical results to make a decision about further ablation.

Conclusion

Data about procedural and long-term outcomes of CFS catheters in scar-related ventricular arrhythmias are scarce. The application of a CFS catheter in endocardial and epicardial ablation of scar-related ventricular arrhythmias in ARVD/C have not been reported with long-term results. The use of contact sense catheters in the ablation of ARVD/C patients seems promising. However, the limited patient number and follow-up duration of our study necessitates its verification with randomized large-sample studies that have a longer follow-up.

Conflict of interest: None declared.

Peer-review: Externally peer-reviewed.

Authorship contributions: Concept – D.A., F.Ö.; Design – F.Ö.; Supervision – S.T.; Fundings – None; Materials – D.A., M.K.; Data collection &/or processing – S.Ç., Ö.Ö., M.K.; Analysis &/or interpretation – D.A., F.Ö.; Literature search – F.Ö., S.T.; Writing – F.Ö.; Critical review – D.A., F.Ö., S.Ç., Ö.Ö., S.T.

References

1. Thiene G, Basso C, Calabrese F, Angelini A, Valente M. Pathology and pathogenesis of arrhythmogenic right ventricular cardiomyopathy. Herz 2000; 25: 210-5. [CrossRef]
2. Santangeli P, Dello Russo A, Pieroni M, Casella M, Di Biase L, Burkhardt JD, et al. Fragmented and delayed electrograms within fibrofatty scar predict arrhythmic events in arrhythmogenic right ventricular cardiomyopathy: results from a prospective risk stratification study. Heart Rhythm 2012; 9: 1200-6. [CrossRef]
3. Dalal D, Jain R, Tandri H, Dong J, Eid SM, Prakasa K, et al. Long-term efficacy of catheter ablation of ventriculartachycardia in patients with arrhythmogenic right ventricular dysplasia/cardiomyopathy. J Am Coll Cardiol 2007; 50: 432-40. [CrossRef]
4. Verma A, Kilicaslan F, Schweikert RA, Tomassoni G, RossilloA, Marrouche NF, et al. Short- and long-term success of substrate-based mapping and ablation of ventricular tachycardia in arrhythmogenic right ventricular dysplasia. Circulation 2005; 111: 3209-16. [CrossRef]
5. Bai R, Di Biase L, Shivkumar K, Mohanty P, Tung R, Santangeli P, et al. Ablation of ventricular arrhythmias in arrhythmogenic right ventricular dysplasia/cardiomyopathy: arrhythmia-free survival after endo-epicardial substrate based mapping and ablation. Circ Arrhythm Electrophysiol 2011; 4: 478-85. [CrossRef]
6. Philips B, te Riele AS, Sawant A, Kareddy V, James CA, Murray B, et al. Outcomes and ventricular tachycardia recurrence characteristics after epicardial ablation of ventricular tachycardia in arrhythmogenic right ventriculardysplasia/cardiomyopathy. Heart Rhythm 2015; 12: 716-25. [CrossRef]
7. Santangeli P, Zado ES, Supple GE, Haqqani HM, Garcia FC, Tschabrunn CM, et al. Long-Term Outcome With Catheter Ablation of Ventricular Tachycardia in Patients With Arrhythmogenic Right Ventricular Cardiomyopathy. Circ Arrhythm Electrophysiol 2015; 8: 1413-21.
8. Sosa E, Scanavacca M, d'Avila A, Pilleggi F. A new technique to perform epicardial mapping in the electrophysiology laboratory. J Cardiovasc Electrophysiol 1996; 7: 531-6. [CrossRef]
9. Garcia FC, Bazan V, Zado ES, Ren JF, Marchlinski FE. Epicardial substrate and outcome with epicardial ablation of ventricular tachycardia in arrhythmogenic right ventricular cardiomyopathy/dysplasia. Circulation 2009; 120: 366-75. [CrossRef]
10. Polin GM, Haqqani H, Tzou W, Hutchinson MD, Garcia FC, Callans DJ, et al. Endocardial unipolar voltage mapping to identify epicardial substrate in arrhythmogenic right ventricular cardiomyopathy/dysplasia. Heart Rhythm 2011; 8: 76-83. [CrossRef]
11. Berruezo A, Fernandez-Armenta J, Mont L, Zeljko H, Andreu D, Herczku C, et al. Combined endocardial and epicardial catheter ablation in arrhythmogenic right ventricular dysplasia incorporating scar dechanneling technique. Circ Arrhythmia Electrophysiol 2012; 5: 111-21. [CrossRef]
12. Mizuno H, Vergara P, Maccabelli G, Trevisi N, Eng SC, Brombin C, et al. Contact force monitoring for cardiac mapping in patients with ventricular tachycardia. J Cardiovasc Electrophysiol 2013; 24: 519-24.
13. Ariyarathna N, Kumar S, Thomas SP, Stevenson WG, Michaud GF. Role of Contact Force Sensing in Catheter Ablation of Cardiac Arrhythmias: Evolution or History Repeating Itself? JACC Clin Electrophysiol 2018; 4: 707-23. [CrossRef]
14. Yokoyama K, Nakagawa H, Shah DC, Lambert H, Leo G, Aeby N, et al. Novel contact force sensor incorporated in irrigated radiofrequency ablation catheter predicts lesion size and incidence of steam pop and thrombus. Circ Arrhythm Electrophysiol 2008; 1: 354-62.
15. Ikeda A, Nakagawa H, Lambert H, Shah DC, Fonck E, Yulzari A, et al. Relationship between catheter contact force and radiofrequency lesion size and incidence of steam pop in the beating canine heart: electrogram amplitude, impedance, and electrode temperature are poor predictors of electrode-tissue contact force and lesion size. Circ Arrhythm Electrophysiol 2014; 7: 1174-80. [CrossRef]
16. Jesel L, Sacher F, Komatsu Y, Daly M, Zellerhoff S, Lim HS, et al. Characterization of contact force during endocardial and epicardial ventricular mapping. Circ Arrhythm Electrophysiol 2014; 7: 1168-73.
17. Afzal MR, Chatta J, Samanta A, Waheed S, Mahmoudi M, Vukas R, et al. Use of contact force sensing technology during radiofrequency ablation reduces recurrence of atrial fibrillation: a systematic review and meta-analysis. Heart Rhythm 2015; 12: 1990-6. [CrossRef]
18. Shurrab M, Di Biase L, Briceno DF, Kaoutskaia A, Haj-Yahia S, Newman D, et al. Impact of Contact Force Technology on Atrial Fibrillation Ablation: A Meta-Analysis. J Am Heart Assoc 2015; 4:e002476.