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Long-term Outcomes of Ablation for Ventricular Arrhythmias in Mitral Valve Prolapse

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Abstract

Objective: Prior studies reporting efficacy of radiofrequency catheter ablation for complex ventricular ectopy in mitral valve prolapse (MVP) are limited by selective inclusion of bileaflet MVP, papillary muscle only ablation or short-term follow-up. We sought to evaluate the long-term incidence of hemodynamically significant ventricular tachycardia (VT) or fibrillation (VF) in patients with MVP after initial ablation.

Methods and Results: We studied consecutive patients with MVP undergoing ablation for complex ventricular ectopy between 2013 and 2017 at our institution. Of 580 patients with MVP, we included 15 (2.6%, 10 women; mean age 50 ± 14 years, 53% bileaflet) with complex ventricular ectopy treated with initial ablation. Over a median follow-up of 3,406 (1,875-6,551) days or 9 years, 5 of 15 (33%) patients developed hemodynamically significant VT/VF after their initial ablation, and underwent placement of an implantable cardioverter defibrillator (ICD). Three of 5 also underwent repeat ablations. Sustained VT was inducible prior to index ablation in all 5 who developed VT/VF, compared to none of the 10 patients who did not develop VT/VF after index ablation (p = 0.002). Complex ventricular ectopy at index ablation was multifocal in all 5 patients who underwent repeat intervention versus 4 of 10 patients (40%) who did not (p = 0.04). All 3 patients with subsequent VT/VF who underwent repeat ablation had a new clinically

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dominant focus of ventricular arrhythmia and 3 of the patients with ICD had appropriate VT/VF therapies.

Conclusions: In the long-term, a subset of MVP patients treated with ablation for ventricular arrhythmias, all with multifocal ectopy on initial EP study, develop hemodynamically significant VT/VF. Our findings suggest the progressive nature of ventricular arrhythmias in patients with MVP and multifocal ectopy.

Keywords

Ventricular arrhythmia ablation procedures; ventricular tachycardia; premature ventricular beats; valvular heart disease; mitral regurgitation

INTRODUCTION

Every year, 0.4-1.9% of patients with mitral valve prolapse (MVP) will develop sudden cardiac arrest, and 7% of sudden deaths in the young are caused by MVP [1–5]. However, primary prevention implantable cardioverter defibrillators (ICDs) are currently not indicated in this population, largely because the optimal predictors of MVP-related clinically significant ventricular arrhythmias remain poorly understood. Sudden cardiac arrest has been reported to be more prevalent in MVP with flail mitral leaflet and severe mitral regurgitation (MR) [6]. More recently, sudden cardiac arrest risk has been linked to a bileaflet phenotype with mild MR, inverted/biphasic inferior T waves on ECG, and complex ventricular ectopy (ComVE - defined as frequent premature ventricular contractions [PVCs], bigeminy, or non-sustained (NS)/sustained ventricular tachycardia [VT]) [2, 7–9]. In this phenotype, left ventricular (LV) focal fibrosis in the papillary muscles (PMs) or the inferolateral base has been described on cardiac magnetic resonance (CMR)-late gadolinium enhancement (LGE) images [2, 10], often in association with mitral annular disjunction (MAD), a separation between the left atrial wall at the level of the MV junction and the LV wall [2, 11, 12].

During electrophysiology (EP) studies, MVP-related ComVE commonly arises from one or both papillary muscles (PMs) or the Purkinje/fascicular system [9]. However, other foci have been reported, often concomitant with the PM/fascicular origin, consistent with a multifocal arrhythmogenic process in MVP [9, 13]. Indeed, diffuse fibrosis identified by CMR T1 mapping has been identified in MVP with ComVE, even in the absence of LGE and even without significant MR [14]. Furthermore, an autopsy study of cases of sudden cardiac death with MVP found multifocal left and right ventricular microscopic fibrosis [15]. The sum of these findings suggests a primary diffuse myopathic process that may have important therapeutic implications.

Symptomatic ComVE in MVP is generally treated with antiarrhythmic medications or radiofrequency catheter ablation. Prior studies reporting efficacy of ablation are limited by selective inclusion of bileaflet MVP with mild MR [9], PM only ablation [16, 17], and/or median follow-up of less than 2 years [9, 17]. Recently, another study reported a 26% prevalence of recurrent ventricular arrhythmias over a mean of 2.5 years in a sample of 43 MVP patients (30 treated with ablation and 13 with ICD), although the proportion of

patients with PVCs versus hemodynamically significant VT or VF following initial ablation was not specified [13].

We sought to evaluate the long-term success of ablation in consecutive MVP patients by quantifying the burden of hemodynamically significant VT or VF after initial ablation. We also sought to investigate the clinical and electrophysiological characteristics of those MVP cases that developed VT or VF following initial ablation.

METHODS

Study Population

We cross-linked the echocardiography and EP databases at the University of California, San Francisco Medical Center from 2013 to 2017 to identify consecutive MVP patients treated with ablation for symptomatic ComVE. The latter was defined as high burden (> 5%) of PVCs, bigeminy, or NSVT/sustained VT. ComVE was considered symptomatic when associated with palpitations, chest pain, shortness of breath, pre-syncope, or syncope. All MVP cases referred to undergo ablation had failed medical therapy.

Because our objective was to evaluate the long-term incidence of hemodynamically significant VT or VF after index ablation, we excluded prevalent cases with a cardiac arrest prior to index ablation. We also excluded cases with prior myocardial infarction or stent placement, ischemic or non-ischemic cardiomyopathies with severely reduced LV ejection fraction (35%), and sarcoidosis.

We assessed how many MVPs developed either hemodynamically significant VT (defined as VT with hypotension or syncope) or VF after index ablation by examining Holter/event monitor data, hospitalization records, ECG recording during stress testing, and subsequent EP studies. We compared the patients who developed life-threatening arrhythmias (hemodynamically significant VT or VF) with an overall group of more benign presentations (cases with repeat ablation for PVCs/NSVT or without repeat intervention). All patients underwent 2-dimensional transthoracic echocardiography, 12-lead ECG, a 48-hour Holter or 2-week event monitor, and one or more EP studies. CMR was available in selected cases based on clinical indication (either ventricular arrhythmias, quantification of MR, or assessment of cardiac chamber dimensions).

All participants gave written informed consent. The study was approved by the University of California, San Francisco's Institutional Review Board.

Arrhythmia Detection

Holter or 2-week event monitor recordings were reviewed for burden and site of origin of PVCs, and for the presence of bigeminy or sustained/NSVT.[2, 7–9] Sustained VT was defined as tachycardia of ventricular origin with a rate >100 bpm and lasting >30 seconds. NSVT was defined as 3 PVCs with a rate >100 bpm lasting <30 seconds. All available 12-lead ECGs were analyzed for biphasic or inverted T waves, and QRS and QT interval durations.

Standard Echocardiography

MVP patients underwent routine 2-dimensional echocardiography studies using a variety of commercially available cardiovascular ultrasound machines as part of standard clinical evaluation. Echocardiograms were obtained within 1 year of the index ablation in the majority of cases.

MVP was diagnosed as systolic leaflet displacement of one or both leaflets > 2 mm beyond the mitral annulus in a parasternal or apical 3-chamber long-axis view.[18, 19] When quantitative assessment of MR was not available, its severity was based on visual estimation of the regurgitant jet.[20] The presence of MAD was assessed qualitatively as a separation between the left atrial wall at the level of MV junction and the LV free wall.[21] LV end-diastolic/end-systolic volumes, LV ejection fraction, LV mass, and left atrial volume were quantified and indexed to body surface area as previously described.[20] Right ventricular dilatation was defined as a basal diameter 4.2 cm. Right ventricular systolic dysfunction was assessed qualitatively.

Cardiac Magnetic Resonance Imaging

CMR was performed in selected individuals based on clinical indications using a 3-T magnetic resonance imaging scanner (Discovery MR750w, General Electric Healthcare, Milwaukee, WI, USA) prior to ablation. At 10 minutes after injection of 0.1 mmol/kg gadobutrol, late gadolinium enhancement images were obtained with a high-resolution breath-hold 2-dimensional sequence at three separate levels in the short axis plane.

Electrophysiology Study and Radiofrequency Catheter Ablation

EP studies were performed using standard protocol with conscious sedation.[22] Standard multielectrode intracardiac catheters were introduced via femoral venous access and positioned under fluoroscopic guidance in the right atrium, coronary sinus, and/or right ventricle. The LV was accessed either through a transseptal or retrograde aortic approach via femoral arterial access. The CARTO system (Biosense Webster) was used to create detailed 3-dimensional electroanatomic mapping, supplemented as needed with intracardiac echocardiography to distinguish between papillary and fascicular origin of ComVE. Mapping and ablation were performed with a 3.5- or 4.0-mm tip catheter (NaviStar ThermoCool; Biosense Webster, New Brunswick, NJ). Radiofrequency was delivered in unipolar fashion from the catheter tip with temperature and impedance monitoring.

If ComVE was not present at baseline, it was induced either by isoproterenol infusion or ventricular burst pacing, or both. The presence or absence of sustained VT, spontaneously or with stimulation, was noted prior to ablation. Induced ComVE was compared with stored surface ECG tracings to determine clinical relevance. Multifocal origin of ComVE was defined as the presence of more than one dominant PVC or VT on EP study.

Success of index ablation was defined as non-inducibility of the targeted clinically dominant ventricular arrhythmias. Lack of success of index ablation was defined as residual hemodynamically significant VT or VF after ablation.

Statistical Analysis

Continuous variables were compared using either Student t tests or Wilcoxon rank sum tests when not normally distributed. Fisher exact tests were used to compare frequency distributions between dichotomous groups. Multivariate analyses were not performed due to the small sample size and low number of outcomes. Two-tailed p-values of <0.05 were considered statistically significant. Analyses were performed using standard statistical software (Stata/SE 15.1).

RESULTS

Baseline Clinical Characteristics

Of 580 patients with MVP on transthoracic echocardiogram from 2013-2017, 20 (3%) underwent ablation for ComVE. Of these, 5 were excluded due to history of myocardial infarction with stent placement (n = 1), sarcoidosis (n = 1), non-ischemic cardiomyopathy (n = 2), and cardiac arrest with secondary prevention ICD implantation prior to index ablation (n = 1) (Figure 1). Of the 15 patients included in the study, over a median follow-up of 3,406 (1,875-6,551) days or 9 years, 5 (33%) developed hemodynamically significant VT or VF after index ablation. The remaining 10 patients did not develop VT or VF. The group that did not develop VT or VF included patients who had no further procedures (7), and those that had repeat ablation for symptomatic PVCs/NSVT (3). The 2 groups had similar demographic and clinical characteristics (Table 1). Among the 5 MVP patients that developed hemodynamically significant VT or VF, 2 (40%) had a prior MV repair. There was 1 valve repair and 1 replacement in the group without subsequent VT/VF. All patients with prior valve surgery had trace or mild MR at the time of their ablation.

Surface and Ambulatory Electrocardiographic Data

On surface ECG, the majority of patients had inverted or biphasic T waves in the inferior leads (Table 1). All patients had a QRS < 120 msec, with a similar QRS measurement across groups. The longest QTc recorded for any patient was 480 msec, without a significant difference between groups. On ambulatory ECG prior to index ablation, patients in both groups had a high burden of PVCs, with multiple PVC morphologies in 60% of MVP patients who ultimately developed VT or VF (Table 1).

Cardiac Imaging

Echocardiographic parameters were similar between the two groups. Overall, only half (8/15 or 53%) of the patients had bileaflet MVP and MAD (Figure 2a). Notably, at the time of the index ablation, most patients (14/15 or 93%) had trace or mild MR (Table 1).

LV ejection fraction, volumes and mass indexes were similar between groups. (Table 1). There was no evidence of right ventricular dilatation or systolic dysfunction in any of the patients studied.

CMR was available in 9 patients. None of the 5 patients who ultimately developed hemodynamically significant VT or VF with available CMR had LGE (Table 1 and Figure 2c–d). LGE was seen in 2 (33%) of the 6 patients with available CMR in the group that did

not develop VT or VF (Table 1 and Figure 2b). In both cases, MVP was bileaflet, LGE was located in the PMs, the index ablation target corresponded to the LGE sites, and the patient had no further procedures after index ablation.

Index Radiofrequency Catheter Ablation

The indication for index ablation was the presence of symptomatic PVCs with NSVT in the majority of patients (14/15 or 93%, Table 2).

All 5 patients that went on to develop hemodynamically significant VT or VF after index ablation had sustained VT that was inducible during the EP study prior to index ablation compared to 0 of 10 patients in the group that did not require repeat procedures in our follow-up period (p = 0.0003).

ComVE seen during the EP study at index ablation was multifocal in 5 (100%) of the 5 patients that subsequently developed hemodynamically significant VT or VF compared with 4 (40%) of the 10 patients who did not (p = 0.04). The sites of ComVE ablated at the index procedure included the anterolateral and posteromedial PM, left anterior and posterior fascicles, right and LV outflow tracts, and basal LV (Tables 2 and 4). The PM/fascicles were a common site of ablation (9/15 or 60%). However, six patients had an ablation target different than the PM/fascicles at index procedure (Tables 2 and 4).

Thirteen out of 15 index ablations had acute procedural success. Both of the index ablations that did not meet criteria for acute procedural success were in the group that eventually developed hemodynamically significant VT or VF (Table 2)

Development of hemodynamically significant VT or VF

Of the 5 patients that developed hemodynamically significant VT/VF after their index ablation, 3 developed VT and 2 developed VF. The median time from index ablation to ICD placement for hemodynamically significant VT/VF was 313 (283-709) days. Over a median follow-up of 3,058 days (153 – 7,870) or 8.4 years after ICD implantation, 3 patients had subsequent appropriate ICD therapy for VT (1) and VF (2). Further details of clinical course and ICD therapies are available in the patient descriptions included in the Supplemental Information. There were no deaths.

Repeat radiofrequency catheter ablation and ICD implantation

A total of 6 patients had repeat ablation (2 had repeat ablation only, 4 had repeat ablation either prior or after ICD implantation) and a total of 6 patients had ICD (2 had ICD only, 4 had repeat ablation and ICD) (Figure 1). The indications for repeat ablation or ICD placement are shown in Table 3 and are expanded in the patient descriptions in the Supplemental Information.

The sites of ComVE targeted during repeat procedures are shown both in aggregate in Table 3 and at the patient level in Table 4. Notably, in the majority (6/9 or 67%) of repeat ablations, the site of ComVE ablation was different than the site ablated at the index or prior repeat ablation (Figure 3)

DISCUSSION

In the long-term, a subset of MVP patients who undergo ablation for ComVE develop hemodynamically significant VT or VF, even if the index ablation was acutely successful. Among those with ICD implantation following index ablation, appropriate ICD therapies for VT/VF may occur. Our findings contrast those from prior studies that reported high efficacy of ablation in MVP [9, 16]. This discrepancy may be explained by a longer follow-up in our sample, with a median of 9 years, while the follow-up was limited to 1-2 years in prior publications [9, 13, 16, 17]. Moreover, prior selected samples of MVP cases treated with ablation only included patients with PM ComVE, or only patients with bileaflet MVP [9, 16].

Our work adds to the literature by including all MVP subtypes and all foci of ventricular arrhythmias with long-term follow-up. We found a significant and novel association between multifocality of ComVE at index ablation and hemodynamically significant VT/VF. In those patients with available EP study at repeat intervention, the origin of ComVE was different than the ventricular arrhythmias induced and successfully targeted at index ablation. This finding argues that multifocality and progression of ComVE, rather than procedural failure, explain the development of malignant arrhythmias in MVP post-index ablation. Our results align with multiple prior studies that have reported multifocal ComVE in MVP [2, 9, 16, 23]. In addition, we demonstrate that induction of sustained VT with standard ventricular pacing maneuvers at index ablation was significantly associated with hemodynamically significant VT/VF requiring repeat procedures. Thus, induction of sustained VT during EP study may represent, together with multifocal ComVE, a useful indicator of risk for progressive arrhythmias [24].

The multifocal ComVE observed at index and repeat ablation, absence of focal fibrosis in the majority of cases with available CMR, and absence of severe MR at time of ablation challenge many of the previously proposed explanations for ComVE in MVP. A recent autopsy study revealed left and right ventricular multifocal microscopic fibrosis in MVP cases with sudden cardiac death [15]. Another study demonstrated mutations in Filamin-C, a cardiomyopathy gene, in family members with arrhythmogenic MVP [25]. Overall, these findings suggest a primary, diffuse myopathic process that may act as a "substrate" for ventricular arrhythmias in the presence of bileaflet MVP, MAD and myocardial stretch (the "trigger") or alone, when bileaflet MVP and associated arrhythmic features are absent. Interestingly, bileaflet MVP with MAD was present in only half of our patients (Figure 2a), indicating that a large proportion of our study subjects fall outside of the bileaflet phenotype proposed in recent studies [2, 7]. While fibrosis of the LV PMs and inferolateral base related to myocardial stretch from prolapsing leaflets has been previously postulated to contribute to the development of ComVE in these patients [2], only 2 of the patients in our study had LGE on CMR (Figure 2b), and none of the patients who developed hemodynamically significant VT or VF had this finding (Figure 2c–d). Diffuse fibrosis by CMR T1 mapping has been linked to a higher risk of ventricular arrhythmia in MVP patients [14], even in the absence of LGE and even without significant MR. The presence of diffuse fibrosis may also explain arrhythmic risk in our patient population, although this could not be confirmed due to the lack of CMR T1 mapping data.

There were no significant differences in demographics or traditional risk factors for arrhythmic complications such as hypertension, diabetes, smoking, or drug use between the 2 groups. LV mass, dimensions, and systolic function (overall normal or borderline) by echocardiography were also similar between the 2 groups. These findings suggest that the occurrence of hemodynamically significant VT/VF was not driven by comorbidities or significant LV systolic dysfunction, hypertrophy, or cavity dilatation.

Prior studies reported that MV surgery lowers the risk of ComVE and appropriate ICD therapies for VT/VF in MVP [26, 27]. However, such studies are limited by selection of bileaflet MVP alone. In our long-term study of all MVP subtypes, we showed, albeit based on a very small sample size, that MV surgery may not protect from hemodynamically significant ventricular arrhythmias, even when followed by a catheter ablation and even if significant residual MR is absent.

Study Limitations

Our study has several important limitations. As in other studies on ablation of ComVE in MVP, the absolute number of patients is small due to the rarity of the condition. Such number has ranged between 9 and 30 in prior literature [7, 9, 11, 13, 16, 17]. Hence, there may be differences between our study groups that we are unable to detect (e.g. in the site of ComVE at index ablation).

Our data was obtained from a single tertiary referral center and may not represent the experience at other institutions. Due to the retrospective design of our study, not all subjects were assessed uniformly. CMR was not available for all patients. In addition, our study included ablations that occurred at different time points over the duration of our study, which may have had differences in available diagnostic and therapeutic technology.

CONCLUSIONS

In the long-term, radiofrequency catheter ablation of ComVE in MVP may not prevent hemodynamically significant VT/VF requiring repeat ablation and/or ICD, particularly in MVP with multifocal ComVE or with sustained VT induced at index EP study. In the absence of guideline recommendations, decision to implant a primary prevention ICD in MVP patients with ComVE remains operator-dependent. Larger prospective studies including clinical, imaging, and EP data are needed to better select MVP patients who may require earlier ICD implantation.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

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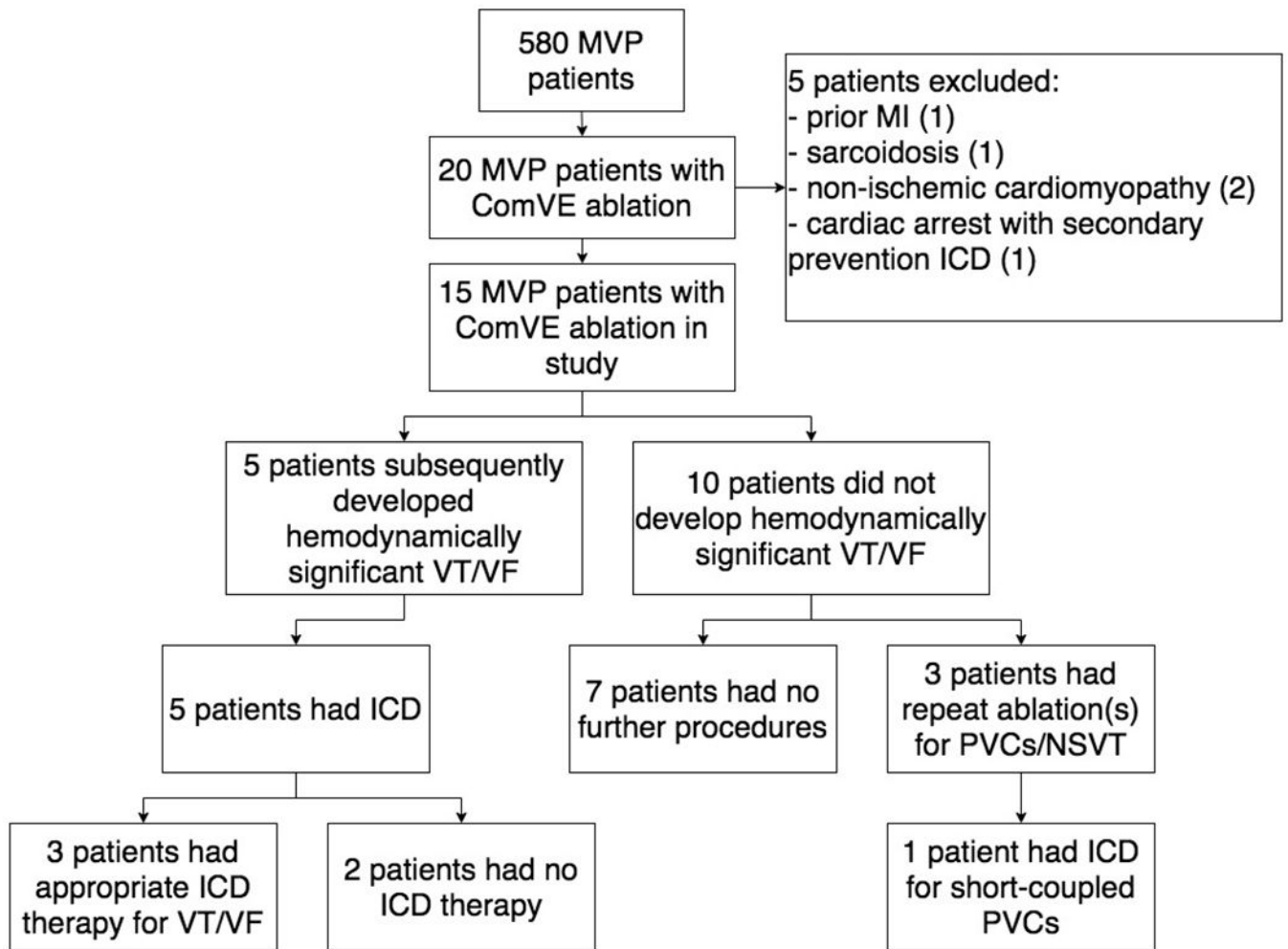
Fig. 1. Patient selection, exclusion and outcomes.
MVP = Mitral valve prolapse; ComVE = Complex ventricular ectopy; ICD = implantable cardioverter defibrillator; MI = myocardial infarction
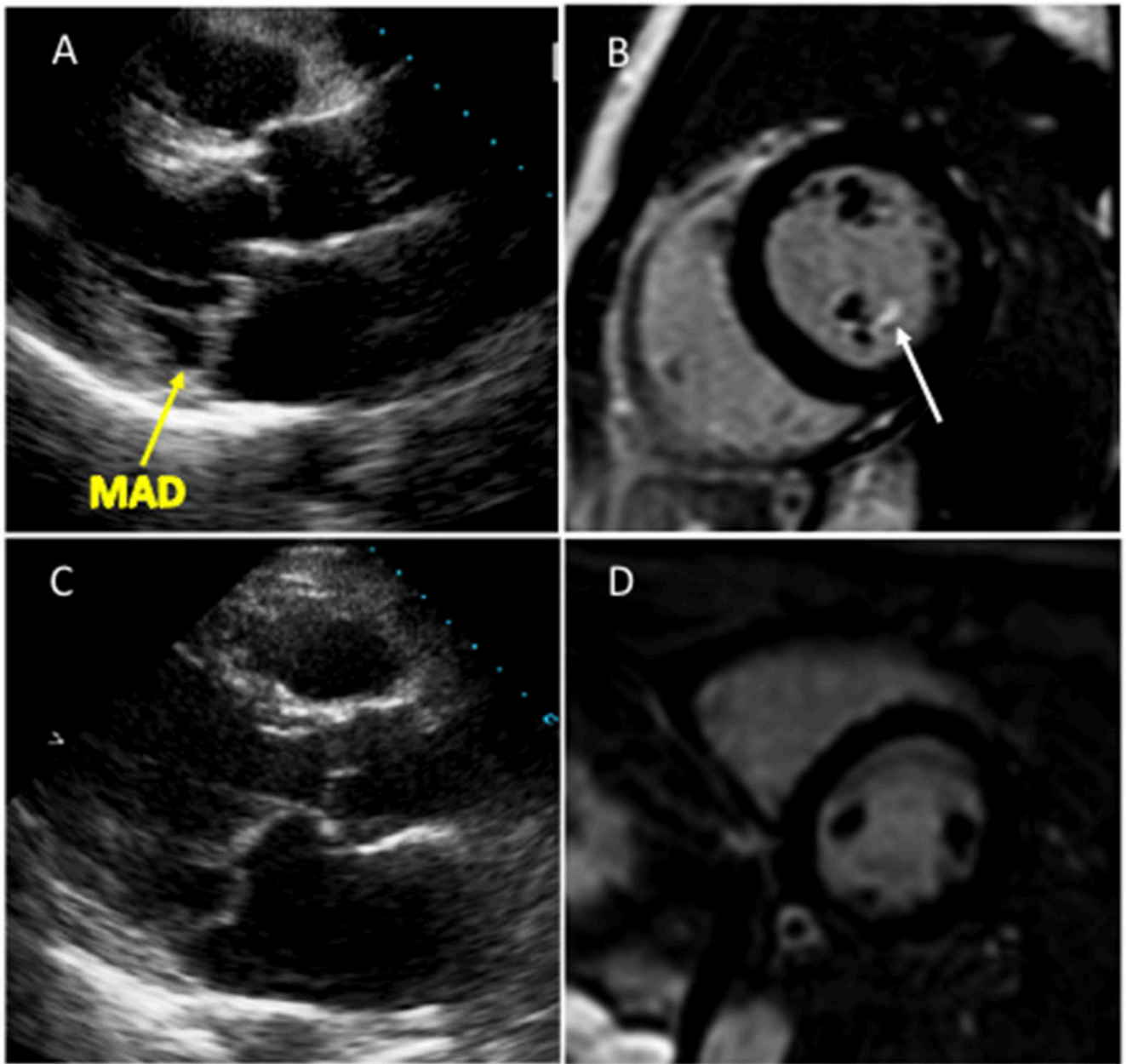
Fig. 2. Imaging of patients with mitral valve prolapse (MVP) undergoing ablation.
(a) Bileaflet MVP and mitral annular disjunction (MAD) (yellow arrow) shown on a 2-dimensional transthoracic echocardiogram. **(b)** In the same patient, cardiac magnetic resonance (CMR) short-axis view demonstrating late gadolinium enhancement (LGE) (white arrow) of the papillary muscles. **(c)** In a separate patient, posterior MVP shown on transthoracic echocardiogram **(d)** In the same patient as (c), CMR short-axis view with no evidence of LGE
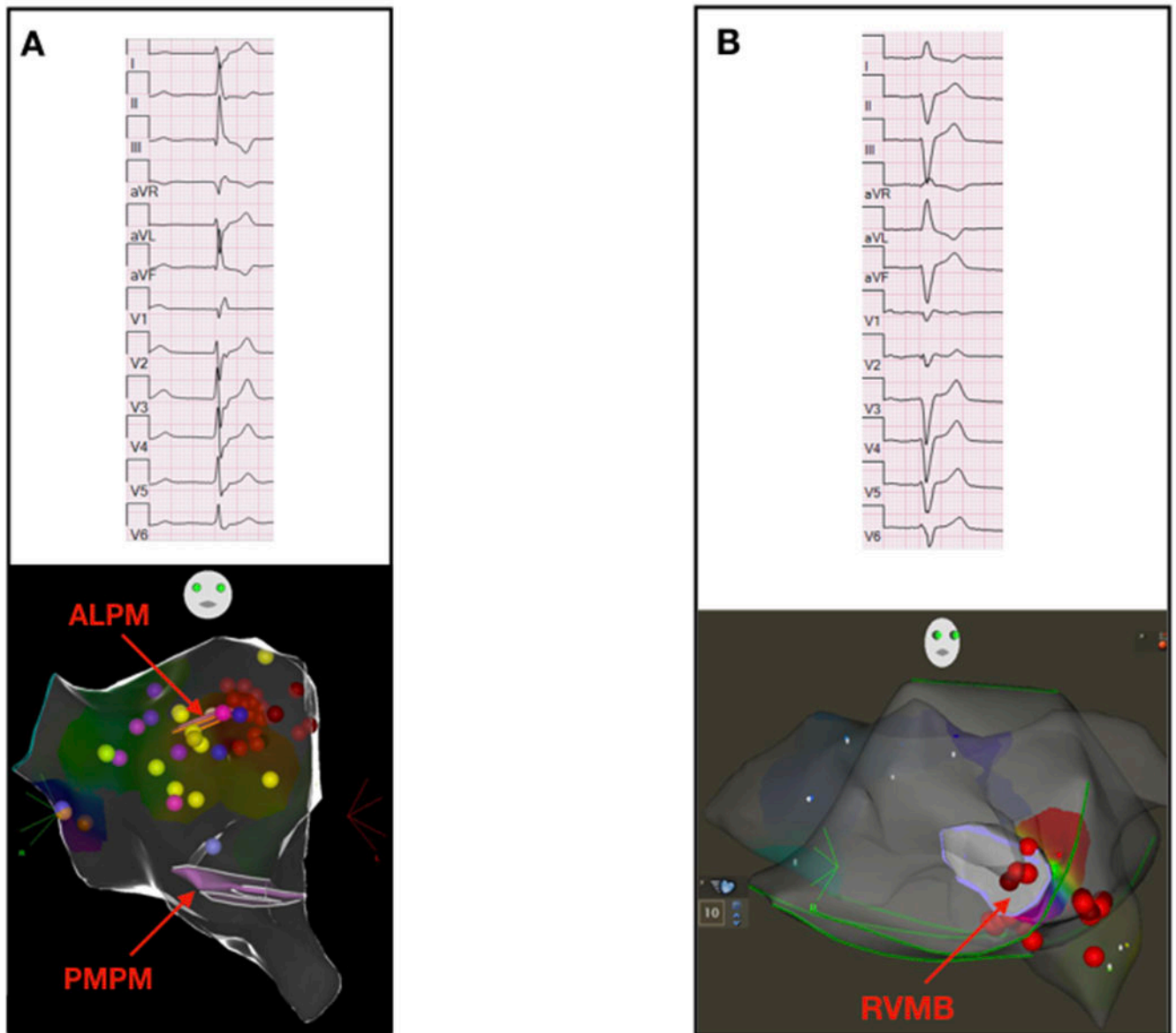
Fig. 3. Single patient example of complex ventricular ectopy with electroanatomical maps at index and repeat ablation.

(a) Right bundle, rightward inferior axis premature ventricular complex (PVC) originating from the anterolateral papillary muscle (ALPM). Electroanatomical map of the left ventricle shows successful ablation lesions (red tags) on the ALPM. PMPM = posteromedial PM. **(b)** Left bundle, leftward superior axis PVC originating from the right ventricular moderator band (RVMB). The map shows successful ablation lesions (red tags) on the RVMB.

Table 1.

Baseline demographic, clinical, electrocardiographic, and imaging characteristics.

		VT/VF after index ablation (n = 5)	No VT/VF after index ablation (n = 10)
,			
Demographics			
	Age at index ablation (years)	44 ± 16	53 ± 13
	Female sex	3	7
	White self-identified ethnicity	5	10
	BMI (kg/m^2)	23	24
Medical History			
	Diabetes	0	0
	Hypertension	1	3
	Atrial fibrillation or flutter	2	2
	Smoking history	2	3
	Drug use	0	0
	Family history of MVP	1	3
	Family history of SCD	1	0
Medication Use			
	Beta blocker	4	9
	Calcium channel blocker	1	2
	Anti-arrhythmic medication	5	6
	Flecainide	2	3
	Propafenone	1	2
	Sotalol	2	0
	Amiodarone	1	3
	Dofetilide	0	2
Surgical History			
	Mitral valve repair/replacement	2	2
ECG			
	Inverted or biphasic T waves	3	6
	QRS duration (msec)	99 ± 11	95 ± 14
	QTc (msec)	462 ± 42	435 ± 18
Ambulatory ECG			
	PVC burden (%)	17 ± 2	20 ± 12
	Multiple PVC morphologies	3	6
	Presence of bigeminy	5	10
	Presence of NSVT	5	5
Echocardiography			
	Prolapsing leaflet		

		VT/VF after index ablation (n = 5)	No VT/VF after index ablation (n = 10)
,			
	Anterior	0	0
	Posterior	2	4
	Bileaflet	3	5
	Mitral annular disjunction	3	7
	Degree of mitral regurgitation		
	Trace	1	3
	Mild	4	6
	Moderate	0	1
	Severe	0	0
	Chamber measurements		
	LV mass index (g/m^2)	80 ± 47	92 ± 17
	LVEDVI (ml/m^2)	69 ± 16	72 ± 17
	LVESVI (ml/m^2)	30 ± 7	31 ±11
	LA volume index (ml/m^2)	40 ± 9	41 ± 22
	LVEF (%)	55 ± 6	56 ± 8
	RV dilatation	0	0
	RV systolic dysfunction	0	0
CMR			
	CMR performed	4	6
	LGE	0	2

ICD = Implantable cardiac defibrillator; ECG = electrocardiogram; BMI = Body mass index; MVP = Mitral valve prolapse; SCD = sudden cardiac death; PVC = Premature ventricular contraction; NSVT = Non-sustained ventricular tachycardia; LV = left ventricular; LVEDVI = Left ventricular end-diastolic volume index; LVESVI = Left ventricular end-systolic volume index; LA = left atrial; LVEF = Left ventricular ejection fraction; RV = right ventricular; CMR = Cardiac magnetic resonance imaging; LGE = Late gadolinium enhancement. Categorical variables are expressed as number of patients. Continuous variables are expressed as mean ± SD. P values were all non-significant at > 0.05.

[*] Degree of mitral regurgitation by echocardiographic assessment within 1 year of index ablation when available, or within 1 year of repeat ablation if data time of index ablation not available.

Table 2.

Details of index ablation

	Subsequent VT/VF after index ablation (n = 5)	No subsequent VT/VF after index ablation (n = 10)	
Indication for index ablation			
PVC/NSVT + symptoms	4	10	
Sustained VT	1	0	
Index ablation details			
Sustained VT inducible prior to ablation	5	0	p = 0.0003
Multifocal origin of ComVE	5	4	p = 0.04
Number of ablations	20 ± 6	18 ± 10	
Mean power per ablation, W	38	32	
Ablation time, s	1058 ±309	867 ± 508	
VT inducible after ablation	2	0	
Sites of ComVE ablated at index ablation			
LV anterior/posterior papillary muscle	3	6	
LV anterior/posterior fascicle	0	1	
RVOT	1	2	
LVOT	0	3	
Basal anterolateral LV	0	2	
Basal inferoseptal LV	2	0	
Superolateral mitral annulus	1	1	
RV His	0	1	

[*] Categorical variables are expressed as number of patients. Continuous variables are expressed as mean ± SD. P values were all non-significant at > 0.05, unless shown. All data obtained from index ablation or from first ablation with available data.

ICD = Implantable cardiac defibrillator; PVC = Premature ventricular contraction; NSVT = Non-sustained ventricular tachycardia; VT = ventricular tachycardia; ComVE = complex ventricular ectopy, W = Watts, s = seconds, LV = left ventricular, RVOT = right ventricular outflow tract, LVOT = left ventricular outflow tract, RV = right ventricular

Table 3.

Details of repeat ablation and/or ICD placement.

Repeat Ablation	
No. of patients with repeat ablation	6
No. of total repeat ablation procedures	9
Following index ablation and prior to ICD	5
Following ICD	4
Range of no. of repeat ablations per patient	1–2
Median time (interquartile range) from index ablation to first repeat procedure (days)	499 (294 – 1,255)
Indication for Repeat Ablation	
PVC/NSVT + symptoms	6
Sustained VT	1
ICD therapy for VF	1
ICD therapy for VT	1
Site of ComVE at repeat ablation different than index ablation	6
Sites of ComVE ablated	
LV anterior/posterior papillary muscle	3
RVOT	1
LVOT	1
Basal inferolateral LV	2
Septal tricuspid papillary muscle	1
RV moderator band	1
ICD	
No. of patients with ICD	6
Indication for ICD	
Sustained VT with hypotension or syncope	2
VT arrest during ablation	1
VF arrest	1
Inducible VF after ablation	1
R on T PVCs and history of exertional pre-syncope	1
Subsequent ICD therapy	
Patients with ICD therapy for VT	1
Patients with ICD therapy for VF	2

No. = number, d = days, PVC = Premature ventricular contraction; NSVT = Non-sustained ventricular tachycardia; VT = ventricular tachycardia; VF = ventricular fibrillation; ComVE = complex ventricular ectopy, LV = left ventricular, RVOT = right ventricular outflow tract, LVOT = left ventricular outflow tract, RV = right ventricular; ICD = Implantable cardiac defibrillator. Categorical variables are expressed as number of patients. Continuous variables are expressed as mean ± SD. Four patients had both repeat ablation and ICD implantation.

Table 4.

Sites of initial and repeat ablations by patient. First ablation is denoted with green x, followed by second ablation with red x and third ablation with blue x.

	Anterior papillary muscle	Posterior Papillary muscle	Posterior Fascicle	RVOT	LVOT	Basal anterolateral LV	Basal inferoseptal LV	Basal inferolateral LV	Superolateral mitral annulus	RV his	Septal tricuspid papillary muscle	RV moderator band
Patient 1	x	x				x					x	x
Patient 2					xx							
Patient 3				xxx								
Patient 4		x										
Patient 5	x	x										
Patient 6	x	x	x									
Patient 7							x					
Patient 8						x		x	x			
Patient 9		x										
Patient 10										x		
Patient 11				x	x				x			
Patient 12							x					
Patient 13		x		x								
Patient 14	x											
Patient 15	x											

RVOT = right ventricular outflow tract; LVOT = left ventricular outflow tract; LV = left ventricle; RV = right ventricle