Epicardial access for ventricular tachycardia and premature ventricular complexes ablation: An institutional experience

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BACKGROUND Epicardial access for ventricular arrhythmia (VA) ablation is a challenging and relatively uncommon procedure during ventricular ablation.

OBJECTIVE This study aimed to assess the outcomes, predictors of success, and complications associated with pericardial access during these procedures.

METHODS This multicenter, retrospective, observational study included data collected over 20 years (2004–2024) from all Mayo Clinic sites performing VA ablation with epicardial access.

RESULTS A total of 265 patients were included in the analysis: 196 for VT ablation and 69 for PVC ablation. Among them, 184 (69%) had at least 1 previous VA ablation, 51 (19.2%) had ischemic cardiomyopathy, 53 (20%) had structurally normal hearts, and 164 (61.9%) had nonischemic cardiomyopathies (NICMs). Three presented with concomitant ischemic cardiomyopathy and NICM. Within the NICM group, the most common diagnoses were dilated cardiomyopathy (n = 80 [30.2%]), arrhythmogenic right ventricular cardiomyopathy (n = 34 [12.8%]), and sarcoidosis (n = 15 [5.7%]). Acute success, defined as noninducibility, was achieved in 100 (61.7%) of 162 patients tested, while partial success (clinical arrhythmia noninduciblility) was observed in 47 (29%). Before discharge, VT recurred in 20 patients (10.2%). During a median follow-up of 61 months, events were observed as follows: 60 (35.5%) patients died, 26 (13.3%) underwent heart transplantation, and 62 (31.6%) required a repeat ablation for VAs. The event-free survival rates were 50% (95% confidence interval 43%–58%) at 1 year.

CONCLUSION Successful VT ablation with epicardial access can be achieved in select cases, though event-free survival remains suboptimal. Advanced disease stage and persistent inducibility at the end of the procedure are predictors of poor outcomes.

KEYWORDS Ventricular tachycardia; Premature ventricular complexes; Epicardial mapping; Catheter ablation; Epicardial ablation

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Introduction

Sudden cardiac death is a notable public health concern, with data indicating an annual occurrence rate of 60 per 100,000 individuals in the United States alone.[1] Of this, a considerable proportion is associated with VAs like ventricular tachycardias (VTs) and premature ventricular complexes (PVCs).

There are several etiologies of VAs. The most common form of idiopathic VA is PVC, which accounts for ~90%.[2] Some cases of PVCs require detailed mapping that includes epicardial mapping to pinpoint safe sites for ablation.[3]

In most VTs of nonischemic origin, the arrhythmogenic substrate is most commonly in the epicardial, perivalvular, or intramural site and this poses a significant challenge and may lead to worse outcomes when relying solely on endocardial catheter ablation.[4]

Some studies prove that endo-epicardial ablation is better than just an endocardial ablation procedure in select patients, especially if they have had a previously failed endocardial ablation.[3]

Methods

Study design

This was an observational, multicenter, retrospective study aiming to document population characteristics, outcomes, and predictors for successful VA ablation of patients who underwent epicardial mapping. We reviewed patients who underwent ablation for VAs (PVCs and VTs) from November 2004 to April 2024 across the 3 centers of Mayo Clinic. Patients who were 18 years of age and older, had successful pericardial access, and underwent epicardial

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KEY FINDINGS

- Epicardial ventricular arrhythmias are becoming more prevalent due to increased recognition and improved prognosis in patients diagnosed with nonischemic dilated cardiomyopathy. Despite the potential benefits, epicardial ablation remains infrequently performed in many centers due to associated complications, challenges in electrogram interpretation, and the restrictive nature of ablation near critical structures.
- While successful epicardial ablation is feasible, the long-term outcomes for epicardial and intramural ventricular tachycardia remain suboptimal. Factors such as in-hospital ventricular tachycardia recurrence, left axis deviation indicative of a septal source, and the presence of diabetes are associated with poorer outcomes.
- Given these complexities, patients undergoing epicardial ablation may benefit from comanagement with advanced heart failure services.

mapping were included in the study. The first ablation in our institution achieving successful epicardial access and mapping was defined as the index procedure. Three investigators manually completed a full chart review for patients who met the inclusion criteria between May and July 2024. Baseline data, procedure characteristics, and outcomes were extracted and collected for further analysis. This study was reviewed and approved by the Institutional Review Board (IRB23-003361). Written informed consent was obtained from all patients before the intervention. The research reported in this paper adhered to Helsinki Declaration as revised in 2013.

Procedure characteristics

Epicardial mapping was conducted at the operators' discretion. Previous unsuccessful endocardial ablations, inability to document endocardial substrate during the procedure, or epicardial origin suggested by electrocardiographic or radiologic evaluation motivate most epicardial approaches. Patients underwent the procedure under conscious sedation or general anesthesia. In most cases, a subxiphoid approach with an anterior or inferior puncture was performed. Five patients underwent thoracotomy for pericardial access due to concomitant open surgery procedures or due to unfeasible pericardial access related to adhesions. One patient required a computed tomography–guided parasternal approach. Intracardiac echocardiography was used as an intraprocedural guide. A 3-dimensional electroanatomic map was created, and activation and/or entrainment mapping was used for patients with inducible and tolerated VT. Otherwise, pace and substrate mapping were performed. Coronary angiography and phrenic nerve mapping were performed before ablation in the case of proximity of any of these structures to the target tissue. Most patients underwent radiofrequency ablation, while cryoablation was employed for some patients via an open approach. All patients who were planned for epicardial access had a preprocedure coronary angiogram. For patients in whom the decision to gain epicardial access was made during the procedure, an interventional cardiologist was consulted to perform a coronary angiogram. All patients that developed postprocedure pain were treated with nonsteroidal anti-inflammatory drugs and colchicine. In patients with persistent pericardial effusion without hemodynamic compromise, a drain was left in place until echocardiographic follow-up.

Endpoints and follow-up

For patients who underwent VT ablation, long-term outcomes included VT recurrence after discharge (sustained VT or ventricular fibrillation documented by an implantable device or external electrocardiography after discharge), new VA ablation, heart transplantation, and mortality. Short-term outcomes included complete acute success (defined as the absence of any inducible VT at the end of the procedure), partial acute success (defined as the absence of inducible clinical VT at the end of the procedure), and mortality before discharge. The primary endpoint was survival free of recurrence, new VT ablation, or heart transplantation.

For patients who underwent PVC ablation, the primary endpoint was recurrence, which was defined as a $>5\%$ PVC burden during the 24-hour Holter recording. Acute success was defined as the absence of spontaneous PVCs and no inducibility with isoproterenol infusion at the end of the procedure.

Data from office visits, implantable cardioverter-defibrillator interrogations, and emergency room visits were reviewed. If none of the long-term outcomes occurred, the period from the procedure to the last office or emergency room visit was considered the follow-up period. For patients who presented any of the long-term outcomes, the follow-up time was defined as the time from the procedure to the event occurrence.

Statistical analysis

Continuous variables are presented as the mean $\pm$ SD and were compared with Student's t test. Categorical variables are presented as absolute and relative frequencies and were compared with the chi-square or Fisher exact test (if the frequency of an observation was <5 in the contingency table). Recurrence-free survival was evaluated through Kaplan-Meier analysis. Time-to-event endpoints comparing subgroups were analyzed with log-rank tests. The associations of potential variables with the composite outcome were tested with Cox regression analyses. For non–time-dependent events, logistic regression analyses were performed. After univariate analysis, variables with a P value $<.1$ were included in the multivariate model. Statistical significance was established with a P value of $<.05$. Statistical analyses were performed with R statistical software (version 4.2.3; R Foundation for Statistical Computing).

Table 1 Baseline characteristics

Variable	Total (N = 265)	VT (n = 196)	PVC (n = 69)	*P*
Age, y	54.1 ± 15.2	55.6 ± 15.2	50 ± 14.5	.01
Male	201 (75.8)	159 (81.1)	42 (60.9)	<.01
Obesity	115 (43.4)	86 (43.9)	29 (42)	.79
BMI, kg/m^2	29.9 ± 6.6	29.9 ± 6.6	29.9 ± 6.6	.99
Smoking status				
Never	186 (70.7)	138 (70.4)	48 (71.6)	.85
Former	61 (23.2)	46 (23.5)	15 (22.4)	.86
Current	16 (6.1)	12 (6.1)	4 (6)	.96
Diabetes	43 (16.2)	37 (18.9)	6 (8.7)	.05
CKD	54 (20.4)	47 (24)	7 (10.1)	.01
Hypertension	109 (41.1)	89 (45)	20 (29)	.02
CAD	77 (29.1)	63 (32.1)	14 (20.3)	.06
ICD	178 (67.2)	165 (84.2)	13 (18.8)	<.01
Concomitant ICM and NICM	3 (1.1)	3 (1.5)	0	—
Structurally normal heart	53 (20)	20 (10.2)	33 (47.8)	<.01
Only ICM	48 (18.1)	41 (20.9)	7 (10.1)	.05
Only NICM	161 (60.8)	132 (67.4)	29 (42)	<.01
All ICM	51 (19.3)	44 (22.5)	7 (10.1)	.03
All NICM	164 (61.9)	135 (68.9)	29 (42)	<.01
Sarcoidosis	15 (5.7)	12 (6.1)	3 (4.4)	.77
ARVC	34 (12.8)	30 (15.3)	4 (5.8)	.06
Amyloid	1 (0.4)	1 (0.5)	0	-
Hypertrophic	9 (3.4)	8 (4.1)	1 (1.5)	.45
Dilated cardiomyopathy	80 (30.2)	68 (34.7)	12 (17.4)	<.01
Previous myocarditis	6 (2.3)	4 (2)	2 (2.9)	.65
Other/idiopathic	19 (7.2)	12 (6.1)	7 (10.1)	.27
CHF	151 (57)	129 (65.8)	22 (31.9)	<.1
TICM	11 (4.2)	2 (1)	9 (13)	<.01
Previous cardiac arrest	45 (17)	42 (21.4)	3 (4.4)	<.01
Previous thoracotomy	13 (4.9)	11 (5.6)	2 (2.9)	.52
Previous ablation	184 (69.4)	135 (68.9)	49 (71)	.74
Number of previous ablations	1.6 ± 0.9	1.5 ± 0.8	1.7 ± 1.1	.33
Previous epicardial ablation	13 (4.9)	11 (5.6)	2 (2.9)	.52
LVEF, %*	44.4 ± 15.0	41.3 ± 15.1	53 ± 10.5	<.01
Normal (≥50%)	114 (43.9)	63 (33)	51 (73.9)	<.01
Mildly reduced (41%–49%)	38 (14.6)	30 (15.7)	8 (11.6)	.41
Reduced (≤40%)	108 (41.5)	98 (51.3)	10 (14.5)	<.01
LVIDd, mm†	58.3 ± 9.0	59.4 ± 9.4	55.2 ± 7.2	<.01
LVPWd, mm‡	9.7 ± 1.7	9.8 ± 1.7	9.5 ± 1.5	.16
IVDs, mm§	10.4 ± 2.1	10.6 ± 2.2	9.8 ± 1.6	<.01
Antiarrhythmics at baseline	194 (73.2)	163 (83.2)	31 (44.9)	<.01
Amiodarone	117 (44.2)	109 (55.6)	8 (11.6)	<.01
Mexiletine	64 (24.2)	59 (30.1)	5 (7.3)	<.01
Sotalol	44 (16.6)	37 (18.9)	7 (10.1)	.09
Quinidine	3 (1.1)	2 (1)	1 (1.5)	1
Procainamide	1 (0.4)	1 (0.5)	0	—
Propafenone	2 (0.8)	2 (1)	0	—
Dofetilide	2 (0.8)	0	2 (2.9)	—
Calcium-channel blockers	7 (2.6)	5 (2.6)	2 (2.9)	1
Flecainide	15 (5.7)	8 (4.1)	7 (10.1)	.06
Number of antiarrhythmics	1.3 ± 0.5	1.4 ± 0.5	1.0 ± 0.2	<.01
Beta-blockers at baseline	180 (67.9)	149 (76)	31 (44.9)	<.01
Anticoagulation at baseline	74 (27.9)	64 (32.7)	10 (14.5)	<.01

Values are mean ± SD or n (%).

ARVC = arrhythmogenic right ventricular cardiomyopathy; BMI = body mass index; CAD = coronary artery disease; CHF = chronic heart failure; CKD = chronic kidney disease; ICD = implantable cardioverter-defibrillator; ICM = ischemic cardiomyopathy; IVDs = interventricular septal thickness in diastole; LVEF = left ventricular ejection fraction; LVIDd = left ventricular internal end-diastolic diameter; LVPWd = left ventricular posterior wall thickness in diastole; NICM = nonischemic cardiomyopathy; PVC = premature ventricular complex; TICM = tachycardia-induced cardiomyopathy; VT = ventricular tachycardia.

*260 patients with available data.

†247 patients with available data.

‡236 patients with available data.

§237 patients with available data.

Table 2 Procedure characteristics

Variable	Total	VT	PVC	*P* value
Sample	265 (100)	196 (74)	69 (26)	
VT ablation	196 (74)	—	—	
ICD shocks	118 (44.5)	—	—	
VT refractory to antiarrhythmics and/or highly symptomatic	50 (18.9)	—	—	
VT storm	18 (6.8)	—	—	
Other	10 (3.8)	—	—	
General anesthesia	148 (55.9)	122 (62.24)	26 (37.9)	<.01
Conscious sedation	117 (44.2)	74 (63.2)	43 (36.8)	<.01
RFA	257 (97)	189 (96.4)	68 (98.5)	.37
Cryoablation	12 (4.5)	10 (0.05)	2 (0.03)	.45
Alcohol ablation	1 (0.4)	0	1 (0.4)	
Procedure time, min	398 ± 129	411 ± 132	358 ± 112	<.01
Ablation time, min	156 ± 101	158 ± 101	152 ± 100	.68
Energy delivery time, s	2518 ± 1595	2818 ± 1656	1598 ± 917	<.001
Fluoroscopy time, min	67 ± 35	67.8 ± 36.6	65.2 ± 28.3	.55
Ablations delivered	29 ± 23	33.5 ± 24	18.4 ± 11.9	<.001
Epicardial ablation	224 (84.5)	169 (86.2)	55 (79.7)	.2
Endocardial ablation	229 (86.4)	167 (85.2)	62 (90)	.33
Epi-endocardial ablation	190 (71.7)	142 (72.4)	48 (69.6)	.65
No ablation	2 (0.8)	2	0	
Subxiphoid access	259 (97.7)	191	68	
Thoracotomy access	5 (1.9)	4	1	
CT-guided parasternal access	1 (0.4)	1	0	
VT ablation–specific characteristics	n = 196	—	—	
Number of induced clinical VTs	0.86 ± 0.78	—	—	
Number of induced VTs	2.35 ± 1.87	—	—	
Clinical VT induced	138 (70.4)	—	—	
Clinical VT with RBBB morphology	82 (41.8)	—	—	
Induced clinical VT with LBBB morphology	50 (25.5)	—	—	
Induced clinical VTs with both LBBB and RBBB	6 (3.1)	—	—	
Induced clinical VT mean CL, ms	415 ± 106	—	—	

Values are n (%), mean ± SD, or n.

CL = cycle length; CT = computed tomography; LBBB = left bundle branch block; ICD = implantable cardioverter-defibrillator; PVC = premature ventricular complex; RBBB = right bundle branch block; RFA = radiofrequency ablation; VT = ventricular tachycardia.

Results

Baseline characteristics

The patients' baseline characteristics are summarized in Table 1. A total of 265 patients were included for review. The mean follow-up period was 31.5 ± 45.5 months. The mean age was 54.1 years, and 75.8% were males. The mean left ventricular ejection fraction (LVEF) was 44.4 ± 15%. Ischemic cardiomyopathy (ICM) was present in 51 (19.2%) patients; 53 (20%) patients had structurally normal hearts and 164 (61.9%) had nonischemic cardiomyopathies (NICMs). Three patients had concomitant ICM and NICM. Among patients with NICM, dilated (n = 80 [30.2%]), arrhythmogenic right ventricular cardiomyopathy (n = 34 [12.8%]), and sarcoidosis (n = 15 [5.7%]) were the most common diagnoses. Nineteen (7.2%) patients presented with idiopathic cardiomyopathy.

A history of previous ablation for VA was present in 184 (69.4%) patients, with a mean of 1.6 ablations among this population. Thirteen (4.9%) patients had a history of previous epicardial ablation for VA at other institutions.

With respect to medical therapy before ablation, 180 (67.9%) patients were taking beta-blockers and 194 (73.2%) were receiving antiarrhythmic drugs (AADs), with a mean number of AADs of 1.3 ± 0.5. Amiodarone, mexiletine, and sotalol were the most commonly used drugs, with prescription rates of 44.2%, 24.2%, and 16.6%, respectively.

Procedure characteristics

The procedure characteristics are summarized in Table 2. Frequent and symptomatic PVCs without sustained VT were the indications for the index procedure in 69 (26.0%) of the patients. Sustained VT was present in 196 (74.0%) of the patients, and implantable cardioverter-defibrillator shocks were the most common indication for ablation (n = 118 [44.5%]), followed by VT refractory to medical therapy without shocks and/or highly symptomatic arrhythmia (n = 50 [18.9%]) and VT storm (n = 18 [6.8%]). Ten patients had other indications, such as intolerance to AADs or primary prevention of device shocks.

General anesthesia was employed in 55.8% of the patients at any point during the procedure. In general, patients who underwent conscious sedation instead of general anesthesia were younger (mean age 51.76 vs 55.99 years, $P = .02$), had healthier hearts with better LVEF (48.71% vs 40.97%, $P \leq .01$), and had structurally normal hearts (35% vs 8%, $P \leq .01$). A detailed description of the baseline and procedural differences between patients who underwent general anesthesia and conscious sedation is shown in Supplemental Table 1. In the overall cohort, the mean procedure time was 398 ± 129 minutes, the mean fluoroscopy time was 67 ± 35 minutes, and the mean number of energy deliveries was 29 ± 23. Epicardial ablation was performed in 221 (83.4%) patients and combined epi-endocardial ablation was performed in 189 (71.3%) patients. In 6 patients, epicardial ablation was not pursued due to proximity to the coronary arteries/phrenic nerve. Of the 13 patients who underwent previous epicardial ablation, 2 patients had significant adhesions, and in 1 patient adhesions could not be lysed preventing access to the best pace-match site.

Among the 196 patients who underwent VT ablation, 138 (70.4%) had at least 1 clinical VT induced during the procedure. Among those patients, 82 (59.4%) had left bundle branch block (LBBB) clinical VT induced, 50 (36.2%) had right bundle branch block clinical VT induced, and 6 (4.3%) had both LBBB and right bundle branch block clinical VT morphology. In the majority of the patients with ICM, the location of the abnormal substrate was found to be in the septal (n = 25 [49%]), inferoseptal (n = 11 [25%]), and inferolateral (n = 17 [33%]) regions.

Complications

Table 3 summarizes the complications encountered during and after the procedure. A total of 69 (26%) patients developed postablation pericarditis. A total of 45 (17%) patients from the study experienced complications due to the procedure. Minor complications were seen in 22 (8.3%) patients, of which pericardial effusion was the most common minor complication (14 patients). Cardiac tamponade was the most common major complication seen in 18 (8.7%) of the 23 patients who experienced major complications due to the procedure. Non–procedure-related complications were seen in 18 (6.8%) patients. Three patients died during the hospitalization period following the procedure due to either acute heart failure or recurrent VT, while 3 patients required sternotomy due to procedure-related complications.

VT outcomes

The outcomes of the 196 patients who underwent VT ablation are summarized in Table 4. Postablation testing was performed in 162 (82.7%) of the patients. Acute success was achieved in 100 (61.7%) patients and partial success in 47 (29%) patients. Before discharge, 20 (10.2%) patients presented VT recurrence. In 65 (33.2%) patients, AAD regimens were reduced. During follow-up, 60 (35.5%) patients died, 26 (13.3%) had heart transplantation, and 62 (31.6%) required a new ablation for VA. The results of the Kaplan-Meier survival analysis for event-free survival and overall survival are presented in Figure 1. Event-free survival rates were 50% (95% confidence interval [CI] 43%–58%) at 1 year. At 96 months, 19% (95% CI 13%–27%) of the patients at risk survived free from the combined outcome, and 63% (95% CI 55%–72%) were still alive.

Univariate Cox regression analysis (Supplemental Table 2) revealed significant association between event-free survival and VT storm at presentation, dilated cardiomyopathy, structurally normal heart, LVEF, left ventricular internal end-diastolic diameter, number of induced VTs, ablation in the left ventricular (LV) anterior segments, acute success in postprocedure tested patients, recurrence before discharge, and postprocedure initiation of sotalol, amiodarone, and mexiletine. After multivariate analysis, clinical VT with left axis deviation (hazard ratio [HR] 4.13, 95% CI 2.16–7.9, P

Table 3 Complications

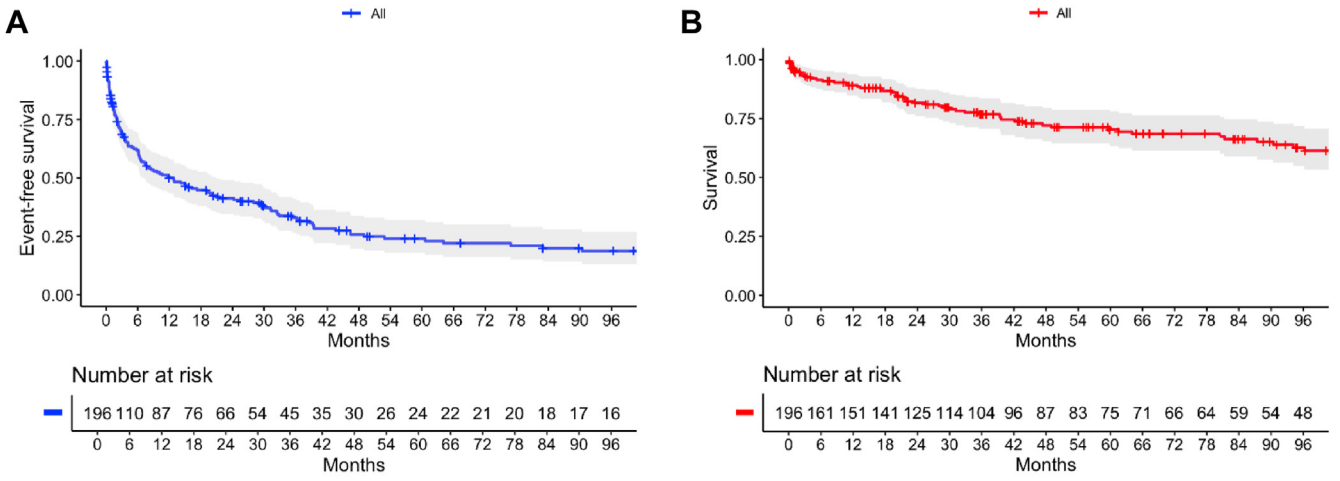
Type of complication	Events
Postablation pericarditis	69/265 (26%)
Procedure-related complications	45 (17%)
Minor	22 (8.3%)
Pericardial effusion	14
Pleural effusion	3
Phrenic nerve Injury	3
Femoral pseudoaneurysm	1
Contained abdominal hematoma	1
Major	23 (8.7%)
Cardiac tamponade	18
Major bleeding	3
Coronary artery injury	1
Severe limb ischemia	1
Non–procedure-related complications	18 (6.8%)

Values are n/n (%), n (%), or n.

Table 4 Outcomes

Variable	Events
PVC ablation	n = 69
Acute success	47 (68)
Intrahospital mortality	1 (1.5)
Antiarrhythmic drug reduction	19 (28)
Mortality	4 (6)
Repeat ablation	12 (17)
VT ablation	n = 196
Postablation testing performed	162 (82.7)
Complete acute success	100 (61.7)
Partial acute success	47 (29)
Unsuccessful procedure	15 (9.2)
No postablation resting	34 (17.4)
Intrahospital recurrence	20 (10)
Intrahospital mortality	3 (1.5)
Antiarrhythmic drug reduction	65 (33)
Mortality	60 (31)
Heart transplantation	26 (13)
Repeat ablation	62 (32)

Values are n (%).
PVC = premature ventricular complex; VT = ventricular tachycardia.



Event-free survival			Survival		
Time	Survival	95% CI	Time	Survival	95% CI
12	50%	43%-58%	12	89%	85%-94%
24	41%	35%-49%	24	82%	76%-88%
36	33%	27%-41%	36	77%	71%-84%
48	26%	20%-34%	48	72%	65%-80%
60	24%	18%-32%	60	70%	63%-78%
72	22%	16%-30%	72	69%	61%-77%
84	20%	14%-28%	84	66%	59%-75%
96	19%	13%-27%	96	63%	55%-72%

Figure 1 Ventricular tachycardia ablation survival analysis: Kaplan-Meier curves illustrating (A) event-free survival (blue line) and (B) overall survival (red line) for patients who underwent ventricular tachycardia ablation. CI = confidence interval.

$\leq$ .01) and VT recurrence before discharge (HR 3.21, 95% CI 1.5–6.88, $P \leq .01$) were associated with lower event-free survival. Postprocedural sotalol (HR 0.27; 95% CI 0.12–0.6, $P \leq .01$) was associated with higher event-free survival rates. For acute complete success, coronary artery disease was the only significant predictive independent variable (Supplemental Table 3). Epicardial, epi-endocardial, and endocardial ablation did not present significant different event-free survival rates ($P = .37$) (Figure 2).

PVC outcome

Acute success for PVC ablation was achieved in 47 (68%) patients; 1 patient died before being discharged because of a non–procedure-related complication. After the procedure, 19 (28%) patients had a reduction of AADs, and during follow-up 4 (6%) patients died and 12 underwent a new ablation (Table 4). Event-free survival rates were 76% (95% CI 66%–87%) at 1 year, 70% (95% CI 59%–83%) at 2 years, 64% (95% CI 52%–78%) at 4 years, and 59% (95% CI 47%–74%) at 8 years following the procedure (Figure 3).

Patients who underwent epicardial ablation (alone or combined with endocardial) presented lower event-free survival than patients with endocardial ablation alone ($P = .039$) (Figure 4). However, the type of ablation was not a significant independent predictor in the multivariate Cox analysis (Supplemental Table 4). For acute success, there were no significant independent predictors (Supplemental Table 5).

Discussion

The main findings of our study in patients who undergo epicardial access and mapping are the following: (1) the event-free survival rates in patients who undergo epicardial access were suboptimal; (B) postprocedure noninducibility of clinical VT was associated with better long-term survival outcomes; and (3) intrahospital recurrence, diabetes mellitus, and LAD on clinical VT were significant predictors for mortality.

Indications

In our retrospective analysis of 265 patients who underwent epicardial access ablation, we found that the majority had

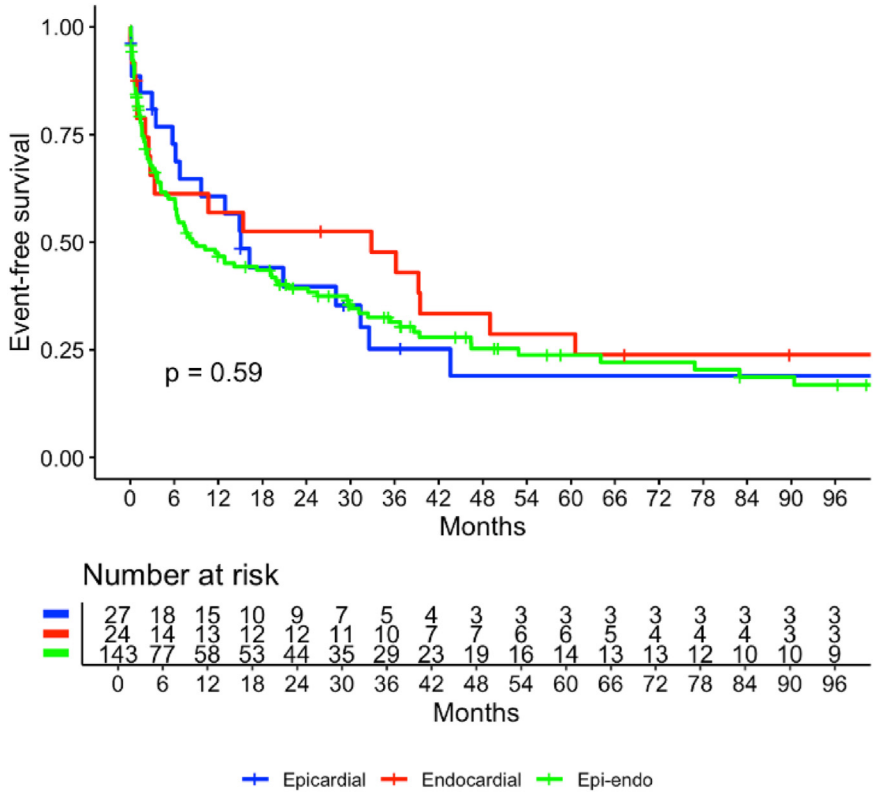


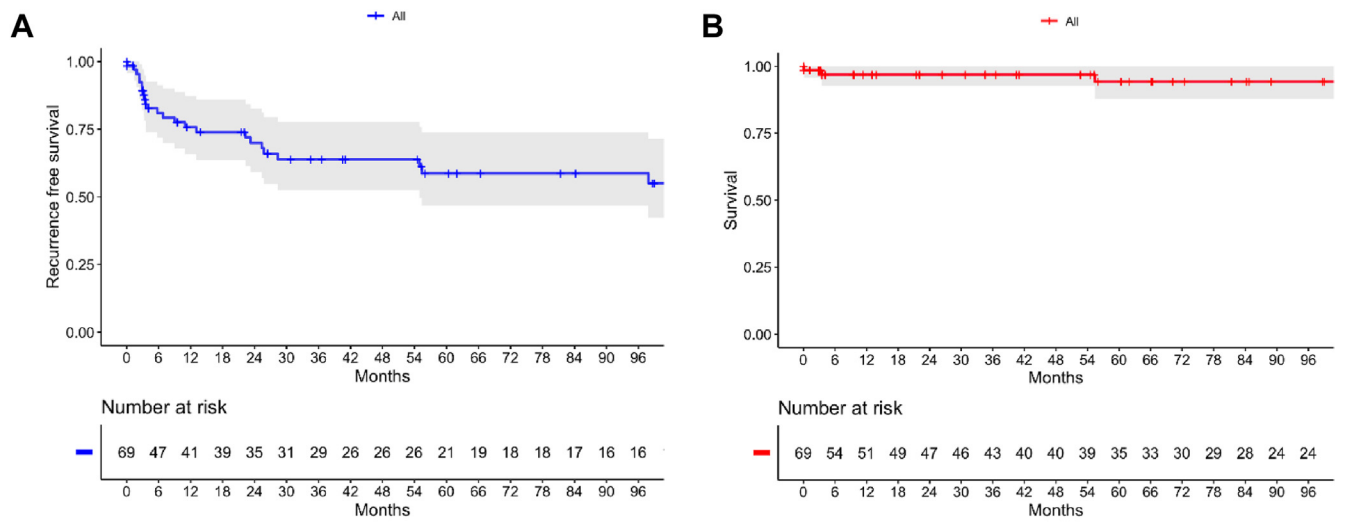
Figure 2 Kaplan-Meier curves illustrating event-free survival after ventricular tachycardia ablation for patients who underwent epicardial ablation (blue line), endocardial ablation (green line), and combined epi-endocardial ablation (green line). *P* value for log-rank test.

previously undergone unsuccessful ablations for VT. Additionally, those who had epicardial ablation as their index procedure were found to have an epicardial focus on prior investigations. Most patients who underwent epicardial access had NICM (61.9%), with dilated cardiomyopathy (30%) being the most common type. This may be because, in NICMs, the substrates tend to be heterogeneous, characterized by patchy interstitial fibrosis with epicardial and/or intramural distribution,[5] which often necessitates an epicardial approach to isolate/identify the extent of the substrate. Identification of an epicardial focus can be done by various methods, including imaging such as Intracardiac echocardiography, computed tomography, or magnetic resonance imaging to locate the scar region. Bogun and colleagues[6] demonstrated that delayed-enhanced magnetic resonance imaging can effectively identify VT substrate in NICM to guide ablation. Similarly, intracardiac echocardiography can be used to identify areas of increased echogenicity during the VT ablation to identify epicardial substrate in NICM patients.[7] Electrocardiographic criteria are not always reliable in diagnosing epicardial VT, as they are often highly substrate and region specific.[8] In cases in which the location of the VT substrate is unclear, it might be beneficial to conduct an epicardial mapping to better identify the focus. In our cohort, 39 patients required endocardial ablation alone. This highlights the need for improved strategies to identify epicardial substrates and minimize unnecessary epicardial access in such patients.

Acute outcomes

In patients undergoing VT ablation, 91% had no inducible clinical VT (92% in ICM and 88% in NICM) at the end of the procedure among the patients who were tested, and there were no significant differences between patients with ICM and NICM. This is slightly higher than what Darma and colleagues[9] reported in a study with similar number of patients.

Major complications occurred in 14 patients, with ventricular perforation and cardiac tamponade being the most common. Of these, 3 patients experienced severe complications necessitating emergency sternotomy due to right coronary artery injury, LV perforation, and double right ventricular perforation. Additionally, 3 other patients developed hemoperitoneum, hemothorax, and abdominal hematoma because of vessel perforation. This is consistent with other studies, which showed major complication rates of 5% to 10%.[10] Of note, the incidence of epicardial vessel injury was extremely rare in our cohort. This is likely due to our collaboration with interventional cardiology, who performed coronary angiograms to clarify the location of the coronary arteries when the VT focus was suspected to be nearby. Most complications associated with obtaining epicardial



Recurrence-free survival			Survival		
Time	Survival	95% CI	Time	Survival	95% CI
12	76%	66%-87%	12	97%	93%-100%
24	70%	59%-83%	24	97%	93%-100%
36	64%	52%-78%	36	97%	93%-100%
48	64%	52%-78%	48	97%	93%-100%
60	59%	47%-74%	60	94%	88%-100%
72	59%	47%-74%	72	94%	88%-100%
84	59%	47%-74%	84	94%	88%-100%
96	59%	47%-74%	96	94%	88%-100%

Figure 3 Kaplan-Meier curves illustrating (A) recurrence-free survival (blue line) and (B) overall survival (red line) for patients who underwent premature ventricular complex ablation. CI = confidence interval.

access, for instance, right ventricular perforation or damage to the coronaries can be minimized by using novel techniques like CO_2 insufflation through the coronary veins.[11] A potential factor that increases the difficulty in obtaining epicardial access is the presence of adhesions due to prior cardiac surgeries or previous pericarditis.

In our cohort, those who underwent only endocardial PVC ablation after pericardial access had a better event-free survival rate than those who underwent epicardial ablation. One possible explanation for this is that the latter group had substrates that were difficult to identify with endocardial mapping alone, necessitating epicardial mapping.

Out of the 3 intrahospital mortalities, 1 was associated with acute heart failure and the other 2 with recurrent VT.

Long-term outcomes

Patients in our cohort had a mean follow-up of 31.5 months, the longest follow-up period in any American study involving catheter ablation with epicardial mapping.

For patients undergoing VT ablation, the event-free survival rate at 12 months was 50%. Because our study's primary endpoint included survival free from VT recurrence, new ablation procedures, or heart transplantation, direct comparisons to other large studies that report only VT recurrence-free survival are limited. However, our study demonstrates an event-free survival rate consistent with the other available literature for long-term outcomes after VT ablation.[12] A meta-analysis done on 22 studies including 1138 patients demonstrated that epi-endocardial mapping and ablation provided superior VT recurrence–free survival compared with endocardial-only strategy (odds ratio 0.52, 95% CI 0.39–0.71, $P < .01$) in select patient populations. Further subgroup analysis found that this effect was more pronounced in ICM (odds ratio 0.39, 95% CI 0.18–0.83, $P = .01$).[13]

The overall survival rate in patients undergoing VT ablation with epicardial access was 89% at 12 months, 82% at 24 months, and 63% at 96 months. Based on the available literature, while event rates may differ between different ablation

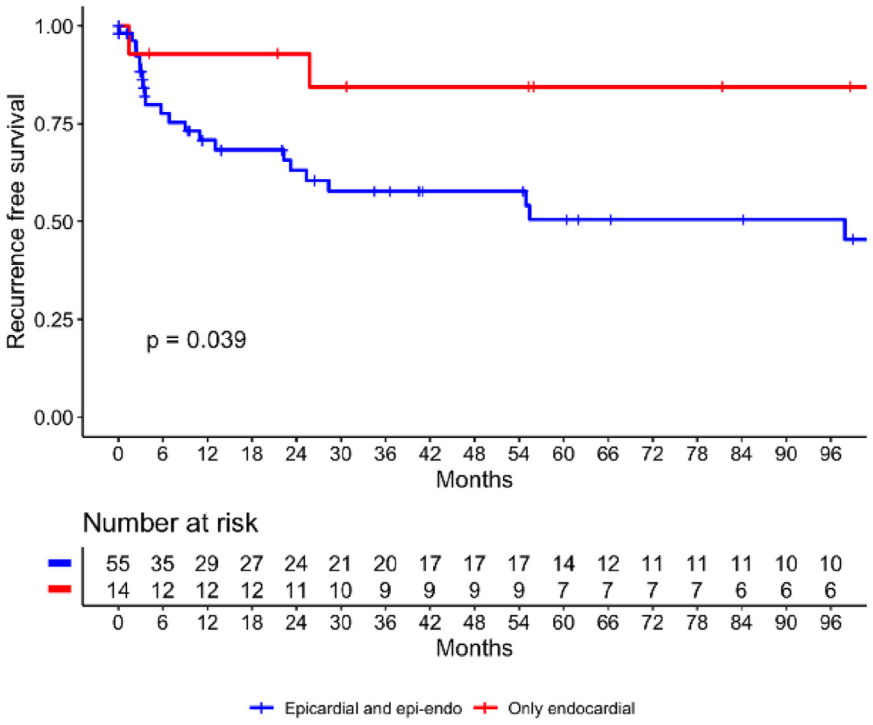


Figure 4 Kaplan-Meier curves illustrating recurrence-free survival after premature ventricular complex ablation for patients who underwent combined epi-endocardial or only epicardial ablation (blue line) and only endocardial ablation (red line). *P* value for log-rank test.

approaches, overall patient survival remains consistent among patients undergoing VT ablation.[14–17]

In our study, the follow-up at 24 months revealed an event-free survival rate of 70% and an overall survival rate of 97% in patients undergoing PVC ablation. Although PVCs may recur in some patients after a successful procedure, they do not significantly impact overall patient survival. Our findings are comparable to the existing literature,[18] regardless of epicardial access.

Predictors of outcomes

In our multivariable analysis of patients who underwent VT ablation, Intrahospital recurrence was associated with an increased risk of presenting the combined outcome (HR 3.18, $P \leq .01$). Interestingly, in our cohort, patients who experienced an intrahospital recurrence had a mean of 3.25 induced VTs, as compared with 2.3 in the entire cohort. This may indicate a more severely ill population with diseased myocardium, partly explaining the poorer survival outcomes.

In our univariable analysis, postprocedure complete success was associated with better chances of event-free survival (HR 0.66, $P = .03$). This aligns with literature showing that noninducibility after VT ablation predicts better outcomes for both ICM and NICM regarding VT recurrence and mortality.[19] Patients who had a successful outcome likely had relatively healthier hearts with less arrhythmogenic substrate. This could have facilitated better recovery of LV function, leading to improved long-term, event-free survival.

Diabetes mellitus was associated with an increased risk of mortality (HR 3.05, $P < .01$) in our patient population, potentially due to elevated epicardial adipose tissue volume and inflammatory changes linked to diabetes.[20,21] Studies have shown that epicardial adipose tissue secretes proinflammatory factors that impair cardiomyocyte function and reduce SERCA,[21,22] which may predict poorer outcomes in diabetic patients undergoing epicardial ablation.

Our analysis identified LAD in clinical VT induced during the procedure as a significant predictor of increased event occurrence (HR 4.13, $P < .01$). This finding is likely due to LAD indicating a septal substrate, a region notoriously challenging to ablate. These results warrant further validation through prospective studies or larger sample sizes.

Limitations

This study's cohort comprised a highly selective group of patients with diverse diagnoses and underlying substrates, most of whom had undergone prior unsuccessful ablations, with substrates predominantly identified as epicardial. As a result, these findings may not be generalizable to a broader population. Furthermore, the retrospective nature of the study constrains our ability to compare success rates between endocardial ablation alone and those requiring epicardial access. Additionally, our data were derived from centers with extensive experience in obtaining epicardial access, which may result in complication rates that differ from those at less experienced centers.

Conclusion

This study highlights the complexities and outcomes of epicardial mapping and ablation for ventricular arrhythmias, demonstrating that while the approach is critical for addressing challenging arrhythmogenic substrates, particularly in nonischemic cardiomyopathies, event-free survival rates remain suboptimal. Key predictors of poor outcomes, such as intrahospital recurrence, diabetes mellitus, and left axis deviation in clinical VT, emphasize the importance of patient selection and procedural precision. Postprocedure noninducibility was associated with improved long-term survival, underscoring the significance of thorough mapping and ablation techniques. Despite its challenges, epicardial mapping plays a vital role in treating patients with refractory arrhythmias, and collaborative, multidisciplinary approaches, along with advancements in procedural strategies, are essential for reducing risks and improving outcomes. Future prospective studies are needed to refine techniques, optimize patient selection, and enhance overall care for this high-risk population.

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Ethics Statement: This study was reviewed and approved by the Institutional Review Board (IRB23-003361). The research reported in this paper adhered to Helsinki Declaration as revised in 2013.

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