

Surgical Endoepicardial Linear Ablation

for Ventricular Tachycardia With
Postinfarction Left Ventricular Aneurysm

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Key words: Tachycardia, ventricular/surgery; heart aneurysm; epicardial mapping; radiofrequency ablation

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This retrospective study evaluated the feasibility of surgical endoepicardial linear ablation for ventricular tachycardia in patients with postinfarction left ventricular aneurysm.

Sixty-four patients with multivessel coronary artery disease and left ventricular aneurysm but no mural thrombosis of the aneurysm or valve disease were treated at our institution from March 2012 through July 2015. All underwent off-pump coronary artery bypass grafting and left ventricular aneurysm repair by linear plication. Twenty-three patients (35.9%) had ventricular tachycardia and underwent surgical endoepicardial linear ablation on the beating heart guided by epicardial substrate mapping with the Carto 3 system. The remaining 41 patients (64.1%) composed the no-ablation group. The effectiveness of surgical linear ablation in the ablation group was evaluated. Safety and clinical outcomes were evaluated and compared between the groups.

The ventricular tachycardia recurrence rate in the ablation group was 17.4% in the immediate postoperative period and 23.8% at last follow-up (39 ± 21 mo). Early (<30-d) mortality rates were 8.7% in the ablation group and 4.9% in the no-ablation group ($P=0.41$); the respective late mortality rates were 19.1% and 18% ($P=0.70$). Multivariate Cox regression analysis indicated that preoperatively poor left ventricular function was an independent risk factor for early and late death in both groups. The groups were similar in terms of the need for postoperative mechanical circulatory support, intensive care unit stay, and cumulative survival rate.

We conclude that, for carefully selected candidates, surgical endoepicardial linear ablation combined with off-pump coronary artery bypass grafting and left ventricular aneurysm linear plication is a feasible treatment for ventricular tachycardia with postinfarction left ventricular aneurysm. (***Tex Heart Inst J* 2020;47(3):194-201**)

Ventricular tachycardia (VT) is a major cause of reduced quality of life and sudden cardiac death in patients with left ventricular aneurysm (LVA) secondary to acute transmural myocardial infarction.^{1,2} An LVA can become an arrhythmogenic substrate, and the substrate for LVA-related VT is usually located in the aneurysm's border zone.³

Scar-related VT is usually controlled by catheter ablation. However, catheter ablation for LVA-related VT can be challenging. The LVA may consist almost entirely of hyaline fibrotic scar tissue; the associated VT may be unstable, noninducible, or multifocal.^{4,5} In this circumstance, surgical ablation is a potential therapeutic option. For decades, the main surgical treatment for LVA-related VT has been subendocardial resection and cryoablation combined with LVA repair and myocardial revascularization.^{6,7} However, extensive myocardial incision and cryoablation may cause additional myocardial damage, which may impair cardiac function and increase operative risk. Associated perioperative mortality rates are high, ranging from 5% to 30%.⁸⁻¹¹

To minimize risks and maximize benefits, the optimal surgical technique provides a compromise between modifying the VT substrate and preserving cardiac function. One possibility is surgical linear ablation, which has proved effective in treating refractory atrial fibrillation.¹² In this study, we retrospectively evaluated the feasibility of surgical endoepicardial linear ablation during off-pump myocardial revascularization and LVA repair in patients with postinfarction LVA-related VT.

This study was supported by funding from the National Natural Science Foundation of China (81370436), Beijing Municipal Commission of Science and Technology (Z131107002213003), and Beijing Municipal Commission of Education (KZ201410025028).

Patients and Methods

In a chart review, we retrospectively identified 82 patients with multivessel coronary artery disease and post-infarction LVA who were treated at our institution from March 2012 through July 2015. Eighteen of these patients had mural thrombosis of the LVA or valve disease and were excluded from this study. The remaining 64 patients met the study criteria. All had undergone off-pump coronary artery bypass grafting (OPCABG) and LVA repair. Twenty-three patients (35.9%) also underwent surgical endoepicardial linear ablation; 41 patients (64.1%) had no additional surgery and served as a control group. Table I summarizes the baseline characteristics of the study population.

Surgical ablation had been performed only when the patient had 1) sustained VT of at least 3 months' duration after last myocardial infarction, as documented by Holter monitoring; 2) refractoriness to at least 2

antiarrhythmic agents; and 3) operative indications for OPCABG and LVA repair. To determine whether the additional procedure caused severe myocardial damage, thus increasing the risk of postoperative death, we compared the safety and clinical outcomes between the 2 groups of patients.

This study was conducted in compliance with the guidelines for human studies at our institution and was approved by an institutional ethical review board. All patients gave written informed consent.

Operative Technique

All 64 patients underwent median sternotomy and pericardiotomy under general anesthesia, and all operations were performed on the beating heart. In both treatment groups, OPCABG was performed before LVA repair. The LVA was repaired by using the off-pump linear plication approach, as described previously.¹³ The linear plication paralleled the left anterior descending coronary

TABLE I. Baseline Characteristics of the 64 Patients

Characteristic	Overall (N=64)	Ablation (n=23)	No Ablation (n=41)	P Value
Age (yr)	58.2 ± 8.1	58.0 ± 7.1	58.4 ± 8.8	0.94
Male	44 (68.8)	16 (69.6)	28 (68.3)	0.99
Body mass index	25.7 ± 3.2	25.5 ± 3.1	25.8 ± 3.3	0.71
Smoking	37 (57.8)	16 (69.6)	21 (51.2)	0.19
Hypertension	26 (40.6)	9 (39.1)	17 (41.5)	0.99
Diabetes	38 (59.4)	10 (43.5)	28 (68.3)	0.07
Dyslipidemia	23 (35.9)	6 (26.1)	17 (41.5)	0.28
LVEF (%)	42.9 ± 10.2	39.6 ± 8.1	44.8 ± 10.9	0.10
LVEDD (mm)	57.7 ± 7.1	60.0 ± 6.9	56.4 ± 6.9	0.02
NYHA class	—	—	—	0.0042
II	10 (15.6)	0	10 (24.4)	0.01
III	46 (71.9)	17 (73.9)	29 (70.7)	0.99
IV	8 (12.5)	6 (26.1)	2 (4.9)	0.02
LVA location	—	—	—	0.56
Anterolateral	51 (79.7)	19 (82.6)	32 (78.1)	0.76
Apical	11 (17.2)	4 (17.4)	7 (17.1)	0.99
Posterior	2 (3.1)	0	2 (4.9)	0.53
LVA size, diameter (mm)	—	—	—	0.87
Long axis	35.9 ± 10.9	35.7 ± 11.9	35.9 ± 10.4	0.94
Short axis	32.4 ± 10.6	33.2 ± 9.2	32.0 ± 11.4	0.66
Medications	—	—	—	<0.001
Amiodarone	10 (15.6)	5 (21.7)	5 (12.2)	0.48
β-blocker	33 (51.6)	3 (13)	30 (73.2)	<0.001
Amiodarone + β-blocker	21 (32.8)	15 (65.2)	6 (14.6)	<0.001

LVA = left ventricular aneurysm; LVEDD = left ventricular end-diastolic dimension; LVEF = left ventricular ejection fraction; NYHA = New York Heart Association

Data are presented as mean ± SD or as number and percentage. *P* < 0.05 was considered statistically significant.

artery and eliminated abnormal scar tissue while restoring normal ventricular size and shape. All 23 ablation patients underwent intraoperative electroanatomic mapping and surgical ablation before undergoing OPCABG.

Intraoperative Substrate Mapping

Patients in the ablation group underwent electroanatomic 3-dimensional mapping of VT substrates (Fig. 1).¹⁴ This was done during sinus rhythm with use of the Carto[®] 3 system (Biosense Webster, a Johnson & Johnson company) with a 3.5-mm irrigated-tip catheter (NaviStar[®] ThermoCool[®]; Biosense Webster), a fill threshold of 10 mm, and intracardiac electrogram

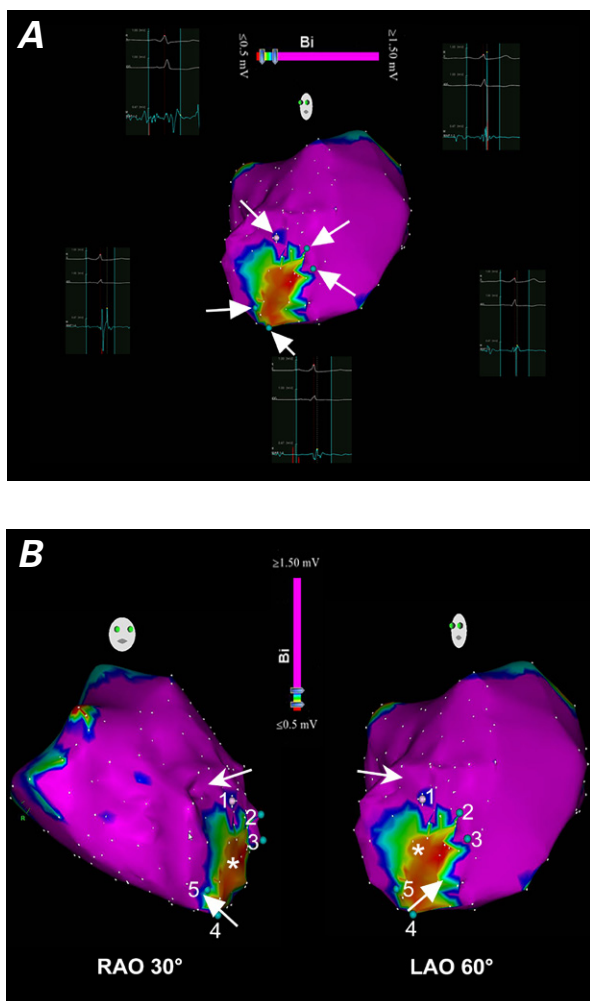


Fig. 1 **A**) Representative epicardial substrate map (60° LAO projection) shows the location of abnormal potentials (arrows) in a patient with left ventricular aneurysm-related ventricular tachycardia. **B**) Epicardial bipolar voltage maps show the location of normal tissue (notched arrows); dense scar (asterisks); and the distinct electroanatomic border of a left ventricular aneurysm and the location of ventricular tachycardia substrates (1–5), primarily in the border zone (arrows).

Bi = bipolar voltage; LAO = left anterior oblique; RAO = right anterior oblique

filtering at 30 to 400 Hz.¹⁵ The resulting epicardial electroanatomic maps were based on bipolar voltage (BV) criteria (dense scar, BV < 0.5 mV; border zone, 0.5–1.5 mV; and normal area, > 1.5 mV). The VT substrates were determined by abnormal potentials, including fractionated electrograms,¹⁶ isolated potentials,¹⁷ and late potentials.¹⁸

After epicardial substrate mapping, programmed electrical stimulation (PES) was performed from the surface of the right ventricle in an attempt to induce sustained VT. The stimulation protocol included pacing at the basic cycle length (400–500 ms) with up to 3 extrastimuli down to the refractory period, a current strength of 10 mA, a pulse width of 2 ms, burst pacing, and intravenous isoproterenol (up to 5 µg/min).¹⁹ Sustained VT was defined as tachycardia lasting more than 30 seconds or requiring countershock because of hemodynamic intolerance.²⁰

Surgical Ablation Technique

Surgical endoepicardial linear ablation was performed on the beating heart with an AtriCure[®] Isolator[®] bipolar radiofrequency ablation (BRFA) device (AtriCure, Inc.) The BRFA device consisted of 2 opposing jaws, each containing a pair of parallel linear electrodes, for clamping and ablating the target endoepicardial tissue.

The ablative procedure began with locating the LVA by direct inspection and palpation. Next, a traction line was attached with horizontal mattress sutures to 2 pieces of felt at the core of the LVA. Then, a 5-mm incision was made between the 2 horizontal mattress sutures securing the traction line. One jaw of the BRFA device was inserted into the endocardium through the incision; the other jaw was placed opposite on the epicardium. The target tissue was then clamped between the 2 jaws. Radiofrequency energy (30 W) was delivered constantly from the ablation system to the BRFA device according to an algorithm based on tissue conductance measurements obtained every 0.1 second.^{21,22} The constant power was terminated when tissue conductance reached the algorithm-based transmural threshold (resistance, < 2.5 Ω). The ablative lesions were extended radially across the border zone from the dense scarred areas of the LVA (BV, < 0.5 mV) to normal areas (BV, > 1.5 mV) previously identified by epicardial substrate mapping. To standardize this technique, 8 ablative lines were evenly distributed across the border zone of the LVA (Fig. 2A). Each ablative line was continuous and was repeated 3 times to ensure a transmural lesion. Finally, the incision between the sutures was closed.

The effectiveness of the ablative procedure was evaluated by performing PES again, according to the same protocol and at the same sites. Acute ablation success was defined as the noninducibility of sustained monomorphic VTs before and after intravenous administration of isoproterenol (up to 5 µg/min).

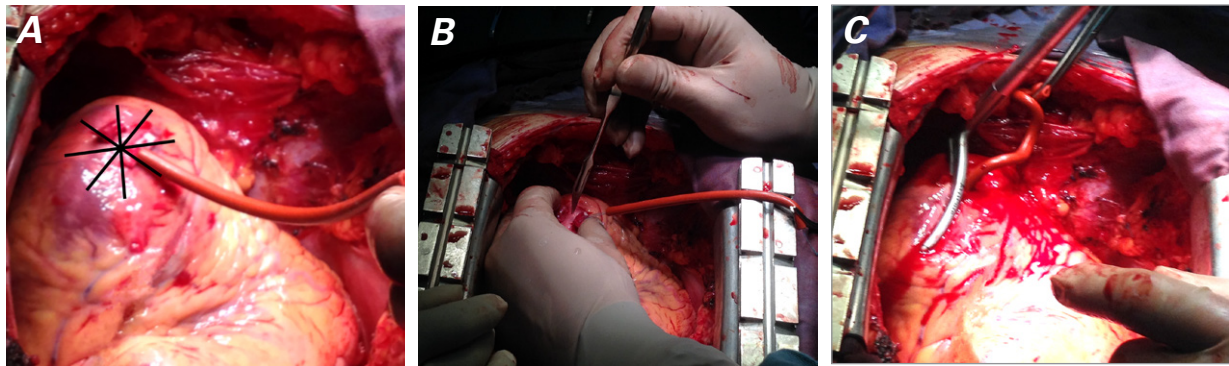


Fig. 2 Intraoperative photographs show the technique for surgical endoepicardial linear ablation for left ventricular aneurysm–related ventricular tachycardia. **A** The 8 black lines radiating to the border zone indicate the planned location of ablative lesions. **B** A 5-mm incision is made in the core of the aneurysm. **C** The 2 jaws of a bipolar radiofrequency ablation device clamp endocardial and epicardial tissue for transmural and linear ablation.

After surgical ablation was completed, OPCABG and LVA repair were performed as described above.

Postoperative Evaluation and Follow-Up

To evaluate the recurrence of sustained VT postoperatively in ablation patients after they were discharged from the hospital, we chose Holter monitoring. Implantable cardioverter-defibrillator (ICD) placement was recommended for patients who had postablative inducible VT or postoperative VT documented by Holter monitoring. Most patients returned for a follow-up visit every 6 months. Patients with an ICD returned for a follow-up visit every 3 months. The endpoint of follow-up was death or recurrent VT.

Statistical Analysis

Data were presented as mean \pm SD for continuous variables and as number and percentage for categorical variables. The Student *t* test was used to compare normally distributed parametric variables, the Wilcoxon test to compare nonparametric variables, and the χ^2 test to compare categorical variables. Multivariate Cox regression analyses were performed to calculate hazard ratios (HRs) and 95% confidence interval (CIs) to identify independent predictors of postoperative death. A *P* value <0.05 was considered statistically significant. All data were analyzed with SAS version 9.4 (SAS Institute Inc.).

Results

When compared with patients in the no-ablation (control) group, those in the ablation group had significantly more severe LV remodeling (mean LV end-diastolic dimension, 60.0 ± 6.9 vs 56.4 ± 6.9 mm; $P=0.02$) and worse functional status (New York Heart Association class III or IV, 100% [23/23] vs 75.6% [31/41]; $P=0.0042$) (Table I).

In the ablation group, epicardial substrate mapping revealed scarred areas according to BV criteria (Fig. 1A).

Abnormal potentials on the epicardial maps were tagged; regions containing VT substrates were located mainly in the border zone of the LVA (Fig. 1B). Surgical endoepicardial linear ablation was performed without complications. In each case, 8 radial, evenly distributed ablative lines transected the border zone from the LVA core to the viable myocardium (Fig. 2). Inducible VT occurred in 19 patients (82.6%) before ablation and in 4 patients (17.4%) after ablation (Table II). No spontaneous VT was identified by postoperative Holter monitoring. Three of the 4 patients who had postablative inducible VT received ICDs; the 4th patient declined the option.

Total myocardial revascularization was achieved in both patient groups; the mean number of grafts was 3.43 ± 0.84 in the ablation group and 3.5 ± 0.8 in the no-ablation group. The 2 groups were similar in terms of the need for mechanical circulatory support after surgery, time in intensive care unit, and early mortality rate (Table III).

The mean follow-up time in the ablation group was 39 ± 21 months. Of the 23 patients in the ablation group, 5 (23.8%) had recurrent VT; all 5 patients received optimal medical therapy according to established guidelines for the management of VT.²³ Only one of these patients experienced sudden cardiac death. Late mortality rates were 19.1% (4/21) in the ablation group and 18% (7/39) in the no-ablation group. The main cause of death in both groups was congestive heart failure. Although the percentage of patients receiving antiarrhythmic medications differed significantly between the 2 groups at baseline (Table I), these differences had little effect on the main outcome of this study. Multivariate Cox regression analysis indicated that preoperatively poor LV ejection fraction and increased LV end-diastolic dimension were independent risk factors for early and late death; surgical ablation did not increase the risk of death (Table IV). Kaplan-Meier survival analysis showed no significant difference in the cumulative survival rate between groups (Fig. 3).

Discussion

In this study, we found that surgical ablation guided by epicardial substrate mapping was a feasible treatment for LVA-related VT and produced results comparable to those reported for catheter ablation.²⁴ We also found that adding surgical linear ablation to OPCABG and LVA repair did not appear to cause additional myocardial damage, further impair cardiac function, or worsen the perioperative prognosis.

Left ventricular aneurysm is a distinct type of thin, transmural scar. The border zone is composed of circular islands of viable fibrotic and necrotic myocardial tissue staggered between dense scar and normal myocardial tissue. This zone may supply the structural and electrophysiologic substrate for LVA-related VT.^{25,26} As our mapping results showed, regions containing abnormal

potentials—and thus possible substrates—were located mainly in the LVA border zone. In previous studies, VT substrates were located mainly in the border zone between dense scar tissue and normal myocardium.^{27,28}

The VT substrate is 3-dimensional and so may not always be located in the endocardium; in some cases, it originates in the mid-myocardium or epicardium.^{29,30} Furthermore, VT substrates may involve a large area within the border zone. In one study,¹⁹ the critical isthmus of the VT substrate alone was an average of 27.3 ± 7 mm long and 9.2 ± 5 mm wide, suggesting the feasibility of linear ablation for VT.

Absolute interruption of abnormal channels in the VT substrate requires that continuous, deep ablative

TABLE II. Perioperative Characteristics of the 23 Patients Who Underwent Ablation

Variable	Value
Inducible VT before ablation	19 (82.6)
VT cycle length (ms)	439.5 ± 60.3
Epicardial mapping points	184 ± 47
Inducible VT after ablation	4 (17.4)
ICD implantation after ablation	3 (13)

ICD = implantable cardioverter-defibrillator; VT = ventricular tachycardia

Data are presented as number and percentage or as mean \pm SD.

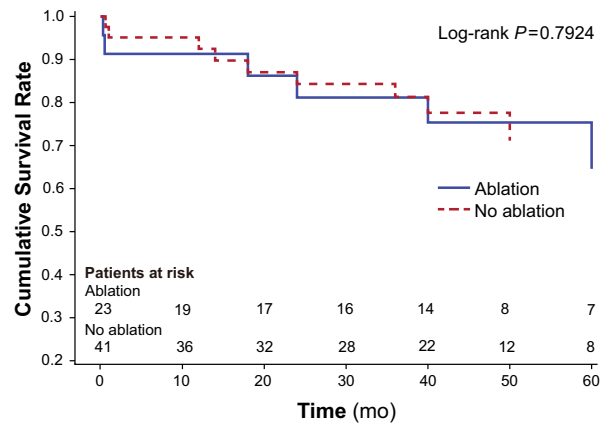


Fig. 3 Kaplan-Meier cumulative survival was similar between treatment groups. $P < 0.05$ was considered statistically significant.

TABLE III. Comparison of Procedural Details and Outcomes Between Groups

Variable	Overall (N=64)	Ablation (n=23)	No Ablation (n=41)	P Value
Grafts	3.5 ± 0.8	3.4 ± 0.8	3.5 ± 0.8	0.90
MCS after surgery				
IABP	11 (17.2)	4 (17.4)	7 (17.1)	0.99
ECMO	2 (3.1)	1 (4.4)	1 (2.4)	0.99
ICU stay (d)	2.0 ± 1.3	1.7 ± 1.1	2.1 ± 1.5	0.44
Early death (<30 d postoperatively)	4 (6.3)	2 (8.7)	2 (4.9)	0.41
Low cardiac output syndrome	3 (4.7)	1 (4.4)	2 (4.9)	0.99
Multiple organ failure	1 (1.6)	1 (4.4)	0	0.36
Late death	11 (17.2)	4 (17.4)	7 (17.1)	0.99
Congestive heart failure	7 (10.9)	3 (13)	4 (9.8)	0.70
Sudden cardiac death	3 (4.7)	1 (4.4)	2 (4.9)	0.99
Noncardiac death	1 (1.6)	0	1 (2.4)	0.99

ECMO = extracorporeal membrane oxygenation; IABP = intra-aortic balloon pump; ICU = intensive care unit; MCS = mechanical circulatory support

Data are presented as mean \pm SD or as number and percentage. $P < 0.05$ was considered statistically significant.

TABLE IV. Multivariate Cox Regression Analysis of Risk Factors for Death in Patients

Variable	Hazard Ratio (95% CI)	P Value
Age	1.10 (0.94–1.28)	0.25
Sex	0.28 (0.02–2.97)	0.29
Body mass index	0.95 (0.73–1.24)	0.72
Hypertension	1.00 (0.19–5.19)	0.99
Diabetes	0.39 (0.09–1.81)	0.23
Dyslipidemia	0.62 (0.13–3.05)	0.56
LVEF	0.74 (0.59–0.94)	0.01
LVEDD	1.35 (1.04–1.75)	0.02
LVA size	1.00 (0.99–1.01)	0.40
NYHA class	0.42 (0.07–2.59)	0.35
MI (within previous 3 mo)	5.98 (0.51–70.19)	0.15
No. of distal anastomoses	1.21 (0.34–4.39)	0.77
ICU stay	1.16 (0.66–2.05)	0.60
Surgical ablation	1.97 (0.42–9.34)	0.39

CI = confidence interval; ICU = intensive care unit; LVA = left ventricular aneurysm; LVEDD = left ventricular end-diastolic dimension; LVEF = left ventricular ejection fraction; MI = myocardial infarction; NYHA = New York Heart Association
P < 0.05 was considered statistically significant.

lesions be created. The BRFA device that we used can produce continuous, endoepicardial ablative lesions. When compared with sequential (point-to-point) catheter ablation via the endocardium or epicardium,³¹ surgical endoepicardial linear ablation may be a more efficient means of creating transmural lesions. This may be one reason for the effectiveness of surgical linear ablation in our study. Another reason may be our operative technique in which we create 8 radial ablative lines that evenly transect the LVA border zone, thus ensuring discontinuity of the VT substrate.

Excellent results can be achieved with VT surgery, endocardectomy, and cryoablation with or without electrophysiologic guidance.²⁰ However, that approach necessitates anatomic resection of the whole scar, which can cause severe tissue damage and increase the risk of postoperative death.¹¹ In our study, we focused on treating the VT substrate instead of the entire LVA scar, thus limiting damage to the adjacent normal myocardium. We also limited myocardial damage by using a BRFA device that could measure tissue impedance. Once impedance reached a transmural threshold, the BRFA system automatically terminated the power output, thus minimizing the risk of overablation. This may partly explain the similar outcomes in our 2 treatment groups. In a preclinical porcine study,³² we established the safety and efficacy of surgical endoepicardial linear ablation with the BRFA device. In a previously reported case,³³ we used mapping-guided surgical linear ablation with a BRFA device to treat a patient with refractory ventricular arrhythmia related to LVA, with good results. The

patient remained angina-free at follow-up 26 months after ablation.

Off-pump linear plication of LVA and OPCABG are feasible and effective,³⁴ especially in high-risk patients.³⁵ Surgical linear ablation is also easily performed on the beating heart, which enables its concomitant use in patients with LVA-related VT undergoing OPCABG and LVA repair. The combination of surgical linear ablation, OPCABG, and LVA plication achieves multiple goals by modifying VT substrates located in the LVA border zone, accomplishing total revascularization, and reshaping the LV.

Limitations

This retrospective study has several limitations. First, the study cohort was relatively small because it was highly selective; we excluded patients with concomitant valvular disease and mural thrombosis of the LVA. Second, surgical linear ablation was performed on a transmural scar located in the LV free wall, which may have led to bias toward a higher success rate because the ablation procedure involved less complicated septal scars. However, creating the 8 radial linear lesions across the LVA border zone appeared to be sufficient to restrain electrical conduction between the LVA substrate and the septal scar. Moreover, surgical linear ablation in this study produced results comparable to those reported for catheter ablation.²⁴ In addition, ICD placement was recommended for several patients whose LVA-related VT recurred after ablation. Third, because this was a single-center study, the ablation technique was

the same in all patients. Fourth, treatments were not randomly assigned, patients were not matched, and the no-ablation group was larger than the ablation group. However, the pathologic basis for treatment was similar in both groups: multivessel coronary artery disease and LVA. Also, establishing the no-ablation group as a control enabled us to evaluate the safety of additional surgical ablation. Fifth, because the LVA was visible on the epicardium, endocardial mapping was not performed before surgical ablation. Therefore, clinical trials with a larger sample size, more in-depth electrophysiologic study, and longer-term follow-up are warranted to investigate the risks and benefits of surgical linear ablation.

Conclusion

We conclude that, for carefully selected candidates, surgical endoepicardial linear ablation combined with OPCABG and LVA linear plication is a feasible treatment for LVA-related VT.

Acknowledgments

We thank all participating colleagues, nurses, and imaging and laboratory technicians.

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