Options for ventricular tachycardia ablation after double valve replacement

Samuel H. Baldinger, MD, Saurabh Kumar, MD, PhD, Alan D. Enriquez, MD, Piotr S. Sobieszczyk, MD, Roy John, MD, PhD, FHRS, William G. Stevenson, MD, FHRS

From the Cardiac Arrhythmia Center, Cardiovascular Division, Brigham and Women's Hospital, Boston, Massachusetts.

Introduction

Transcoronary ethanol ablation (TCEA) for ventricular tachycardia (VT) is a treatment option for selected patients when endocardial and epicardial ablation have failed. We present a case of successful TCEA for treatment of nearly incessant VT in a patient in whom access to the left ventricle (LV) was impeded because of prosthetic double valve replacement.

Case report

A 52-year-old man was referred for treatment of recurrent sustained monomorphic VT with variable cycle lengths (CLs) of 360 to 480 ms (125–166 bpm) causing recurrent implantable cardioverter-defibrillator (ICD) shocks despite increasing doses of amiodarone. He had rheumatic heart disease and had undergone mitral and aortic valve replacements with mechanical St. Jude prostheses as well as tricuspid valve repair 14 years previously. Two years after surgery, he had an embolic lateral wall myocardial infarction and subsequently developed recurrent episodes of heart failure, fluctuating renal dysfunction, and monomorphic VT for which an ICD was implanted. In the past year, VT episodes increased despite therapy with amiodarone at doses of up to 600 mg daily. The patient was admitted for nearly incessant slow VT with a CL of 430 ms, right bundle branch block-like configuration in lead V_1, and rightward frontal plane axis, consistent with an origin in the lateral LV scar. His left ventricular ejection fraction was 20%, with LV end-diastolic diameter of 7.3 cm. Serum creatinine was 1.07 mg/dL.

A sestamibi rest myocardial perfusion single-photon emission computerized tomographic study showed a severely dilated LV with a large region of myocardial scar in the distribution of the distal left circumflex coronary artery (Figure 1). Although he had a small paravalvular leak around the aortic valve, hemodynamic function was deemed satisfactory and not warranting repeat surgery. Warfarin was held, and the patient was started on intravenous heparin. He was subsequently taken to the electrophysiology laboratory.
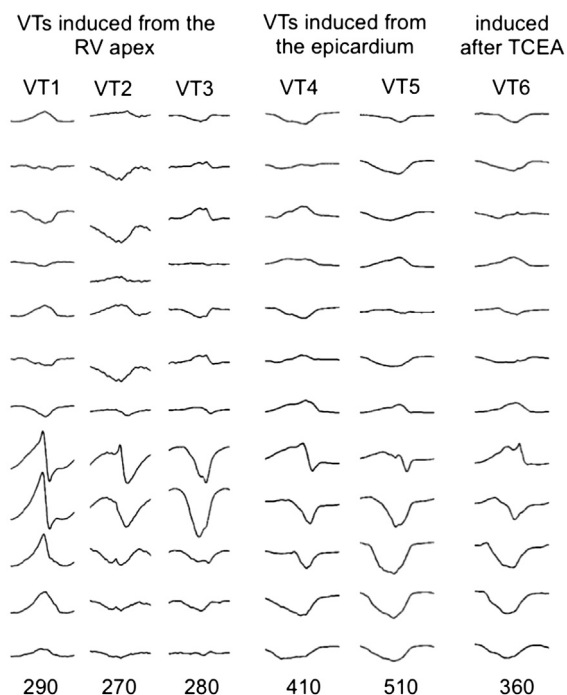
Programmed ventricular stimulation from the right ventricular (RV) apex with the patient under general anesthesia induced 3 different morphologies of sustained monomorphic VT (VT1–VT3), all of which had a shorter CL than the documented VTs (Figure 2). None were bundle branch reentry, and all appeared remote from the RV apex and basal septum, where the His-bundle catheter was located.

Mechanical prosthetic aortic and mitral valves precluded either a retrograde aortic or transseptal approach to the LV endocardium. Epicardial access was attempted using a subxiphoid percutaneous approach as described by Sosa et al,[1] and the pericardial space was entered beneath the RV. There was no significant pericardial bleeding or evidence of inadvertent ventricular puncture. However, pericardial adhesions from prior cardiac surgery limited catheter mapping to the apical aspect of the LV. An electroanatomic voltage map of the accessible epicardial surface was created using a mapping system (CARTO 3, Biosense Webster, Diamond Bar, CA) and a 3.5-mm irrigated-tip ablation catheter. An extensive area of low bipolar voltage (<1.5 mV) consistent with scar was present in the inferolateral LV apex extending to the accessible mid-LV, consistent with perfusion imaging (Figures 1 and 3). With pacing there was no left phrenic nerve capture in the area. Monomorphic VT4 with CL of 410 ms, right bundle branch block-like configuration in lead V_1, and right inferior frontal plane axis was induced with burst pacing from the ablation catheter (Figure 2). It terminated spontaneously. Substrate modification of the scar was then performed targeting regions of slow conduction evident as late or fractionated potentials or stimulus-to-QRS delays >40 ms during pace-mapping.[2] Radiofrequency (RF) ablation was performed with power of 30–50 W aiming for a minimum impedance drop of 10 Ω at each site in an attempt

KEY TEACHING POINTS

- After double valve replacement, an initial epicardial approach to radiofrequency ablation for treatment of ventricular tachycardia (VT) can be considered when VT morphology is not suggestive of a septal origin. However, percutaneous epicardial access often is limited in patients with prior cardiac surgery.

- Options for left ventricular (LV) access after double valve replacement include direct percutaneous LV puncture, percutaneous interventricular septal puncture, and open chest surgical ablation.

- Transcoronary ethanol ablation can provide effective treatment of recurrent VT for selected patients when access to the LV endocardium and epicardium is impeded.

Figure 2 Three fast ventricular tachycardia (VT) morphologies different from the documented VT were induced from the right ventricular (RV) apex (VT1–VT3). Another 3 VTs with different morphologies consistent with origin in the lateral scar area, all with right bundle branch block-like morphology in lead V_1 and right frontal plane axis, were induced by pacing in the epicardial scar area (VT5–VT6). VT6 was induced after the first transcoronary ethanol ablation (TCEA) and remained inducible.

to render the region electrically unexcitable to pacing (10 mA and 2-ms pulse width). During pace-mapping within the scar, sustained monomorphic VT5 with a cycle length of 510 ms, right bundle branch block-like configuration in lead V_1, and right superior frontal plane axis was induced (Figure 2). It was initially hemodynamically tolerated, allowing activation and entrainment mapping. Earliest activation (10 ms presystolic) was noted in the most posterior portion of the accessible scar. However, activation was early over a broad area. Entrainment mapping in that region produced concealed fusion with a postpacing interval of 25 ms (Figure 3). The VT did not terminate despite multiple RF applications up to 120 seconds in duration. The epicardial unipolar voltage map also showed extensive low unipolar voltage (Figure 2).[3] These findings suggested either an endocardial

or intramural VT origin with a broad epicardial breakout. However, lack of accessibility to the endocardial LV prompted a detailed bedside interdisciplinary discussion among electrophysiologists, interventional cardiologists, and cardiac surgeons about the next approach. Coronary angiography was performed with the ablation catheter
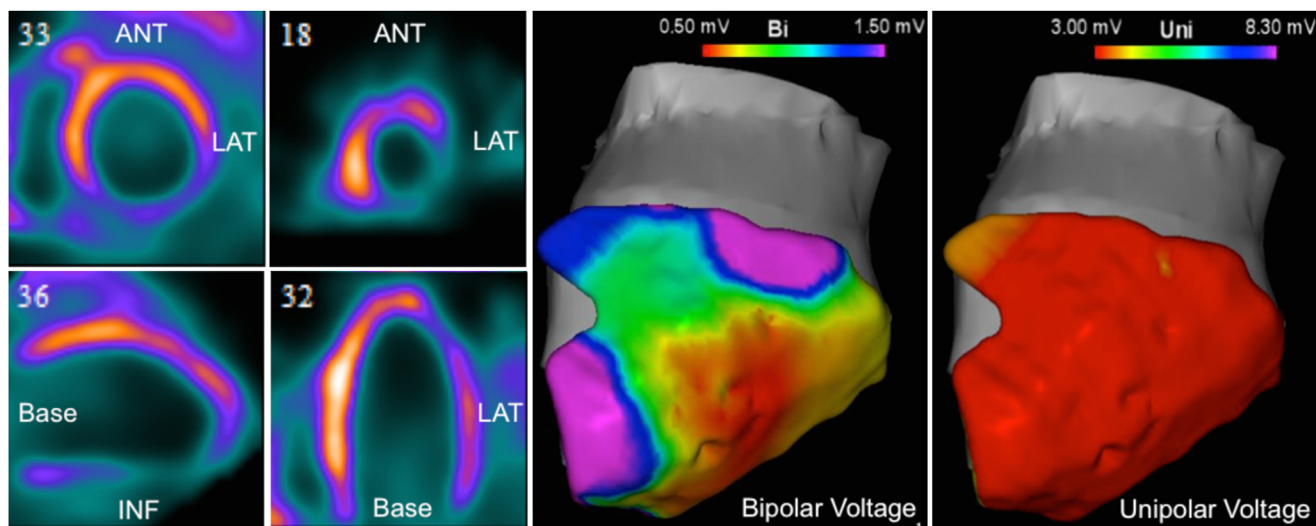
Figure 1 Tc-99m sestamibi rest myocardial perfusion single-photon emission computerized tomographic study showing a severely dilated left ventricle with a large region of myocardial scar in the distribution of the distal left circumflex coronary artery **(left panels).** The epicardial voltage map (left lateral view) shows a large inferolateral area of low bipolar voltage <1.5 mV **(middle panel)** and unipolar voltage <8.3 mV **(right panel).** ANT = anterior; Bi = bipolar voltage; INF = inferior; LAT = lateral; Uni = unipolar voltage.
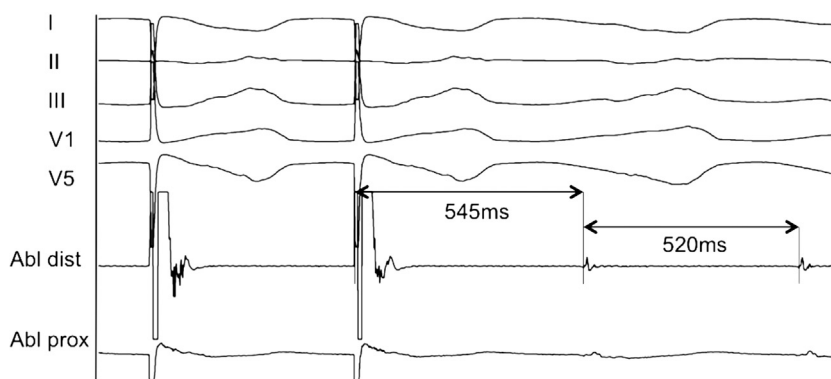
Figure 3   Entrainment mapping from the ablation catheter positioned in the inferolateral epicardial scar, where transcoronary ethanol ablation was subsequently performed, showing concealed fusion with a postpacing interval of 25 ms. Abl = ablation catheter; dist = distal; prox = proximal.

positioned in the inferolateral LV scar at the site of earliest activation and shortest postpacing intervals consistent with the VT exit (Figure 4). Distal branches of a marginal branch appeared to be supplying the scar (Online Supplementary Material, Cine 1). The decision was made to perform TCEA. An angioplasty balloon was inserted into 1 of the marginal branches over the wire. The patient's ongoing VT terminated as soon as contrast was injected into the branch. The balloon was positioned proximal to a bifurcation and inflated with contrast to occlude the vessel. Ethanol 3 mL was then injected down the vessel over several minutes. The balloon remained inflated for 10 minutes (Online Supplementary Material, Cine 2). Repeat angiography showed successful occlusion of the targeted marginal branch (Online Supplementary Material, Cine 3). Ventricular stimulation was repeated from the epicardial LV. The previous VTs were no longer inducible. However, another morphology of VT (VT6) was induced (Figure 2). Therefore, a second small marginal branch inferior to the previous vessel was cannulated and injected with ethanol after balloon occlusion. VT6 remained inducible, however, and no further ablation was performed.

During follow-up, the patient remained free of VT. His device was subsequently upgraded to provide AV synchrony and biventricular pacing, with subsequent re-exploration for atrial lead dislodgment. He had no further ICD shocks or documented VT, but he died 2 months later from complications of sepsis, possibly related to device infection.

Discussion

Catheter ablation of VT originating from the LV endocardium typically is performed using an atrial transseptal or retrograde aortic approach. However, neither approach is feasible in the setting of mechanical aortic and mitral valves given the high risk of catheter entrapment. When VT morphology is not suggestive of a septal VT, an initial epicardial approach for RF ablation can be considered. In our experience, percutaneous epicardial access in patients with prior cardiac surgery is successful in only about 30% of

patients.[4] Tschabrunn et al[5] reported obtaining access that was limited but allowed for ablation in 8 patients with prior surgery for valvular heart disease. As in our case, extensive mapping was limited by adhesions. A surgical subxiphoid approach that allows blunt dissection of adhesions under direct vision has been performed successfully and offers an alternative.[6]

In our case, epicardial ablation abolished some of the inducible VTs, but others remained likely because of intramural, endocardial, or epicardial substrate that was out of reach beneath adhesions. Hsieh et al[7] reported 2 cases in which direct percutaneous LV puncture and catheter ablation were used effectively to treat scar-related VT in patients with prosthetic aortic and mitral valves. Hemorrhage into the left pleural space occurred in the first case and was prevented in the second by using a minithoracotomy and pursestring ties to control bleeding from the LV puncture site. Vaseghi et al[8] reported successful percutaneous interventricular septal puncture to gain access to the LV endocardium for VT ablation in a patient with dual mechanical valve replacement. Open chest surgical ablation is also an option but was not
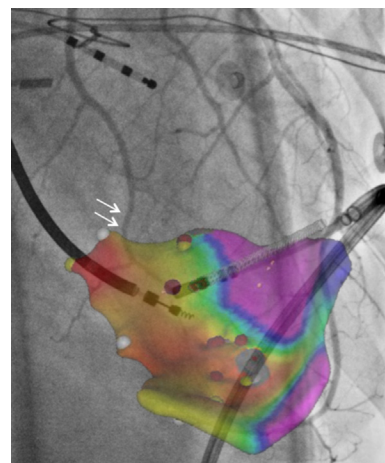
Figure 4   Overlay of coronary angiography and epicardial voltage map showing distal branches of a marginal branch *(arrows)* supplying the inferolateral area where the ablation catheter is located.

selected in our patient because of his comorbidities and absence of need for surgical revision of his valves.[9]

TCEA is an option for selected patients when endocardial and epicardial ablation have failed.[10] It can provide effective termination of VT storm and reduce the number of arrhythmic episodes.[11] Our patient was challenging because routine endocardial LV access was not possible, epicardial access was hampered by adhesions from prior cardiac surgery, and comorbidities increased the risks of surgical cryoablation and transventricular access. The case demonstrates the feasibility of TCEA for treatment of VT when access to the LV endocardium and epicardium is impeded.

Appendix

Supplementary data

Supplementary material cited in this article is available online at http://dx.doi.org/10.1016/j.hrcr.2015.02.007.

References

1. Sosa E, Scanavacca M, d'Avila A, Oliveira F, Ramires JA. Nonsurgical transthoracic epicardial catheter ablation to treat recurrent ventricular tachycardia occurring late after myocardial infarction. J Am Coll Cardiol 2000;35:1442–1449.
2. Stevenson WG, Sager PT, Natterson PD, Saxon LA, Middlekauff HR, Wiener I. Relation of pace mapping QRS configuration and conduction delay to ventricular tachycardia reentry circuits in human infarct scars. J Am Coll Cardiol 1995;26:481–488.
3. Tokuda M, Tedrow UB, Inada K, Reichlin T, Michaud GF, John RM, Epstein LM, Stevenson WG. Direct comparison of adjacent endocardial and epicardial electrograms: implications for substrate mapping. J Am Heart Assoc 2013;2:e000215.
4. Sarkozy A, Tokuda M, Tedrow UB, Sieria J, Michaud GF, Couper GS, John R, Stevenson WG. Epicardial ablation of ventricular tachycardia in ischemic heart disease. Circ Arrhythm Electrophysiol 2013;6:1115–1122.
5. Tschabrunn CM, Haqqani HM, Cooper JM, Dixit S, Garcia FC, Gerstenfeld EP, Callans DJ, Zado ES, Marchlinski FE. Percutaneous epicardial ventricular tachycardia ablation after noncoronary cardiac surgery or pericarditis. Heart Rhythm 2013;10:165–169.
6. Soejima K, Couper G, Cooper JM, Sapp JL, Epstein LM, Stevenson WG. Subxiphoid surgical approach for epicardial catheter-based mapping and ablation in patients with prior cardiac surgery or difficult pericardial access. Circulation 2004;110:1197–1201.
7. Hsieh CH, Thomas SP, Ross DL. Direct transthoracic access to the left ventricle for catheter ablation of ventricular tachycardia. Circ Arrhythm Electrophysiol 2010;3:178–185.
8. Vaseghi M, Macias C, Tung R, Shivkumar K. Percutaneous interventricular septal access in a patient with aortic and mitral mechanical valves: a novel technique for catheter ablation of ventricular tachycardia. Heart Rhythm 2013;10:1069–1073.
9. Anter E, Hutchinson MD, Deo R, et al. Surgical ablation of refractory ventricular tachycardia in patients with nonischemic cardiomyopathy. Circ Arrhythm Electrophysiol 2011;4:494–500.
10. Sacher F, Sobieszczyk P, Tedrow U, Eisenhauer AC, Field ME, Selwyn A, Raymond JM, Koplan B, Epstein LM, Stevenson WG. Transcoronary ethanol ventricular tachycardia ablation in the modern electrophysiology era. Heart Rhythm 2008;5:62–68.
11. Tokuda M, Sobieszczyk P, Eisenhauer AC, Kojodjojo P, Inada K, Koplan BA, Michaud GF, John RM, Epstein LM, Sacher F, Stevenson WG, Tedrow UB. Transcoronary ethanol ablation for recurrent ventricular tachycardia after failed catheter ablation: an update. Circ Arrhythm Electrophysiol 2011;4:889–896.