Introduction

We present a case of a patient who underwent surgical ablation of ventricular tachycardia (VT) and 36 years later required catheter ablation for VT recurrence.

Case report

A 76-year-old patient with ischemic cardiomyopathy and prior coronary artery bypass grafting was awakened by an implantable cardioverter-defibrillator (ICD) shock. Device interrogation showed delivery of a single successful shock for monomorphic VT with a cycle length (CL) of 300 ms. He was also found to have multiple episodes of VT, CL 360 ms, terminated with antitachycardia pacing. In 1980 he had suffered an anterior wall myocardial infarction and soon after developed medically refractory VT. He was treated with mexiletine at that time, which led to major depression. He traveled to the University of Pennsylvania in 1982 and underwent a “Pennsylvania peel,” consisting of a subendocardial resection and aneurysmectomy, by Drs Mark Josephson and Alden Harken. In 2003 he received a dual-chamber ICD, which was later upgraded to a biventricular ICD in 2012. He remained free from ventricular arrhythmias for 36 years.

He believed his ICD shock to be secondary to significant stressors in his life and therefore he declined to make changes to his medical therapy. Five months after the first ICD shock he presented with 2 additional ICD shocks. He was treated with amiodarone, and percutaneous coronary intervention to a saphenous vein graft to an obtuse marginal was performed. Two months into treatment with amiodarone he received another ICD shock, and the decision was made to proceed with electrophysiology study and VT ablation.

Single photon emission computer tomographic myocardial perfusion imaging prior to ablation showed severe and extensive infarction involving 50% of the left ventricle (Figure 1A). Endocardial mapping using an Orion mapping catheter and Rhythmia Hdx electroanatomic mapping system (Boston Scientific, Natick, MA) showed extensive scar and low voltage involving the apex, mid anterior, mid inferior, mid lateral, and mid septal wall regions with late potentials within the scar (Figure 1B). Multiple VTs were induced, CLs of 220 ms and 360 ms. The VT with CL of 360 ms was believed to be the clinically observed VT (Figure 1C). Local abnormal ventricular activity was observed and recorded from the scar border zones during right and left ventricular pacing (Figure 2). Knowing that he had VT with at least 2 different CLs, the decision was made to proceed with endocardial substrate modification targeting scar and its border zones. Radiofrequency ablation was performed using an IntellaNav MiFi open-irrigated ablation catheter (Boston Scientific, Natick, MA) with power titrated between 40 and 48 watts (Figure 3). Specific attention was given to the inferoseptal region of the scar, the likely exit site of the perceived clinical VT. Aggressive ventricular programmed stimulation post ablation was performed and VT was no longer inducible. His amiodarone dose was reduced to 100 mg daily. During 12 months of follow-up he has remained arrhythmia free.

KEY TEACHING POINTS

- The treatment of patients with ventricular tachycardia (VT) has evolved significantly over the past 4 decades.
- Prior to the development of catheter-based, percutaneous ablation of myocardial tissue, surgical resection of subendocardial scar was performed in patients with refractory VT. Our case report highlights an extraordinary example of a patient with ischemic cardiomyopathy and recurrent VT who underwent successful surgical resection of subendocardial scar and aneurysmectomy and remained VT free for 36 years.
- We report on the electroanatomic findings from an electrophysiology study of this patient who developed VT.
Discussion

Surgical myocardial resection and left ventricular aneurysmectomy, later referred to as the “Pennsylvania peel,” for recurrent, medically refractory VT was first reported from the University of Pennsylvania by Drs Josephson, Harken, and Horowitz in 1979.\(^1\)\(^2\) Subsequent reports included an increasing number of patients in whom successful surgical treatment of VT was performed.\(^3\)\(^4\) It is noteworthy that our patient remained stable and arrhythmia free for so many years without VT recurrence.

It is possible that a minor new ischemic insult led to changes in the myocardial substrate just enough to support new reentrant VT. Our case report highlights an amazing journey in the treatment of VT. We believe this patient to be the longest known survivor following successful surgical ablation of VT. In conclusion, cardiac electrophysiologic care over the past 40 years has evolved dramatically and technological advancements continue to bring new options for patients with medically refractory ventricular arrhythmias.

**Figure 1** A: Single photon emission computer tomographic myocardial perfusion imaging showing severe and extensive infarction involving 50% of the left ventricle. B: High-definition endocardial electroanatomic map of the left ventricle showing extensive scar. C: Induced ventricular tachycardia during electrophysiology study.

**Figure 2** Local abnormal ventricular activity and late potentials recorded from endocardial scar border zones during right ventricular pacing (A, B) and left ventricular pacing (C, D).
References

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4. Josephson ME, Harken AH, Horowitz LN. Long-term results of endocardial resection for sustained ventricular tachycardia in coronary disease patients. Am Heart J 1982;104:51–57.

Figure 3  Electroanatomic map showing radiofrequency ablation lesions targeting late potentials and scar border zones.