Ablation of Ventricular Tachycardia in Patients with Ischemic Cardiomyopathy

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KEYWORDS

• Ablation • Ventricular tachycardia • Ischemic cardiomyopathy • Ventricular arrhythmia

KEY POINTS

- Ventricular tachycardias (VTs) occurring after prior myocardial infarction are usually caused by reentrant circuits formed by surviving myocardial bundles.
- Although part of the reentrant circuits may be located in the midmyocardium or epicardium, most of the VTs can be safely and successfully ablated by endocardial ablation targeting the late potentials/local abnormal ventricular activation, which are surrogates for the surviving myocardial bundles.
- A combination of activation, substrate, pace mapping, and entrainment mapping, as well as the use of contact force catheters, further improves ablation success and safety.

The management of ventricular arrhythmias following myocardial infarction is often limited by medication toxicities or treatment failure. Catheter ablation for ventricular tachycardia (VT) has emerged as a viable treatment of patients with recurrent arrhythmias, which often lead to shocks or antitachycardia pacing delivered by implantable cardiac defibrillators (ICDs). In the last 10 years, the use of VT ablation has increased from 2.8% in 2002 to 10.8% in 2011, reinforcing the importance of this option for managing such patients.[1]

CASE INTRODUCTION

A 70-year-old man with ischemic heart disease, previous 3-vessel coronary artery bypass graft, and ICD implantation for primary prevention developed recurrent monomorphic VT and multiple ICD shocks. He had a known basal-inferior scar. After maximizing antiarrhythmic therapy and ensuring no significant progression of his coronary artery disease, he was referred for ablation. His electrocardiogram (ECG) is shown in **Fig. 1**.

PREABLATION ASSESSMENT

Patients presenting for ischemic VT ablation are often ill and have several comorbidities, and optimization of medical management is often required. The presence of right heart failure also substantially increases procedure-related complications. At the authors' institution, reviewing the echocardiography and 12-lead ECGs of all clinical VTs constitutes the most important preablation assessment. Aneurysms, areas of wall motion abnormality, and wall thickness are carefully evaluated, and correlate with the scar identified by the voltage map acquired during the ablation procedure. The exit point of each clinical VT is predicted based on the criteria described later to guide pace mapping.

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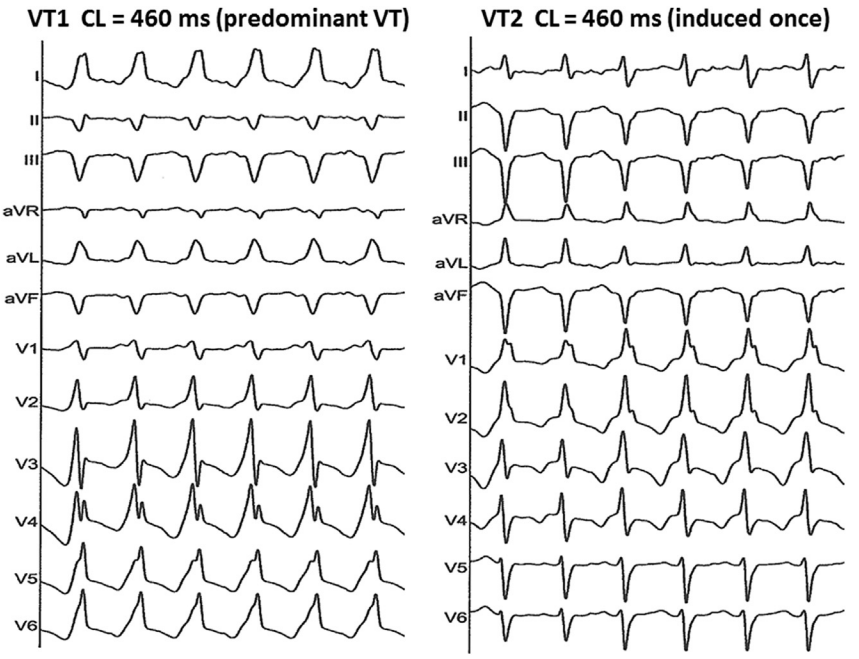

Fig. 1. Two VTs were induced during an electrophysiologic study. The QRS morphology predicted that the exit point of VT1 was septal, inferior, and basal left ventricle (LV). The exit point of VT2 was inferior and apical LV.

USING THE 12-LEAD AND QRS MORPHOLOGY TO DETERMINE EXIT OF VENTRICULAR TACHYCARDIA

Miller and colleagues[2] created an algorithm that gives a specific region of origin for ischemic VTs based on the 12-lead QRS characteristics. They divided the left ventricle (LV) into regions of origin, as seen in **Fig. 2**. Their data only included patients with a single site of infarction and was not perfect or universally applicable for VTs, such as those with multiple sites of infarction. Clinicians should also be suspicious for an epicardial exit because this can dramatically change the procedural plan. Berruezo and colleagues[3] reported on 3 specific characteristics that identify an epicardial exit of VT. These are a pseudodelta wave of greater than or equal to 34 milliseconds on the 12-lead ECG, an intrinsicoid deflection time of greater than or equal to 85 milliseconds, and an RS complex duration of greater than or equal to 121 milliseconds.

The QRS morphology of the recorded VT helps identify the exit of the ventricular tachycardia and can help plan the ablation approach and direct pace mapping maneuvers, if applicable. For example, as the exit site progresses closer to the left arm leads I and aVL, the lead polarity becomes more negative (progressive absence of an R wave). A VT with a left bundle branch block–like configuration usually originates in either the right ventricle (RV), LV septum, or is a form of bundle branch reentry. A VT with a right bundle branch block pattern (R > S in V1) originates from the LV, with few exceptions. A superiorly directly frontal plane axis (S > R in II, III, aVF) correlates with an exit at the inferior aspect of the ventricle. QRS progression is also used as a guide for exit. For example, dominant S waves in leads V3 and V4 suggest a more apical exit (away from the atrioventricular [AV] groove), whereas dominant R waves are associated with a more basal exit (near the AV groove).

CASE REVISITED

With this information, and by reviewing our 12-lead ECG, the exit point of VT1 and VT2 was predicted to be inferior-basal LV and inferior-apical LV, respectively.

MECHANISM OF ISCHEMIC VENTRICULAR TACHYCARDIA

Original research performed by de Bakker and colleagues[4,5] using Langendorff perfusion setup clarified the mechanisms and circuits for VT. Many VTs originate from circumscribed areas that can be as small as 1.4 cm^2. This finding is in contrast with the minority of cases in their electrophysiologic and histologic studies that showed only macroreentry around scar. Within the scar, multiple,