Sustained Ventricular Tachycardia in Apparently Normal Hearts

Ablation Should Be the First Step in Management

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KEYWORDS

• Catheter ablation • Ventricular tachycardia • Implantable cardioverter defibrillator
• Antiarrhythmic drugs

KEY POINTS

- Patients without structural heart disease tend to have fewer morphologies of ventricular tachycardia (VT), with automaticity and triggered activity a more common mechanism than re-entry, associated with extremely low risk of sudden death.
- Ablation can be curative in patients with a single morphology of VT that is focal in origin, particularly in patients without overt structural heart disease.
- There are limited data in secondary prevention implantable cardioverter defibrillator (ICD) literature to support the routine implementation of ICD in normal hearts.
- Antiarrhythmic drugs have not been shown to reduce all-cause mortality in patients with or without structural heart disease.
- Data examining the incidence of arrhythmic death in patients with structural heart disease with lower risk features such as tolerated VT and ejection fraction greater than 35% demonstrate a low incidence of annual sudden death. This data may potentially be extrapolated to patients without overt structural heart disease.

Catheter ablation has been demonstrated to be an effective therapy for ventricular arrhythmias across the entire spectrum of patients, with structurally normal hearts to advanced fibrosis with systolic dysfunction.[1,2] In the latter group, ablation is typically performed as an adjunct to antiarrhythmic therapy, with background implantable cardioverter-defibrillator (ICD) therapy. Although ICD is merely an abortive therapy, and antiarrhythmic drug (AAD) therapy is suppressive therapy, catheter ablation is the only curative treatment modality in the armamentarium for ventricular arrhythmias, short of cardiac transplantation. The ideal clinical setting for catheter ablation as a standalone therapy is in patient profiles without structural heart disease (SHD) that are at low risk for subsequent sudden death. In this article, the role of catheter ablation as first-line therapy in patients with apparently normal hearts is discussed. The comparative evidence for ICD and effectiveness of

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antiarrhythmic drugs in patients with normal ejection fraction (EF) are reviewed, as well as highlighted areas of uncertainty-advanced imaging methods have the potential to identify "concealed" or subtle fibrosis and/or inflammation not previously recognized by angiography and echocardiography as "overt" SHD.

COMMON TYPES OF VENTRICULAR TACHYCARDIA IN STRUCTURALLY NORMAL

Sustained ventricular tachycardia (VT) in patients without overt SHD is unrelated to myocardial scar and typically thought to be the result of automaticity or triggered activity. Patients with myocardial scar substrates harbor the ideal scenario for re-entrant mechanisms and typically have multiple circuits, yielding on average 2 to 3 VT morphologies seen during catheter ablation.[3,4] The curative potential for catheter ablation of VT is logically highest in patients with a single focus, represented by a singular electrocardiographic morphology.

In patients without overt SHD, the most common ventricular arrhythmias are the adenosine-sensitive VTs, accounting for approximately 90% of all idiopathic VTs.[5] The mechanism of these arrhythmias is triggered activity due to cyclic adenosine monophosphate mediated-delayed afterdepolarizations, with characteristic features of initiation and termination.[6,7] Two phenotypic forms of adenosine-sensitive VT are commonly described, with the more common presenting as frequent premature ventricular complexes (PVCs), couplets, and salvos of nonsustained VT that often occur at rest or after exercise. The less common form is precipitated by exercise or emotional stress, rather than being suppressed by it, and often presents as sustained VT.

The commonest location of VT in patients without overt SHD, historically called repetitive monomorphic VT, arises from the right ventricular outflow tract (RVOT). In one series, 75% of patients with sustained monomorphic idiopathic VT and 89% of with nonsustained VT were found to have an RVOT site of origin.[8] The arrhythmia classically occurs in younger age with adrenergic triggers, often at peak exercise or during the cool-down period. Sudden death is exceedingly rare, although short-coupled variants resulting in R-on-T phenomenon have been reported with polymorphic VT and ventricular fibrillation in rare cases.[9–11] Ablation success and elimination of this condition can be achieved with a single targeted radiofrequency application. After successful curative ablation, medications are typically discontinued. Structural abnormalities in the right ventricle need to be considered and excluded because the outflow tract comprises 1 of the 3 corners in the "triangle of dysplasia" seen in arrhythmogenic RV cardiomyopathy.[12]

Over the past 10 years, a greater appreciation of outflow tract arrhythmias with precordial lead transition at V3 and earlier on 12-lead ECG has prompted successful mapping and ablation approaches in the aortic root, within the cusps adjacent to the sinuses of Valsalva.[13,14] Focal mechanisms from myocardial fibers at the top of the left ventricular (LV) ostium at the junction of the valves are thought to be the pathophysiologic basis. The risk of sudden death does not appear to be higher compared with patients with RVOT origins. In a recent large-scale, multicenter analysis of outcomes of ablation of idiopathic PVCs, the overall acute success rate was 84%. Success rates were highest (93%) for patients with an RVOT site of origin, with the same patients exhibiting a trend toward the lowest major complication rate (2.1%).[15]

Idiopathic left VT, most commonly arising from the posterior fascicle, is the second most common cause of VT in the absence of overt SHD. Re-entry has been proposed as the common mechanism, with an antegrade limb in a slow verapamil-sensitive zone of the septum with exit and retrograde activation using the fascicle.[16,17] The mechanism of these tachycardias has been demonstrated via entrainment maneuvers.[18–20] This form of VT also has an excellent long-term prognosis,[21,22] although cases of tachycardia-mediated cardiomyopathy and sudden cardiac death have been infrequently reported.[23] VTs originating from the fascicular system are highly curable with ablative therapy, even when the clinical tachycardia cannot be induced and mapped in the electrophysiology laboratory.[24,25] Current guidelines support the primary role for ablation, and ICD implantation is not indicated in this population.

Based on the low overall risk for sudden cardiac death and high success rate of ablative therapy, the 2008 American College of Cardiology/American Heart Association/Heart Rhythm Society Guidelines for Device-based Therapy of Cardiac Rhythm Abnormalities classifies ICD therapy as class III (not indicated) for patients with outflow tract VT, idiopathic VT, or fascicular VT in the absence of SHD.[26] For these abovementioned reasons, catheter ablation is clearly the first-line approach for these VT phenotypes because it is curative in the vast majority of patients.

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