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Short Review **Clinical Cardiology Research and Reports** **Open Access**

Management of Idiopathic Ventricular Tachycardia

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Abstract

Idiopathic ventricular tachycardia in patients with an anatomically normal heart is a distinct entity whose management and prognosis differs from ventricular tachycardia associated with structural heart disease. The tachycardia's QRS morphology on surface electrocardiogram (ECG) predicts the site of origin and is commonly classified as right ventricular tachycardia or left ventricular tachycardia. Patients generally tolerate the tachycardia and sudden cardiac death is rare in this patient population. Treatment options include pharmacotherapy or catheter ablation.

Keywords: Ventricular tachycardia; electrocardiogram; catheter ablation; bipolar electrograms

Introduction

Ventricular tachycardia (VT) most commonly occurs in the setting of structural heart disease. However, when VT is discovered in a structurally normal heart (i.e., the absence of ischemia, valvulopathies, or other cardiomyopathies), it is termed idiopathic VT. Traditionally, idiopathic VT was thought to compromise around 10% of all VTs. Recently the overall incidence of idiopathic VT was found to be around 14/100,000 individuals, with similar rates between men and women. In general, idiopathic VT carries a more benign prognosis relative to VT associated with structural heart disease, though it remains an important cause of morbidity and mortality in otherwise healthy individuals.

The most common location for idiopathic VT to arise from is the right ventricular outflow tract (RVOT-VT). Cyclic AMP mediated, calcium-dependent, triggered activity is thought to be the mechanism of this arrhythmia. RVOT-VT commonly manifests in the third to fifth decades of life and is about twice as likely to occur in women compared to men.

ECG Signatures

The classic RVOT-VT exhibits a left-bundle pattern (QS in V1) consistent with RV activation followed by LV activation. The inferior leads (II, III, and aVF) characteristically have tall R waves, while leads aVL and aVR will exhibit deep S waves given the superior to inferior activation pattern. The precordial transition from negative (QS) to positive (rS/RS) in VT compared to that in sinus may help distinguish between RVOT and left ventricular outflow tract (LVOT-VT). If the precordial transition is later than that seen in sinus, the VT is likely to originate from the RVOT.

Important features which contribute to the ECG characteristics of RVOT-VT are the presence of supra-valvular muscle bundles, distal variable conduction (below the pulmonary valve) from extensions of muscle from these bundles, and the possibility of “dead-end tracts” or remnants of the conduction system which persist beyond the bifurcation of the bundle branches. These complex interconnections can result in varied exits from a single focus and subtle beat-to-beat variation in morphology.

Catheter Ablation

Activation mapping is the current gold standard for localizing an RVOT-VT exit. However, this VT can be quite difficult to induce in the electrophysiology (EP) lab given the mechanism of this arrhythmia and the implications and limitations occurring during the procedure. To help with induction, sedation can be kept to a minimum during vascular access, and it may even be required that the patient be minimally sedated or kept awake during mapping. Pharmacologic adjuncts can be used including caffeine, isoproterenol, epinephrine, and/or phenylephrine to help with induction. Finally, both atrial and ventricular burst pacing can stimulate catecholamines and calcium loading to augment induction of VT. Pace-mapping can be used as an adjunctive strategy, especially when non-inducible, but it is important to remember a closely matched pacing morphology near the endocardial exit may be some distance from the successful ablation site.

Idiopathic VT can arise from the left ventricular outflow tract (LVOT), and regions in close proximity which include the aortic valve cusps, sinus of Valsalva, aorto-mitral continuity, and LVOT epicardium. Compared to the RVOT-VT, LVOT-VT appears to be more common in males and presents about a decade later in life. Like with RVOT-VT, the underlying mechanism appears to be cyclic AMP mediated, calcium-dependent triggered activity.

Catheter Ablation of LVOT-VT

Similar to RVOT-VT, the LVOT-VT can be difficult to induce due to the underlying mechanism. Often, strategies discussed in the RVOT-VT section can be of similar use in these cases, i.e., reducing or avoiding sedation, utilizing pharmacologic adjuncts for stimulation, and attempting rapid burst pacing.

Ablation within the distal coronary sinus and its distal branches has the issue of high impedance and coronary artery proximity which limits the ability to deliver adequate power, if at all, and may preclude ablation. One should always perform a coronary angiogram to delineate coronary artery anatomy and proximity prior to ablation from this region. Increasing irrigation flow prior to coming on ablation and gradual uptitration from a lower power can be helpful given the

high impedance in this region. Subxiphoid epicardial access has at times been utilized but is usually not successful given the location relative to the coronary artery which precludes ablation, as well as the epicardial fat that precludes adequate ablation energy delivery to the key region.

Conclusion

Even though in the majority of circumstances, idiopathic VTs are not considered life-threatening arrhythmias, prevention of recurrences by means of antiarrhythmic drugs is often ineffective and, the patients' quality of life remains unaffected. Different options of treatment need to be discussed with the patient, portraying the whole scenario. The overall acute success rate of catheter ablation is about 85-90% with a long–term prevention of arrhythmia recurrence of about 75-80%. The overall complication rate is about 2%, with severe complication rate < 1%.

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