Idiopathic Ventricular Tachycardia: Transcatheter Ablation Or Antiarrhythmic Drugs?

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Abstract

Introduction: Ventricular tachycardia or frequent premature ventricular contractions (PVCs) can occur in the absence of any detectable structural heart disease. In this clinical setting, these arrhythmias are termed idiopathic. Usually, they carry a benign prognosis and any potential ablative intervention is carried out if patients are highly symptomatic or, more importantly, if frequent ventricular arrhythmias can lead to ventricular dysfunction.

Methods: In this paper, different forms of idiopathic ventricular tachycardia are reviewed. Outflow tract ventricular tachycardia from the right ventricle is the most frequent form of the so-called idiopathic ventricular tachycardia. Other forms of idiopathic ventricular arrhythmias include ventricular tachycardia/PVCs arising from tricuspid annulus, from the mitral annulus, inter-fascicular ventricular tachycardia and papillary muscle ventricular tachycardia. When interventional treatment is deemed necessary, detailed mapping (earliest activation during VT/PVC, pace mapping) is crucial as to identify the successful ablation site. Catheter ablation more than antiarrhythmic drug treatment is usually highly effective in eliminating idiopathic ventricular arrhythmias and providing prevention of recurrence.

Conclusions: Idiopathic VTs are not considered life-threatening arrhythmias and, prevention of recurrences is often achieved by means of catheter ablation that provides an improvement of quality of life. The overall acute success rate of catheter ablation is about 85-90% with a long-term prevention of arrhythmia recurrence of about 75-80%. It is advisable that the procedure is carried out by electrophysiologists with expertise in VT catheter ablation and extensive knowledge of cardiac anatomy as to ensure a high success rate and reduce the likelihood of major complications.

Introduction

We define idiopathic ventricular tachycardia (VT) as all arrhythmias originating from the ventricles not linked to any detectable structural heart disease. They are usually due to focal triggered activity or re-entry between fascicular bundles. Detailed mapping of these arrhythmias are crucial because it can guide subsequent ablative intervention, that is often curative. Idiopathic VTs are quite a large group of ventricular arrhythmias that comprises different varieties:

a. Outflow Tract Ventricular Tachycardia
b. Tricuspid Annulus Ventricular Tachycardia
c. Mitral Annulus Ventricular Tachycardia
d. Interfascicular Ventricular Tachycardia
e. Papillary Muscle Ventricular Tachycardia

The scope of this paper is to highlight how to properly deal with VTs and/or frequent premature ventricular contractions (PVCs) in the absence of any detectable organic heart disease. These arrhythmias usually carry a benign prognosis, but patients are frequently symptomatic and, therefore therapy is warranted. Antiarrhythmic drugs (AADs) therapy may constitute a potential strategy of treatment, but generally they need to be administered chronically. Side effects and specific contraindications may limit the use of pharmacologic treatment without reducing the patients' symptoms. Furthermore, some drugs could induce significant adverse effects that includes negative inotropism and extra-cardiac toxicities, especially harmful if used in young patients.

Therefore, catheter ablation can offer an alternative, effective therapy for preventing arrhythmias.

Outflow Tract Ventricular Tachycardia

Among the group of idiopathic VTs, outflow tract VTs are probably the most frequent occurring arrhythmias due to a discrete focus. Classically, their origin is suggested by QRS morphology and axis. Patients are often symptomatic for non-sustained episodes or long period of premature ventricular contractions (PVCs) with the
same ECG morphology of VT. Almost 80% of outflow tract VTs arise from the RV outflow tract and the majority from site 1-2 cm below the pulmonary valve. Typically, a LBBB pattern with transition in the precordial leads at V3-V4 occurs. Transition at the precordial leads V1-V2 is suggestive of left side origin. Precise mapping of single PVCs is required to specifically identify the site of origin and guide successful ablation. Earliest local activation at successful ablation site precedes surface QRS by 20-45 ms; bipolar electrograms often show sharp rapid deflections whilst unipolar recordings typically demonstrate QS morphology. The presence of PVCs of the same VT morphology is critical to mapping and successful ablation; in those cases of sporadic occurrence of PVCs, pace-mapping can be employed to identify the successful ablation site. In case of paucity of PVCs or difficulty to induce VT during mapping at baseline, incremental pacing, burst pacing and sometimes atrial high rate pacing at baseline and during isoproterenol infusion are required to promote arrhythmia occurrence.

Less frequently, outflow tract VTs – PVCs originate from the left ventricle, aortic valve cusps or great arteries. On the other hand, some varieties can originate from the aorto-mitral continuity, the base of the septum or LV epicardium. In these circumstances, QRS complexes show inferior axis, but prominent R waves in V1-V2. Detailed mapping is often improved by using intracardiac echocardiography (ICE) as adjunctive means to properly localize the anatomic structures potentially involved. Proximity to coronary arteries may limit RF energy applications in these locations and, in some circumstances coronary angiograms is warranted. Aortic cusps VTs originate from sleeves of left ventricular myocardium above the aortic annulus. Even in these circumstances, activation mapping is an excellent approach (more than pace-mapping), but it is important, in order to achieve the highest success rate, to map all the different cardiac chambers before proceeding to ablate the earliest site. Usually, the results of catheter ablation in these locations are quite similar to those obtained in ablation of RV outflow tract VT. On the other hand, it is important to remind ourselves that possible complication rate related to this procedure include damage to the coronary arteries and, damage to the aortic valve, especially when RF current is delivered between the right and left commissures. Again, the use of ICE may be helpful in successfully guiding ablation and reducing the complication rate.

As far as the energy levels required to ablate, it is advisable to adopt different energy levels of radiofrequency current (RF) in relation to the anatomic location. Usually, we like to carry out the procedure by delivering RF current through an irrigated tip electrode catheter. In the RVOT, not more than 25 W-30 W are used, while less energy power is usually employed for sites like aortic root, aortic cusps and ablation within the coronary venous system. In some patients, ARVC/D at initial phase with prominent epicardial involvement and scarce endocardial signs, may produce ventricular arrhythmias mimicking idiopathic RV outflow tract VTs. In those circumstances, an attentive EKG morphology evaluation at baseline is of pivotal importance, since any signs of ventricular repolarization abnormalities may suggest more specific investigations, such as the need to provide a cardiac magnetic resonance imaging (MRI) to rule out an underlying pathologic substrate (i.e. occurrence of late gadolinium enhancement as sign of fatty/fibrous tissue infiltration). Along with non-invasive investigations, 3-D electroanatomic mapping is advisable to improve the likelihood to make the final diagnosis. (Tab.1) In some occasions, endomyocardial biopsy may
be required to properly distinguish different pathologic entities (RV dysplasia/cardiomyopathy, amyloidosis, sarcoidosis, etc.).

**Tricuspid Annulus Ventricular Tachycardia**

Spontaneous PVCs or VTs (approximately 8% of idiopathic VTs) could arise from the lower body of RV or regions 1–2 cm below the tricuspid valve. All these arrhythmias present LBBB pattern. Accurate mapping and subsequent ablation can be effective in promoting prevention of recurrence in > 80% of cases thus, highlighting the critical role of catheter ablation in these forms of arrhythmias. These arrhythmias are often resistant to antiarrhythmic drugs and, ablation is usually considered the most effective strategy of therapy. Also in these patients, it is of pivotal importance to rule out the occurrence of areas of RV scars, as possible signs of arrhythmogenic cardiomyopathy. Therefore, detailed voltage maps of the right ventricle is warranted as to identify additional signs of possible RV cardiomyopathy.

**Mitrail Annulus Ventricular Tachycardia**

Less frequently, idiopathic VTs/PVCs arise from the mitral annulus (about 5% of all idiopathic ventricular arrhythmias). In these circumstances, the most frequently involved area is the anteroseptal region of the annulus. Similar to aortic cusp VTs, a delayed potential is recorded during sinus rhythm at the valve annulus and, this potential precedes the QRS electrogram by 50–70 ms during VT or spontaneous PVCs (11). Endocardial ablation is highly effective in suppressing the arrhythmia and, it should be considered the first line strategy of treatment. Occasionally, an epicardial approach via coronary sinus system may be required. Even in this clinical setting, patients are quite symptomatic and the use of AADs is rarely effective for the control of the arrhythmias over time.

**Interfascicular Ventricular Tachycardia (Belhassen VT)**

This form of VT classically involves the posterior fascicles of left ventricle and demonstrates a RBBB pattern, left axis deviation pattern. It occurs in otherwise healthy patients, sometimes elicited by physical stress and could be terminated by verapamil infusion. Two other forms of verapamil-sensitive VT have been described; a left anterior fascicular VT with a narrow QRS complex, RBBB and right axis deviation and an upper septal fascicular VT with narrow QRS complex and normal axis. Due to the possible beneficial effect of intravenous verapamil, it has been hypothesized to be a Ca-dependent mechanism. Fascicular VT can usually be initiated with atrial extrastimulation, atrial pacing or ventricular pacing and, sometimes isoproterenol is required to reproduce the arrhythmia. During VT, activation propagates anterogradely, from the basal to the apical part of the LV septum, over the abnormal Purkinje tissue, giving rise to an anterograde late diastolic potential. Subsequently, the reentrant wavefront turns around in the lower third of the septum and activates the conduction Purkinje fibers. Therefore, for the classic form of posterior fascicular VT, RF current is directed at the anterograde apical Purkinje potential; the success rate of ablation for the reentrant wavefront turns around in the lower third of the septum and activates the conduction Purkinje fibers. Therefore, for the classic form of posterior fascicular VT, RF current is directed at the anterograde apical Purkinje potential; the success rate of ablation is > 85–90%. Intravenous verapamil can slow the tachycardia and then terminate, while long-term oral therapy with verapamil is not as effective as RF catheter ablation. Response of VT to lidocaine, sotalol, procainamide and amiodarone is less consistent and none of these drugs appear to be effective in the long run.

**Papillary Muscle Ventricular Tachycardia**

This is a distinct form of LV-VT arising form the papillary muscles and it can demonstrate a QRS pattern similar to fascicular VT. This arrhythmia can be catecholamine-sensitive and exercise-sensitive and clinically characterized by frequent PVCs rather than run of sustained VT. It is thought they have a focal automatic mechanism with spontaneous QRS variations that lacks in fascicular VTs. Intracardiac echocardiography is the ideal guide to detect the region of the papillary muscle that is the location of the early activation and for guiding the ablation catheter. Like fascicular VT, this variety of ventricular arrhythmia is poorly sensitive to long-term antiarrhythmic treatment.

**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%. It is advisable that the procedure is carried out by electrophysiologists with expertise in VT catheter ablation and extensive knowledge of cardiac anatomy as to ensure a high success rate and reduce the likelihood of major complications.

**References**

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