Catheter Ablation of Idiopathic Right Ventricular Tachycardia

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Catheter ablation of ventricular tachycardia has become an alternative to pharmacological, electrical, or surgical therapy of severe ventricular tachyarrhythmias. It may be successful in about two thirds of patients with ventricular tachycardia of various origins.1-7 Though catheter ablation of ventricular tachycardia is still an evolving, and thus experimental, technique, it has proven indispensable in those patients in whom pharmacological therapy has failed and in whom implantation of an antitachycardia pacemaker/cardioverter/defibrillator device is inadvisable (for example, patients in incessant ventricular tachycardia and/or those in whom map-guided antitachycardia surgery bears a high risk due to severely compromised ventricular function). It is in these patients that catheter ablation bears a procedure-related in-hospital mortality risk of 3.3% (based on our experience with a total of 92 patients [65 with direct current ablation and 27 with radio frequency current ablation]) (M. Borggrefe, C.H. Mief, U. Karbenn, G. Breithardt, 1990; unpublished observations).

However, this low risk-to-benefit ratio is irrelevant if the risk of the ventricular tachycardia to be treated is low, as in patients with idiopathic sustained right ventricular tachycardia. This rare entity has been reported to have a benign prognosis during a moderately long follow-up period.8 In this situation, therapeutic interventions cannot be used to improve the prognosis of these patients but to abolish symptoms. In patients with idiopathic right ventricular tachycardia, symptoms may range from palpitations to recurrent episodes of syncope.8 Experience with catheter ablation of idiopathic ventricular tachycardia has been lacking. Therefore, the report by Morady et al9 in this issue of Circulation represents a new and important contribution.

The authors9 report their experience with a total of 10 relatively young patients (mean age, 39±14 years; seven women and three men) with recurrent symptomatic, idiopathic sustained monomorphic ventricular tachycardia of one configuration originating in the right ventricular outflow tract and presenting with a left bundle branch block pattern and an inferior axis. In each patient, ventricular tachycardia was associated with symptoms of rapid palpitations, lightheadedness, and chest pain or dyspnea, as well as with syncope in one patient. No patient had previously been resuscitated. During baseline electrophysiological study, ventricular tachycardia was inducible by two premature stimuli in only three patients; in another three patients, isoproterenol was additionally necessary to facilitate induction of ventricular tachycardia. In the remaining patients, either atrial and ventricular pacing (n=3) or isoproterenol alone (n=1) was necessary. Catheter ablation was guided by pace mapping. One to three direct current shocks were delivered to the target sites. At electrophysiological study 7–9 days after ablation, ventricular tachycardia was inducible in only one patient. Another patient had a recurrence of ventricular tachycardia 3 weeks after ablation, whereas the remaining eight patients were free of recurrences and off antiarrhythmic drugs during a follow-up period of 15–68 months. It is noteworthy that no acute or long-term complications occurred.

At first glance, these impressive results may suggest that catheter ablation is the treatment of choice in relatively young but symptomatic patients with idiopathic ventricular tachycardia, eliminating the drawbacks of long-term antiarrhythmic therapy even if drug treatment has not failed. However, because of the lower risk-to-benefit ratio of patients without underlying heart disease compared with that of patients with coronary artery disease, careful consideration of the various factors involved is required.

**Diagnosis**

Morady et al9 state that their study population was composed of patients with idiopathic right ventricular tachycardia. Exclusion of structural heart disease, which was of utmost importance, was based only on echocardiography and the presence of a normal right ventricular ejection fraction by multigated radionuclide angiography. Left ventricular angiography and coronary angiography were normal in each patient.
However, right ventricular angiography was not performed in any case.

Two other major entities have to be considered in the presence of right ventricular tachycardia; morbus Uhl, a rare congenital disorder that manifests itself with right ventricular failure in childhood, and arrhythmogenic right ventricular disease or dysplasia, which usually manifests itself in adolescence. In the latter disorder, the diagnosis is based on the presence of right ventricular tachycardia, an abnormal regional contraction pattern of the right ventricle, and the detection of fibrolipomatous myocardial replacement ("dysplasia"). (In patients with right ventricular tachycardia in whom these histological features cannot be demonstrated in the presence of contraction abnormalities, the disorder should be called "arrhythmogenic right ventricular disease."

Although this definition of arrhythmogenic right ventricular disease seems to be straightforward, differentiation of a diseased right ventricle from a normal one may be difficult. The range of variability of angiographic and echocardiographic appearance of the normal right ventricle has been poorly defined. Kisslo reported a high degree of concordance between measurements of diffuse right ventricular changes by angiography and echocardiography, but the patients in that study had been selected on the basis of an abnormal angiogram. However, though the methods agreed as to the presence or absence of localized lesions, there was agreement as to their precise location in only eight of 14 instances. Other studies have also demonstrated a high correlation between a positive finding on echocardiography in patients with suspected right ventricular dysplasia and the likelihood of a positive angiographic finding, but the location of contraction abnormality found by echocardiography did not always correspond to the one detected by angiography. In another study, Illiceto et al reported that two-dimensional echocardiography was able to detect abnormalities of the right ventricle in 25% of cases with suspected right ventricular dysplasia but in none of the normal subjects. Conversely, 33% of cases with a negative echocardiographic study had a positive angiogram. Even with optimal recording conditions, detection of contraction abnormalities may vary considerably. The typical regional lesions with aneurysmal accumulations, dyskinesia, and isolated dilatation may be difficult to detect using echocardiography, particularly in minor cases. Echocardiography is of limited value for the assessment of the right ventricular free wall because of its proximity to the sternum. Newer echo systems using transducer frequencies of 5 MHz or more may improve echo visualization of involved segments of the right ventricle close to the chest wall. Thus, echocardiography has obvious limitations in detecting localized lesions. The sensitivity and/or specificity of two-dimensional echocardiography for detection or exclusion of right ventricular abnormalities needs further careful evaluation in a larger spectrum of cases. It is therefore doubtful whether a normal echocardiogram (like those of the patients in the study by Morady et al) excludes abnormalities of the right ventricle with sufficient certainty.

Because regional contraction abnormalities of the right ventricle do not necessarily result in a reduction of global right ventricular function, a normal right ventricular ejection fraction determined by multigated radionuclide angiography does not exclude the presence of arrhythmogenic right ventricular disease as the underlying heart disease in right ventricular tachycardia.

Clinical Course

Another obvious problem is related to the course of arrhythmogenic right ventricular disease, which has a wide spectrum of manifestations and may show progression during long-term follow-up. The possibility cannot be excluded that at least some of the patients in the study by Morady et al may have had an underlying structural abnormality not (or not yet) apparent on radionuclide angiographic or echocardiographic evaluation. Perhaps Morady and coworkers will be able to follow their patients on an extended long-term basis to identify those who might later progress to manifestation of right ventricular disease with or without evidence of fibrolipomatous infiltration of the right ventricular myocardium.

Correct differentiation of patients with right ventricular dysplasia from those with idiopathic ventricular tachycardia is important because the former has a worse prognosis than the latter. The 10-year mortality rate of right ventricular dysplasia ranges from 15% to 25%, which does not include those patients in whom sudden death is the first manifestation of the disease, in contrast to the low mortality rate of idiopathic ventricular tachycardia. However, in the latter study (M. Borggrefe, C.H. Mief, U. Karbenn, G. Breithardt, 1990; unpublished observations), methodological approach on excluding an underlying cardiac disorder has not been defined.

Catheter Mapping

Morady et al were able to show that catheter mapping techniques, mostly developed in patients with sustained ventricular tachycardia after myocardial infarction, can also be employed in patients with idiopathic right ventricular tachycardia. They correctly stress that the electrographic signals of their patients did not demonstrate the same type of early electrical activity as that typical of patients with previous myocardial infarction. Though they did not use pacing during sustained ventricular tachycardia at the site of subsequent ablation to demonstrate involvement of an area of slow conduction, they were able to obtain favorable results. However, any comparison of their data with findings in post–myocardial infarction patients may be difficult because of the unknown anatomical and electrophysiological peculiarities in idiopathic right ventricular tachycardia patients.
Complications of Catheter Ablation

Major complications include induction of a more severe, a more frequent, or a new type of sustained ventricular tachycardia, as well as wall perforation. Because all patients studied by Morady et al9 had ventricular tachycardia originating from the right ventricular outflow tract, the latter issue is of critical importance. Wall perforation of the right ventricle has been reported in a few instances, occurring even in institutions with a large-scale experience.5,23 Wall perforation may occur as a consequence of catheter manipulation and positioning technique, the type of electrical power source used, or anatomical reasons. The straight course that the ablation catheter may take from the inferior vena cava through the orifice of the tricuspid valve to the outflow tract of the right ventricle may make the catheter exceptionally stiff. The vigorous contraction of the ventricles that occurs during direct current ablation may then favor protrusion of the catheter through the free wall of the right ventricle. Theoretically, this type of complication may be avoidable by use of other power sources (such as radio frequency current) that do not cause contraction of the myocardium. After preceding experimental studies we never observed perforation of the free wall of the atria or of the ventricles with radio frequency ablation in 76 patients. These favorable results may be due in part to control of the temperature at the tip of the electrode catheter to avoid overheating and subsequent “cutting” effects.24,25 Increasing experience with the use of radio frequency currents for catheter ablation of myocardium suggests that wall perforation might be less frequent with this technique than with the use of direct current.

Wall perforation during application of direct current electrical shocks did not always occur at the site of ablation but also at remote sites, for example at the site of a pacing catheter positioned in the apex of the right ventricle.5 The apex of the right ventricle is a site with a very variable thickness that may range from 0.3 mm to 1.2 mm.26 The vigorous contraction of the ventricles during direct current shocks may favor protrusion of the catheter at such sites.

These considerations raise the important issue of whether catheter ablation in patients such as those treated by Morady et al9 should be done with surgical standby. The limited number of patients in their study does not, of course, exclude the possibility of catheter perforation occurring in a larger group of patients, which suggests that at the least, immediate pericardial drainage by needle puncture must be available. This should leave sufficient time to prepare for surgical intervention if still necessary. In the dramatic situation of wall perforation, it should not be forgotten that patients are usually receiving intravenous heparin, which should immediately be counteracted by administration of protamine sulfate.

Advantages of Catheter Ablation

If successful, catheter ablation has the clear advantage that the patient will not need further antiarhythmic medication. This may be important in female patients who wish to become pregnant or in those patients who have responded only to amiodarone, which has well-known long-term side effects. In a few patients with arrhythmogenic right ventricular dysplasia, we have observed, after initially successful catheter ablation, progression of the disease as evidenced by the appearance of a new type of right ventricular tachycardia previously not observed spontaneously and not inducible (M. Borggrefe, U. Karbenn, C.H. Mief, G. Breithardt, 1990; unpublished observations). In contrast to antitachycardia surgery, there are no major restrictions on performing another catheter ablation procedure if necessary.

Conclusions

Morady et al9 should be congratulated for their efforts to carefully describe their results of direct current catheter ablation in idiopathic right ventricular tachycardia. This study may serve as a stimulus to refinement of our diagnostic abilities by the use of newer approaches such as magnetic resonance imaging. In cases of right ventricular dysplasia, typical findings of magnetic resonance imaging are regional wall thinning, aneurysmal sacculations, and regional dilatation, as well as circumscribed increases in signal intensity as an indicator of fatty tissue infiltration.22-30 In addition, right and left ventricular volumes and ejection fractions can be assessed using dynamic cine imaging.31 These findings may help to noninvasively separate idiopathic ventricular tachycardia from right ventricular dysplasia.

References


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