Transcoronary Chemical Ablation of Ventricular Tachycardia

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After identification of the artery supplying blood to the arrhythmogenic area, transcoronary chemical ablation of ventricular tachycardia was undertaken in three patients with incessant tachycardia in whom the other therapeutic options had failed. Sterile ethanol (96%) was given at a dose of 1.5 ml in two patients and a total of 6 ml in the third. The arrhythmia was cured in two patients and suppressed during a 1-month period in the third until new collateral blood supply to the arrhythmogenic area developed and ventricular tachycardia recurred. The procedure was then repeated successfully. After administration of ethanol in the high interventricular septum, one patient developed temporary complete atrioventricular block and a pacemaker was implanted. No other complications occurred. We observed that in patients with ventricular tachycardia after myocardial infarction, it is possible to identify and catheterize small coronary arteries responsible for blood supply to the site of origin or pathway of ventricular tachycardia. After careful transcoronary mapping with saline, chemical ablation can prevent further episodes of the arrhythmia in selected patients. (Circulation 1989;79:475–482)

Ventricular tachycardia can be treated by antiarrhythmic drugs, electrical devices, surgery, and percutaneous electrical ablation.1–5 Experimentally aconitine-induced arrhythmias have been successfully treated by means of chemical ablation.6 Antiarrhythmic drugs and electrical devices result in successful control of ventricular tachycardia in a substantial number of patients.1–3,4 However, surgical techniques and percutaneous ablation are the only forms of treatment directed to destroy, remove, or isolate the arrhythmia substrate leading to definitive cure. Surgical treatment of ventricular tachycardia is associated with appreciable preoperative and postoperative mortality rates because of the extensive cardiac damage present in these patients. Percutaneous electrical ablation of ventricular tachycardia is still in an experimental phase. Major problems in the development of percutaneous electrical ablation are the small size of lesions created by the electrical shock requiring exact localization of the site of origin or pathway of the ventricular tachycardia.7,8 An arrhythmogenic area in the heart requires blood supply to preserve electrical activity of the myocardial cells involved in the mechanism of the arrhythmia. Evidence has been presented supporting reentry in surviving cells in the infarcted area as the mechanism of ventricular tachycardia after myocardial infarction.9,10 By using selective coronary angiography and cold isotonic saline, we have demonstrated that it is possible to localize the arterial blood supply to the site of origin of ventricular tachycardia after myocardial infarction.11 After identifying that artery, transcoronary chemical ablation of ventricular tachycardia was subsequently undertaken in three patients.

Patients and Methods

Three patients with incessant ventricular tachycardia in the chronic phase after myocardial infarction were studied (Table 1). The rate of incessant ventricular tachycardia varied between 130 and 160 beats/min while on antiarrhythmic drugs (amiodarone only for two patients, in combination with propafenone for one patient). Antiarrhythmic drugs were continued during the catheterization to control the rate of ventricular tachycardia. The first patient had incessant ventricular tachycardia with two QRS morphologies that were uncontrollable by antiarrhythmic drugs. Other morphologies of ventricular tachycardia that were not documented clinically at the time of the first study were also initiated by programmed electrical stimulation. Because of over-
all poor left ventricular function with a left ventricular ejection fraction of 11% and a New York Heart Association functional Class for dyspnea of III, the patient was considered a high-risk surgical candidate. The incessant character of ventricular tachycardia excluded the use of antitachycardia pacing or a cardioverter-defibrillator. Despite many attempts to control ventricular tachycardia with almost every available antiarrhythmic drug alone or in combination, the patient remained in incessant ventricular tachycardia. Patient 2 had undergone apicoseptal aneurysmectomy with placement of a septal patch because of multiple embolic phenomena arising from a post–myocardial infarction apical thrombus. At the time he was studied at our institution, he had been in incessant ventricular tachycardia for 1 month. Antiarrhythmic drugs slowed the rate of ventricular tachycardia, but the arrhythmia remained incessant. Both propafenone and mexilitine resulted in acute pulmonary edema. The patient was receiving amiodarone at a dosage of 400 mg/day when seen in our institution. Because of the previous surgery and poor left ventricular function, the patient was considered a poor candidate for reoperation. Patient 3 was sent to our institution because of incessant ventricular tachycardia uncontrollable by antiarrhythmic drugs. The arrhythmia was present for more than 1 month at the time of admission to our hospital. He had been receiving amiodarone for 7 years since he developed episodes of sustained ventricular tachycardia after an anterior wall myocardial infarction. Three months after the acute myocardial infarction, the patient underwent aneurysmectomy. Ventricular tachycardia recurred more and more frequently during this 7-year period until it became incessant 1 month before admission. His left ventricular ejection fraction was 20% and his functional Class for dyspnea was IV during incessant ventricular tachycardia. The patient was considered a poor surgical candidate. All patients gave informed consent to the different parts of the study.

Electrophysiologic Study

The three patients were continuously in ventricular tachycardia during the study. During ventricular tachycardia, endocardial mapping was performed with unipolar and bipolar recordings to localize the site of earliest electrical activity during the arrhythmia. Localization of the site of origin of ventricular tachycardia was done with results from endocardial mapping, results from pace mapping, the morphology and axis of the QRS complex during ventricular tachycardia, and the electrocardiographic location of myocardial infarction during sinus rhythm.12–14

Once the site of origin of ventricular tachycardia had been identified, coronary angiography was performed by the Judkins technique.

To identify the coronary artery providing blood supply to the site of origin of ventricular tachycardia, previously described methodology was used.11 The results of transcoronary termination of ventricular tachycardia with isotonic saline are described in detail elsewhere.11

Transcoronary Chemical Ablation of Ventricular Tachycardia

After demonstration of reproducible termination of ventricular tachycardia by administration of isotonic saline,11 transcoronary chemical ablation of ventricular tachycardia was undertaken in these three patients. For that purpose, 96% ethanol was used based on results from previous clinical studies in patients with renal tumors15,16 and recent observations by Inoue et al6 in the dog heart. Ethanol was given at a dose of 1.5 ml in two patients. The other patient received a total dose of 6 ml, as will be described later. After administration of ethanol, a 10-minute period was allowed before selective angiography of the same coronary artery was repeated to assess patency. Thereafter, programmed electrical stimulation was repeated to assess whether the arrhythmia substrate had been destroyed.

Results

Acute Termination of Ventricular Tachycardia by Ischemia or Isotonic Saline

As previously described,11 termination of ventricular tachycardia by occlusion of the vessel and administration of isotonic saline was attempted during the arrhythmia. In all three patients, repeated termination of ventricular tachycardia was obtained (an example is given in Figure 1).

Transcoronary Ablation of Ventricular Tachycardia

After demonstration of the reproducible termination of the two ventricular tachycardias by means of administration of iced saline through the first septal branch (Figure 2), definitive occlusion of this branch was planned. Occlusion occurred during manipulation of a balloon catheter. That was followed by immediate disappearance of ventricular tachycardia. A programmed electrical stimulation study performed immediately after occlusion and 7 days later failed to initiate any ventricular tachycardia. The patient was discharged on the same medication (amiodarone 400 mg/day and propafenone 900 mg/day) he was receiving unsuccessfully before occlusion.

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age (yr)/sex</th>
<th>Clinical diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>61/M</td>
<td>Inferior+anterior MI, incessant VT</td>
</tr>
<tr>
<td>2</td>
<td>44/M</td>
<td>Inferior+anterior MI, aneurysmectomy, incessant VT</td>
</tr>
<tr>
<td>3</td>
<td>62/M</td>
<td>Anterior MI, aneurysmectomy, incessant VT</td>
</tr>
</tbody>
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M, male; MI, myocardial infarction; VT, ventricular tachycardia.
The first septal branch has been selectively catheterized (Panel A), and retrograde blood supply to the posterior descending coronary artery (from the right coronary artery) is observed (arrows) (Panel A). Panel B: Termination of ventricular tachycardia by iced saline is illustrated. Four electrocardiographic leads were recorded simultaneously with the aortic pressure.

Sion of the septal branch. One month after discharge from hospital (5 weeks after occlusion of the septal branch), the patient returned to hospital with recurring incessant ventricular tachycardia of the same morphologies and a new morphology that also originated in the interventricular septum that had been documented at the time of programmed stimulation, although not spontaneously. A repeat coronary angiography (Figure 3) demonstrated persistent occlusion of the septal branch. However, collateral circulation to the left anterior descending coronary artery had developed from a conus branch.
FIGURE 2. Patient 1: in Panel A, the left coronary artery is shown (left anterior oblique projection). The first septal artery was selectively catheterized (Panel B). The open arrow points to the metal marker of the 2.5F catheter. Ventricular tachycardia was terminated by administration of iced saline or temporary occlusion of this septal branch (Panel C).
and a right ventricular branch from the right coronary artery (Figure 3). Both branches were occluded using ethanol in two successive sessions. Ventricular tachycardia lost its incessant character with the patient now having infrequent short-lasting episodes of slow, asymptomatic, and well-tolerated nonsustained ventricular tachycardia, which completely disappeared after 1 week. At the 9-month
follow-up, he had no recurrences of ventricular tachycardia.

In patient 2, occlusion or administration of isotonic saline through the first septal branch (Figure 4) reproducibly terminated ventricular tachycardia. Ethanol was given in a dose of 1.5 ml through the septal branch. Ventricular tachycardia disappeared and the patient developed, as expected, complete atrioventricular block with a junctional escape rhythm. A permanent rate-responsive VVI pacemaker was implanted 3 days later. Atrioventricular conduction recovered after a few days. Ventricular tachycardia was not inducible by programmed stimulation after ablation with ethanol. At the 6-month follow-up, no recurrences of the arrhythmia have occurred, and atrioventricular conduction is preserved.

After repeat demonstration of termination of incessant ventricular tachycardia by iced isotonic saline on the very distal part of the posterior descending coronary artery, transcoronary ablation with 1.5 ml ethanol was successfully performed through that vessel in patient 3 (Figure 5). No ventricular tachycardia was inducible immediately after ablation or 1 week later. At the 2-month follow-up without antiarrhythmic drugs, no recurrences of the arrhythmia had occurred. The functional Class of the patient improved to II.

Complications

During transcoronary termination of ventricular tachycardia, patient 1 complained of short-lasting chest pain after administration of isotonic saline. Interestingly, no patient complained of chest pain during selective occlusion of the coronary artery providing blood supply to the site of origin of ventricular tachycardia, and no patient developed a fall in blood pressure or showed an enzyme rise.
During administration of ethanol, no changes in blood pressure occurred. All three patients complained of short-lasting chest pain. The pain was acute and initially severe but disappeared within a few seconds probably because of immediate destruction of the nerve terminals. After a total dose of 6 ml ethanol, patient 1 had a rise of oxalacetic transaminase to 180 units. After 1.5 ml ethanol, patient 2 had a maximal value of the oxalacetic transaminase of 89 units (normal at our laboratory is less than 40 units). This value was 68 units in patient 3. Patient 2 developed transient complete atrioventricular block because of occlusion of the septal artery. No other complications occurred. The functional Class of the three patients did not worsen and improved in patient 3. A technetium pyrophosphate (and a MIBI nuclear study in two patients) did not show an area of myocardial infarction in any of the patients.

**Discussion**

The results of our study suggest that by using the presently available coronary angiographic and percutaneous transluminal coronary angioplasty techniques, combined with information from programmed electrical stimulation of the heart, it is possible to identify the coronary artery branch providing blood supply to the site of origin or pathway of ventricular tachycardia. Occlusion of the “tachycardia-related vessel” or administration of isotonic saline resulted in termination of the arrhythmia, most likely because of changes in the electrophysiologic properties of the cells involved in the arrhythmia mechanisms. Intermittent occlusion of the coronary vessel or administration of iced saline makes it possible to identify, or “map,” the coronary artery providing blood flow to the area of abnormal impulse formation; this is a prerequisite before definitive ablation of that area can be undertaken. Destruction of the cells responsible for the arrhythmia by ethanol resulted in cure of ventricular tachycardia in all three patients.

Although much more work is required before the real value and limitations of this technique for the treatment of ventricular arrhythmias are known, the data from the present study do demonstrate the feasibility of this approach. An important refinement will be to catheterize the smallest possible coronary artery allowing ablation of the area of
abnormal impulse formation. Ethanol, like similar alcohols used in the experimental situation, produces immediate dehydration and necrosis of myocardial cells. Depending on its concentration at the target site, a transmural necrosis occurs. At the same time, occlusion of the coronary artery in which ethanol is given occurs immediately (Figures 3 and 5). Detailed studies on the exact mechanism of action on both the coronary artery and the myocardium are lacking at the present time. The risks of creating an infarction at the area where ventricular tachycardia originates have to be carefully balanced against the possible benefits. Size of myocardial infarction should be as limited as possible.

Also, it will have to be assessed whether the lesion created is homogeneous and does not result in new arrhythmias. A small-to-moderate enzyme rise occurred after transcoronary ablation with ethanol. In neither the electrocardiogram or the nuclear study was a new myocardial infarction observed in any of the patients. The atrioventricular block in patient 2 was an expected complication because of the necrosis of the conduction system produced by the ethanol. Another important question to be answered is how often new collateral blood supply (as in patient 1) will lead to recurrence of the arrhythmia. Risks and benefits of transcoronary chemical ablation have to be compared with the risks of surgery or percutaneous electrical ablation of ventricular tachycardia in a particular patient. Surgery may destroy much more myocardium and has the additional risks of thoracotomy, cardiopulmonary bypass, and cardioplegia. Percutaneous electrical ablation probably results in too small a lesion to control ventricular arrhythmias or cause (in case of multiple shocks) generalized myocardial damage.

Clarification of these points will require further study to establish in the future the best possible therapy—surgery or chemical or electrical ablation. The data presented and results from experimental studies in the dog heart suggest a new concept in the treatment of tachycardias. Interruption of blood supply or chemical ablation of an arrhythmogenic pathway by way of the coronary arteries may result in cure of the arrhythmia.

References

Key Words • ventricular tachycardia • transcoronary ablation of tachycardias • percutaneous ablation
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