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Short Communications

Torsade de Pointes Secondary to d,l-Sotalol after Catheter Ablation of Incessant Atrioventricular Reentrant Tachycardia—Evidence for a Significant Contribution of the "Cardiac Memory"

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Summary: Radiofrequency catheter ablation of a right septal accessory pathway was performed in a 66-year-old patient with incessant orthodromic atrioventricular reentrant tachycardia. Intravenous administration of flecainide, ajmaline, verapamil, and d,l-sotalol had been ineffective in controlling the tachycardia. After the ablation procedure, precordial T-wave inversion was observed during sinus rhythm. These repolarization abnormalities persisted and were suggested to represent "cardiac memory." Three days later, atrial fibrillation with a fast ventricular response developed and oral d,l-sotalol, which had been well tolerated previously on a long-term basis, was started again. However, at this time, and in the presence of the persisting repolarization abnormalities, the T waves became deeper and broader within a few hours after the introduction of d,l-sotalol. Marked QT prolongation that was paralleled by the occurrence of repeated episodes of torsade de pointes developed. Serum electrolytes were normal. Direct current cardioversion was necessary due to the degeneration of torsade de pointes into ventricular fibrillation. Further sustained arrhythmia episodes were suppressed by temporary endocardial ventricular pacing. The patient recovered without any sequela. This case demonstrates that repolarization abnormalities after catheter ablation, which may be due, at least in part, to the "cardiac memory," are not always benign but may contribute significantly to proarrhythmia.

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Introduction

Time-dependent T-wave changes after abrupt alterations of the cardiac activation sequence are well documented. These abnormalities are thought to be due to the presence of a "cardiac memory"[1,2] and may occur with intermittent bundle-branch block,[3] after cessation of ventricular pacing,[4,5] after termination of a wide QRS tachycardia,[6] or after successful ablation of overt ventricular preexcitation due to an accessory pathway.[7,8]

We report a patient in whom repolarization abnormalities, presumably induced by the "cardiac memory," seem to have contributed to the occurrence of repeated episodes of torsade de pointes that developed shortly after administration of oral d,l-sotalol.

Case Report

A 66-year-old man was referred to our hospital for evaluation and treatment of incessant supraventricular tachycardia. Intravenous administration of verapamil, ajmaline, flecainide, and sotalol had been ineffective in controlling the tachycardia that, at the time of admission, had lasted for 8 days. The electrocardiogram (ECG) (Fig. 1) recorded during sinus rhythm 2 weeks before admission showed marked ventricular preexcitation. Eight years before, the patient had undergone mitral valve replacement due to the presence of mitral stenosis.

After written informed consent was obtained and with the patient in the postabsorptive state, an electrophysiologic study was performed by using conventional techniques of intracardiac recording and stimulation previously reported by our laboratory.[9] Intracardiac recordings obtained during ongoing incessant supraventricular tachycardia confirmed the diagnosis

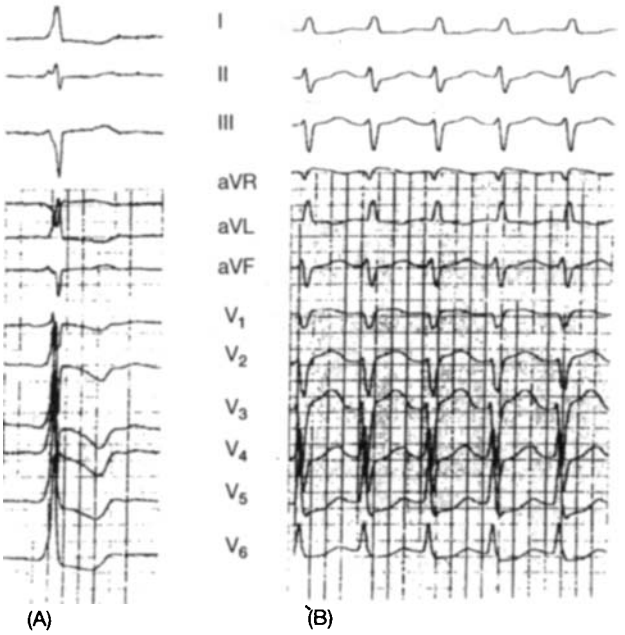

FIG. 1 (A) Baseline electrocardiogram demonstrating ventricular preexcitation. (B) Orthodromic reentrant tachycardia. 50 mm/s.

of orthodromic atrioventricular reentrant tachycardia involving a right septal accessory pathway. Programmed stimulation failed to terminate the tachycardia permanently.

During the ongoing arrhythmia and after detailed mapping of the tricuspid ring, a radiofrequency impulse was delivered via the distal pole of a 7F Polaris catheter (Mansfield Scientific, Inc., Watertown, Mass., U.S.). As radiofrequency generator, the HAT 200 (Dr. Osypka GmbH, Grenzach-Whylen, Germany) was used in a bipolar mode. This radiofrequency impulse resulted in abrupt termination of the tachycardia. Due to the presence of transient complete atrioventricular (AV) block, ventricular pacing was performed. Approximately 5 min later, 1:1 AV conduction resumed and pacing was stopped. Besides right-bundle branch block, regularly conducted sinus beats showed marked precordial T-wave inversion (Fig. 2A). The QT and QTc intervals in lead II measured 450 and 503 ms, respectively. QT interval dispersion, defined as the difference between the maximal and minimal QT interval occurring in any of the 12 leads on the standard ECG, was 100 ms. Rate-corrected QT interval dispersion (QTc interval dispersion) measured 110 ms. Conduction through an accessory pathway was ruled out by programmed atrial and ventricular stimulation. The patient had no symptoms of myocardial ischemia and there was no enzymatic evidence of myocardial damage.

After 48-h, anterograde and retrograde AV conduction were again assessed. There was no ventricular preexcitation during incremental atrial stimulation, and retrograde conduction was completely absent. No supraventricular tachycardia could be induced.

Repeated ECG recordings showed persisting precordial T-wave inversion. The duration of the QT interval remained almost unchanged.

Three days after the ablation procedure, the patient developed repeated episodes of self-terminating atrial fibrillation with ventricular rates ranging between 140 to 160 beats/min. Therapy with oral sotalol was reinitiated. The drug had been previously taken by the patient (daily dose 320 mg) on a long-term basis to prevent paroxysms of supraventricular tachycardia. The drug had been well tolerated, but due to inefficacy, sotalol was stopped 1 year before admission.

At this time, after the second dose of 80 mg, the patient complained of episodes of dizziness. The ECG showed an increase in T-wave negativity and further QT-prolongation (Fig. 2B). The QT interval in lead II was 520 ms (QTc: 548 ms). QT interval dispersion increased to 200 ms (QTc interval dispersion: 211 ms). A few minutes later, recurrent episodes

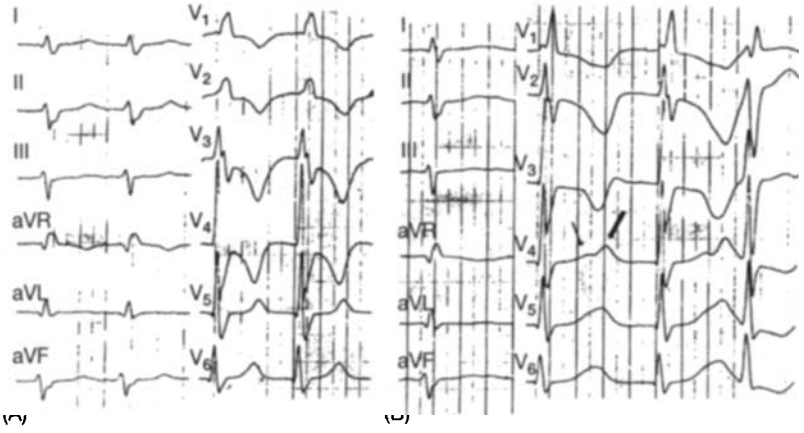

FIG. 2 Electrocardiograms recorded after ablation of incessant orthodromic reentrant tachycardia. (A) Immediately after ablation, there were marked T-wave abnormalities with deep T-wave inversion in the precordial leads. QRS duration: 140 ms. QT interval (lead II): 450 ms (QTc: 503 ms). (B) Deepening of the T-wave, further QT prolongation (small arrow) and U-waves (broad arrow) after initiation of sotalol therapy. QT interval (lead II); 520 ms (QTc: 548 ms). Premature beats were observed and recurrent episodes of torsade de pointes developed. 50 mm/s.

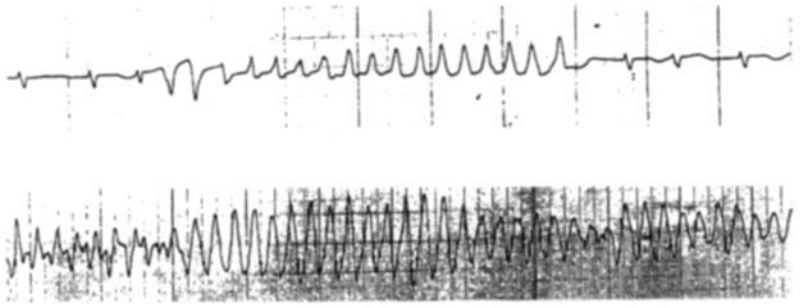

FIG. 3 Discontinuous rhythm strips showing torsade de pointes after initiation of sotalol therapy. 25 mm/s.

of torsade de pointes developed (Fig. 3). Due to degeneration into ventricular fibrillation, direct current cardioversion was necessary. Further sustained arrhythmia episodes were prevented by temporary right ventricular pacing at a rate of 100 beats/min. D,l-sotalol was immediately discontinued. Serum electrolyte levels were within normal limits: potassium 4.4 mEq/l (normal: 3.6 to 5.5 mEq/l), magnesium 1.68 mEq/l (normal: 1.3 to 2 mEq/l), sodium 139 mEq/l (normal: 132 to 155 mEq/l), calcium 4.4 mEq/l (normal: 4.2 to 5.8 mEq/l). Serum-creatinine measured 1,1 mg/dl and creatinine clearance according to the formula of Cockcroft and Gault was 75 ml/min.

Temporary right ventricular pacing was continued for 2 days. Serial ECGs recorded during sinus rhythm revealed persistence of right bundle-branch block (QRS duration: 140 ms) but slow regression of the T-wave changes and QT shortening over time (Fig. 4). Three weeks after the ablation procedure, the patient was discharged. At this time, QT and QTc intervals in lead II were 460 and 517 ms, respectively. QT and QTc dispersion markedly decreased to 40 and 46 ms, respectively. The patient received digoxin and verapamil for slowing of the atrioventricular conduction during episodes of atrial fibrillation. He was advised not to take sotalol or other repolarization-prolonging antiarrhythmic drugs in future. No further ventricular tachyarrhythmia recurred during a follow-up period of 24 months. Accessory pathway conduction did not resume.

Discussion

"Cardiac memory" refers to the persistence of abnormal T-wave polarity after reversion of the ventricular activation from abnormal to normal.[1,2] The resulting repolarization abnormalities have been considered as a physiologic response to an altered activation sequence. In the present case, incessant supraventricular tachycardia, abolition of conduction via an overt accessory pathway, and, although short but present, a period of ventricular stimulation due to transient AV block may all have contributed to the occurrence of the repolarization abnormalities observed after termination of incessant orthodromic tachycardia by successful catheter ablation.

Other than precordial T-wave inversion, a complete right bundle-branch block was present after catheter ablation. It can be suggested that the latter results from ablation of the proximal part of the right bundle branch due to catheter displacement during radiofrequency energy delivery. Distribution and morphology of the repolarization abnormalities observed after ablation differed from those that can be found in the presence of a right bundle-branch block. The occurrence of bundle-branch block and the development of T-wave inversion can be considered as separate entities.

In our case, the repolarization abnormalities markedly increased after oral administration of d,l-sotalol. It seems to be of particular interest that the drug was administered before and was well tolerated on a long-term basis. However, at this time, repeated episodes of torsade de pointes developed a few hours after intake of the second dose. Electrolyte abnormalities, especially hypokalemia and/or hypomagnesemia, which are known factors that may predispose or contribute to the development of torsade de pointes, or therapy with other repolarization-prolonging drugs were not present.

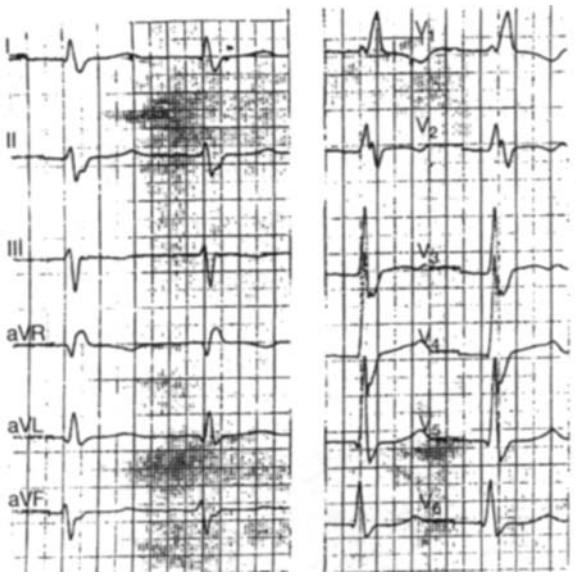

FIG. 4 Follow-up 12-lead electrocardiogram. Three weeks later, there is almost complete resolution of the repolarization abnormalities present after ablation. Right bundle-branch block persist. QRS (lead II): 140 ms, QT interval (lead II): 460 ms (QTc: 517 ms). 50 mm/s.

It seems conceivable that the cardiac memory-related repolarization abnormalities may have set the stage for the generation of torsade de pointes during sotalol exposure. Sotalol is a well known potassium-channel blocker, and it has been demonstrated that cardiac potassium channels are responsible or at least involved in the cardiac memory. It has been shown in intact dogs that the cardiac memory is abolished by 4-aminopyridine, which blocks both the transient outward potassium current, I(to), and the potassium current I(K).[5] Recently, Darpö *et al.*[10] described a patient with Wolff-Parkinson-White syndrome who developed torsade de pointes tachycardia after infusion of the class III agent almokalant. In their case, orthodromic tachycardia was repeatedly induced and terminated by programmed transesophageal stimulation before and after administration of the drug. It seems conceivable that similar mechanisms may have been operative in their case.

Our observation shows that repolarization abnormalities after catheter ablation, which may be due, at least in part, to cardiac memory, are not always benign but may contribute significantly to arrhythmogensis and proarrhythmia induced by antiarrhythmic drugs. Thus, careful monitoring is recommended when potassium-channel blocking antiarrhythmic agents are used in patients with repolarization abnormalities resulting from cardiac memory. Although speculative, similar effects may have played a role at least in some of the reported cases of unexplained sudden arrhythmic death occurring early after catheter ablation,[11] particularly after either direct current or radiofrequency current catheter ablation of the atrioventricular junction.[12]

References

1. Rosenbaum MB, Blanco HH, Elizari MV, Lazzari JO, Davidenko JM: Electrotonic modulation of the T wave and cardiac memory. *Am J Cardiol* 1982;50:213–222
2. Katz AM: T wave "memory:" Possible causal relationship to stress-induced changes in cardiac ion channels? *J Cardiovasc Electrophysiol* 1992;3:150–159
3. Engel TR, Shah R, DePodesta LA, Frankl WS, Krause RL: T-wave abnormalities of intermittent left bundle-branch block. *Ann Intern Med* 1978;89:204–206
4. Tsutsumi T, Izumo K, Sekiya S, Harumi K: Post-pacing T loop abnormalities. *Jpn Heart J* 1985;26:897–908
5. Del Balzo U, Rosen MR: T wave changes persisting after ventricular pacing in canine heart are altered by 4-aminopyridine but not by lidocaine: Implications with respect to phenomenon of cardiac "memory." *Circulation* 1992;85:1464–1472
6. Kernohan RL: Post-paroxysmal tachycardia syndrome. *Br Heart J* 1969;31:803–816
7. Hirai M, Tsuboi N, Hayashi H, Ito M, Inden Y, Hirayama H, Ito T, Yanagawa T, Sano H, Kondo T, Tomita Y, Tondoroki H, Tsuda M, Saito H: Body surface distribution of abnormally low QRST areas in patients with Wolff-Parkinson-White syndrome: Evidence for continuation of repolarization abnormalities before and after catheter ablation. *Circulation* 1993;88:2674–2684
8. Kalbfleisch SJ, Sousa J, El Atassi R, Calkins H, Langberg J, Morady F: Repolarization abnormalities after catheter ablation of accessory atrioventricular connections with radiofrequency current. *J Am Coll Cardiol* 1991;18:1761–1766
9. Chen X, Borggrefe M, Shenasa M, Haverkamp W, Hindricks G, Breithardt G: Characteristics of local electrogram predicting successful transcatheter radiofrequency ablation of left-sided accessory pathways. *J Am Coll Cardiol* 1992;20:656–665
10. Darpö B, Allared M, Edvardsson N: Torsades de pointes induced by transesophageal atrial stimulation after administration of almokalant. *Int J Cardiol* 1996;53:311–313
11. Evans G, Scheinman M, Bardy G, Borggrefe M, Brugada P, Fisher J, Fontaine G, Huang SKS, Huang WH, Josephson M, Kuck KH, Hlatky MA, Levy S, Lister JW, Marcus F, Morady F, Tchou P, Waldo AL, Wood D: Predictors of in-hospital mortality after DC catheter ablation of atrioventricular junction: Results of a prospective, international, multicenter study. *Circulation* 1991;84:1924–1937
12. Olgin J, Scheinman M: Comparison of high energy direct current and radiofrequency catheter ablation of the atrioventricular junction. *J Am Coll Cardiol* 1993;21:557–564