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CASE REPORT

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Transient atrioventricular block following catheter radiofrequency sinus node modification

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Abstract

Complete atrioventricular block, second-degree Mobitz type II and first-degree atrioventricular block with right bundle branch block were observed consecutively following successful radiofrequency ablation in close proximity to the sinus node. This resulted in the modification and disappearance of the inappropriate sinus tachycardia that had previously been present. Neither tachycardia nor conduction disturbances have been recorded in the 9-year follow-up, implying that ventricular pacing standby should also be considered in high atrial ablation cases. (Cardiol J 2007; 14: 504–507)

Key words: catheter ablation, radiofrequency, inappropriate sinus tachycardia, atrioventricular block

Introduction

Twelve years have passed since Waspe et al. [1] and Lee et al. [2] published the first works on sinus node modification in inappropriate sinus tachycardia (IST), but no data have been reported concerning subsequent atrioventricular (AV) conduction disturbances. We report on transient complications unique to IST ablation. In the case of the successful modification here presented, complete heart block, followed by Mobitz type II second-degree AV block and right bundle branch block (RBBB) with first-degree AV block occurred unexpectedly. Its mechanism cannot be clearly explained. Although it later reverted spontaneously to normal conduction, a less favorable clinical outcome is also likely. This makes ventricular lead introduction reasonable not only in ablation proximal to the AV node [3, 4], but also in cases of sinus node modification.

Case description

A 47-year-old female office clerk, the mother of three healthy children, had presented with fully symptomatic IST and remained in ambulatory follow-up for eight years. She had experienced long-lasting palpitations, dizzy spells, spots before the eyes and multiple presyncopal and syncopal states. Her resting heart rate had rarely dropped below 100/min except at night, showing markedly excessive acceleration during even moderate physical effort, and was concomitant with decreased exercise tolerance. Ambulatory, as well as detailed in-hospital diagnosis had been carried out in order to establish the etiopathological background. All potential extra-cardiac disorders were excluded as proven causative factors. Invasive and non-invasive cardiac examination, including ECHO, coronarography, ventriculography and myocardial biopsy failed to reveal any significant heart disease except for sinus arrhythmia. Treadmill tests were performed twice but discontinued at an early stage owing to presyncope after a heart rate of 164/min

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had been reached. Ambulatory ECG monitoring showed long-lasting sinus tachycardia of up to 220/min. In addition, antiarrhythmic pharmacotherapy, using all available drugs, appeared to be ineffective and resulted in the development of transient first-degree AV block. An ischemic or inflammatory etiology and AV node pathology were excluded, as, with a high degree of probability, was an ectopic origin. The electrophysiology (EP) studies performed twice revealed neither signs of sinus automaticity nor conductive insufficiency. Retrograde ventriculoatrial conduction was absent and the Wenckebach point appeared to be very high at over 220/min. We found no sign of dual node physiology or pre-excitation. Sinus node re-entry was excluded as, with a high degree of probability, was right atrial focal tachycardia. We performed sinoatrial node catheter radiofrequency (RF) modification on 6 January 1998. Following a detailed EP study, sinus tachycardia at a rate of 176/min, lasting over 20 min was easily initiated during slow intravenous administration of orciprenaline at a dose of 0.5 mg. Programmed atrial and ventricular pacing neither initiated nor terminated sinus tachycardia. All basic measurements, including AH, HV and QRS, remained within normal limits before and after the onset of arrhythmia. Endocardial mapping was performed using four electrodes placed at the following sites: the coronary sinus (Csp, Csd), the His bundle region (HBE), the high right atrium (HRA1, HRA2, HRA3, HRA4) and the ablative 7 F electrode (ABL) in the high right atrium. The first was introduced via the left subclavian vein and the others using the right femoral vein approach. Analysis of atrial impulse propagation before and during tachycardia revealed a physiological sequence. We observed the earliest atrial activation potential recorded from the HRA1 and HRA2 electrodes. At the last one we found the atrial potential to be prolonged and fractionated with a multicomponent appearance during tachycardia. The ablative electrode was placed in this vicinity, close to the sinus node region, at the junction of the superior vena cava with the posterolateral right atrium wall. We targeted RF energy at the site of earliest activation with fractionation of atrial potential during tachycardia. The second RF delivery of 25 W × 60 s resulted in abrupt termination of the sinus tachycardia of 170/min with restoration of a sinus rhythm of 75/min and normalization of the atrial potential morphology (Fig. 1). Two consecutive "consolidation" RF pulses were delivered, each of 60 s × 25 W. Shortly afterwards an unexpected 10-second episode of transient complete AV block was noticed with a junctional rhythm

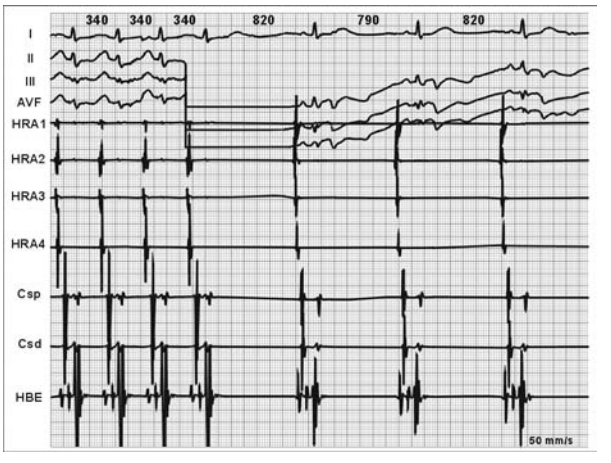

Figure 1. Surface and intracardiac electrogram recorded during radiofrequency energy delivery shows termination of sinus tachycardia of 170/min with restoration of sinus rhythm of 75/min.

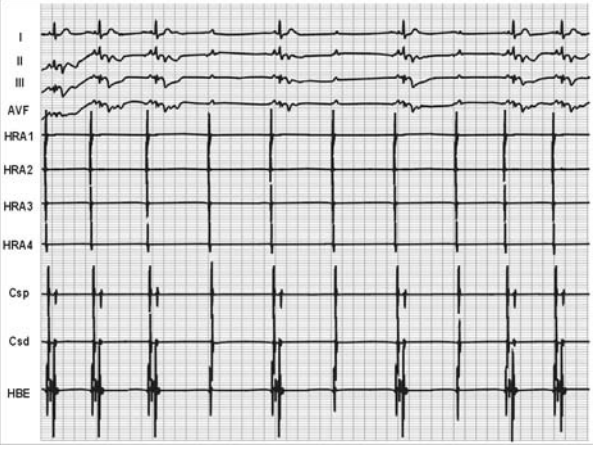

Figure 2. Surface and intracardiac electrogram recorded soon after radiofrequency sinus node modification. Second-degree Mobitz type II atrioventricular block.

of 40/min (not recorded on paper for technical reasons) and later second-degree block of the Mobitz II type (Fig. 2) and afterwards one-degree AV block with RBBB, which spontaneously disappeared after two days.

Owing to the nature of IST, there are no standard methods of assessment of the efficacy of ablation, although several findings have been noted after the procedure. In contrast to the initial state, slow sinus rhythm acceleration up to 135/min, and a gradual moderate slowing down was brought about by intravenous orciprenaline administration at a dose 1.0 mg. The atrial potential recorded from

the high right atrium electrodes, initially prolonged and fractionated during tachycardia, did not demonstrate such characteristics afterwards. The EP studies performed shortly afterwards and on the following day revealed that the basic parameters were unchanged. Hemodynamically essential sinus tachycardia remained non-inducible, either with pacing or with a beta-sympathomimetic agent. Holter monitoring showed none of the arrhythmia that had hitherto prevailed and a significant decrease in heart rate.

All antiarrhythmics, except for small doses of beta blocker, were withdrawn. During the nine-year follow-up we have found the patient to be in a good clinical state with a physical condition, presenting neither severe tachycardia nor the AV conduction disturbances she had experienced formerly.

Discussion

Inappropriate sinus tachycardia is an uncommon and still poorly defined chronic arrhythmia, which occurs in otherwise healthy people with no underlying structural heart disease. It is characterized by a positive P wave in II, III and aVF in ECG, typical impulse propagation and a high resting heart rate (usually over 100/min), with exaggerated acceleration on even moderate exertion. This rare disorder occurs mainly in young females and may lead to recurrent syncopal states [5–7]. Its mechanism and anatomic substrate are unknown.

There are some probable etiologic factors including the following: enhanced sinus node automaticity, automatic right atrial tachycardia in the closed vicinity of the sinus node, a depressed efferent cardiovagal reflex, beta-adrenergic hypersensitivity and excess sympathetic tone as well as a diminished parasympathetic tone regarding sinoatrial node innervation [5, 7]. Although IST is usually considered an idiopathic disorder, it must be pointed out that it is frequently observed as a result of atrioventricular nodal re-entry tachycardia and RF ablation of the para-Hisian accessory pathways [8–10]. This mechanism probably involves transient parasympathetic denervation [11]. EP studies carried out in IST cases have revealed a physiologic sequence of impulse propagation, with its earliest activation in the region of the sinus node. Although it is possible to differentiate between IST and sinus node re-entry, the exclusion of ectopic atrial tachycardia might be troublesome, since the response of both arrhythmias to programmed pacing is similar. The management of IST is still the clinical problem to be solved. Pharmacologic therapy with beta-blockers, calcium channel blockers, amiodarone and propafenone is empiric and usually not fully efficient [3]. For the remaining group of patients who have failed pharmacotherapy ablation should be considered. There is no precise method of verifying ablation efficacy, which is why the effects of both treatment methods, pharmacological as well as non-pharmacological, remain unsatisfactory.

Apart from the more aggressive RF destruction of sinus node function or the AV node with subsequent pacemaker implantation [12], the publications draw attention to sinus node RF modification [13, 14] with its sophisticated innovations [15, 16] as the potential therapy of choice in IST. The first descriptions of RF sinus node ablation in IST were published by Waspe et al. [1] and Lee et al. [2] in 1994, and since that time numerous cases have been published worldwide. To our knowledge, AV conductive disturbances following sinus node modification have not previously been reported, although they were described in cases of AV nodal re-entry and bypass-tracts [3, 4]. This and the long distance between the ablative electrode and the AV junction make this phenomenon difficult to explain. From the theoretical background the conclusion may be derived that the causative factors for these disturbances can be mechanical compression of the electrode over the AV junction and right bundle or transient excess parasympathetic or reduced sympathetic tone. The Bezold-Jarish reflex should not be excluded either.

Regardless of the exact localization of the arrhythmia substrate and its relationship to the sinus node central zone, the observation of AV block following RF application in the high right region is unexpected and difficult to explain.

Conclusion

Introduction of a back-up temporary pacing lead prior to supraventricular tachycardia ablation procedures should be considered even in a high atrial RF application.

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