Case Report

Atrioventricular block following prolonged focal cryoablation for parahisian premature ventricular complexes

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1. Introduction

Ventricular tachycardia originated from the parahisian region represents the 3%-9% of the total of idiopathic ventricular arrhythmias [1,2]. Catheter ablation is an effective treatment option in these patients, and cryoablation is often preferred as energy source over radiofrequency (RF) due to its capability for creating reversible lesions before established, permanent tissue damage [3–7]. Focal cryoablation – target temperature −80 °C – is often preceded by cryomapping – target temperature −30 °C – in regions close to the His bundle area. Although this helps the operator predict the effect of subsequent cryoablation, transient atrioventricular block (AVB) during cryoablation despite prior uneventful cryomapping has been reported in the literature [8,9]. Focal applications often last 6–8 minutes due to the dynamics of cryoenergy lesion formation [4,10].

We report the case of a patient with severely symptomatic parahisian premature ventricular complex (PVCs) who showed an unusual behavior following 6 minutes of cryoablation.

2. Case report

A 54-year-old woman with exertional dyspnea and incessant palpitations at rest was referred for electrophysiological study and ablation after treatment with beta-blockers failed due to poor intolerance even at low dose. Twelve-lead ECG during sinus rhythm showed very frequent PVCs, often with a trigeminal pattern (one PVC after every two sinus beats). The PVCs had a left bundle branch block pattern with a relatively narrow QRS, left axis, precordial transition after every two sinus beats (Fig. 1). Holter monitoring showed a high burden monomorphic PVCs (22% of beats in 24 hours), with a normal left ventricular function on the transthoracic echocardiography. After obtaining informed consent, electrophysiological study was performed under mild sedation. Baseline ECG on the day of the procedure showed a similar PVC density as was previously documented on the Holter monitoring.

Activation mapping was performed using a 7-Fr irrigated tip ablation catheter (Flexability, Abbott Laboratories, Chicago, IL) under electroanatomical mapping with Ensite Precision (Abbott Laboratories, Chicago, IL). Window of interest limits were set between −70 ms before QRS onset as measured in lead aVL and the peak of the R wave in lead aVF.

The right ventricular outflow tract (RVOT) and right ventricular septum were initially mapped. The area of interest was located on the mid septal region of the tricuspid annulus. Earliest local activation improved when positioning the catheter at locations where atrial electrogram (EGM) was present, immediately adjacent to a point where His deflection was observed. There, a ventricular EGM preceding QRS onset by 20 ms and with QS pattern on unipolar signal was recorded (Fig. 2).
Due to the high risk of AVB inherent to ablation in this region, the left ventricular outflow tract (LVOT) and aortic root were explored retrogradely. Earliest local activation - at the non-coronary cusp (NCC) - was 16 mm away from His signal, preceding QRS onset by 14 ms. At the deepest and most posterior portion of the NCC, a radiofrequency lesion titrated to a maximum of 30 Watts (17 cc/min irrigation with normal saline) showed no impact on PVC density, nor induced junctional beats.

The mapping catheter was then replaced by a 6-mm-tip cryoablation catheter (Freezor, Medtronic Inc., Minneapolis, MN) that was positioned at the site of earliest activation on the mid-septal tricuspid annulus (Fig. 3). After cryomapping at −30 °C showed no impact on atrio-ventricular (AV) conduction and a slight decrease in PVC density (Fig. 4, panel A), we proceeded with cryoablation at −80 °C for a planned total duration of 8 minutes and under PR interval monitoring. PVCs were abolished after the first 15 seconds of cryoenergy administration (Fig. 4, panel B).

AV conduction remained unchanged until 354 seconds of cryoablation, when a mild PR prolongation was observed but with PR value within normal range. Cryoablation was not stopped at that point, and few seconds later progressive PR prolongation leading to 2-to-1 AV block appeared (Fig. 5). Immediate termination of cryoablation led to prompt recovery with normal PR interval. After a 30-min waiting period, PVCs were still suppressed and did not reappear after isoproterenol infusion.

Follow-up visits were scheduled at three months, one year and two years. All of them included 12-lead ECG and 24-h Holter monitoring. After two years, the patient has remained asymptomatic, and no significant PVC has been reproducible detected.

3. Discussion

The main finding of this case is the presence of very late 2-to-1 AVB despite previously uneventful cryomapping and prolonged (beyond 350 seconds) cryoablation.
vicinity of the conduction system [4,10,11]. Nonetheless, adverse effects in atrioventricular conduction, although mostly transient, have been reported. Fischbach et al. [9] described a series of five patients that presented with conduction system injury during cryoablation of supraventricular tachycardia despite no evidence of deleterious effects during cryomapping. In their sample, AV block appeared within the first 15 seconds of cryoablation with rapid tissue recovery during thaw. Miyamoto et al. [8] described a series of ten patients with ventricular arrhythmias originating from the para-Hisian region treated with cryoablation. In their study, one patient with baseline first-degree AVB developed complete AVB that eventually required pacemaker implantation. In this case, AV conduction was unaltered during cryomapping at −30 °C but complete AVB appeared 5 s after reaching −80 °C.

In our case, we planned to perform a prolonged (8 minutes) cryoablation lesion because, after reaching the target temperature (−80 °C), PVCs remained present for 15 seconds. The patient presented PR prolongation after almost 6 minutes of cryoenergy application with subsequent progression to 2-to-1 AV block. Complete recovery was observed after thaw.

Previous experimental studies showed that the main determinant of lesion formation in focal cryoablation is the duration of applications [12]. The explanation proposed for this is simple: cryotherapy lesion formation is based on conductive transmission of cold temperatures, leading to intra and extracellular disruption and irreversible loss of function, followed by microvascular hemorrhage and further intracellular damage occurring during thaw [10]. This transmission of freeze requires time to reach depths beyond 5–6 mm, influenced by a proximity gradient from the catheter tip. This would imply that, in cases where cryoablation is performed directly on the conduction system or few millimeters from its structures, AV block appears in the first few seconds after

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**Fig. 3.** Site of earliest activation is displayed in white on the mid-septal tricuspid annulus.

**Fig. 4.** Panels A and B show reduction and complete suppression of PVCs during cryomapping (−30 °C) and cryoablation (−80 °C) respectively.
reaching the −80 °C target temperature even if previous cryomapping yielded no disturbances, as has been widely reported [7,9,13–15]. This behavior, where conductive transmission results in damage of neighboring structures, is also seen during atrial fibrillation cryoablation, explaining right phrenic palsy and oesophageal lesions [16,17].

Our case illustrates that conductive freezing/cooling is a dynamic phenomenon that continues to evolve even several minutes after its onset, and therefore should compel electrophysiologists to stay alert throughout the whole duration of cryoablation, even if conduction disturbances do not appear during the initial phase of cryoenergy application. To the best of our knowledge, this has not been previously reported in the literature.

4. Conclusion

Focal cryoablation is a particularly valued strategy when applications must be delivered in the vicinity of the conduction system. Our case illustrates that its effects evolve throughout the whole duration of the application and that the absence of conduction disturbances during the first minutes does not fully guarantee its safety.

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Credit statement

Carla Lázaro: conceptualization; Álvaro Marco: writing, reviewing & editing; Daniel Rodríguez: methodology.

Declaration of competing interest

No conflict of interest exists for any of the authors.

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