Radiofrequency ablation of an epicardial ventricular tachycardia through the great cardiac vein in a patient with mitro-aortic mechanical prostheses

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Introduction
Cardiac surgery is a source of epicardial adhesions which can subsequently limit access into the pericardial space for epicardial ventricular tachycardia (VT) ablation.

Furthermore, mechanical mitro-aortic prostheses preclude catheter access to the left ventricle.

In this setting, we describe the radiofrequency (RF) catheter ablation of an epicardial VT through the coronary venous system, in a patient who had undergone mitro-aortic valves replacement using mechanical prostheses.

Clinical presentation
A 60-year-old woman was referred to our institution for RF catheter ablation of a presyncopal, drug resistant, recurrent, and monomorphic VT with a cycle length of 380 ms. The patient underwent mitro-aortic valves replacement using mechanical prostheses coupled to surgical atrial fibrillation ablation 3 months prior to admission. In addition, she had a prior history of amiodarone-related hyperthyroidism. Laboratory samples and preoperative (mitro-aortic valves surgery) coronary angiogram were deemed normal. Left ventricular ejection fraction and other echocardiographic parameters were within the normal range. The VT had right bundle branch block morphology, inferior axis, and a slurred initial deflection consistent with a pseudo-delta-wave (Figure 1A). Three days before the procedure, warfarin was stopped and heparin was initiated. Anti-arrhythmic medications were discontinued five half-lives before the procedure. After written informed consent was obtained, an electrophysiological study was performed in the postabsorbable state under light sedation. Four hours before the procedure, aPTT, PT, and INR were 76 s, 58%, and 1.5, respectively. From the beginning of the study, frequent monomorphic premature ventricular contractions (PVCs) with the same characteristics of the VT, including couplets and triplets, were recorded. Of note, the patient had a rate-controlled left atrial flutter since the surgical AF ablation procedure was not completely effective. A 7 F irrigated-tip catheter (SPRINKLAR™ Medtronic, Inc., Minneapolis, MN, USA) was inserted through the right femoral vein and used for mapping and ablation purpose. Surface ECG and bipolar endocardial electrograms were continuously monitored and stored on a computer-based digital amplifier/recorded system (LabSystem Pro, Bard Electrophysiology, Lowell, MA, USA). Intracardiac electrograms were filtered from 30 to 500 Hz and measured with online calipers at 25–200 mm/s. Targeting PVCs was chosen as the ablation strategy due to the poor tolerance of the clinical VT. Mapping was performed at the RVOT and above the pulmonary valves. Both activation analysis during PVCs and pacemapping at those sites were poor. Indirect mapping of the LV was then performed through the CS. During PVCs, local activation was the earliest at the distal aspect of the great cardiac vein (GCV) (Figure 2), preceding the onset of the QRS complex by 15 ms. A fragmented potential was recorded at that site during PVCs (Figure 3) and pacemapping provided an identical (12/12) match

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with the VT morphology (Figure 1B). A single RF application at the distal aspect of the GCV (40°C, 20 W, 20 ml/mn) definitively eliminated PVCs (Figure 4). Afterwards, ventricular stimulation with and without isoproterenol infusion failed to induce any VT. ECG monitoring showed no ST-segment changes during or after RF energy application, and an echocardiographic control showed no pericardial effusion. Warfarin was re-initiated 6 h after the procedure. Heparin was stopped 3 days later when INR was 3.5. After the procedure, 24 h Holter monitoring showed neither PVCs nor VT. The patient has remained arrhythmia-free during the 9 months period of follow-up.

**Discussion**

Although the VT of our patient fulfilled the criteria described by Berruezo et al.\(^1\) (pseudo-delta-wave \(\geq 34\) ms in precordial leads, intrinsicoid deflection time \(\geq 85\) ms in V2, and RS complex duration \(\geq 121\) ms in any precordial lead), suggesting an epicardial origin, pericardial access, as described by Sosa et al.\(^2\) was limited in our case by a prior cardiac surgery source of epicardial adhesions. Further, activation analysis and pacemapping at the left coronary cusps were not performed due to the risk of catheter entrapment within the aortic mechanic valve. The subxiphoid surgical approach described by Soejima et al.\(^3\), which is feasible and safe,\(^3,4\) could have been applied in our case. However, before tempting a surgical approach, the good electrophysiological criteria obtained from the distal aspect of the GCV led us to attempt at first VT RF ablation through the coronary venous system based on the publication of Obel et al.\(^5\). Indeed, these authors recently described five cases of idiopathic epicardial LVOT VT, in which ablation through the coronary venous system was successful in all the cases without acute or long-term complications after a mean follow-up of 24 months, thus assessing the feasibility and the safety of such an approach. In addition, the absence of transition of the R wave of the VT in the precordial leads was consistent with an origin of the VT (or at least its possible exit) in the vicinity of the mitral annulus,\(^6\) thus in a possible close relationship with the great veins of the coronary venous system. Although a fragmented signal was recorded at the successful ablation site, and no late potential could be recorded, the exact mechanism of the VT and its aetiology is lacking. Indeed, neither adenosine injection nor entrainment manoeuvre was performed and no warm-up or cool down phenomena were observed during RF application. We suggest, however, a post-surgical fibrotic substrate source of micro-reentry as the possible mechanism of the VT based on VT behaviour.

In patients referred for left-sided VT catheter ablation, when the ECG is suggestive of a pericardial circuit, after confirming the fact that the VT has no critical endocardial participation, it could be interesting to map at first through the coronary venous system in order to determine if the critical component of the circuit can be reached through the coronary venous system. If this is the case, one can attempt to ablate the VT by this approach, which is feasible and safe.\(^5\) Conversely, if the mapping is disappointing or the circuit not reachable due to anatomical constraints, one could consider a surgical approach through a subxiphoid approach described by Soejima et al.\(^3\), which is feasible and safe.\(^3,4\)
Figure 3 Effective site of radiofrequency application. Surface ECG II, III, aVF, V1, and endocardial recordings from the distal tip (ABL 1–2) of the ablation catheter showing a fragmented potential during a premature ventricular contraction preceding the onset of the QRS by 15 ms. Note that the patient presents a regular and fast atrial activity (left atrial flutter). Sweep speed is 100 mm/s.

Figure 4 Effective radiofrequency application at the distal aspect of the great cardiac vein. Are shown surface ECG II, III, aVF, V1, and endocardial recordings from the distal tip of the ablation catheter (ABL 1–2). Sweep speed is 12.5 mm/s.
limitations, then a pericardial approach as described by Sosa et al.\textsuperscript{2} or another type of pericardial approach\textsuperscript{3} can be applied depending on the characteristics of the patient and his clinical history.

**Conclusion**

In selected patients, especially after cardiac surgery, the coronary venous system can be considered as a potential approach for the ablation of epicardial VT. However, it is paramount to recognize that epicardial VT RF ablation through the coronary venous system is limited by the accessibility of only certain segments of the LV and can only be applied to a limited number of patients.

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