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Catheter Ablation of Premature Ventricular Contractions From the Left Ventricular Summit

Ablación con catéter de extrasístoles ventriculares del summit ventricular izquierdo

To the Editor,

Approximately 12% of idiopathic left ventricular (LV) arrhythmias (VA) originate from the LV summit: a triangular region of the epicardial LV outflow tract with the apex at the bifurcation between the left anterior descending and left circumflex coronary arteries with its base formed by an arc connecting the first septal perforator branch of the left anterior descending coronary artery with the left circumflex coronary artery. It is transected laterally by the great cardiac vein (GCV) at its junction with the anterior interventricular vein (AIV) into an area accessible to ablation inferiorly and an inaccessible area superiorly. Electrocardiographically, right bundle branch block morphology with inferior rightward axis is typically observed. During recent years, ablation of LV summit VA has received increasing attention in the literature, given its significant frequency and the challenging technical aspects of catheter ablation.1–4

We report the case of a 59-year-old man with a history of ischemic heart disease and normal LV function who presented with palpitations and high density (30%) premature ventricular contractions (PVC) on 24-hour Holter recording. Beta-blockers and amiodarone were ineffective and he was scheduled for an

Figure. Kaplan-Meier survival curves according to body mass index.

0.57–1.54; P = .805; obesity, HR = 0.837; 95%CI, 0.49–1.42; P = .507). Similarly, there were no differences in the number of admissions for cardiovascular causes (obesity, HR = 0.986; 95%CI, 0.547–1.468; P = .663; overweight, HR = 0.981; 95%CI, 0.611–1.575; P = .936).

The conclusion drawn from this study, based on BMI analysis, is that obesity and overweight show no prognostic differences compared with normal weight for cardiovascular mortality, cardiovascular hospitalization, and appropriate and inappropriate therapies in this population of patients with HF and an ICD implant for primary prevention of SD.

However, the interpretation of these study results should take into account the limitations of the study. First, the conclusions are drawn from BMI analysis, which does not differentiate body fat from lean body mass. Second, we did not analyze distribution of body weight (peripheral vs abdominal) or other measurements of adiposity such as body fat percentage. In addition, no information was available on the proinflammatory and nutritional status of the study population. Furthermore, the available information on BMI was taken from the time of implantation only; therefore, possible changes in this parameter at follow-up were not considered. Lastly, the retrospective design of the study increased the risk of bias.
electrophysiologic study that was performed free from antiarrhythmic drugs in a conscious state. The PVC exhibited a QRS complex with a maximum duration of 147 ms, right bundle branch block morphology, QRS morphology in V1 and inferior axis of +90° (Figure 1A), suggesting a possible LV summit/outflow origin.1 Mapping and ablation were carried out using a 3.5 mm tip irrigated catheter (ThermoCool SF; Biosense Webster) facilitated by an electroanatomic mapping system (CARTO 3). The ablation catheter was first placed in the septal region of the right ventricular outflow tract where poor activation and pace maps were observed. The same catheter was then inserted in the coronary sinus but could only be advanced to the lateral mitral annulus region and consequently it was replaced by a 5 F multipolar catheter, which was successfully advanced out to the GCV/AIV junction. Activation mapping at this site showed a local ventricular signal of 15 ms pre-QRS with a near perfect pace mapping (97% concordance). Adjacent structures, such as the aortic-mitral continuity and the left coronary cusp (LCC) and right coronary cusp, were subsequently assessed with the LCC showing the second best activation (0 ms pre-QRS) and pace map (91% concordance) (Figure 1B and Figure 1C). Next, a safe distance (> 10 mm) between the ablation catheter in the LCC and the left main coronary artery was confirmed by coronary angiography (Figure 2A). Ablation was then carried out in the LCC with power initially set to 30 W and maximum temperature to 45 °C, aiming for a minimum impedance drop of 10 Ω (Figure 2B). Power was eventually titrated up to 50 W, which promptly terminated the PVC. During follow-up, the patient showed no further PVC.

Figure 1A: Twelve-lead electrocardiogram showing PVC; B: Intracardiac electrograms displaying an early fragmented (arrow) signal 15 ms pre-QRS on the distal bipole of the MAP catheter located at the GCV/AIV junction. A far-field rounded signal 0 ms pre-QRS was recorded on the distal bipole of the ABL catheter positioned at the left coronary cusp; C: Spontaneous PVC and pace-maps at the GCV/AIV junction and LCC with 97% and 92% concordance, respectively. ABL, ablation; AIV, anterior interventricular vein; GCV, great cardiac vein; LCC, left coronary cusp; MAP, mapping; PVC, premature ventricular contractions; RVA, right ventricular apex.
et al. reported successful ablation in 9 of 16 patients who had VA that were mapped marginally closer to GCV/AIV, describing an aVL/aVR Q-wave ratio < 1.45 to be predictive of successful ablation from the LCC (aVL/aVR Q-wave ratio was 1.33 in our patient). Finally, when ablation from the endocardium or the coronary venous system fails, a percutaneous epicardial approach may be considered. In a study of 23 such patients, epicardial ablation was attempted in 14 patients, being successful in only 5 of them, and in the remaining 9 patients, the VA origin was in the inaccessible area in close proximity to the major coronary vessels.

In summary, we present a patient with PVC from the LV summit with best activation and pace maps at the GCV/AIV junction. However, due to anatomical restrictions, ablation was carried out successfully from the LCC.

CONFLICTS OF INTEREST

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