Chapter 20
GCV (Great Cardiac Vein) PVC

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Abstract LV summit PVCs are not infrequent and sometimes could be ablated via coronary sinus in the great cardiac vein.

The patient was a 44-year-old man, with complaints of palpitation, and dyspnea on exertion. Frequent PVCs were determined in ECG; with an inferior axis; LBBB pattern with the early precordial transition, and breakthrough pattern in precordial leads (Fig. 20.1).

In ECG Holter monitoring there was about 20% PVC count. LVEF by echocardiography was about 40%. Ablation recommended.

EPS and ablation was done; using quadripolar catheter for RV potential recording and pacing; one decapolar catheter for CS; and an arterial line for retrograde aortic approach; and another vein for GCV mapping via CS.

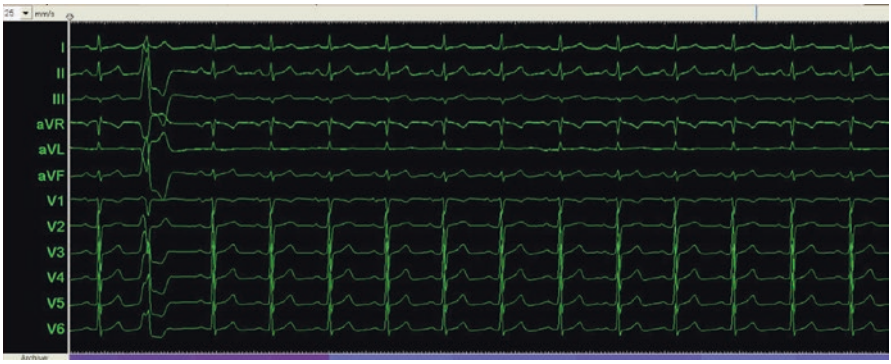

Fig. 20.1 PVC with breakthrough in V2, inferior axis, iso-neg in I, negative in aVL

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The procedure was done during conscious sedation using 3D Carto mapping (CARTO; Biosense Webster) (Fig. 20.2). A 3.5 mm open irrigation tip catheter was used. With the retrograde mapping of the aortic cusp, the earliest site of activation was an LCC with about 30 ms earliest signal but ablation in that area was unsuccessful.

So RVOT (right ventricular outflow tract) and GCV (Great cardiac vein) mapping was done. The earliest signal in GCV was found and was about 34 ms earlier than surface ECG (Fig. 20.3); and RF ablation in that area with 20 W and target temperature 43 °C resulted in the elimination of the PVCs abruptly, but because of the impedance rise in the coronary sinus, ablation was interrupted; and intermittent titrated RF currents were applied. PVCs were eliminated without recurrence after 30 min waiting period.

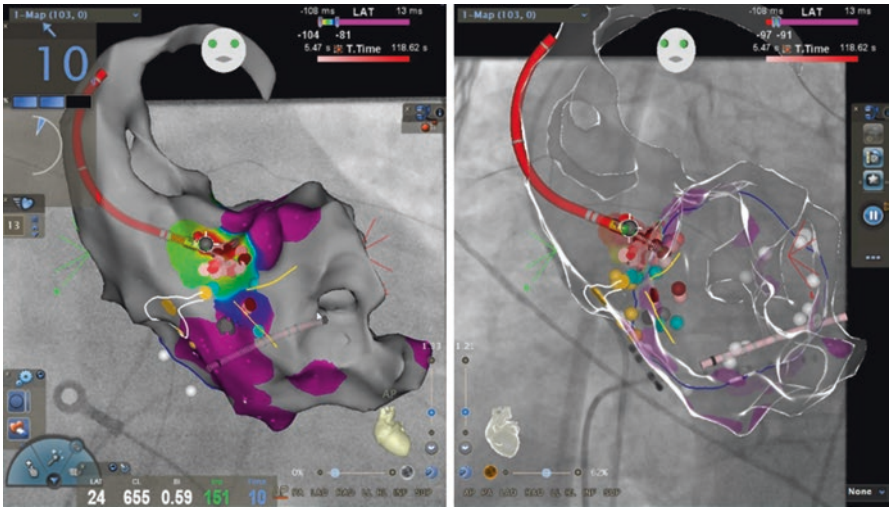

Fig. 20.2 CARTO mapping of PVCs (a left aortic cusp ablation was not successful)

Fig. 20.3 Earliest signal about 32 ms (in GCV)

Discussion

The most common site of idiopathic PVCs is in right and left ventricular outflow tract. Patients with a high PVC burden could develop a reversible from of LV cardiomyopathy. So PVC ablation could reverse the process of cardiomyopathy and cardiomyopathy could regress after PVC ablation [1, 2].

The classic ECG profile of outflow tract PVC is LBBB pattern and tall R waves in leads II, III, aVF, and QS pattern in aVR and aVL.

Idiopathic PVC could be ablated from aortic cusps. ECG criteria for RCC (right coronary cusp) PVC is a QS or rS pattern in lead V1, but LCC (left coronary cusp) PVC overall has multiphasic components with a positive complex in V1 [3, 4].

Target site for ablation in LCC is a site with very small presystolic up to 80 ms earlier than QRS onset with isoelectric segment before QRS and a delayed signal in NSR which reverse during PVC and a QS pattern in unipolar ablation recording, with an M or W shaped QRS complex. In RCC PVCs there is a QS or rS pattern in V1 and in right–left junction the potential in V1 has a notching on downstroke or a QS pattern [5, 6].

AMC (Aortomitral continuity) PVCs have RBBB, inferior axis, and q R pattern in V1. The LV summit is a triangular portion of the LVOT epicardium between LAD and LCX bifurcation. Epicardial LV summit PVC, have a maximum deflection index in the precordial leads. LVS is dissected by GCV into the basal and apical portion of the triangle [7–9].

In this PVCs, there is a QS pattern in lead I about 30%, and early precordial R wave transition in 70%. Intrinsicoid deflection time is also prolonged. LV summit arrhythmia could be ablated via GCV or via the epicardial approach [9].

Epicardial approach is only successful for ablation of the PVCs arising from the basal and lateral aspects of the LV summit, because of proximity to coronary vessels.

Because of epicardial fat, basal, and apical LV summit epicardial ablation is mostly unsuccessful.

ECG criteria predictive for the basal or lateral summit are: *Q* wave ratio >1.8 in aVL/aVR, $R/S > 2$ in lead V1 and absence of *q* wave in lead V1 [9].

Ablation with irrigated tip catheter in the cusps and GCV should be done with caution and start with 10–15 W and increase slowly to 30 W.

In PVCs originating for GCV, coronary angiography should be done before an attempt for ablation.

The acute success rate of ablation in idiopathic PVC is about 80 and 70% with and without antiarrhythmic drugs, respectively.

Complications during ablation consist of the development of bundle branch block or AV block, or aortic regurgitation or pulmonic regurgitation or coronary damage.

Sometimes the origin of the PVC is midmyocardial and longer duration lesions are required.

Bipolar ablation could be done for eliminating mid myocardial foci.

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