Chapter 27
Epicardial Approach for VT Ablation in an ARVC Case

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Abstract A young old man, with frequent episodes of VT, referred for ablation suspected for sarcoidosis and ARVC in CMR, after ICD implantation presented with VT storm, without any endocardial scar and with extensive epicardial scar in voltage mapping, so epicardial VT ablation was done.

Patient was a 36 y/o man, referred from elsewhere with diagnosis of RVOT–VT and hemodynamic compromise and termination of the arrhythmia with D/C shock. Echocardiography revealed moderate RV dysfunction and mild LV dysfunction, so cardiac MRI was recommended for patient.

Cardiac MRI revealed patchy areas of Late gadolinium enhancement (LGE) suggesting fibrosis in the RV inflow and outflow tract and another site of LGE in the mid posterolateral wall of LV with LVEF about 45%, and moderate RV dysfunction.

Patient was infertile.

The first diagnosis in cardiac MRI was Sarcoidosis and ARVC was recommended as the second diagnosis (Fig. 27.1a, b).

ICD was implanted for patients.

He refused ablation so was referred for Rheumatology consult.

Three weeks later he presented with VT storm.

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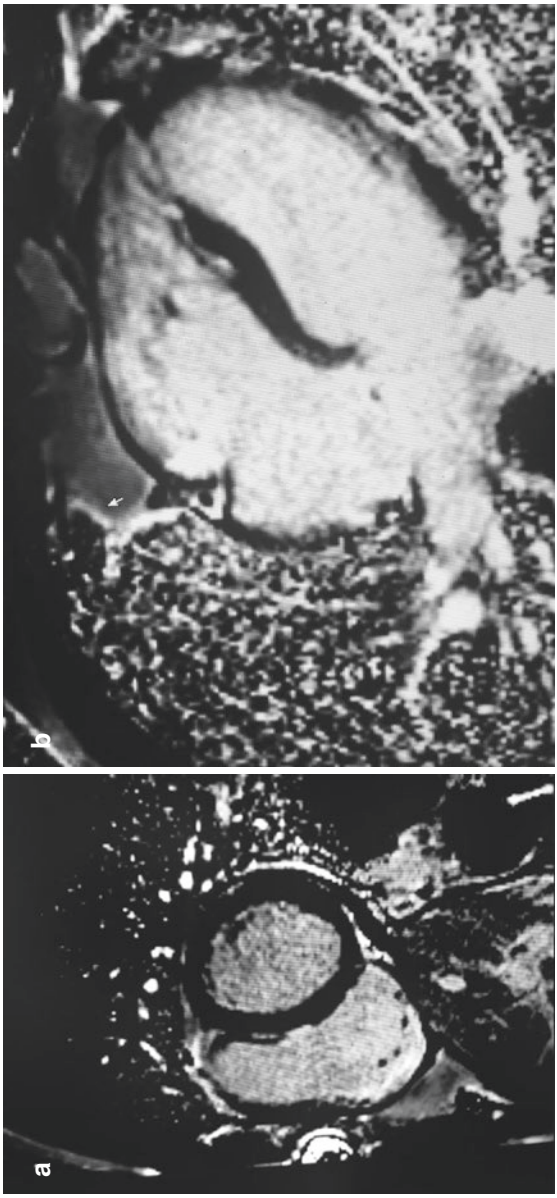

Fig. 27.1 (**a**, **b**) LGE in CMR in the septum, RV free wall and a small portion in the LV posterolateral are

Ablation

We tried to do endocardial biopsy using guide of 3D voltage mapping, but interestingly there was not any low voltage area in bipolar voltage mapping, but unipolar mapping revealed large scar burden, so ARVC with RV epicardial involvement was considered for him.

We did not do an endocardial biopsy because of the absence of any site of the endocardial scar.

So we tried to do an epicardial approach for VT ablation.

Subxiphoid puncture was done and 3D electroanatomical mapping of the epicardium revealed multiple sites of patchy scars in the RVOT and RV inflow areas.

Because of hemodynamic deterioration with VTs (Figs. 27.2 and 27.3), substrate modification was done for patients (Fig. 27.4a, b).

Before ablation coronary angiography was done determination of the position of the coronary arteries (Fig. 27.5).

We ablated all of the entrance conducting channels and late potentials in the RVOT and RV inflow (Figs. 27.6 and 27.7).

No arrhythmia was inducible after ablation with three ventricular extrastimuluses and Isuprel infusion (Fig. 27.8), so ablation was terminated and no arrhythmia was present in the 1 and 3 months follow-ups later.

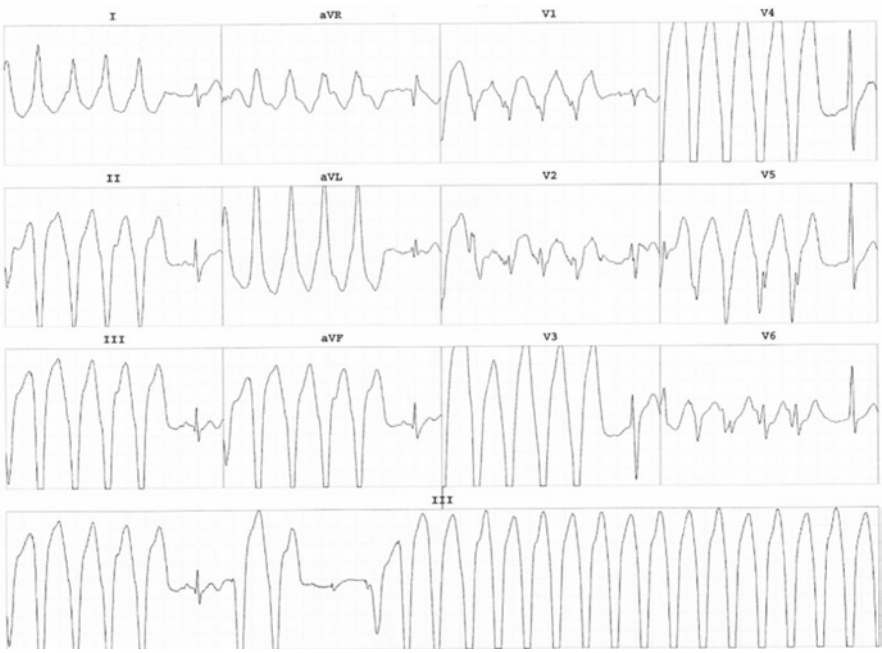

Fig. 27.2 VT1, Induced by VES (ventricular extra stimulation), the superior axis with negative concordance and positive I, aVL

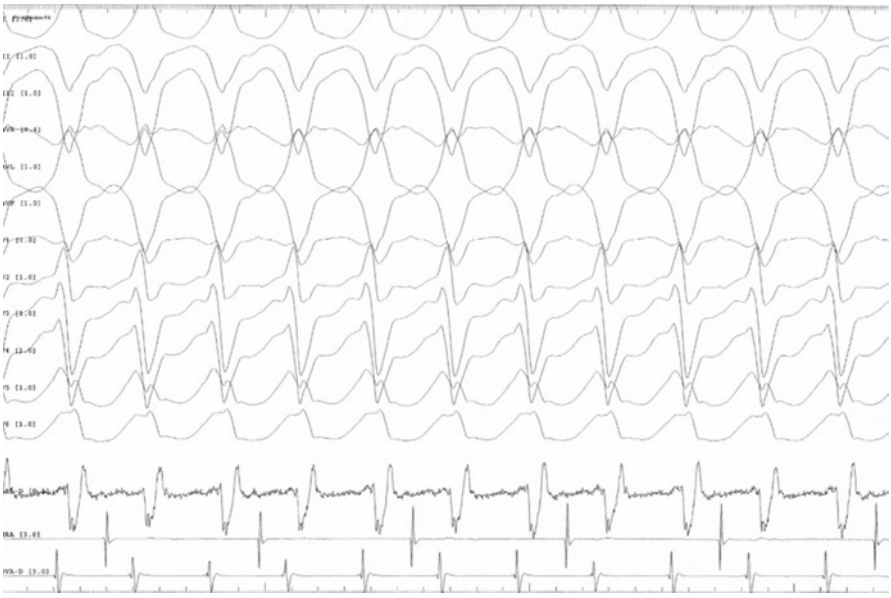

Fig. 27.3 VT2. Superior axis, breakthrough in V2, positive I, aVL

Discussion

Scar distribution patterns in cardiac MRI may be helpful by indicating disease-specific patterns of fibrosis on LGE images. Cardiac Sarcoidosis is a diagnostic challenge and work up for it requires biopsy [1–3].

A scar pattern of basal septal RV involvement may be infavor of Sarcoidosis, but does not prove it.

In ARVC, the disease process starts in the epicardial RV where fibrofatty tissue replaces myocardial tissue.

In about 50% of the patients with ARVC, for cardiac MRI and voltage mapping was done, endocardial voltage mapping failed to detect areas of scar, especially in the inferobasal part of RV [4–6].

Involvement of LV is present in up to ¾ of the ARVC patients [7–9].

An epicardial approach is often necessary to eliminate VT in ARCV patients. The presence of epicardial scar could be assessed by unipolar mapping.

Low amplitude electrograms in bipolar mapping are defined in the endocardium by less than 1.5 mv voltage and in the epicardium by less than 1 mv voltage.

Endocardial unipolar signals suggesting midmyocardial or epicardial substrates are defined as less than 5.5 mv in RV and 8.3 mv in LV [10, 11].

In this patient we did scar dechanneling with ablation of the conducting channels entrance sites within scar, characterized by earliest late potentials following global ventricular activation and end point of the ablation was the elimination of all conducting channels into scar. Following targeted ablation at entrance sites and

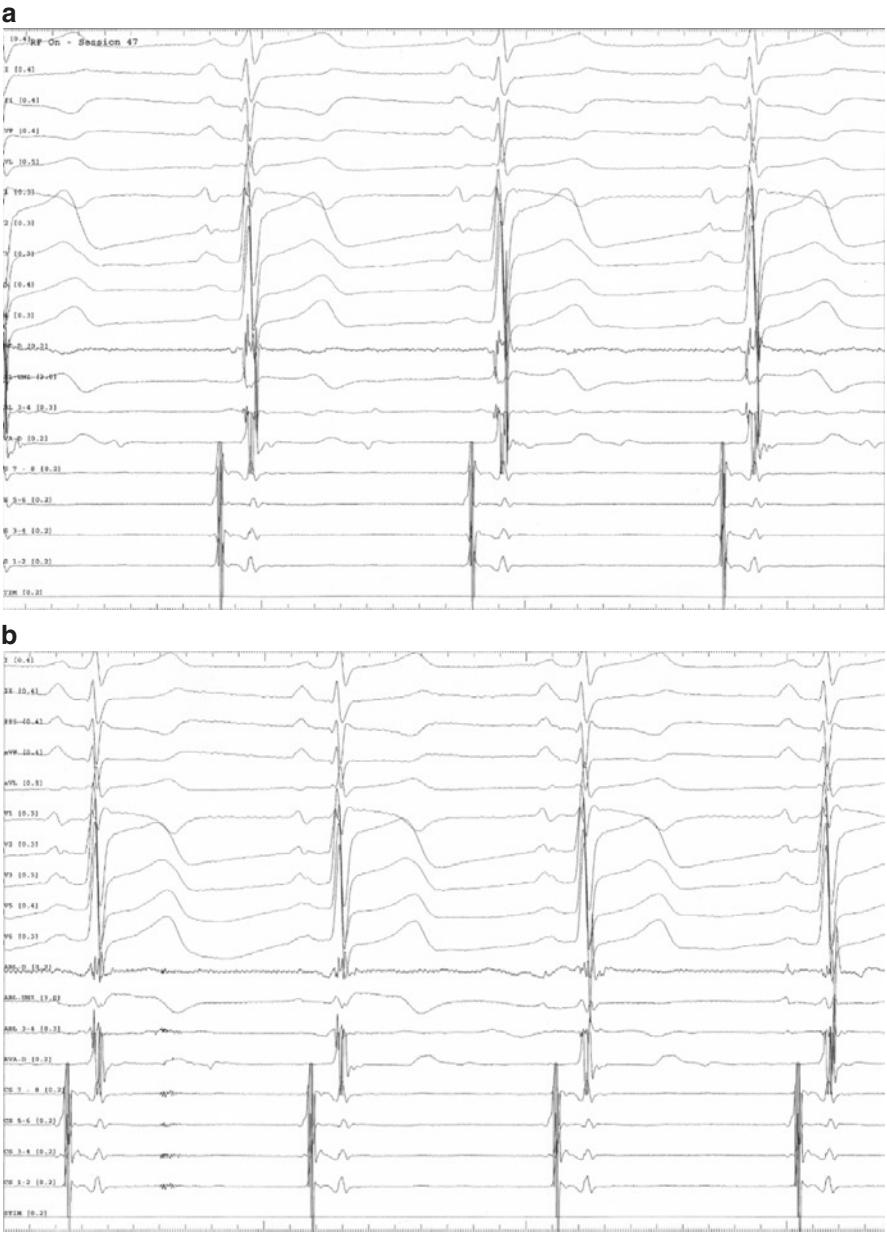

Fig. 27.4 (**a**) LAVAs (Local abnormal ventricular activity) in RV epicardium. (**b**) Disappearance of late potentials (LPs) after ablation

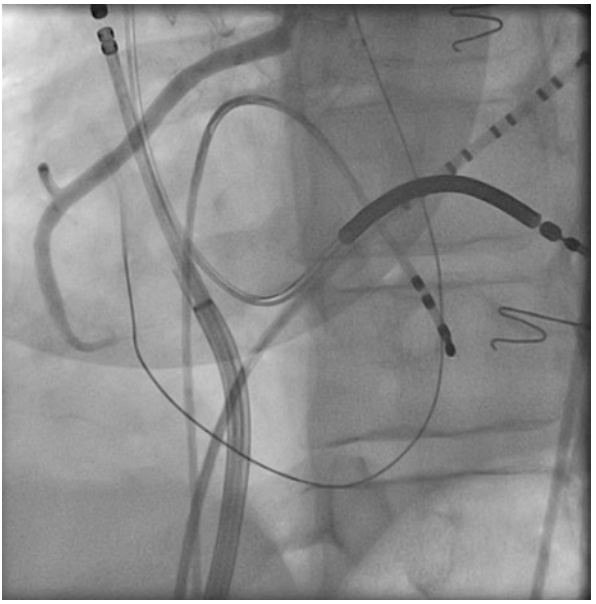

Fig. 27.5 Coronary angiography before epicardial RFA, epicardial ablation catheter is present

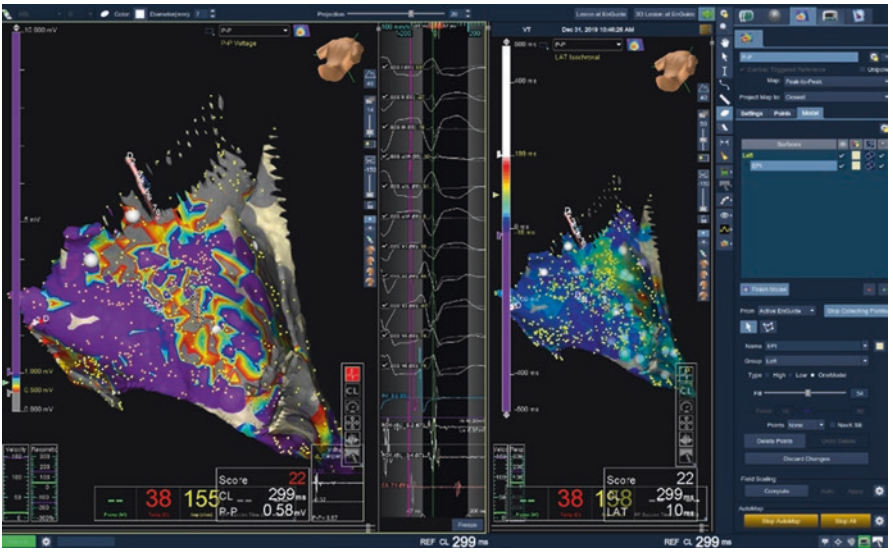

Fig. 27.6 Extensive ablation of RV epicardium, targeted entrance conducting channels, using LAT of late potentials

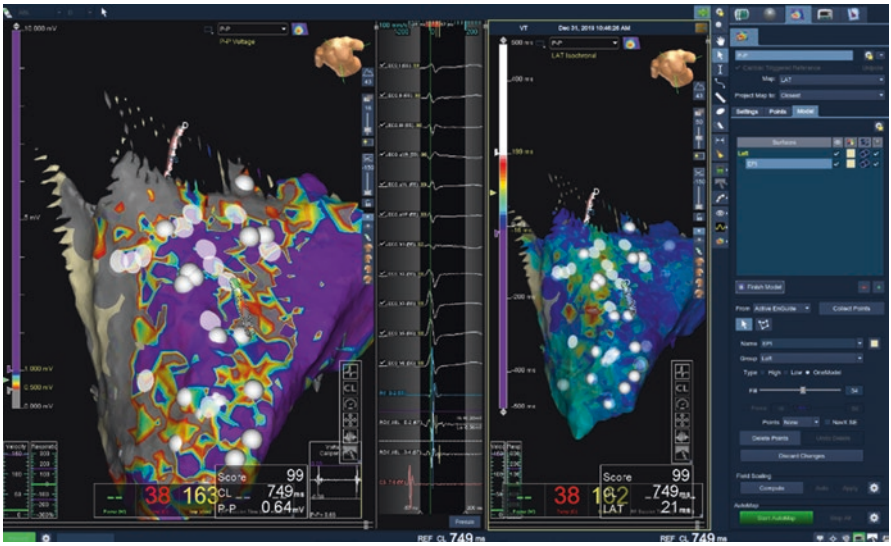

Fig. 27.7 Extensive ablation of RV epicardium, targeted entrance conducting channels, using LAT of late potentials

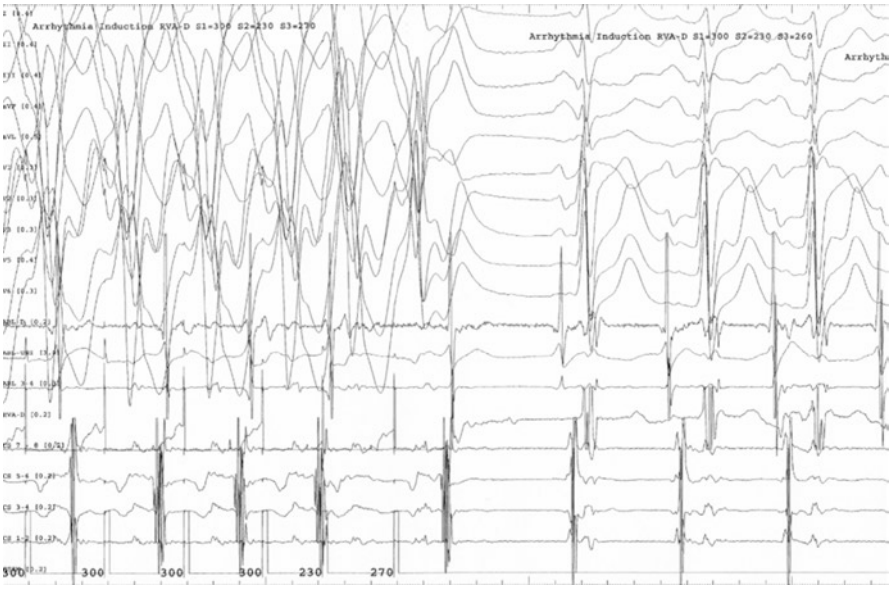

Fig. 27.8 No inducible arrhythmia post-ablation with VES

noninducibility of VT with three ventricular extrastimuluses with and without Isuprel infusion.

IN this patient ablation was successfully terminated, and intrapericardial triamcinolone was administered before long sheet withdrawn. Procedure was done without any complication [12, 13].

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