Case report

Three dimensional epicardial mapping and ablation of recurrent non-ischaemic ventricular tachycardia

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KEYWORDS

Electroanatomic mapping
Epicardial approach
Substrate based mapping
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ABSTRACT

Radiofrequency ablation is a therapeutic option for recurrent ventricular tachycardia (VT) in both ischaemic and non-ischaemic subsets. Usually this is attempted by mapping endocardially; however, in some situations epicardial approach may be needed to access the VT circuit. We report two cases in which epicardial approach was used to successfully ablate the VT, when endocardial ablation was ineffective.

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Case 1

A 50-year-old female was evaluated for recurrent symptomatic ventricular tachycardia (VT). Ventricular tachycardia was of right bundle branch block (RBBB) type morphology with superior axis, QRS duration of 204 ms, pseudo-delta wave and intrinscoid deflection of 132 ms suggesting a possible epicardial origin (Figure 1). Left ventricular (LV) function was preserved on echo and angiogram revealed normal major epicardial coronaries. Contrast-enhanced cardiac magnetic resonance imaging (MRI) demonstrated a mid-myocardial and epicardial scar in anterolateral LV wall. A diagnosis of non-ischaemic scar VT was made and patient was taken up the patient for electrophysiology study.

An initial endocardial approach was used using electroanatomic mapping system (CARTO, Biosense Webster). The equipment used for recording was an EP-TRACER (Cardio Tek, Maastricht, Netherlands). Ventricular tachycardia was induced by programmed extra stimuli (PES) from right ventricular (RV) apex. The VT was haemodynamically unstable necessitating prompt cardioversion. A substrate map of LV endocardium in sinus rhythm (SR) revealed low voltage potentials (<0.5 mV) in anterolateral wall with inability to pace at 10 mA, 2 ms pulse width suggestive of electrically unexcitable scar. Signals in the vicinity of this scar were fragmented suggestive

of far field potentials and no mid or late diastolic potentials or areas of continuous electrical activity were recorded. Pace-maps from endocardial surface were unsatisfactory. All this pointed towards a likely epicardial location of the re-entry circuit and a decision to approach the pericardial space was made.

Heparin was reversed using intravenous protamine and the pericardial space was accessed using a percutaneous subxyphoid approach using technique previously described by us.¹ A standard 8F sheath mounted over a guidewire was then introduced into the pericardium and an externally irrigated 4 mm ablation catheter (F curve, Thermocool Celsius, Cordis Webster) was introduced through the sheath and carefully advanced into the pericardial space.

At an epicardial location facing the presumed site of endocardial scar (anterolateral LV), voltage map during SR showed multiple areas of low voltage electrograms with mid-diastolic potentials (MDP) (Figure 2) representing slow conducting channels. A good 12/12 pace map from these areas with a short stimulus to QRS suggested exit site of the VT circuit (Figure 3). A coronary angiogram was done to ensure the tip of the ablation catheter was far away from the coronary arteries. Tachycardia was induced and radio frequency (RF) ablation was performed at the mapped epicardial site which promptly terminated the tachycardia. Radio frequency was, then, applied at all the low voltage channels in the scar area using 40 W and 20 mL/min irrigation to create a linear lesion. Post ablation, the tachycardia could not be induced on PES even after isoprenaline challenge. A pigtail catheter was left

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Figure 1 Electrocardiogram showing ventricular tachycardia with right bundle branch block, superior axis. QRS duration is 204 ms, pseudo-delta (PD) wave of 75 ms and intrinsic deflection (ID) of 117 ms suggest epicardial location.

Figure 2 Electrogram demonstrating epicardial mapping catheter showing mid-diastolic potentials (MDP) during sinus rhythm. From top to bottom, surface electrograms I, II, III, aVF, and V6; intracardiac electrograms from distal epicardial mapping catheter (MAPD).

in the pericardial space for 24-hours to drain any residual pericardial fluid.

Case 2

A 46-year-old female with history of recurrent symptomatic VT was evaluated. Clinical VT had RBBB, left superior axis morphology (Figure 4). Two dimension echo and MRI showed normal biventricular function with no evidence of myocardial scar.

Patient was taken up for electrophysiology study using electroanatomic mapping system. Ventricular tachycardia of

cycle length 370 ms which resembled the clinical VT was induced by PES by a quadripolar catheter placed at the RV apex. Activation mapping of the LV during VT was performed using a Navistar (F curve) catheter. Endocardial activation map failed to reveal any good early activation signal. Earliest signal was noted in postero-apical region of the LV (–18 ms to QRS onset). Radio frequency lesions given at this area failed to terminate the tachycardia. After termination of tachycardia, pace-mapping was attempted in this region; however, a satisfactory pace-map was not obtained.

In view of failure to achieve an early activation map from endocardium, it was decided to undertake an epicardial approach. A percutaneous epicardial access was obtained as

Figure 3 Epicardial left ventricular voltage map left lateral view recorded during sinus rhythm demonstrating a large anterolateral scar. Areas in purple display normal voltage (>1.5 mV); areas in red display more dense scar (<0.5 mV). Pink dot represents site with mid-diastolic potentials and good pace-mapping and short S-QRS consistent with exit site of circuit. Red dots indicate site of ablation. Black dots indicate scar (absence of voltages).

Figure 4 Ventricular tachycardia with right bundle branch block, left superior axis morphology.

described in previous case. The Navistar (F curve) catheter was introduced in the epicardial space and LV was mapped during the VT. A sharp potential with early activation (-40 ms to QRS) was mapped to LV postero-apical area on epicardial activation map, which corresponded to the endocardial site mapped earlier (Figure 5). Efficient local capture using 10 mA and 2 ms pulse duration allowed entrainment mapping. A good pacemap was obtained at this site after termination of tachycardia. Left coronary angiogram was done to exclude any major branch near to the mapping catheter. Ventricular tachycardia was reinduced after PES and RF lesions were

delivered at this site which promptly terminated the tachycardia within 7 seconds. The lesions were consolidated by giving RF energy for 2 minutes using 50 W and 20 mL/min irrigation. Post ablation, no clinical VT was inducible at baseline or with isoprenaline and aggressive ventricular pacing.

Follow-up

At a follow-up of 1 year, both the patients were free of any episodes of VT and antiarrhythmics were discontinued.

Figure 5 Epicardial left ventricular (LV) activation map. Left: Epicardial activation map during ventricular tachycardia (VT) shows pre-systolic signals (-70ms to QRS). From top to bottom, surface electrograms I, II, III, aVF, and V6; intracardiac electrograms from distal epicardial mapping catheter (MAPD). Right: Epicardial LV activation map left lateral view showing sites of early activation. Ablation at this marked site terminated the VT in 7 seconds. Red dots indicate site of ablation.

Discussion

Monomorphic VT of non-ischaemic and idiopathic aetiology can have critical parts of the VT circuit in the epicardium far away from the endocardium and epicardial ablation is required in such cases.² We have described two cases of VT (non-ischaemic scar, idiopathic) successfully ablated via an epicardial approach.

Epicardial mapping and ablation were first introduced by Sosa et al.³ in 1996 for Chagas cardiomyopathies. Percutaneous epicardial approach was used by Schweikert et al. in 17 of 18 epicardial VTs occurring in various clinical settings after failure of endocardial ablation.⁴

In our first case, electrocardiogram was suggestive of epicardial origin of the VT as per published criteria⁵ and prompted us to target the arrhythmia through epicardial aspect after a brief endocardial attempt. The patient had evidence of a scar in inferolateral LV with preserved LV function. Subepicardially located circuits are probably more frequent in cases of non-transmural infarction,⁶ so that epicardial VT should particularly be suspected when occurring in patients with preserved LV systolic function. A substrate based approach targeting low voltage channels was used. Similar approach for an endocardial substrate based ablation has been well described for post ischaemic scar VT.⁷ In the second patient, activation mapping was utilised and areas demonstrating sharp presystolic potentials during VT were targeted for ablation. Both substrate based mapping and entrainment mapping were successfully employed by Soejima et al. in 6/7 patients with epicardial VTs related to non-ischaemic dilated cardiomyopathy.⁸

Several concerning issues have been raised regarding the epicardial approach including potential for tamponade,

perforation of the heart and risk of coronary occlusion. These can be easily minimised through some simple precautions. Periodic aspiration from the epicardial catheter and placement of a pigtail catheter post procedure for 24-hours avoids pericardial tamponade. Coronary angiogram to ensure a minimum distance of 12 mm (3 times catheter size of 4 mm) between RF ablation site and epicardial coronary vessels minimises the risk of coronary occlusion. Recently, Grimard et al.⁹ reported a retrospective analysis of a series of 32 patients in which pericardial access could be managed successfully in 89% of the cases for epicardial RF ablation.

Conclusion

Mapping and ablation of VT by percutaneous epicardial approach is safe and feasible therapeutic strategy in some of the patients with symptomatic recurrent VT. It increases the success rates of VT ablation in situations where an endocardial approach is ineffective.

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