Unconventional warfare: Successful ablation of ventricular tachycardia by direct ventricular puncture in a patient with double mechanical heart valves

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Introduction

Catheter ablation of ventricular tachycardia (VT) has improved dramatically over the last 2 decades, with the introduction of new mapping and ablation technologies. The use of epicardial access and transcoronary ethanol ablation (TCEA) has been an important innovation to target arrhythmogenic substrate that is not accessible from the standard endocardial approach.

Mechanical heart valves hinder access to cardiac chambers, and cases with both mechanical mitral and aortic prostheses represent a subset of patients in whom endocardial access to the left ventricle (LV) is not feasible via the retrograde aortic or transseptal approaches, and percutaneous access to the epicardial space is hindered by surgical adhesions. Ablation of left VT in these cases has been successfully performed by surgical epicardial methods and by TCEA. However, TCEA may not be an option in cases without target vessels supplying the area of interest or in the presence of collateral arterial supply to this area.

We describe a case of drug-refractory VT in a patient with mechanical mitral and aortic valves, where successful mapping and ablation of endocardial substrate for VT was performed by accessing the LV through direct transthoracic left ventriculotomy.

Case report

A 63-year-old man with a congenital bicuspid aortic valve suffered infective endocarditis. This required a bioprosthetic aortic valve replacement, at which time he had a single graft to the right coronary artery. He then developed prosthetic valve endocarditis, requiring 4 additional open-heart procedures. He now has mechanical aortic and mitral valves and is on suppressive therapy for endocarditis, without clinical recurrence. In 2005 he had a witnessed cardiac arrest owing to monomorphic VT and underwent implantation of a single-chamber implantable cardioverter-defibrillator (ICD). This was later upgraded to a cardiac resynchronization device because of worsening heart failure symptoms and pacemaker dependency. He continued to experience episodes of VT, which resulted in over 40 episodes of sustained VT and more than 30 ICD shocks. Initial attempts to manage the VT with sotalol and, later, amiodarone were only partly successful. Despite being on amiodarone and mexiletine, he continued to experience slower VT episodes, which were long-lasting, resulting in worsening of his heart failure symptoms and continued ICD shocks.

The patient was then brought for electrophysiology study with a plan to map and, if possible, ablate the VT by TCEA or surgical (thoracotomy) epicardial ablation. As a result of the 5 prior open-heart procedures and resultant pericardial adhesions, percutaneous epicardial access was considered impossible.

Preprocedure, an echocardiogram revealed moderate LV dysfunction and a scar located at the inferior wall, extending from the apex to the mid inferior wall and to the interventricular septum.

During the electrophysiology study, monomorphic VT was easily induced by programmed ventricular stimulation with single ventricular extrastimulus. The cycle length of the clinical VT was 500 msec and was hemodynamically tolerated. Morphology of the VT is shown in Figure 1A. The electrocardiogram suggested an exit point in the inferoapical LV. The initial inscription of the QRS complexes during VT was sharp, suggesting an early endocardial exit of the tachycardia. Mapping from the right ventricle and coronary sinus (CS) revealed delayed activation compared to the onset of the QRS complexes in VT. An attempt to map the VT through the tributaries of the CS was made; however, the catheter could not be advanced into a suitable tributary of the CS.

Epicardial access was gained by anterolateral thoracotomy. Extensive pericardial adhesions were released with digital dissection to facilitate epicardial mapping. Limited activation mapping of the inferolateral apical regions of the

KEYWORDS Ventricular tachycardia; Ablation; Lateral ventriculotomy; Mechanical prosthetic valves; Mapping
(Heart Rhythm Case Reports 2017;3:599–603)

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https://doi.org/10.1016/j.hrcr.2017.10.007

KEY TEACHING POINTS

- Catheter ablation of ventricular tachycardia (VT) can improve morbidity and mortality in patients with structural heart disease with implantable cardioverter-defibrillators implanted for primary as well as secondary prevention.
- Substrate-based ablation is a safe and effective strategy for reentrant VTs, which is the predominant mechanism of tachycardia in these patients.
- Access to the "substrate" will be challenging in some cases, especially in the presence of mechanical prosthetic heart valves.
- Unconventional approaches are needed in those cases where a "hybrid" approach of catheter-based ablation and surgical ablation is useful.

LV was done with the electroanatomic CARTO system (Biosense Webster, Diamond Bar, CA) (Figure 2).

Cryoablation at the site of earliest epicardial activation did not affect the tachycardia, indicating that the VT circuit is not close to the epicardium. TCEA was then planned and a coronary angiogram was performed. The left coronary artery was normal, but it provided collateral supply to the inferobasal LV, in the area of scar observed on echocardiography (Figure 3A, B) (Supplementary Videos 1 and 2), which was just beneath the early points in the epicardial map. The right coronary artery was totally occluded at the mid segment, with bridge collaterals supplying the same area in the inferior wall scar, which appeared like the substrate of the VT. The left anterior descending artery (LAD) supplied a collateral to the posterolateral branch of the right coronary artery, which was the primary source of blood supply to the scarred area. A Pilot 50 Hi Torque Guidewire (Abbott Vascular, Santa Clara, CA) was advanced through the left main and into the LAD and down around the apex. Over the wire, a 2.0 × 8-mm over-the-wire balloon was advanced to the

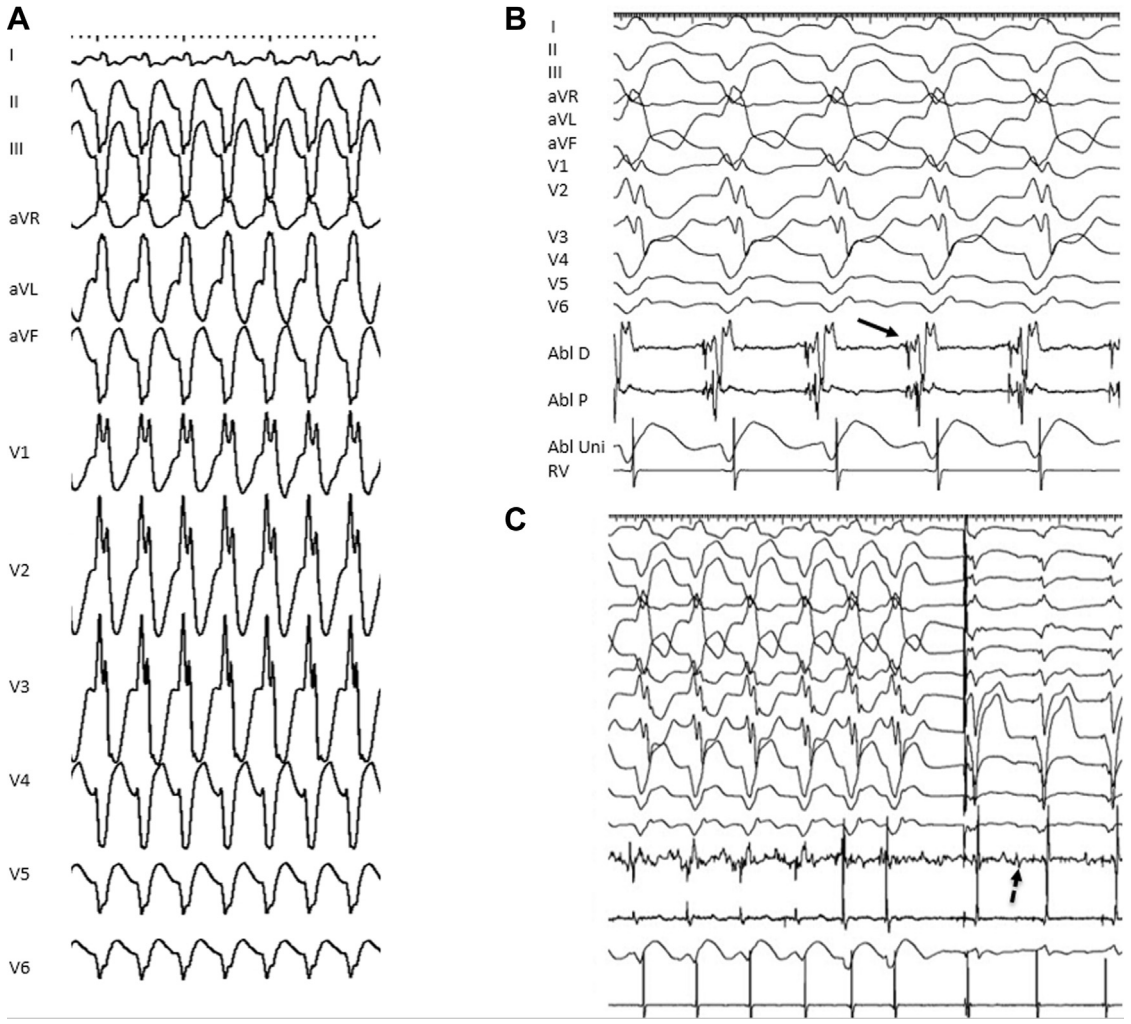

Figure 1 Twelve-lead and intracardiac recordings of clinical ventricular tachycardia (VT). **A:** A 12-lead rhythm strip of VT showing right bundle branch block with left axis deviation. **B:** Intracardiac recordings from the endocardial left ventricle. There is a presystolic potential (*arrow*) preceding the onset of the QRS complex. **C:** Termination of the VT during ablation at the site shown in panel B. Note the late potential in diastole (*dotted arrow*) that preceded the QRS during VT, indicating the slow conducting isthmus involved in the tachycardia circuit. Abl D = ablation distal; Abl P = ablation proximal; Abl Uni = ablation unipolar.

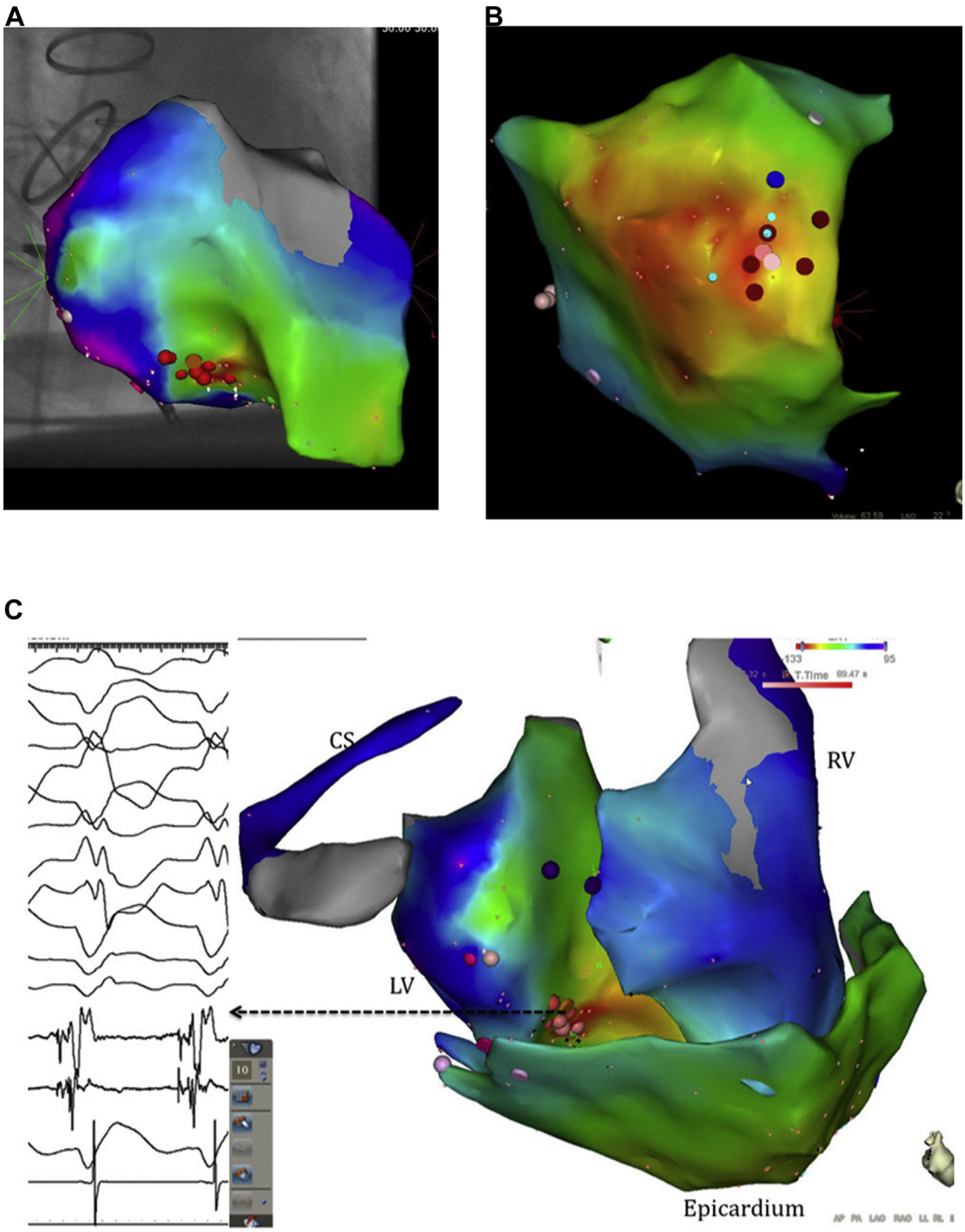

Figure 2 CARTO electroanatomic activation map during ventricular tachycardia (VT). **A:** Endocardial map showing earliest activation occurring at the mid-inferior wall of the left ventricle (LV). **B:** Limited epicardial activation map. Early activation is diffuse, which indicates that the VT exit is not epicardial. **C:** Electroanatomic maps of the right ventricle (RV), LV, coronary sinus (CS), and epicardium, showing the exit of the tachycardia at the inferior endocardial surface of the LV.

LAD and positioned just before the collaterals to the postero-lateral branch. Ice-cold saline, injected through the balloon after inflation of the balloon to prevent any antegrade flow of warm blood through the collateral branch, successfully terminated the VT in reproducible fashion. However, tachycardia promptly restarted after each termination, possibly the result of warm blood perfusion of the infarct zone from the right coronary bridge collaterals. Still, this provided some indication that this particular scar was the source of the VT. However, alcohol ablation was not attempted because of the presence of collaterals. With the failure of epicardial ablation, the inability to attempt TCEA, and the suggestion that

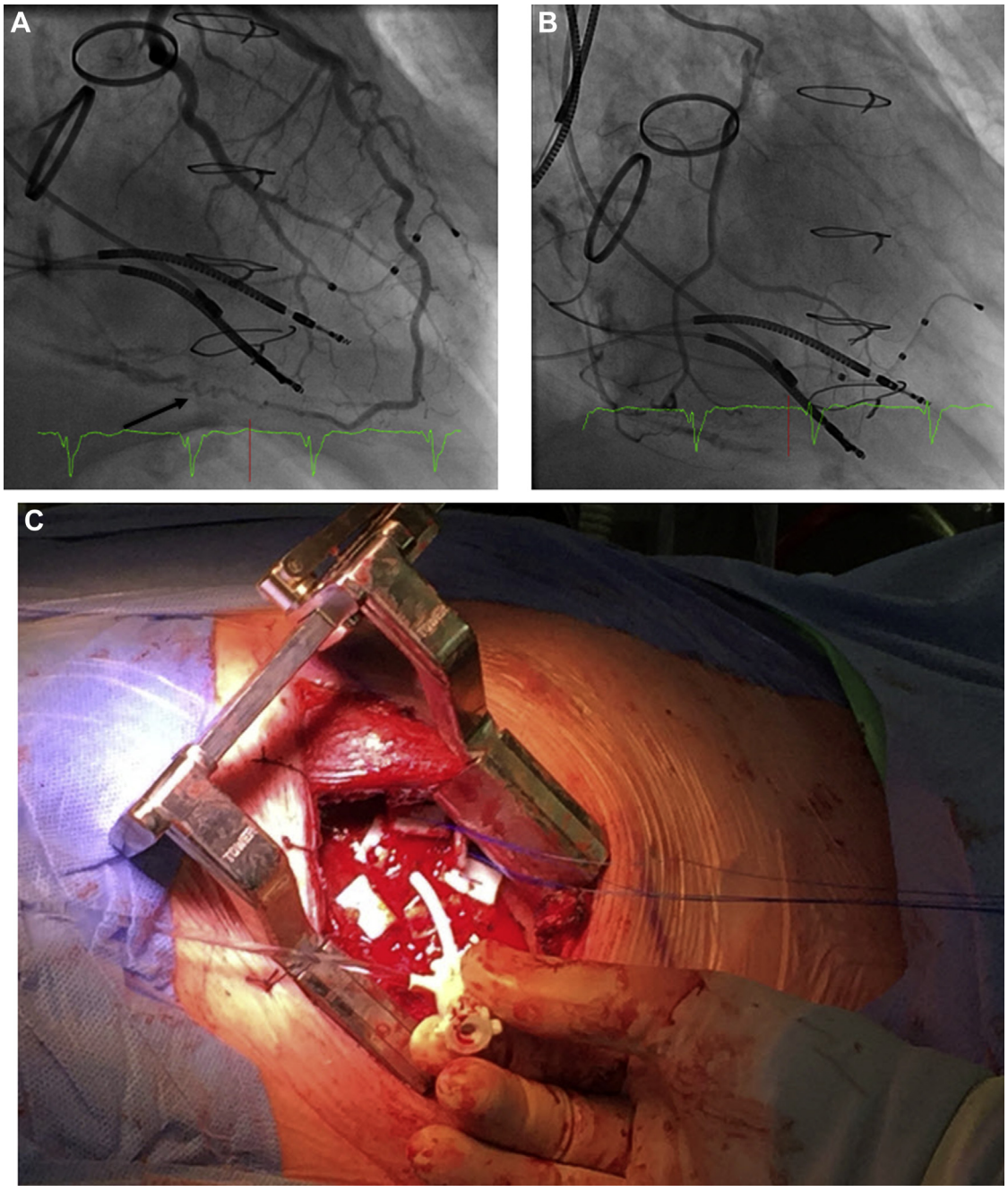

Figure 3 **A:** Coronary angiogram of the left coronary artery system. Collaterals to the right system are seen (*arrow*) overlying the scarred area. Calcification of the scar is also seen (*asterisk*). **B:** Right coronary angiogram. Total occlusion of the distal right system with faint collateralization to the left circumflex artery is seen. **C:** Exposure of the left ventricle (LV) lateral wall and introduction of a SafeSheath Hemostatic Peel Away Introducer to the LV cavity through the free wall puncture.

the VT was originating from the endocardial portion of this posterolateral scar, a decision was made to access the LV through ventriculotomy.

Pledgeted Prolene sutures were placed in a rosette pattern around the apical lateral wall and LV puncture done through the canter. A 17 gauge Tuohy needle was used to puncture the LV lateral wall and to gain access to the LV cavity. A guide wire was introduced into the LV, over which an 8.5F Safe-Sheath Hemostatic Peel Away Introducer (Pressure Products Medical Supplies Inc, San Pedro CA) was advanced to the LV cavity (Figure 3C).

Mapping of the LV endocardium was performed using a bidirectional (D/F) Biosense ThermoCool SmartTouch catheter (Biosense Webster, Diamond Bar, CA). Activation mapping revealed the early site of activation preceded by a presystolic potential at inferoapical location (Figure 1B and C and Figure 2C). Entrainment was performed from this site, which reproducibly resulted in termination of the

tachycardia, which reinitiated after a few paced beats with a ventricular ectopic beat. Ablation of the area resulted in termination of the VT (Figure 1C). Radiofrequency applications were started at a power setting of 20 W with a maximum temperature setting of 41°C and power output was titrated up to 40 W to achieve an impedance drop of more than 10 ohms. More mapping of the endocardium was performed to identify the late potentials and to homogenize the scar. After completion of the substrate modification, VT remained noninducible with programmed ventricular stimulation up to 3 extrastimuli using 2 drive trains (600 msec and 400 msec). Despite some nonsustained VT in the first postprocedure days, the patient has now been off amiodarone and mexiletine for 14 months and has not suffered any further ICD shocks.

Discussion

Success of a VT ablation depends on modifying the substrate of the arrhythmia, for which access to the substrate is the key determinant. Interventional targets may be "excluded" owing to access issues in structural heart disease patients. Direct ventricular puncture provides a useful alternative access site in these patient subsets with acceptable morbidity and complication rates to approach the inaccessible targets using standard approaches.

Ablation of VT in patients with prosthetic mitral and aortic valves is a challenging procedure. Techniques used are epicardial mapping, TCEA, and rare reports of direct LV puncture to map the endocardium.[1–3]

Prosthetic valves prevent access to the ventricles and can pose a significant barrier in ablating the tachycardia. Most of the cases even post cardiac surgery, epicardial surface of the heart can be accessed percutaneously and release the adhesions with the mapping catheter to facilitate mapping and ablation. Epicardial access by thoracotomy is another option in postsurgical cases. In this case, the patient had undergone 5 open-heart procedures and the only option to access the LV epicardium was by a standard thoracotomy and manual release of the adhesions. TCEA is the other nonconventional ablation strategy employed in this patient subset. Accessing the LV cavity by direct ventricular puncture has been reported previously and is a standard procedure in structural heart interventions, especially transcatheter aortic valve replacement.

Transapical LV puncture was used in the past, mostly for diagnostic purposes,[4] but has been abandoned in favor of transvenous/transarterial approaches with low morbidity and mortality rates. However, larger sheaths are required in therapeutic interventions.

Hsieh and colleagues[1] reported 2 cases of direct transthoracic LV access to ablate VT, in which 1 was a direct percutaneous puncture of the LV and the second was with mini-thoracotomy. Direct puncture of the LV was complicated by left hemopericardium because of inadequate hemostasis. The advantage of ventricular puncture with thoracotomy is better control of hemostasis, as purse-string sutures placed around the point of LV puncture would prevent bleeding from the puncture site. To our knowledge this is the first case of direct LV puncture performed with anterolateral thoracotomy and LV lateral wall puncture.

Vaseghi and colleagues[5] have described a percutaneous interventricular transseptal approach in a similar situation. This approach could be an alternative to an open thoracotomy approach when epicardial mapping is not contemplated. A thoracotomy approach would provide more freedom for catheter manipulation than a transseptal approach from the internal jugular vein.

Knowing the substrate would help in selecting the puncture site. The reported cases of VT ablation with direct ventricular puncture were done with apical punctures. This might pose difficulties in accessing the apical scar, as the catheter has to take a reverse loop, thus compromising the stability to deliver adequate lesions. Posterolateral puncture, on the other hand, is more appropriate for accessing septal/inferior and apical targets. Because the puncture is being performed under direct vision, the operator can avoid coronary artery branches and CS tributaries.

Conclusion

We report a case of successful catheter ablation of drug-refractory VT in a patient with 2 mechanical valves. LV endocardial mapping and ablation were performed by direct lateral ventriculotomy. This is a safe and effective technique in targets that are sequestrated and/or inaccessible because of the presence of mechanical valves.

Appendix

Supplementary data

Supplementary data associated with this article can be found in the online version at https://doi.org/10.1016/j.hrcr.2017.10.007.

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