Cardiac magnetic resonance–guided pacemapping for ventricular tachycardia substrate ablation in sinus rhythm in a patient with nonischemic cardiomyopathy

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Introduction
Substrate mapping–guided ablation is a recommended therapeutic option in patients with recurrent episodes of scar-related ventricular tachycardia (VT). Color-coded pixel signal intensity (PSI) maps obtained from preprocedural late gadolinium enhancement cardiac magnetic resonance (LGE-CMR) imaging can be used for identification and characterization of arrhythmic substrate. PSI maps permit visualizing the border zone areas distribution inside the scar as corridors of viable tissue connecting the healthy myocardium, defined as heterogeneous tissue channels (HTCs), that correlate with the conducting channels on the electroanatomical mapping (EAM). Our group previously reported that the integration of PSI maps and HTCs into the navigation system during the ablation procedure help not only in procedure approach planning but also to improve outcomes in both ischemic and nonischemic cardiomyopathy patients.

Case report
We report the case of a 78-year-old man with a nonischemic cardiomyopathy and repetitive monomorphic VT episodes recorded in the 24-hour Holter monitoring despite optimal medical therapy with amiodarone. The patient had moderate left ventricular (LV) systolic dysfunction on the echocardiography. A VT ablation procedure and an implantable cardioverter-defibrillator implant were programmed. Prior to the procedure, a multidetector computerized tomography (MDCT) and an LGE-CMR were obtained for guiding the ablation. The LGE-CMR showed a midmyocardial scar in the basal septum (Figure 1A).

LGE-CMR images were analyzed using a previously described technique. Briefly, full LV volume was reconstructed in the axial orientation, and the resulting images were processed with ADAS 3D LV software (ADAS3D Medical, Barcelona, Spain). The LV was divided into 10 layers from the endocardium to the epicardium. A 3-dimensional shell was obtained for each layer. PSI maps were obtained from LGE-CMR images, color-coded, and projected to each of the shells following a trilinear interpolation algorithm. To identify the scar areas, a PSI-based algorithm was applied to characterize the hyperenhanced area as scar core or border zone, using 40% ± 5% and 60% ± 5% of the maximum signal intensity (SI). Pacemapping from the entrance of the HTCs identified in the PSI maps accurately reproduce the morphology of the induced ventricular tachycardias (VTs).

In patients with hemodynamically not well-tolerated VT, ablating the entrance of HTCs could be an option for VT ablation even in nonischemic patients. This approach can be performed without the need of a complete EAM.

KEY TEACHING POINTS
- A color-coded pixel signal intensity (PSI) map obtained from late gadolinium enhancement cardiac magnetic resonance (LGE-CMR) images allow a complete substrate identification even in a case with small midseptal scar, overcoming the limitations of the electroanatomical map (EAM) owing to the far-field effect of the healthy surrounding tissue in the electrograms.
- LGE-CMR scar maps also allow identification of heterogeneous tissue channels (HTCs) in nonischemic patients. Pacemapping from the entrance of the HTCs identified in the PSI maps accurately reproduce the morphology of the induced ventricular tachycardias (VTs).

KEYWORDS
- Arrhythmogenic substrate
- Cardiac magnetic resonance
- Catheter ablation
- Conducting channels
- Image-guided substrate ablation
- Pace-mapping
- Ventricular tachycardia

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intensity as thresholds. In the LGE-CMR-delivered PSI maps, HTCs were defined as continuous corridors of border zone surrounded by scar core or scar core and an anatomical barrier (mitral annulus) connecting 2 areas of healthy tissue. HTCs were obtained automatically by the ADAS 3D LV software, the entrance of these HTCs being the final targets for ablation. LGE-CMR-delivered PSI maps were merged with the MDCT using the ADAS software for improving the image merge process owing to optimal MDCT spatial resolution.

The ablation procedure was performed under general anesthesia. A quadripolar electrode catheter was advanced via a femoral vein to the right ventricular apex. A right femoral arterial access was also obtained and heparin was given targeting an activated clotting time of 300 seconds. After that, the first step of the procedure was acquiring a fast anatomical map of the aorta for image integration. This fast anatomical map was then used to integrate the MDCT cardiac reconstruction and PSI maps imported from ADAS 3D into the spatial reference coordinates of the CARTO system (Figure 1B). A midmyocardial scar in the basal septum was observed in the PSI map with a multibranch HTC with 4 independent HTC exits. After programmed stimulation protocol from the right ventricular apex, 3 different nontolerated VTs were easily induced (Figure 2 shows the VT morphologies: VT1 had a superior axis with left bundle branch morphology, an early transition in V2, and a cycle length (CL) of 300 ms; VT2 had an inferior axis with positive concordance in precordial leads and a CL of 335 ms; VT3 had a superior axis, positive concordance in precordial leads, and a CL of 323 ms). Activation map, VT entrainment or electrogram (EGM) analysis of the HTC

Figure 1  A: Late gadolinium enhancement cardiac magnetic resonance (LGE-CMR) and pixel signal intensity (PSI) map images showing a midmyocardial scar in the basal septum. B: Integration of the PSI maps within the spatial reference coordinates of the CARTO system, by performing a fast anatomical map of the aortic root.

Figure 2  Three ventricular tachycardia (VT) morphologies induced during the programmed stimulation protocol. VT1 had a superior axis with left bundle branch morphology, an early transition in V2, and a cycle length (CL) of 300 ms; VT2 had an inferior axis with positive concordance in precordial leads and a CL of 335 ms; VT3 had a superior axis, positive concordance in precordial leads, and a CL of 323 ms.
entrances during VT were not attempted owing to hemodynamic instability. During sinus rhythm, the ablation catheter (ThermoCool SmartTouch; Biosense Webster, Diamond Bar, CA) was positioned at the HTC entrances and pacemapping was sequentially performed using a 4 mA output with a pulse duration of 2 ms. An excellent concordance was obtained with the 3 VTs previously recorded (Figure 3). Then, the EGM information was analyzed before applying radiofrequency (RF) in order to avoid applications in EGM with His or fascicular EGM characteristics. A total of 6 ablations were performed (40 W, 45°C, 30 mL/min) at 3 of the 4 HTC entrances, with a mean 460 ablation index and a total 318 seconds ablation time. The fourth HTC entrance was not ablated owing to the presence of His potential at that point. Afterwards, no VTs could be induced despite an aggressive programmed stimulation from the right ventricular apex. Total procedure and fluoroscopy time were 90 and 8 minutes, respectively. Twenty-four hours after the procedure, amiodarone was withdrawn and the patient was discharged. After 9 months follow-up, no VT recurrence was observed.

Discussion
Traditionally, scar-related VT catheter ablation has been performed using classic methods of activation and entrainment mapping during arrhythmia to identify and ablate the critical isthmus of the VT. However, just one-third of patients have only hemodynamically tolerated VT, so a substrate-based
ablation has become a routine part of most VT ablation procedures, with good clinical outcomes. Even if unipolar and bipolar voltage can provide important information regarding scar location and depth, a substrate mapping strategy based only on voltage map has some limitations in the case of non-ischemic cardiomyopathy with midseptal scar. Standard bipolar voltage mapping provides limited information regarding fibrotic tissue that is located deeper with respect to the tip–tissue interface, especially when using multipolar catheters with small interelectrode spacing. In some patients, scar may be mainly midmyocardial and the bipolar voltage maps from the endocardium may underestimate the amount of scar, or even may display completely normal voltage. LGE-CMR has shown to provide additional substrate characterization in these cases. Dickfeld and colleagues previously reported that a 2-mm rim of surviving endocardium was able to prevent the detection of midmyocardial scar using traditional voltage criteria. In these cases, LGE-CMR provided visualization of the midmyocardial scar, functioning as the VT reentrant substrate that had not been detected in the EAM. With the present clinical case we are reporting a new approach for VT ablation in nonischemic patients with basal septal myocardial scar. The arrhythmogenic substrate identification in this case was based only on the LGE-CMR information without performing any EAM. This results in a simplification of the procedure, as EAM is usually tedious and time-consuming. The complete arrhythmogenic substrate identification with the LGE-CMR information is supported by the complete abolition of all 3 previously inducible VTs and the absence of VT recurrences during the follow-up. Moreover, this case is a good example of the safety of this approach. Presence of the conduction system can be a challenge for ablation in the basal septum. Even if a complete EAM was not performed to identify the location of the conduction system in this case, we systematically check for EGMs with conduction system characteristics into the entrance of HCTs before RF delivery. With this approach we identified a His EGM into the entrance of the fourth HTC and ablation was avoided at this point.

Limitations
It is worth bearing in mind some limitations to correctly interpret this case report. First, the lack of activation map or entrainment maneuvers owing to hemodynamic instability of induced VTs does not permit to assure that RF was delivered exactly at the HTC entrance instead of at its neighboring area. Second, lack of EAM prevents performance of a direct comparison between uni-bipolar voltage and LGE-CMR information.

Conclusion
This case further illustrates the utility of cardiac imaging for VT ablation procedure planning and how it permits ablation of nontolerated VTs in a very efficient way without the need for obtaining a detailed electroanatomical map. The case depicts how magnetic resonance imaging–guided ablation can be of great help in nonischemic patients with nontolerated VTs and midseptal scars, in whom substrate characterization is especially challenging owing to the interposition of subendocardial normal muscle and the far-field effect of surrounding tissue on the local electrograms.

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