



Characteristics of the ablation lesions in cardiac magnetic resonance imaging after radiofrequency ablation of ventricular arrhythmias in relation to the procedural success

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Background In human patients, studies about the cardiac magnetic resonance (CMR) appearance of the acute radiofrequency (RF) lesions in relation to the procedural outcomes after catheter ablation (CA) of ventricular arrhythmias (VA) are scarce. We aimed to investigate the RF lesions characteristics in relation to the procedural success.

Methods Patients referred for ablation of VA received CMR (1.5 T) using gadolinium contrast before and after ablation. CA in left ventricle was performed using a 3.5-mm irrigated catheter. The volume and transmural of the RF-induced lesions were measured in early gadolinium-enhanced postablation CMRs. *Acute failure* was defined as persistently inducible VA at the end of the CA.

Results Twenty-five patients (60.7 ± 9.8 years, 19 with sustained ventricular tachycardia) were studied. All RF lesions had nonenhanced core. The volume of the nonenhanced lesions showed positive correlation with the maximal RF power ($r = 0.598$, $P = .002$) and the impedance drop ($r = 0.416$, $P = .038$). Patients with transmural ($\geq 75\%$) lesions had significantly larger impedance drop as compared to those with nontransmural lesions ($< 75\%$): 20.3 ± 9.4 versus 13.5 ± 4.3 , $P = .037$. In the failures, the lesions volume was nonsignificantly larger: $3.86 \pm 3.3\%$ versus $2.6 \pm 1.7\%$, $P = .197$; however, it was considerably deeper: $86 \pm 13\%$ versus $62 \pm 26\%$, $P = .03$.

Conclusions CMR after VA ablation showed nonenhanced lesions resembling the no-reflow phenomenon in myocardial infarction. Although the size and the depth of the RF injury correlated with the ablation energy and impedance drop, they were not associated with acute ablation success. (Am Heart J 2018;204:68-75.)

Cardiac magnetic resonance (CMR) can be useful for evaluation of the anatomical substrate in patients with ventricular arrhythmias (VAs) before radiofrequency (RF) catheter ablation (CA).¹ Scar visualization using late gadolinium enhancement-CMR (LGE-CMR) was validated in histological studies and can be used to define the heterogeneous “gray” border zone containing viable and nonviable tissue.^{2,3} Recent studies demonstrated a good overlap between LGE-CMR signal intensity-based scar characterization and

low-voltage areas in the electroanatomical voltage maps.^{4,8} In patients with ischemic cardiomyopathy, the scar size measured in LGE-CMR was associated with inducibility of sustained VA.^{9,10}

In animal experiments, CMR was used to visualize the stages of tissue injury after CA.¹¹⁻¹⁴ However, there are limited data on the appearance and the transformation of the RF lesions in human patients. In a previous study, hyperenhanced (HE) lesions on sites of RF energy delivery were observed years after CA of ventricular premature beats (VPB).¹⁵ Also, nonenhanced (NE) lesions resembling the no-reflow phenomenon in acute myocardial infarction were observed in atrial walls of patients after CA for atrial fibrillation.¹⁶ Because of the scarce data, we aimed to assess the extent and the characteristics of the myocardial damage caused by the RFCA in human patients ablated for VAs. Because the LGE-CMR can occasionally detect areas of LGE even in patients without overt structural heart disease assessed by conventional imaging techniques, we divided

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the patients into 2 groups: VA with LGE (VA/LGE+) and VA without LGE (VA/LGE-) and examined the acute RF lesions and procedure outcomes separately for both groups. Our working hypothesis is that the size and the depth of the RF lesions in early gadolinium enhancement (EGE)-CMR are associated with the success after catheter ablation of VA.

Methods

Study protocol

Twenty-five patients with either scar-related or idiopathic VA scheduled for RFCA were examined using CMR before and after RF ablation. The study complied with the Declaration of Helsinki. All patients provided written informed consent for RF ablation and CMR before and after CA. All of them underwent CA of VA at the Heart Center of Leipzig between May 2015 and June 2016. Patients aged <18 years, with ongoing ventricular tachycardia (VT), or with metallic implants contraindicated for CMR were not included.

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Imaging protocol

CMR imaging was performed using a 1.5-T MR scanner (Philips Ingenia, Best, the Netherlands) with a dedicated 32-channel coil. Cardiac synchronization was achieved with a vector electrocardiogram. For cine imaging, a balanced steady-state free precession sequence with retrospective gating (>30 phases per cardiac cycle) was used during end-expiratory breath holding (repetition time [TR], 3.4 milliseconds; echo time (TE), 1.7 milliseconds; flip angle 60°, in-plane spatial resolution 1.8 × 1.8 mm, slice thickness 8 mm). All standard cardiac geometries (2-, 3-, and 4-chamber views and multiple, gapless short-axis slices covering the entire left ventricle [LV] from the mitral annulus to the apex) were acquired.

Before RFCA, LGE imaging was carried out 10-15 minutes following administration of the contrast agent (intravenous bolus of 0.2 mmol/kg gadolinium-DTPA) using an inversion-prepared 3-dimensional (3D) spoiled gradient echo sequence (TR/TE/flip angle = 3.6 milliseconds/1.7 milliseconds/15°, reconstructed in-plane spatial resolution 1.6 × 1.6 mm, slice thickness 5 mm); radial *k*-space traversal using an elliptical *k*-space shutter in combination with parallel imaging (ie, a sensitivity-encoding factor of 2.0) was used; total breath-hold duration for 3D-LGE imaging was in the range of 12-16 seconds depending on field of view and heart rate. The inversion delay was individually adapted (typically in the range of 190-250 milliseconds) to completely nullify normal myocardium. All images were visually inspected to rule out the presence of susceptibility artifacts.

Post-RFCA imaging using the abovementioned pre-RFCA protocol was performed by the third postablation day at latest. To visualize the ablation lesions as NE

areas, 3D EGE imaging was performed 3 minutes after gadolinium contrast injection using the following parameters: TR/TE/FA = 3.6 milliseconds/1.7 milliseconds/15°, voxel size: 1.6 × 1.6 mm, slice thickness 5 mm, and fixed IR delay 300 milliseconds. Additionally, T2-weighted CMR imaging was performed to assess myocardial edema resulting from RF application.

Electrophysiological study

Before the RFCA, all patients signed an informed consent for undergoing the procedure. The preparation of the patients was performed according to the clinical routine at our institution. At the beginning of the RFCA, we measured the duration of the unfiltered QRS complex at 200 mm/s. The primary mapping approach was endocardial in most of the cases. After a single transeptal puncture, a steerable sheath (Agilis; St Jude Medical, St Paul, MN) was used to introduce the ablation catheter. All patients were administered heparin intravenously to achieve an activated clotting time ≥300 seconds. At the beginning of the procedure, a programmed ventricular stimulation was performed to induce the clinical VT.

Electroanatomical mapping and catheter ablation

Electroanatomical voltage mapping (EAM) was performed using EAM system (Carto; Biosense Webster, Diamond Bar, CA). In all patients with sustained VT and known structural heart disease, electroanatomical voltage map in sinus rhythm was created using standard filter settings (16-500 Hz) for the bipolar Carto signals. The fill threshold was set at 15. The median number of the points was 200 (quartiles 100-300) points. In particular, higher-density mapping was performed in areas demonstrating low-voltage and fragmented signals. Local bipolar electrograms demonstrating peak-to-peak amplitude ≥1.5 mV were defined as healthy, and low-amplitude signals <0.5 mV were defined as dense scar, whereas potentials with amplitudes in between were defined as border zone. The surface of the bipolar low-voltage areas was measured and presented as absolute figures in cm² and percentage of the left ventricular area. The late potentials and fragmented electrograms were annotated. The analysis of the stimulus-QRS intervals as well as the match between the paced QRS and the clinical VT were used to identify the protected isthmus in unstable VT. In patients with hemodynamically stable VTs, a combination of substrate, activation mapping, and entrainment was performed to characterize the reentry circuit of VT. In patients with idiopathic VPB of focal origin, activation mapping with annotation of the earliest bipolar signal was used to identify their origin. In scar-related VPB, with presumably reentry mechanism, extensive substrate-guided ablation was additionally performed.

Ablation was performed using 7F, 3.5-mm irrigated-tip ablation catheters (Navistar Thermocool; Biosense Webster Inc, Diamond Bar, CA), irrigation rate of 30 mL/min,

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