Ablation Lesion Characterization in Scarred Substrate Assessed Using Cardiac Magnetic Resonance

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ABSTRACT

OBJECTIVES This study examined radiofrequency catheter ablation (RFCA) lesions within and around scar by cardiac magnetic resonance (CMR) imaging and histology.

BACKGROUND Substrate modification by RFCA is the cornerstone therapy for ventricular arrhythmias. RFCA in scarred myocardium, however, is not well understood.

METHODS We performed electroanatomic mapping and RFCA in the left ventricles of 8 swine with myocardial infarction. Non-contrast-enhanced T1-weighted (T1w) and contrast-enhanced CMR after RFCA were compared with gross pathology and histology.

RESULTS Of 59 lesions, 17 were in normal myocardium (voltage >1.5 mV), 21 in border zone (0.5 to 1.5 mV), and 21 in scar (<0.5 mV). All RFCA lesions were enhanced in T1w CMR, whereas scar was hypointense, allowing discrimination among normal myocardium, scar, and RFCA lesions. With contrast-enhancement, lesions and scar were similarly enhanced and not distinguishable. Lesion width and depth in T1w CMR correlated with necrosis in pathology (both; r² = 0.94, p < 0.001). CMR lesion volume was significantly different in normal myocardium, border zone, and scar (median: 397 [interquartile range (IQR): 301 to 474] mm³, 121 [IQR: 87 to 201] mm³, 66 [IQR: 33 to 123] mm³, respectively). RFCA force-time integral, impedance, and voltage changes did not correlate with lesion volume in border zone or scar. Histology showed that ablation necrosis extended into fibrotic tissue in 26 lesions and beyond in 14 lesions. In 7 lesions, necrosis expansion was blocked and redirected by fat.

CONCLUSIONS T1w CMR can selectively enhance necrotic tissue in and around scar and may allow determination of the completeness of ablation intra- and post-procedure. Lesion formation in scar is affected by tissue characteristics, with fibrosis and fat acting as thermal insulators. (J Am Coll Cardiol EP 2019;5:91–100) © 2019 by the American College of Cardiology Foundation.

Ischemic cardiomyopathy frequently leads to lethal ventricular arrhythmia and sudden cardiac death (1). Radiofrequency catheter ablation (RFCA) is increasingly performed for treatment of scar-related ventricular tachycardia (VT), and substrate modification based on electroanatomic mapping has become the cornerstone therapy for VT (2,3). Ablation lesion formation in the presence of...
Ablation Lesion Characterization in Scar

**ABBREVIATIONS AND ACRONYMS**

3D = 3-dimensional
BZ = border zone
CMR = cardiac magnetic resonance
CNR = contrast-to-noise ratio
EGE = early gadolinium-enhanced
IQR = interquartile range
LGE = late gadolinium-enhanced
LV = left ventricle
MI = myocardial infarction
RFCA = radiofrequency catheter ablation
TI = inversion time
VT = ventricular tachycardia

scar tissue, however, is not well understood, and the outcomes are suboptimal, especially for hemodynamically unstable VT (4).

Cardiac magnetic resonance (CMR) offers significant advantages over other imaging modalities for identification of arrhythmic substrates due to superior soft-tissue contrast (5). CMR can also visualize acute and chronic RFCA lesions with or without contrast agent (6-8). Non-contrast-enhanced T1-weighted (T1w) imaging can separate the necrotic core from the surrounding edematous rim in acute RFCA lesions (9,10). Recently, non-contrast-enhanced T1w imaging with long inversion time (TI) has demonstrated improved delineation of acute necrotic lesion cores, surrounding hema
toma, and chronic scar, exploiting differences in intrinsic T1 relaxation times (11). This could benefit target selection and acute evaluation of ablative treatment.

We hypothesize that inability to access the necrotic extent of ablation lesions may lead to incomplete ablation of arrhythmogenic substrate, allowing subsequent recovery of tissue with restoration of conduction and recurrent arrhythmia. We further hypothesize that non-contrast-enhanced T1w CMR with long TI can detect acute RFCA lesions within and around scar, without the overestimation typically seen in late gadolinium-enhanced (LGE) CMR (12). In this study, we utilize CMR techniques to examine RFCA lesion formation within and around scar in swine 8 weeks after myocardial infarction (MI). We compare imaging results to gross pathology and histology.

**METHODS**

**ABLATION PROCEDURE IN SWINE WITH MI.** Animal experiments were approved by the Institutional Animal Care and Use Committee. Swine were initially anesthetized with mechanical ventilation using a combination of ketamine and xylazine and maintained under sedation using 1% to 2% isoflurane. We created MI in 8 swine (weighing 35 to 45 kg) by occluding the mid-left anterior descending coronary artery for 2 h using an angioplasty balloon (13).

We performed an electrophysiological study 8 weeks after MI. In heparinized animals, endocardial mapping of the left ventricle (LV) was performed during sinus rhythm via retrograde approach using a duodecapolar catheter (AFocus II, Abbott Inc., Minnetonka, Minnesota) with the EnSite Velocity mapping system (Abbott). A bipolar voltage map was created, where normal myocardium voltage was >1.5 mV, scar <0.5 mV, and border zone (BZ) 0.5 to 1.5 mV (3). Endocardial RFCA in the LV was performed at 2 to 3 sites each in scar, BZ, and normal tissue using an irrigated 3.5-mm tip contact force-sensing ablation catheter (TactiCath, Abbott). Each RFCA application was delivered at 30 W for 60 s (17 ml/min irrigation) with a temperature limit of 48°C, and targeting contact force was 10g to 40g. Force-time integral and changes of local impedance and amplitude of bipolar electrograms were measured during each ablation.

**CMR IMAGING PROTOCOL.** CMR imaging was performed in all swine, 1 week before and immediately after the RFCA procedure. Four swine were imaged at 1.5-T and 4 others at 3-T (Avanto and Prisma, Siemens Healthcare, Erlangen, Germany). Typical imaging parameters for non-contrast-enhanced 3-dimensional (3D) inversion-recovery T1w sequence were (Online Appendix): TI = 700 to 800 ms (2RR triggering), flip = 25°, repetition time/echo time = 5.4/2.7 ms, reconstructed pixel size = 1.1 x 1.1 x 1.1 mm (acquired slice thickness was 2.2 mm with 2 x interpolation), field of view = 300 x 270 mm, matrix = 272 x 216, bandwidth = 200 Hz/pixel, scan time = 15 to 20 min. Contrast-enhanced imaging was performed both at early phase (early gadolinium-enhancement [EGE]: 1 to 20 min after injection) and late phase (LGE: 20 to 30 min after injection). These were acquired with the same resolution as non-contrast-enhanced imaging, but with TI = 400 ms and 1RR triggering (scan time = 8 to 10 min). Early phase EGE (up to 20 min) was captured with shorter scans, using one-half the number of slices (same slab thickness).

**HISTOPATHOLOGICAL ANALYSIS.** After imaging, swine were euthanized using potassium chloride. The hearts were removed and filled with polyurethane elastomer within 2 h of sacrifice. Specimens were fixed in buffered formaldehyde and then sectioned into 2-mm slices to contain the areas of ablation for processing. Slides from these areas were stained with hematoxylin and eosin and Masson trichrome. The specimens and slides were digitally scanned (ImageScope; Aperio, Sausalito, California) to measure lesion size. Because histopathological processing causes tissue shrinkage by 10% to 15% (10), we used gross pathology to measure width and depth of necrotic lesions (Figure 1). Lesion size measurements were performed, blind to other measurements, by a trained cardiac pathologist. Aspect ratio (depth/width) was calculated to compare the shapes of ablation lesions.
DATA ANALYSIS. RFCA lesion size and signal intensity were measured from CMR images and analyzed with quantitative analysis software (OsiriX, Pixmeo, Bernex, Switzerland). Contrast-to-noise ratio (CNR) was estimated as the signal difference between lesion or scar and neighboring normal myocardium, divided by the SD of the background noise. RFCA lesion width and depth measured in CMR images were compared with those from gross pathology in a matched slice orientation, using multiplanar reformatting if needed. In T1w images, we measured the width and depth of enhanced core and width of hypointense rim. In contrast-enhanced images, we measured the width and depth of hypointense core, described as no-reflow region (7), and enhanced rim. Lesion volume was measured in 3D CMR images using open-source software (Seg3D 2.3.2, National Institutes of Health National Center for Research Resources, Salt Lake City, Utah).

STATISTICAL ANALYSIS. Continuous variables are expressed as mean ± SD, if normally distributed, or otherwise as median (interquartile range [IQR]: 25th, 75th percentile). We used the Student's t-test or Mann-Whitney U test for parametric or nonparametric continuous variables, respectively. We used
A total of 59 RFCA lesions were created in the LV of 8 swine: 17 lesions in normal myocardium, 21 in BZ, and 21 in scar. Table 1 shows the comparison of each ablation procedure’s parameters. Force-time integral of RFCA in normal myocardium (1,019 ± 448 s) was significantly lower than in BZ (1,437 ± 672 s, p = 0.043) and scar (1,702 ± 675 s, p = 0.001). The decrease of local amplitude and impedance during ablation in normal myocardium (median: 1.28 [IQR: 0.67, 1.88] mV, 0.04 [IQR: 0.01, 0.14] Ω, respectively) was significantly larger than in BZ (median: 0.38 [IQR: 0.19, 0.57] mV, p < 0.001; 0.01 [IQR: 0.00, 0.03] Ω, respectively) and scar (median: 0.27 [IQR: 0.15, 0.42] mV, p < 0.001; 0.03 [IQR: 0.01, 0.06] Ω, p = 0.006, respectively).

**RESULTS**

**ABLATION PROCEDURE.** A total of 59 RFCA lesions were created in the LV of 8 swine: 17 lesions in normal myocardium, 21 in BZ, and 21 in scar. Table 1 shows the comparison of each ablation procedure’s parameters. Force-time integral of RFCA in normal myocardium (1,019 ± 448 s) was significantly lower than in BZ (1,437 ± 672 s, p = 0.043) and scar (1,702 ± 675 s, p = 0.001). The decrease of local amplitude and impedance during ablation in normal myocardium (median: 1.28 [IQR: 0.67, 1.88] mV, 0.04 [IQR: 0.01, 0.14] Ω, respectively) was significantly larger than in BZ (median: 0.38 [IQR: 0.19, 0.57] mV, p < 0.001; 0.01 [IQR: 0.00, 0.03] Ω, respectively) and scar (median: 0.27 [IQR: 0.15, 0.42] mV, p < 0.001; 0.03 [IQR: 0.01, 0.06] Ω, p = 0.006, respectively).

<table>
<thead>
<tr>
<th></th>
<th>Normal (n = 17)</th>
<th>Border Zone (n = 21)</th>
<th>Scar (n = 21)</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Force-time integral, g s</td>
<td>1,019 ± 448</td>
<td>1,437 ± 672</td>
<td>1,702 ± 675</td>
<td>0.005*</td>
</tr>
<tr>
<td>Amplitude before RFCA, mV</td>
<td>2.83 (2.06, 3.30)</td>
<td>1.17 (0.84, 1.34)</td>
<td>0.16 (0.13, 0.29)</td>
<td>&lt;0.001</td>
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<td>Amplitude decrease, mV</td>
<td>1.47 (0.77, 1.74)</td>
<td>0.46 (0.20, 0.66)</td>
<td>0.03 (0.00, 0.08)</td>
<td>&lt;0.001*</td>
</tr>
<tr>
<td>Impedance before RFCA, Ω</td>
<td>102 (100, 108)</td>
<td>96 (94, 102)</td>
<td>96 (93, 98)</td>
<td>0.008*</td>
</tr>
<tr>
<td>Impedance decrease, Ω</td>
<td>11.0 (10.0, 16.5)</td>
<td>9.0 (7.0, 11.0)</td>
<td>8.0 (6.0, 11.0)</td>
<td>0.009*</td>
</tr>
<tr>
<td>Lesion volume, mm³</td>
<td>397 (301, 474)</td>
<td>121 (87, 201)</td>
<td>66 (33, 123)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Lesion width, mm</td>
<td>9.6 ± 1.5</td>
<td>6.8 ± 1.6</td>
<td>6.2 ± 2.4</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Lesion depth, mm</td>
<td>6.5 ± 1.2</td>
<td>2.8 ± 1.4</td>
<td>2.0 ± 1.0</td>
<td>&lt;0.001*</td>
</tr>
<tr>
<td>Transmurality, %</td>
<td>51.5 ± 7.2</td>
<td>40.5 ± 12.6</td>
<td>38.6 ± 11.3</td>
<td>0.0001</td>
</tr>
</tbody>
</table>

Values are mean ± SD or median (interquartile range: 25th, 75th percentile). *One-way analysis of variance was used to compare each parameter among 3 groups. Kruskal-Wallis test was used to compare each parameter among 3 groups.

CMR = cardiac magnetic resonance; RFCA = radiofrequency catheter ablation.

ACUTE T1W AND CONTRAST-ENHANCED CMR IMAGING. Non-contrast-enhanced T1w and contrast-enhanced CMR imaging were performed at medians of 172 (IQR: 146, 292) and 279 (IQR: 251, 373) min after RFCA procedures, respectively. EGE imaging was started 1 to 2 min, and LGE 24.3 ± 6.3 min, post-contrast injection. The average heart rate during CMR imaging before and after ablation was similar (116 ± 12 vs. 99 ± 24 beats/min, p = 0.089). All RFCA lesions in normal myocardium (n = 17) demonstrated an enhanced core and a hypointense rim on T1w images (Figure 1B). EGE images exhibited hypo-intense lesion core with enhanced rim (Figures 1C and 2E), whereas LGE images showed the contrast-agent entering the lesion core, thus obscuring the borders (Figure 2F). The volume of enhanced core in normal myocardium on T1w images was significantly smaller than the total lesion volume measured in LGE, including the hypointense core and enhanced rim (T1w: 409 ± 138 mm³ vs. LGE: 1,523 ± 568 mm³, p < 0.0001). Correlation between T1w and LGE improved when including the hypointense rim on T1w images (1,321 ± 411 mm³, r² = 0.49, p = 0.001), with no significant difference (p = 0.24).

All lesion cores in BZ and scar were enhanced in T1w images, whereas scar itself was hypointense. This allowed delineation of all RFCA lesions even within and around scar (Figures 1B and 2B). However, with contrast-enhanced images, both the RFCA lesions and pre-existing scar were enhanced and not distinguishable from each other (Figures 1C, 2E, and 2F). As measured in T1w images, the median volume of lesion cores created in normal myocardium (397 [IQR: 301, 474] mm³) was significantly larger than those created in scar (66 [IQR: 33, 123] mm³, p < 0.001) and BZ (121 [IQR: 87, 201] mm³, p < 0.001), despite the lower force-time integral (Table 1). Volume rendering of the 3D T1w images gave clear understanding of the spatial relationship of RFCA lesions in and around scar (Online Figure 1, Online Video 1).

Although lesions were well visualized when imaging at either 1.5-T or 3-T, CNR of lesions was significantly lower when imaging at 1.5-T than at 3-T (23.8 ± 10.6 vs. 42.1 ± 22.1, p < 0.001). CNR of lesions created in BZ and scar was similar to that of lesions created in normal myocardium when imaging at either 1.5-T (22.9 ± 11.7 vs. 25.8 ± 7.7, p = 0.37) or 3-T (40.3 ± 23.7 vs. 47.5 ± 16.8, p = 0.43). The CNR of scar itself was much lower (1.5-T: −7.2 ± 6.5, p < 0.001; 3-T: −14.8 ± 8.7, p < 0.001), allowing clear differentiation between lesion and scar (Online Figure 2).

HISTOPATHOLOGICAL FINDINGS. We analyzed all 59 RFCA lesions of 8 swine. Each lesion was noted on gross pathology to be in the appropriate targeted area-normal myocardium, BZ, or scar. Each lesion exhibited 2 concentric regions with different types of
damage: a core of coagulation necrosis with global eosinophilic changes and hemorrhage (Figure 1E); and a rim of contraction band necrosis with partially intact myocardial cells, hemorrhage, and hemostasis (Figures 1E and 1F). On gross pathology, the coagulation necrosis appears as a gray core, surrounded by a dark rim of contraction band necrosis (Figure 1A). RFCA lesions in normal myocardium demonstrated centrifugal extension from coagulation necrosis, to contraction-band necrosis with hemorrhage, to intact myocardium (Figure 1D).

Among 42 lesions within BZ and scar, coagulation necrosis extended into fibrotic tissue regions in 26 lesions (62%) and beyond fibrotic tissue in 14 lesions (33%). The aspect ratio (depth/width) of necrotic ablation lesion core was lower in the BZ (0.39 ± 0.15, p < 0.0001) and scar (0.33 ± 0.13, p < 0.0001) compared with that in normal myocardium (0.68 ± 0.22). In 12 lesions (29%), necrosis was peripheral to fat within scar. In 7 of them, fat within scar, resulting from infarct healing, appeared to block and redirect the expansion of necrosis created during RFCA (Figure 1H), confining the necrosis to a channel of preserved myocardium (Figure 2C).

CMR imaging compared with pathology. The enhanced lesion core width, depth, and transmurality on T1w images were well correlated with those of necrotic core seen on pathology ($r^2 = 0.94; r^2 = 0.94; r^2 = 0.87$, all $p < 0.001$, respectively) (Figures 3A to 3C), without systematic difference ($p = 0.061, p = 0.052, p = 0.21$, respectively) (Figures 3D to 3F). In T1w imaging of RFCA, the hypointense rim surrounding lesion cores created in normal myocardium was significantly thicker than that of the contraction-band necrosis on pathology samples by 0.55 mm.
Do not hallucinate.

W e could not delineate the hypo-intense rim in and around scar because both were hypo-intense. In normal myocardium, the lesion width of the hypo-intense core in EGE correlated with necrosis width of pathology ($r^2 = 0.82$, $p < 0.001$) without systematic difference ($p = 0.21$), whereas it tended to underestimate in smaller lesions and overestimate in larger lesions (Online Figure 3). Although the lesion width of the hypo-intense core in LGE correlated with necrosis width ($r^2 = 0.66$, $p < 0.001$), it systematically underestimated by 1.4 mm (95% confidence interval: 0.5 to 2.4, $p = 0.005$). The width of the enhanced lesion rim in LGE correlated with that of contraction-band necrosis ($r^2 = 0.61$, $p = 0.001$); however, it systematically overestimated by 3.5 mm (95% confidence interval: 2.7 to 4.3, $p < 0.001$).

**FIGURE 3** Linear Regression Analysis and the Bland-Altman Plots of Ablation Lesion Measurements on Non-Contrast-Enhanced T1w CMR Images and Necrosis on Gross Pathology

Linear regression analysis and the Bland-Altman plots of ablation lesion measurements on non-contrast-enhanced T1w CMR images and necrosis on gross pathology. Lesion width (A), depth (B), and transmurality (C) on T1w CMR images are well correlated with those of necrosis on pathology. The blue dots and red triangles represent the measurements using a 1.5-T and a 3-T scanner, respectively. There was no systematic difference of lesion width (D), depth (E), and transmurality (F) between T1w CMR and pathology. The red and blue dotted lines represent the 95% confidence intervals and limits of agreement (mean ± 1.96 SD), respectively. Abbreviations as in Figure 2.

**LESION VOLUME AND PARAMETERS OF THE ABLATION PROCEDURE.** The relationship between lesion core volume measured on T1w images and each parameter of the ablation procedure is shown in Figure 4. Force-time integral and local impedance decrease during ablation were correlated with lesion volume in normal myocardium ($r^2 = 0.32$, $p = 0.018$; $r^2 = 0.53$, $p < 0.001$, respectively), but they were not correlated with lesion volume in BZ ($r^2 = 0.10$, $p = 0.08$; $r^2 = 0.03$, $p = 0.21$, respectively) and scar ($r^2 = 0.05$, $p = 0.16$; $r^2 = 0.02$, $p = 0.56$, respectively). There was no correlation between the amplitude decrease of bipolar electrograms and lesion volume. Impedance before ablation was not significantly different in BZ and scar with or without fat (95.5 ± 4.4 vs. 97.2 ± 6.7 Ω, $p = 0.56$) and did not correlate with volume and aspect ratio of lesion in BZ and scar.
DISCUSSION

MAIN FINDINGS. This is the first report to study ablation lesions created within and around scar from MI using CMR, gross pathology, and histology. There were 3 main findings. 1) Non-contrast-enhanced T1w CMR imaging selectively enhanced acute lesions, allowing differentiation of acute ablation lesion necrosis from infarct scar. Contrast-enhanced imaging did not allow such differentiation, because both tissues were enhanced. 2) Ablation lesion volumes in BZ and scar were significantly smaller than those made in normal myocardium and not correlated with ablation parameters, such as force-time integral and decrease in impedance and amplitude. 3) Ablation-lesion extent surrounding scar depended on the structure and composition of scar tissue. Fat acted as a thermal insulator, whereas preserved myocardium acted as a conducting channel.

T1W CMR IMAGING. Recent papers (9,10) showed that T1w imaging provided more robust characterization of acute RFCA lesions than either T2-weighted imaging or LGE. In particular, inversion-recovery T1w imaging with TI = 700 to 800 ms could accurately depict the lesion core (10,11). Enhancement of lesion cores on T1w images resulted from T1 shortening of necrotically ablated tissue. Changes in T1 during ablation were likely the result of configuration alterations in heat-denatured proteins (15), including hemoglobin (16) and myoglobin. We found the enhancement to be highly correlated with necrosis. CMR imaging at 3-T provided higher CNR of ablation lesions than 1.5-T, but with higher variability, which might be due to less effective image normalization during image reconstruction, where signals are brighter near the surface coils.

Acute lesion appearance in contrast-enhanced imaging varied with time after contrast injection, a result of the wash-in and wash-out kinetics of the tissue (7). The no-reflow region in EGE tended to underestimate lesion core of smaller lesions and overestimate that of larger lesions, compared with pathology. LGE exhibited additional enhancement of adjacent edematous tissues, causing overestimation of lesion size (12). These results suggest that T1w imaging is superior to contrast-enhanced imaging in the delineation of acute RFCA lesions within and around scar.

ABLATION PARAMETERS TO ASSESS LESION EXTENT. Assessment of necrotic-lesion extent is important for eliminating arrhythmic substrates and for safe performance of RFCA. Although changes in

![Figure 4](image-url)
electrical impedance and electrogram amplitude have been used for lesion assessment (17), the accuracy is controversial (18,19). Recent studies showed that monitoring electrode-tissue contact force is the most reliable and accurate method to ensure lesion creation (19). In this study, however, the force-time integral did not correlate with lesion volume within and around scar. Our results suggest that the composition of scar including fat limit the spread of lesion necrosis, which is not taken into account by the force-time integral. T1w imaging reliably assessed acute lesion creation in the heterogenous environment surrounding infarct scar.

Recent papers showed the utility of impedance to identify scar regions due to significantly lower impedance than in normal myocardium (20). In our study, however, the difference between impedance of BZ or scar and that of normal myocardium was smaller than that reported (5% vs. 33% on average). This is likely because they used different mapping catheters without contact force sensing and continuous scanning of frequencies in the range of 1 to 1,000 kHz, whereas we used contact force-sensing catheter with clinical system scanning only at 500 kHz. Nevertheless, earlier authors reported an impedance difference between scar and normal myocardium of <10 Ω at 500 kHz, which was similar to our result of 8 Ω. Furthermore, in our study, impedance did not reflect the difference of scar composition such as fat. The electrical properties are only relevant very close to the ablation electrode (within a few millimeters), where the energy is converted from RF to heat (21). Fat occupied mid-wall and epicardial layers that were not close enough to the catheter to affect electrical impedance measurement.

**ABLATION LESION FORMATION IN SCAR.** The lesion formation process within and around scar is an important, although not well-understood, issue for VT ablation. There have been conflicting reports of ablation lesion size in normal versus diseased ventricles, which are likely related to differences in the models, ablation parameters, and equipment (22,23).

In the current study, we demonstrated that volumes of lesions created with RFCA, using clinical equipment and settings, were significantly smaller within and around scar than those in normal myocardium. Additionally, the aspect ratio (depth/width) of lesions in BZ and scar was significantly lower than that in normal myocardium. This could result from the fibrosis and fatty scar tissue behaving as a thermal insulator. Fat particularly appeared to block RF lesion spread transmurally and redirecting the expansion of necrosis circumferentially. Liu et al. (24) used phantom and computational models to report a negative exponential correlation between the percentage of fatty tissue and thermal conductivity. Additionally, they demonstrated that as the tissue fat content increased, temperatures increased at the tissue edges, but decreased within the tissue. Our findings are compatible, suggesting that fat acts as a thermal insulator, while preserved myocardium within scar acts as a conducting channel. Wong et al. (25) reported that epicardial RFCA lesions had substantially less penetration into myocardium with underlying areas of fat compared with normal myocardium, using an ovine model. Our results, from both CMR and histopathology, showed that the ablation-lesion extent around scar depended on the structure and composition of the scar.

**CLINICAL IMPLICATION.** Expansion of necrosis beyond the fibrotic tissue was observed in 33% of lesions, suggesting that ablation within or beyond scar borders is feasible, especially with T1w imaging verification. Because myocardial fat in chronic MI can be detected using CMR (26), pre-procedural CMR may be useful not only for detection of arrhythmic substrates but also for prediction of the effects of RFCA. Furthermore, non-contrast-enhanced T1w CMR may allow treatment evaluation intra- or post-procedure because RFCA lesions can be visualized minutes after ablation (6,9,10). T1w imaging is highly advantageous in monitoring treatment progression because noncontrast imaging can be repeated as needed, whereas contrast-enhanced scans are limited by the dosage restrictions (27) and the need to wait for systemic clearance. Thus, the proposed technique may assist in obtaining complete ablation of arrhythmogenic substrate and improve outcomes.

**STUDY LIMITATIONS.** Limitations exist when extrapolating the study results to the clinical setting. First, this study was performed in swine 8 weeks after MI, although previous studies have shown similarity with human MI to demonstrate substrates of VT (13,28). Second, we performed RFCA with only 1 ablation setting. Further study is needed to observe effects of other clinically relevant settings. Third, force-time integral of RFCA in normal myocardium was significantly lower than that in BZ and scar. We believe catheter contact in normal myocardium was less stable than in scar due to the myocardial contraction and ventricular ectopies during ablation. However, it is important to note that lesion in normal myocardium was significantly larger than that in BZ and scar despite the lower force-time integral. Fourth, we did not estimate lesion volumes from pathological samples because typical methods...
assuming half-ellipsoidal shapes are inaccurate, especially for irregularly shaped lesions in or near scar tissue. Finally, we did not analyze the effect of slice orientation on measurement accuracy because it is typical to use a short-axis orientation for clinical imaging of the LV.

CONCLUSIONS

Non-contrast-enhanced T1w CMR imaging can visualize acute RFCA lesion necrosis within or adjacent to scar. The presence of scar and fat alter lesion formation by insulating and redirecting heat propagation from ablation. As a result, ablation lesion volume within and around scar does not correlate with standard ablation parameters including force-time integral and is significantly smaller than that in normal myocardium. Because T1w CMR can detect these effects even in scarred substrate, unlike contrast-enhanced methods, it may be a more effective tool to accurately assess ablative treatment intra- and post-procedure.

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KEY WORDS cardiac magnetic resonance, myocardial infarction, non-contrast-enhanced T1-weighted imaging, radiofrequency catheter ablation, ventricular arrhythmia

APPENDIX For an expanded Methods section as well as supplemental figures and a video, please see the online version of this paper.