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RESEARCH LETTER

Lesion Size and Lesion Maturation After Radiofrequency Catheter Ablation for Ventricular Tachycardia in Humans With Nonischemic Cardiomyopathy

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Human data on the evolution of radiofrequency catheter ablation (RFCA) lesions are limited and largely lacking in nonischemic cardiomyopathy (NICM). Morphological changes after RFCA in postinfarction swine models have been well described and show significantly smaller lesions in scar compared with nonscar areas.[1] Importantly, patterns of fibrosis in NICM are different from postmyocardial infarction scars[2] and may influence lesion size. The importance of understanding radiofrequency lesion evolution is also highlighted by a recent study of histology after stereotactic radioablation (SBRT) in which prior radiofrequency ablation likely complicates interpretation of the effects of SBRT.[3]

The data that support the findings of this study are available from the corresponding author upon request. All patients were treated according to our standard clinical protocol and provided informed consent for mapping and ablation. All patients or next of kin provided informed consent for postmortem analysis. Ten patients with NICM (male, median age 63 years [interquartile range (IQR), 56–66], 9/10 class IV/V variant) and ventricular tachycardia underwent electroanatomical mapping and irrigated-tip RFCA (45–50 W, 30 mL/min normal saline, maximum 43 °C, contact force >9g, duration 60 s) before death (transplantation in one). After death, the hearts were fixed, embedded in gelatin or HistOmer, and sectioned. Integration of EAM data with histology was performed as previously described.[2] Ablation lesions were examined on gross pathology and histology (7-µm sections stained with Sirius red, hematoxylin cross-stained). In each patient, depth and width of the three largest isolated lesions were measured and the volumes calculated as ellipsoids.

The median time between ablation and death was 39 days (range, 1–821). Ablation lesions were visible on pathology and histology within fibrotic areas. Median lesion width was 13.3 mm (IQR, 11.0–15.0), median depth 7.2 mm (IQR, 6.1–8.3), and volume 323.8 mm^3 (IQR, 225.9–479.9). Median wall thickness was 14.3 mm (IQR, 11.2–15.9); thus, importantly, ablation lesions reached depths of 54.5% (IQR, 45.3–64.3) of the wall thickness.

Recent (<30 days) ablation lesions were easily visible on gross pathology as semi-circular, endocardial lesions with hemorrhagic rims (Figure). Older lesions maintained the endocardial semi-circular shape but were fully scarred and white. On histology, the ablation lesions were also easily identified. Fresh lesions showed no fibrosis, rather denucleated, necrotic cells. Lymphocytic infiltration was seen around the lesion. Myocytes outside the rim were easily distinguished from necrotic tissue by the presence of nuclei. In patients with 21-, 28- and 49-day old lesions fibrosis had begun to form around the rim of the lesion. The interior of the lesion remained denucleated myocytes. The fibrotic rim appears to become thicker over time. Older lesions (≥343 days) were fully fibrotic without surrounding lymphocytic infiltration.

We found that lesions after RFCA in NICM patients are of considerable size with lesion maturation over

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several weeks to months. In a recent study, lesion depths after irrigated RFCA with contact force sensing were studied in an swine model of reperfused myocardial infarction.[1] The maximum achieved lesion depths (contact force 30g) were 5.1±0.6 mm, 3.3±0.7 mm, and 2.0±0.5 mm in normal myocardium, scar borderzone, and dense scar, respectively. The lesions created in our study population are considerably larger with a median depth of 7.2 mm. One possible explanation for the difference is different ablation settings used (45–50 W for 60 s versus 30 W for 30 s). However, the greater lesion depth may also be due to the difference in the nature of the scars.

Radiofrequency energy heats the tissue adjacent to the catheter tip by resistive heating, followed by conductive heating to distant myocardium. It has been speculated that postinfarct scars are less prone to conductive heating compared to normal myocardium explaining the difference in lesion size.

NICM scars, which are inhomogeneous and more diffuse,[2] may allow for deeper penetration of radiofrequency energy compared to ischemic scars and thus account for the deeper lesions shown here.

Given that the lesions reached depths of 54.5% of wall thickness, approaching the area of interest from 2 sides may allow for complete transmurality and elimination of deep substrates. However, in areas that cannot be reached from 2 sides (left ventricular summit, regions protected by fat), or areas with greater wall thickness (ie, hypertrophic cardiomyopathy), additional techniques are required. SBRT has been recently suggested if RFCA fails to eliminate the ventricular tachycardia substrate, which is more frequently encountered in patients with nonischemic scars. The course of tissue injury after SBRT is of great interest, but most patients who are treated have had radiofrequency ablation that failed. A recent paper described acute and chronic histological tissue changes following SBRT in humans. Their observations are consistent with our findings of radiofrequency lesions, highlighting the complexity of assessing ablation effects in this population.

Here, we have clearly described the gross pathological and histological features of radiofrequency ablation lesions and described how they mature over time. We hope that this information provides future researchers with a benchmark to discriminate between naturally occurring scars, scar formed by radiofrequency ablation, and scars formed by other interventions (Figure).[4]

ARTICLE INFORMATION

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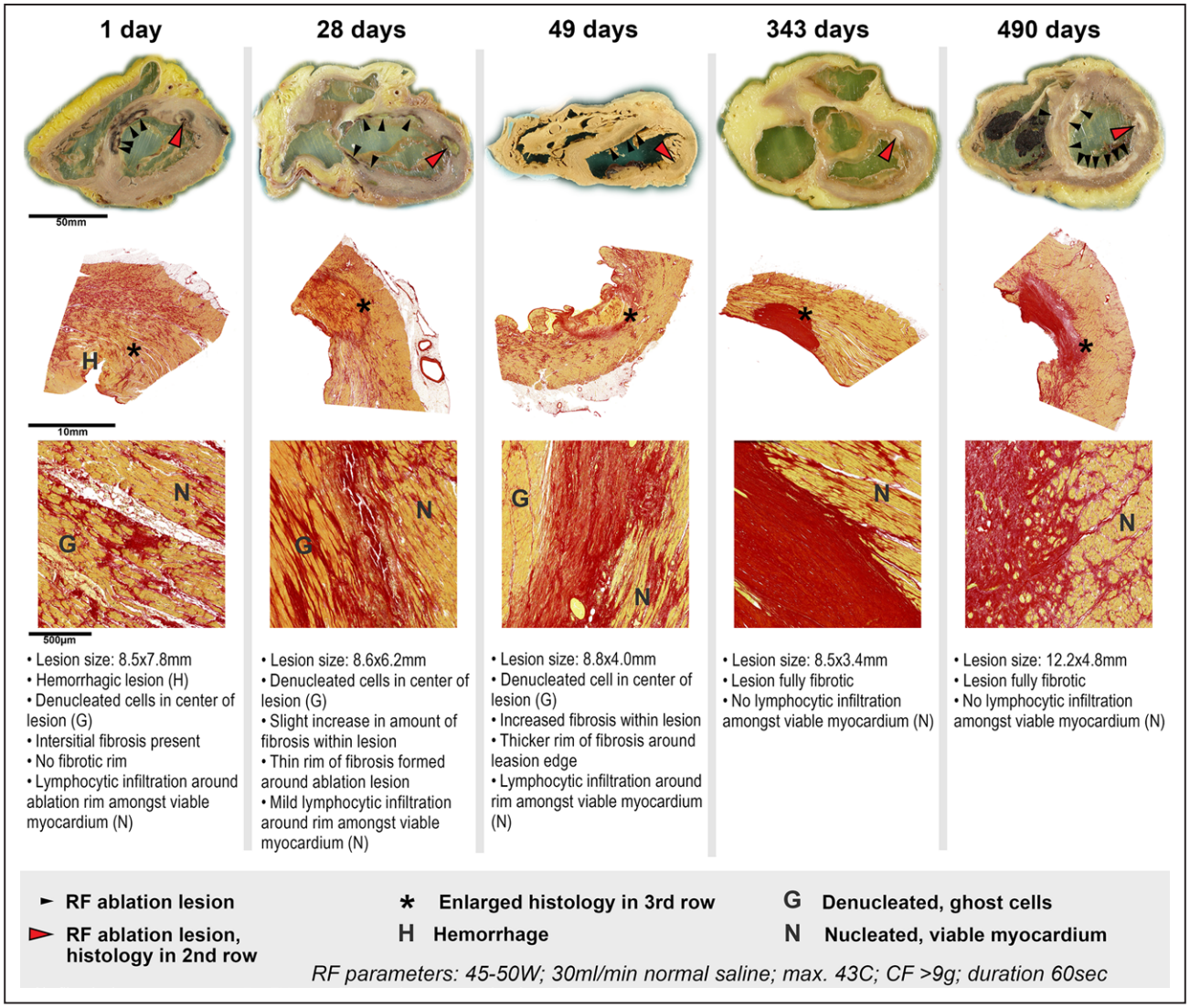

Figure. Pathological and histological changes occuring in NICM tissue after radiofrequency (RF) ablation.
Top, Basal short-axis slices. Number of days between RF ablation and explantation. **Middle** and **bottom**, Histological sections of ablation lesions, collagen-red, cytoplasm-yellow, nuclei-black. CF indicates contact force.

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