

High-Resolution MRI Of Temporal Evolution Of Thermal Ablation Lesions

Benu Sethi¹, Andriy Shmatukha¹, Mohammed Shurrah², Jennifer Barry¹, and Eugene Crystal²

¹Sunnybrook Research Institute, Sunnybrook Health Sciences Centre, Toronto, Ontario, Canada, ²Arrhythmia Services, Schulich Heart Centre, Sunnybrook Health Sciences Centre, Toronto, Ontario, Canada

Target Audience: Medical practitioners and scientists using or aiming to use MRI for the outcome assessment of different types of thermal ablations – especially, the radiofrequency (RF) ones used in electrophysiology (EP) being the only curative treatment available for drug-resistant cardiac arrhythmias (1, 2).

Purpose: Detailed investigation of how the spatial and temporal evolution of ablation lesions is reflected by MRI in order to identify the most appropriate imaging strategies leading to robust reliable spatial and temporal MRI quantification of therapeutic thermal damage without real-time thermal mapping. Such quantification would improve the safety and efficacy of clinical EP procedures currently suffering from high post-procedural arrhythmia recurrence rates attributed to insufficient ablation contiguity and transmurality (3, 4). Previous studies (e.g., 5, 6) did not observe lesion evolution over sufficiently long time periods.

Methods: 32 lesions were created in the Latissimus dorsi muscles of 8 rabbits using clinical-like power and time settings (35 Watt for 45 sec). In 6 animals, the lesions were created 15 min apart of each other and the animals were imaged immediately. 2 of them were sacrificed and the rest was survived and imaged at 2 wks after ablation, when 2 of them were sacrificed, while the other 2 were imaged again at 4 wks after ablation and then sacrificed. In the rest (2 animals), 2 lesions were created and the animals were imaged. 2 wks later, 2 additional lesions were created and the animals were imaged again, survived for another 2 wks, imaged and sacrificed. MRI was conducted at 1.5T using a standard transmit-receive birdcage head coil and consisted of high-resolution (HR) 3D T1w, T2w and LGE scans. Typically, T2w data was acquired with in-plane resolution of 0.31 x 0.31 mm, TR/TE of 900ms/26.8ms, echo train length of 24. T1w and LGE data was acquired with in-plane resolution of 0.31 x 0.38 mm, TI/TR/TE/FA of 200ms/15.7ms/7.5ms/25°, while an injection of a Gd-based contrast agent (0.1 ml/kg) preceded the LGE data acquisition with the centre of the k-space being acquired ~8.5 min. after the injection. In all scans, 22 1.2-mm thick slices with zero spacing were acquired and reconstructed to 72 1.2-mm thick slices with 0.9 mm overlap. After sacrifice and lesion extraction, 2-3 4-µm thick slices were cut out of each lesion parallel to the lesion's outer surface approximately at half the lesion's maximum depth. The samples were preserved in 10% formalin, then dehydrated, embedded in paraffin, stained with hematoxylin and eosin (HE) on glass slides and scanned as digital images.

Results: On all image types, the acute lesions had wide and fuzzy borders, which became thin and sharp 2 wks after ablation, but started widening and losing their

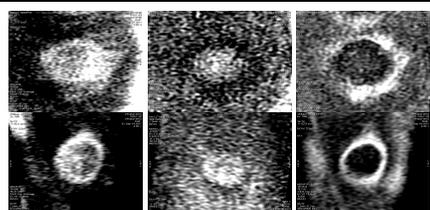


Fig. 1: T2w (left), T1w (middle) and LGE (right) images of the same lesion immediately after (upper row) and 2 wks after the ablation.

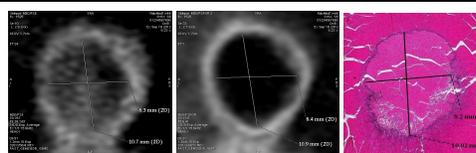


Fig. 3: A 2 wks old lesion on T2w (left), LGE (middle) and histology (right).

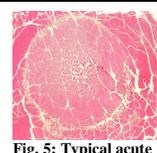


Fig. 5: Typical acute lesion on histology.

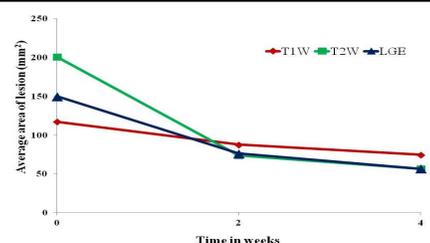


Fig. 2: Reduction in lesion size (area) over time

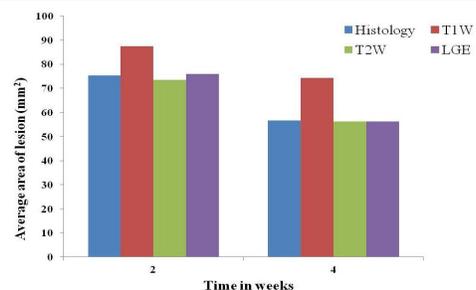


Fig. 4: Comparison of lesion size (area) as observed on T1w, T2w, LGE, Histology

sharpness at 4 wks (Fig. 1). This was accompanied by lesion size reduction with time (Fig. 2). As compared to the normal tissue, the lesions appeared on T2w images as hyper-intense areas (whose cores become darker with time, almost iso-intense with the normal tissue at 4 wks) surrounded at 2 and 4 wks by bright rims (thin and sharp at 2 wks) and almost absent at 4 wks) as bright cores surrounded by dark rims (wide in acute, very narrow at 2 wks and almost absent at 4 wks). On LGE, the lesions appeared as dark cores surrounded by hyper-enhanced rims, which became thin and sharp 2 wks after ablation but wide

and fuzzy at 4 wks. The cores were the darkest at 2 wks, while they looked brighter in acute and almost iso-intense with the normal tissue at 4 wks. On histological examination, both 2 and 4 wks lesions consisted of several zones with different levels of thermal damage.

Typically, each lesion was comprised of a coagulation necrosis core (the area with severe tissue damage consisting of cells with fragmented multifocal vacuolation), surrounded by a zone of relatively preserved (as compared to the core) tissue structure with fibrous tissue and post-haemorrhage hematoma, surrounded by an area of contraction band necrosis. The latter was surrounded by an inflammatory zone of transition to the normal muscle tissue. In 2 wks lesions, a very good correlation was observed between the sizes of dark cores observed on T2w, LGE (Figs. 3, 4) and histology (the contraction band necrosis rim). Lesions, which demonstrated broader fuzzier borders on 2 wks LGE and T2w images, were also found to demonstrate considerably smaller sizes and more diffused borders on 4 wks LGE and T2w images as well as histology as compared to their counterparts demonstrating sharp thin borders on 2 wks T2w and LGE. Some smaller lesions, which demonstrated fuzzy unclear borders at 2 wks T2w and LGE appeared to be (almost) completely healed at 4 wks MRI and histology. The rim areas more bright and wide on T2w are also more bright and wide on LGE and more dark and wide on T1w.

Discussion: MRI characterization and quantification of the initial thermal damage immediately after thermal ablations are desirable but challenging due to the time-transient nature of the ablation lesion borders. A “golden age” exist (probably, specific for each tissue, but around 2 wks in the rabbit skeletal muscle), at which lesion borders become sharp, well-defined, easily identifiable on HR T2w and early LGE, and well-corresponding to histology. At the “golden age”, the hyper-intense rims surrounding ablation lesions on T2w and early LGE correspond to the contraction band necrosis observed on histology, thus high resolution and contrast-to-noise ratio T2w imaging can be used as a substitute for LGE to deliver the same information without injecting Gd-based (and so potentially nephrotoxic) contrast agents. At the “golden age”, the sharpness, thickness and intensity of the hyper-intense rims surrounding ablation lesions on T2w and early LGE can serve as a predictor of approaching or distant lesion healing. Outside the “golden age”, perfusion-based techniques (early LGE and dynamic contrast enhancement) represent the ablation lesion borders (contraction band necrosis) more reliably and accurately than T1w and T2w. The dark rim surrounding the ablation lesion cores on T1w has the T1 relaxation time ~290 ms. It was prominent in acute lesions and was attributed to the prominent fresh haemorrhage (Fig. 5) surrounding the coagulation necrosis core in acute lesions. It was observed on all acute T1w images, but vanished later (2 and 4 wks) on both T1w MRI and histology (replaced by post-haemorrhage hematoma). It can serve for the discriminator between acute and chronic lesions during repeated thermal ablations (e.g., during the treatment of recurrent cardiac arrhythmias).

Conclusion: Ablation lesion formation and evolution follows the initial thermal damage and continues until complete lesion healing. HR MRI (especially early LGE and T2w) are reliable estimator of the contraction band necrosis and lesion healing stage (especially at the “golden age”). The reported observations are in a good concordance with previously reported ones (e.g., 7) except for those failing to represent properly the ablation lesions at more than 24 hrs after ablations (8).

References: 1. Cappato et al; Circulation 2005; 111: 1100-1105 2. John and Stevenson; Cur. Cardiol. Rep. 2011; 13(5): 399-406 3. Deneke et al; Europ. Heart Jour. 2005; 26: 1797-1803 4. Ranjan et al; Circul.: Arrh. Elect. 2011; 4: 279-286 5. Dickfeld et al; Heart Rhythm 2007; 4: 208-214 6. Dickfeld et al; J Am Coll Cardiol 2006; 47: 370-378 7. Shmatukha et al; Proc. of 8th Interv. MRI Symp. 2010; P-25, 237-239 8. Kholmovski et al; Proc. Intl. Soc. Mag. Reson. Med. 20 (2012) 397