High-Resolution MRI Of Temporal Evolution Of Thermal Ablation Lesions
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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 electrophiology (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 transmurlarity (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 hematoxilin 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 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). On T1w, the lesions appeared 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-haemorrhaghe 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 fuzzy 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 thin sharp borders on 2 wks T2w ad 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 thermal mapping. Such quantification 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).
