MR Microscopy in Forensic Medicine: Analysis of Electric Injury Patterns in Human Skin

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Three-dimensional magnetic resonance microscopy (MRM) images of fixed skin specimens provided a complete view of the damaged tissues at the site of an electric injury as well as in neighbouring tissues, consistent with histologic reports. The T_2 of the dermal layer in the central zone was reduced due to thermal damage and increased in the intermediate zone because of cellular necrosis caused by the electric field. A subjacent blood vessel with intravascular thombosis supports the hypothesis that electricity traveled through the vascular system before arcing to ground. This study is the first forensic application of MRM to the analysis of electric injury patterns in human skin.

Introduction: In forensic medicine, whole-body X-ray radiography is used routinely to assess skeletal damage. However, damage assessment in soft-tissues is central to many forensic studies. It is therefore surprising that there are just a few reports on the use of whole-body MRI in post-mortem examinations (1, 2). The purpose of this study was to demonstrate magnetic resonance microscopy (MRM) characterization of electric injury in human skin. The pattern observed for an electric injury depends on the strength and frequency of the electric field, the path of the current, and the histoarchitecture of the tissues. Tissue trauma can result from electric field effects and Joule heating due to the passage of electricity (3). To characterize the electric injury pattern in skin a variety of techniques, ranging from histology to scanning electron microscopy, have been applied (4,5). These techniques give detailed information about changes to cell morphology in sections take at the site of the entrance and exit wounds, but provide little information about the extent of tissue damage in peripheral and deep tissues. Clinical MRI studies can provide some information about vessel patency and muscle necrosis, but the injury pattern is lost due to limited spatial resolution (6). In this work, MRM was used to characterize the microanatomy of an electric injury pattern in human skin.

Materials and Methods: Three skin specimens, with visible epidermal lesions, were dissected from the palm and sole of the foot of a human cadaver that had received a fatal electric shock, then fixed in formalin. For MRM experiments skin samples were rehydrated and imaged in phosphate buffered saline. All MRM experiments were performed on a Bruker Biospec spectrometer (Bruker Instruments, Inc. Billerica, MA) coupled to horizontal magnet operating at 7T (300 MHz for protons). Skin samples were imaged in a 35 mm probe at room temperature with fat suppression. Quantitative T_2 relaxation maps were calculated from 16 images acquired with a multiecho sequence (TR = 5s, 2τ = 12 ms). Magnetization transfer (MT) maps were calculated using the following equation: $[1 - M_{so}/M_o]$, where M_{so}/M_o gives the ratio of image intensities acquired with and without the application of a 5-s, 12-μT saturation pulse 6000 Hz off-resonance. Quantitative 2D images had a slice thickness of 2 mm and an in-plane resolution of 120 μm. 3D images were acquired with a RARE imaging sequence (TR = 2s, TE = 8 ms, NEX = 1, RARE = 8).

Results and Discussion: On gross inspection, lesions were found to be composed of three zones: a central zone, an intermediate zone, and a peripheral zone. In the central zone the epidermal layer was completely destroyed and the underlying dermis was thermally damaged. In the intermediate zone the epidermis had detached from the dermal surface. In the peripheral zone there was little evidence of damage to cutaneous tissues. To assess the damage to cutaneous tissues in all three zones, two-dimensional T_2 and MT maps were acquired perpendicular to the skin surface and directly through the wound site. The T_2 and MT values for the hydrated epidermis were 48 ± 16 ms and 0.71 ± 0.06, respectively except in the central zone where it had been completely destroyed. The thermally damaged dermal tissue in the central zone had reduced T_2 values (14 ± 9 ms) compared to dermal tissue at the periphery (28 ± 4 ms). However, there was very little difference in the MT for these two zones. The reduction in T_2 of the dermal layer in the central zone was consistent with carbonization. In the intermediate zone, with the detached epidermis, T_2 increased from 28 ms in the peripheral zone to 40 ± 9 ms. In addition, there was a marked decrease in MT, from 0.70 ± 0.03 in the peripheral zone to 0.65 ± 0.04 in the intermediate zone. The increase in T_2 and the reduction of MT in the intermediate zone was consistent with cellular necrosis caused by the electric breakdown of cell membranes.

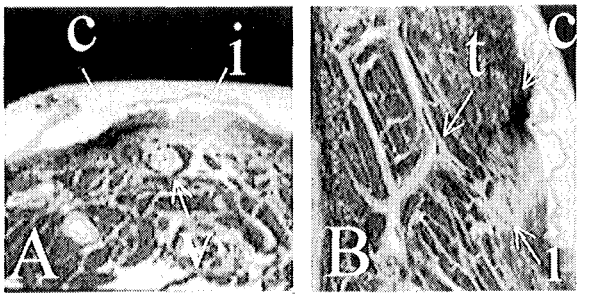

Fig. 1A: T2 weighted cross-section image of skin showing electric injury pattern with carbonized central zone (c) and necrotic intermediate zone (i) with vessel damage (v) Fig. 1B: T2 weighted view parallel to skin surface showing carbonized central zone (c), intermediate zone (i) and intravascular thrombosis (t) in the peripheral zone.

Fig. 1 shows two T_2-weighted images extracted from a 3D data set acquired for a formalin-fixed skin specimen from the plantar surface of the foot. Thermally damaged tissue appears dark in **Fig. 1A** and necrotic tissue appears bright. Necrosis was observed to extend into the hypodermal layer. With fat suppression this layer, which is composed mostly of fat, appears dark except for the necrotic zone, blood vessels running parallel to the skin surface, and connective tissue septa between clusters of adipocytes. And a vessel traversing the wound site had ruptured. The image shown in **Fig. 1B** was extracted from the 3D data set, parallel to the skin surface showing the superficial vascular plexus. Of particular interest was the vessel below the wound site that had ruptured. There was very little damage to the vessel in the peripheral zone. However, there was a significant reduction in image intensity for the vessel lumen, compared to other vessels present in the hypodermis. This reduced signal intensity was consistent with intravascular thrombosis or carbonization of the intimal surface of the vessel. A minor branch of the same vessel was also thrombosed. These observations support our hypothesis that electricity traveled through the superficial vascular plexus until it arrived at a vessel that was closest to the skin surface where it arced to ground generating electrical and thermal damage in the tissues in its path. Another minor lesion, characterized by a local disorganization of the connective tissue septa, was observed deep in the hypodermis but distal to the wound site. This lesion might be attributed to Joule heating by proximal osseous tissue. However, studies of the whole limb are required to answer this question.

Conclusions: Using three-dimensional MRM, we were able to establish the exact current path and resulting electric injury pattern in at least three lesions. The advantage of the MRM technique over traditional histologic studies is that the wound analysis was not limited to the underlying subcutaneous tissue, but included the peripheral and deep tissues of the intact sample. This type of analysis would be especially valuable in the clinical management of these injuries as well as in the development of forensic strategies to discriminate this type of injury from other types of trauma to the skin.

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