Detection and Delineation of Focal Cortical Lesions in Patients with Focal Epilepsy: Preliminary Results at 7T and 3T with 32 Channel Phased Arrays

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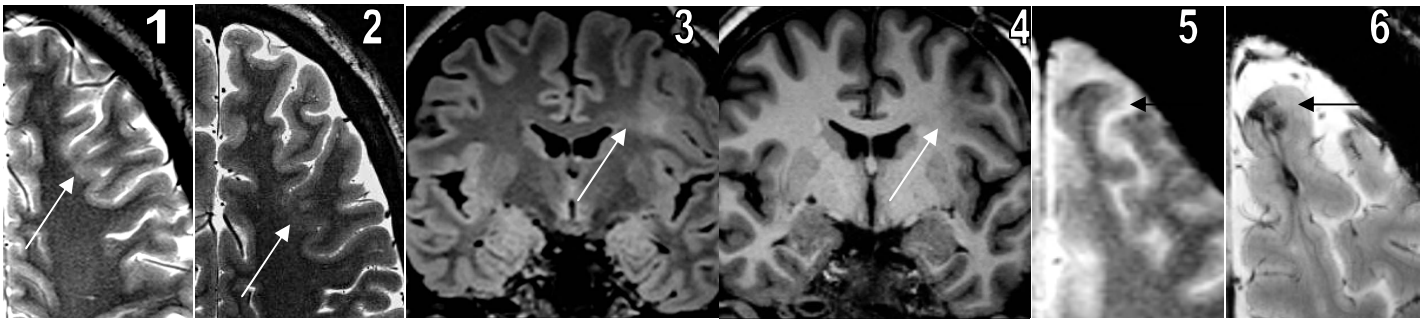
Introduction: MR imaging remains critical in the management of patients with medically refractory epilepsy. These patients continue to seize frequently despite multiple medications and their only hope for cure is surgical resection. Identification of a focal cortical dysplasia (FCD) on MRI minimizes the amount of brain that needs to be resected and dramatically improves the probability of a seizure free outcome. Using conventional MR imaging, either at 1.5 Tesla or 3 Tesla with current 12-channel phased array coils, some patients with lesional epilepsy are likely incorrectly labeled as MR negative. When a FCD is detected, cure is dependant on resecting the entire abnormality. However, the need for complete resection for seizure cure must be balanced with the desire to minimize or avoid functional deficits. Thus, accurate MRI depiction of anatomical extent provides the surgeon with knowledge required for minimal resection and optimal functional outcomes. Although abstracts have commented that 7 Tesla MRI has identified evidence of cortical involvement in Multiple Sclerosis not previously visualized at lower field strengths[1], no abstracts or published reports discuss the role of 7T imaging in the evaluation of patients with focal epilepsy. In this study, patients with known or suspected cortical dysplasias were imaged at 7T to determine if the area of cortical dysplasia could be better characterized and delineated. Results of 32 channel 3T and 7T imaging were compared to each other as well as with prior clinical MR studies to determine the role of each.

Methods: Four patients with known or suspected cortical dysplasia were imaged on a 7T whole-body Siemens Sonata MRI scanner (Siemens Medical Solutions, Erlangen, Germany) with a head only gradient insert and using an in-house developed 32-channel phased array coil. Axial 2D Flash, T2* spoiled gradient-echo weighted images (TR/TE=1200/21.7, flip angle=55°, FOV=192x168 mm^2, bandwidth=30 Hz, 1.0-1.5 mm thick slices with an in-plane resolution of 0.33 x 0.33 mm^2), and T2 turbo spin-echo, TSE, images (TR/TE = 6890/78, flip angle=150°, bandwidth=190 Hz, turbo factor=9, averages=1) with the same resolution and orientation as the Flash-T2* were obtained with coverage targeted to the clinically suspected area of concern. An MPRAGE (TR/TE/TI=2600/3.26/1100, bandwidth=200 Hz, 0.6 mm thick with an in-plane resolution of 0.6 x 0.6 mm^2) with the same orientation as the Flash-T2* and T2 TSE scans was also acquired to improve GM/WM boundary identification. Additionally, all patients were imaged on a 3T TrioTim Siemens MRI scanner utilizing a 32 channel phased array coil. Imaging sequences at 3T included sagittal MPRAGE (TR/TE/TI=2300/3.05/900, FOV= 230x230 mm^2, bandwidth=240 Hz, 0.9 mm thick with an in-plane resolution of 0.9 x 0.9 mm^2), sagittal SPACE T2-weighted FLAIR (TR/TE/TI=5000/352/1800, Flip Angle=120°, FOV=200x200 mm^2, bandwidth=790 Hz, 1 mm thick with an in-plane resolution of 0.5 x 0.5 mm^2 interpolated), axial T2 TSE (TR/TE = 6380/97, flip angle=150°, FOV=190x181 mm^2, bandwidth=150 Hz, turbo factor=11, averages=1) and axial diffusion tensor imaging, DTI, (TR/TE = 7980/84, bandwidth=1,395 Hz/Px, 1.85 mm thick with an in-plane resolution of 1.85 x 1.85 mm^2).

Results: Patient 1 is a 17 year old male with a long history of medically refractory complex partial seizures localized to the left frontal lobe. Two MRIs, including one at 3T with a 12 channel phased array coil, were reported as normal. Given the clinical suspicion for a left frontal focus, the patient was enrolled in the study. Imaging demonstrated an area of gray-white matter blurring, cortical signal abnormality and subtle increased T2 signal extending to the ventricle on both 7T and 3T imaging, consistent with a focal cortical dysplasia (Figure 1 Ax T2 image at 3T with 12 channel head coil, Figure 2 Ax T2 image at 7T). Surgical resection confirmed a FCD, type IIB. The patient has been seizure free since the surgery, with improved cognitive function. Patient 2 is a 38 year old female patient with long-standing, medically refractory seizures who had been extensively evaluated at an outside institution, with an MRI demonstrating a focal area of abnormal signal in the left frontal lobe that was thought to represent a focal cortical dysplasia versus a neoplasm. Patient was referred for evaluation to better delineate and characterize the known lesion. 7T and 3T imaging demonstrated blurring of the gray-white matter interface with abnormally thick cortex and an indistinct cortical ribbon (Coronal reformats from volumetric FLAIR, Figure 3, and MPRAGE, Figure 4, obtained at 3T with 32 channel coil). Abnormal white matter FLAIR hyperintensity extended from the lateral ventricular margin and fanned out radially to the dysplastic cortex. These findings were consistent with a cortical dysplasia and excluded a neoplasm. Patient 3 is a 20 year old male with stereotypic seizures which arise from the left frontal midline region. An initial MRI at 3T with 12 channel phased array coil was initially interpreted as normal, but given the seizure focus, a question was raised as to a possible FCD in the left insular region. Imaging at 7T and 3T with 32 channel coil demonstrated normal parenchyma, with no evidence of a cortical malformation, including the area in question in the left insular region. Patient 4 is a 44 year old male with a history of seizures who had an episode of aphemia, concerning for a partial seizure. MRI demonstrated a non-specific focus of FLAIR abnormality in the left frontal lobe. Additional imaging at 7t and 32 channel 3T demonstrated an area of focal encephalomalacia of the left superior frontal gyrus with hemosiderin in small areas of the cortex and subcortical white matter consistent with a prior hemorrhagic infarct (Figure 5 T2* image obtained as part of patient's clinical workup on a Siemens 1.5T MRI scanner, Figure 6 T2* image obtained at 7T).



Discussion: In two of the patients, advanced imaging with 7T and 3T with 32 channel head coils allowed characterization and delineation of focal cortical dysplasias. In a third patient, this combination of advanced imaging cleared an area of concern based on the initial study. And finally, the 7T T2* imaging was clearly able to identify an area of old infarct with hemosiderin staining that was below the level of detection of traditional MRI imaging. While the 7T imaging was able to demonstrate areas of cortical gray-white matter indistinctness as well as areas of hemorrhage, 3T imaging with volumetric FLAIR imaging helped identify the radial pattern of white matter abnormality. The two technologies complemented each other in defining lesion boundary and characterizing the involved tissue. 7T imaging clearly will be helpful in identifying lesions with iron deposition. As FLAIR imaging is developed at 7T, areas of white matter abnormality will hopefully become more easily identifiable. Until then, 3T will continue to have an important role, with advances in technique allowing more sensitive detection of abnormalities. Both 7T and 3T with 32 channel coil hold great promise as a problem solving tool for lesion identification in those patients with "MR negative" medically refractory epilepsy.

References: 1. Mainero C. et al. ISMRM 2008, Prog 508.