UTE imaging of white matter injury of immaturity

U. E. Svanholm1, M. Mårtensson1, B. Vollmer2, L. Holmström1, A. Takahashi4, B. Nordell,5 and O. Flodmark1

1Department of Hospital Physics, Karolinska University Hospital, Stockholm, Sweden, 2Department of Woman and Child Health, Karolinska Institute, Neuropediatric Research Unit, Stockholm, Sweden, 3Department of Woman and Child Health, Karolinska Institute, Neuropediatric Research Unit, Stockholm, Sweden, 4Global Applied Science Laboratory, GE Healthcare Technologies, Menlo Park, CA, United States, 5Department of Nuroradiology, Karolinska University Hospital, Stockholm, Sweden

INTRODUCTION

MRI with ultra-short echo time (UTE) has been shown to produce signal from tissue where conventional MRI does not. This includes protons bound to macro-molecules in the brain. The presence of short T2 components in white matter has been established (1), and an in-vivo study has shown that the short T2 components can be connected to the myelin in white matter (2). So far very little has been reported on the use of UTE to assess white matter injury in patient groups other than multiple sclerosis. In this work, however, a new patient group, children with injury to the white matter acquired early in life, is introduced as a potential beneficiary from imaging with ultra-short TE.

MATERIALS AND METHOD

Imaging: Imaging was performed on a 1.5 T clinical scanner (Signa Excite TwinSpeed; GE Healthcare Technologies, Waukesha, WI, USA) with an eight channel phased array head coil. A 2D ultra-short echo time sequence (3) was implemented, using a half pulse excitation with the slice selection gradient toggled between the two pulses, and subsequently adding data from the two excitations to create one full line in excitation k-space. Radial ramp sampling from k-space centre was performed. The resolution was 192x193 which is highly under sampled for radial imaging since full coverage of k-space would require n_HR^2 acquisitions (4). This is, however, not a major issue when scanning the brain with axial slices, due to the limited size of the anatomy compared to the field of view (5).

A non-selective inversion pulse was used to cancel the signal from the long T2 components in the white matter of the brain. To further suppress any remaining long T2 components a difference image was created by subtracting an image with the parameters conventional echo time (TE2) from the UTE image (TE1) (6). The sequence parameters used were: TR/TE/TE1/TE2 = 1250/380/0.09/15 ms, FA = 80° and FOV/slice thickness = 220/5 mm. The parameters were selected to yield a high contrast between the short components in white matter and surrounding tissue, while keeping the scan time reasonably low. The total acquisition time was 8 min. 5 sec.

Subject: The subject was a 14 year old male with hemorrhagic-ischemic periventricular white matter injury of immaturity on the left and subsequent right sided hemiplegia. It is expected that this type of injury will have caused not only tissue changes in the primary injury site, but also leads to secondary changes in periventricular white matter distant from the primary lesion (7), which may be visible on a UTE image. An age and gender matched healthy control was scanned for comparison.

RESULTS

Figure 1 shows a T2 weighted FSE (A) and a subtracted UTE image (B) of the subject’s brain, with the lesion clearly visible on both images. Figure 2 shows images of the brain in a location inferior to the lesion; figures A and B show a T2 weighted image (A) and a subtraction UTE image (B) of the subject’s brain, and figures C and D show the same type of images of corresponding anatomy in the healthy control. Figure 2B suggests that in the patient not only the myelin layer at the site of the primary lesion, but the entire myelin layer on the left is thinner than on the right. Furthermore, comparison between figures 2B and D shows that the subject has a thinner layer of short T2 components than the control on levels distant from the primary lesion site, which is supported by the finding that the amount of short T2 components normalized to brain volume is smaller in the subject (28%) than in the control (39%). This is consistent with the notion that in brain injury of immaturity secondary, more global, white matter abnormalities are likely to be present.

DISCUSSION

Our data illustrates the potential of using UTE for imaging atypical white matter. Conventional T2 weighted images rely on changes in water content to detect white matter abnormalities, whereas UTE images provide different and more detailed information on white matter in the brain. Apart from viewing T2 weighted images, changes in white matter are currently examined using MRI diffusion imaging. This, however, has several disadvantages. For example, ambiguity can sometimes arise in diffusion images when crossing fibers give rise to a signal void even where there is white matter present. Furthermore, UTE gives information about myelin content, which means combining these techniques may provide better information about whether loss of function is due to damage of the axon or myelin. Thus, including UTE in imaging of white matter could prove very useful in both refining diagnosis and planning and evaluating treatment. However, more research is needed, both on how to combine findings from different types of images, as well as on developing the pulse sequence to allow for multi-slice acquisitions.

REFERENCES