Diffusional Kurtosis Imaging in Acute Human Stroke

J. A. Helpern[1,2], C. Lo[1], C. Hu[1], M. F. Falangola[1], O. Rapalino[1], and J. H. Jensen[1]

[1]Radiology, NYU School of Medicine, New York, NY, United States, [2]Medical Physics, The Nathan Kline Institute, Orangeburg, NY, United States

Background

The management of acute human stroke is a high-priority medical emergency. Thrombolytic therapy is now part of the standard of care in most major medical centers, and the selection of patients for these treatments depends on several clinical factors as well as brain imaging findings. Diffusion MRI combined with perfusion imaging are accepted imaging protocols used to characterize ischemic stroke and a perfusion-diffusion mismatch has proven clinically important for the triage of patients for thrombolytic therapy. However, variability in the experimental methodology and analysis of the data as well as in the specific criteria used for interpreting the perfusion/diffusion mismatch are not without controversy. The most commonly used diffusion MRI images employed in the characterization of stroke include DWI and the calculated ADC maps. It has long been appreciated, however, that diffusion-weighted MRI is, in principle, capable of yielding considerably more information about the state of tissue than what is derived from presently implemented diffusion methods. Diffusional Kurtosis Imaging (DKI) is a relatively new MRI technique [1-3] for quantifying non-Gaussian water diffusion. Importantly, it is clinically a more practical technique (compared with, e.g., q-space imaging) with acceptable image acquisition times that can be easily implemented on clinical scanners using vendor-provided commercial software. From DKI, one can calculate all of the conventional diffusion metrics as well as a new metric, the mean kurtosis (MK). The MK measures a fundamentally different aspect of the diffusion process, is meaningful in both grey and white matter, and is relatively impervious to CSF partial volume affects. As a consequence, MK may be a more specific marker of tissue microstructural integrity. In this work, we present preliminary results of DKI applied to human ischemic stroke.

Methods

Data from 5 patients admitted to the ER with neurological symptoms compatible with acute ischemic stroke (based on the DWI and ADC maps) were acquired on a 1.5 T Avanto MRI (Siemens) as previously described [2,3] with nine 5 mm slices (no gap) centered on the main DWI hyperintensity using 6 b-values (0 to 2500 s/mm^2 in steps of 500) and 30 encoding directions. The time from initial ictus to MRI ranged from 12 hours to 6 days. DKI raw data were post-processed and MK maps were obtained. MK maps were compared with conventional DWIs and ADC maps.

Results and Discussion

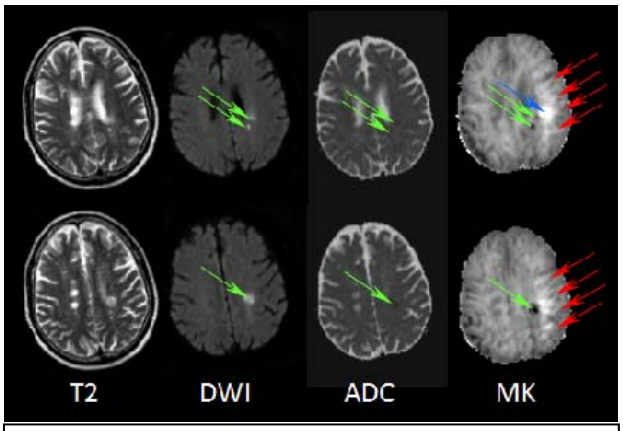

Figure 1: T_2, DWI, ADC and MK maps from two adjacent slices from a 75 y/o male presenting with R-sided weakness. Multiple foci of predominantly left deep WM acute infarction are seen with DWI hyperintensity, ADC decrease and MK decrease (green arrows). The MK map shows additional foci of very high MK (blue arrow, mean MK = 1.84 ± 0.16) surrounded by a region of diffusely increased MK (red arrows, mean MK = 1.23 ± 0.23) within the subcortical white matter of the left parietal lobe located lateral to the region of acute infarct. This region is not identified in the ADC or DWI images.

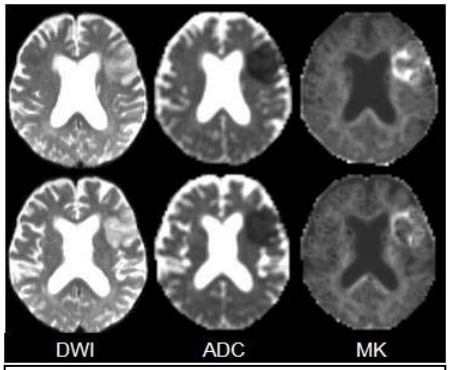

Figure 2: DWI, ADC and MK maps from two adjacent slices from a 74 year old woman with aphasia and weakness of her right hand. Here, DWI, ADC and MK delineate the same anatomical region of acute ischemia in the left frontal-parietal lobe. DWI and ADC are isointense within the region, whereas MK shows a heterogeneous appearance.

Due to the preliminary nature of this work, results are presented descriptively. For all cases, the MK maps identified brain regions of acute ischemia that correlated well with regions of DWI hyperintensity and ADC decrease, but in several patients, the MK maps also identified abnormal brain regions adjacent to the acute infarct that were not apparent on any other MRI image acquired (Figure 1). Within the region of acute ischemia DWI and ADC were generally isointense compared with the MK maps, which showed a heterogeneous appearance, possibly reflecting a differential degree of brain tissue damage (Figure 2). In one case there was evidence of progressive normalization of the MK and eventual relative decrease within days after the initial event exhibiting a faster temporal course in comparison with DWI. Finally, in one patient, subacute infarcts showing normalization of the ADC with persistent DWI signal abnormality showed decreased MK, visually different from the acute infarcts. Although speculative at this point, it is possible that MK maps with regions of abnormal kurtosis extending into the parenchyma adjacent to DWI/ADC-identified regions possibly correspond to reversibly injured ischemic tissue. The temporal behavior of the signal abnormalities on the MK maps may also prove to be more useful than DWI for differentiating acute versus subacute infarcts. The fact that MK identifies brain regions affected by ischemia that are not identified by conventional diffusion-weighted techniques is encouraging. Additional studies are needed to define the specificity/sensitivity of MK as well as its relationship to perfusion images.

References: 1) Jensen JH & Helpern JA, Proc. ISMRM, 2003; 11:2154. 2) Jensen JH, et al., Magn Reson Med, 2005; 53(6):1432-40. 3) Lu H, et al., NMR in Biomed, 2006; 19(2):236-47.