Stroke Analysis by Means of Kurtosis Diffusion Imaging in In Vivo Animal Studies

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Introduction

Diffusion-weighted magnetic resonance imaging (DWI) and diffusion tensor imaging (DTI) are powerful tools for brain visualization. Clinical applications range from the evaluation of stroke to elucidation of degenerative white matter abnormalities in multiple sclerosis and Alzheimer's desease. In particular, DWI has become a major tool for the detection of the ischemic regions and characterization of their development after the onset of stroke [1]. However, the biophysical mechanisms that cause a decrease in the apparent diffusion coefficient (ADC) are only poorly understood, and remain a subject of active debating. The most frequent interpretation refers to intercompartmental water shift and the increased diffusion restrictions in the intracellular space. Many parameters such as intra- and intercellular ADCs, the intracellular water volume fraction, a membrane permeability come into play.

The constraints imposed by a cellular microstructure and anisotropic arrangement on the molecular propagation give rise to deviations from normal isotropic unrestricted diffusion. These deviations cannot be adequately assessed by the standard methods based on the Gaussian model of diffusion. To overcome these limitations, diffusion kurtosis imaging (DKI) has been recently proposed [2] to characterize the non-Gaussian water diffusion in tissues. Although the DKI is still in an early stage of development, promising results in terms of better tissue characterization have been reported [3,4]. At the same time, its practical utility remains to be proven. In this work we report preliminary results of application of the DKI to characterization of ischemic regions in *in vivo* animal studies.

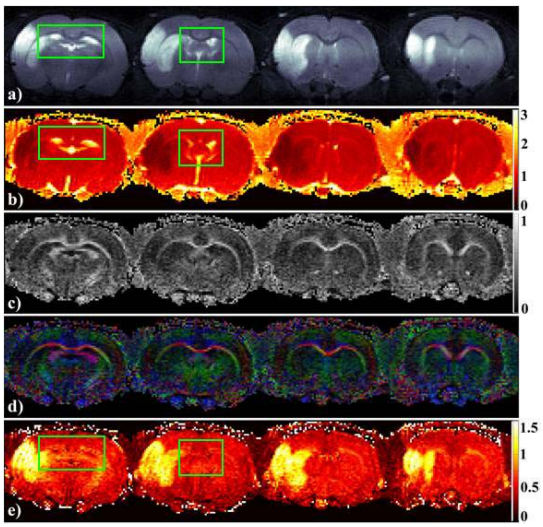

Figure 1. a) anatomical RARE; b) MD maps (units in μ s^{-1}); c) FA maps; d) colour-coded FA maps; e) MK maps.

Materials and Methods

All MR experiments were performed on a 7T system (Bruker, PharmaScan) equipped with magnetic field gradients with maximum strength of 760mT/m using a home built RF surface coil (2.5 cm diameter). The animals (300 g, Sprague-Dawley male rats) were anaesthetised with isoflurane (2%) and maintained at constant temperature (37°C) using a feedback-controlled air heating system. Four segment DW EPI SE images were acquired with the following acquisition parameters: TR/TE = 3000/30 ms, FOV = 3×3 cm^2, matrix size 128×128, slice thickness 1 mm, diffusion gradient duration δ = 5ms, NEX = 4. We used b values ranging from 0 to 3000 smm^{-2} and 20 gradient directions. The diffusion time was 17 ms. The acquisitions were respiration triggered. For the stroke experiments, transient MCAO was induced [5]. The rats underwent a 120 min transient occlusion and were imaged 6 hours after reperfusion. The maps of mean diffusivity (MD), fractional anisotropy (FA), and colour FA were produced according to standard DTI protocols. In terms of excess kurtosis, the attenuation curves are expressed as [2]: $S(b) = \exp(-bD_{app} + \frac{1}{6}b^2 D_{app}^2 K_{app})$, where D_{app} and K_{app} denote the apparent diffusivity and the apparent excess kurtosis, respectively. With respect to the diffusion propagator, excess kurtosis provides a dimensionless quantitative measure of the deviation from Gaussian diffusion. Mean excess kurtosis values (MK) were evaluated as the average over twenty gradient directions and set as a map parameter in the kurtosis maps.

Results and Discussions

Figures 1a - 1e show the anatomical RARE images (a) and the maps of MD (b), FA (c), colour FA (d) and MK (e). One can clearly identify an extended ischemic lesion on the left side in Figures 1a, 1b, and 1e. MD is decreased and MK is enhanced in the affected regions. The contrast produced by the MK maps appears stronger in comparison to that of MD. In difference to images in Figure 1a, MK is not enhanced in the ventricles of free liquid marked by squares. These results will be discussed in the context of complimentary information obtained by the DKI in comparison to that of standard methods. The potentials of this method in assessment of stroke will be addressed.

Conclusion

DKI is a simplest extension of DTI that enables one to quantify deviations from Gaussian diffusion of water molecules in brain tissue. Our results obtained for animals that experienced stroke have shown that DKI has a potential to better characterize pathological brain tissue in MCAO models.

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References

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