Diffusion time dependent kurtosis maps visualize ischemic lesions in stroke patients

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Introduction

Kurtosis maps have previously been proposed for visualization of non-Gaussian diffusion [1,2]. A single numerical value of the kurtosis is difficult to interpret, but in repeated diffusion weighted (DW) measurements using different diffusion times (T_D), changes of the kurtosis (K) may reflect changes of the underlying microstructural properties of the tissue. For example, a prolonged T_D may lead to reduced or increased K, due to either restricted diffusion or exchange of diffusing water molecules over the cell membranes. Furthermore, kurtosis is highly sensitive to the volume fractions of intra- and extracellular water, making it suitable to study changes in cell sizes. Here, we aim to investigate the contribution of differential kurtosis (ΔK) maps to the characterization of ischemic lesions in stroke patients.

Method

Nine ischemic stroke patients were examined between one and five days after the onset of stroke. Measurements were performed at a Siemens 3T Allegra head scanner in six different diffusion encoding directions using a stimulated echo pulse sequence. Signal-versus-b curves were acquired for T_D = 60 ms and T_D = 260 ms, by varying the mixing time (TM). The duration of the pulsed diffusion gradients was 27 ms and TR/TE was 2000 ms/105 ms. For each T_D, the signal curve was sampled with 16 b-values for b_{max} = 7500 s/mm^2. Regions of interests (ROIs) were placed in the lesions, as well as in contralateral normal-appearing brain matter (NAM). Six ROIs were placed in white matter (WM) and six in grey matter (GM). Diffusion tensor imaging (DTI) analysis was performed for both diffusion times, for b-values less than 1500 s/mm^2, giving mean diffusivity (MD) and fractional anisotropy (FA). In addition, K was calculated according to [1], but with b_{max} = 7500 s/mm^2. The change in kurtosis, ΔK, was calculated as $\Delta K = K(T_D{=}60) - K(T_D{=}260\text{ ms})$. For comparison, the differential MD (ΔMD) was calculated as $\Delta MD = MD(T_D{=}60) - MD(T_D{=}260\text{ ms})$.

Results

Figure 1 show a set of maps from one patient and Figure 2 shows the signal-versus-b curves for the two ROIs indicated in Figure 1. A summary of MD and K is presented in Table I for all the lesions and the control ROIs.

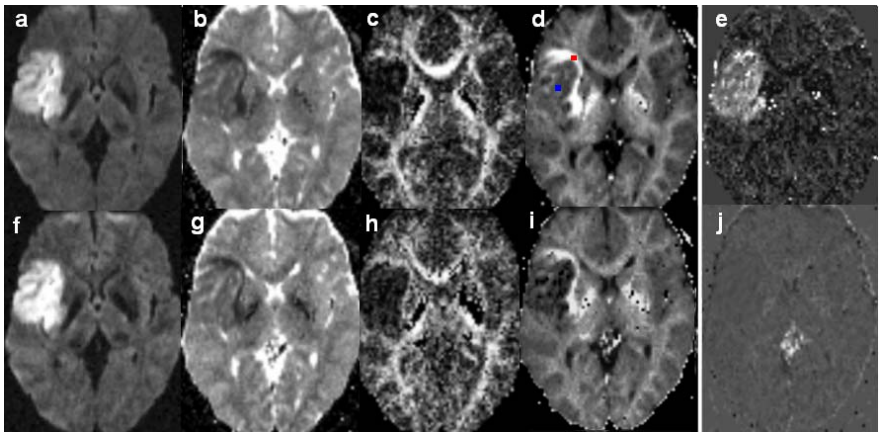
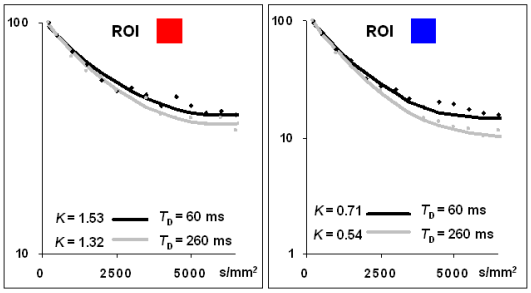


Figure 1. Maps from one patient, approximately 60h after the onset of stroke, (a,f) DW-image for b=1000 s/mm^2, (b,g) MD, (c,h) FA and (d,i) K, for T_D = 60 ms (top row) and T_D = 260 ms (bottom row). The stroke lesion is bright in the DW maps, and particularly the WM is dark in the MD maps. The WM in the periphery of the DW abnormality is present as a bright area in the K maps (high kurtosis). The ΔK map (e) demonstrate clear effects of a prolonged T_D, which is not observed in the ΔMD map (j) The NAM seems to be independent of T_D, both in the ΔK and ΔMD map. Moreover, the ΔK map visualizes the lesion (cf. the DW-image).



	MD(T_D60)	MD(T_D260)	K(T_D60)	K(T_D260)
Lesion WM	0.33±0.08	0.37±0.11	1.33±0.21	1.06±0.23
Normal WM	0.73±0.17	0.71±0.18	0.72±0.13	0.76±0.14
Lesion GM	0.52±0.10	0.53±0.12	0.66±0.13	0.39±0.11
Normal GM	0.76±0.11	0.76±0.14	0.62±0.12	0.65±0.10

Figure 2. Signal-versus-b curves from the red ROI are presented to the left and curves from the blue ROI to the right. The T_D dependence indicates effects of exchange.

Table I. MD [μm^2/ms] and K from the ROIs (±1 standard deviation). Increased K is observed in WM parts of the lesion as compared to NAM, although K decreases in the lesions at longer T_D. The GM parts of the lesion show values rather similar to normal appearing GM, but is severely decreased for prolonged T_D.

Discussion and conclusion

The increase in kurtosis in the WM part of the lesions is likely due to changes in volume fractions and reduced cell membrane permeability. The GM areas of the lesions show only slightly higher K than normal appearing GM for the shortest T_D, but the kurtosis for the GM in the lesion decreases with prolonged T_D. This indicates more normally distributed diffusion, i.e. apparently more freely diffusing, explained by the rapid water exchange between the intra- and the extracellular spaces [3,4]. The entire lesion area stands out in the ΔK map, as compared to the DW image. The ΔK map shows the effect of the transition to more normally distributed diffusion as T_D is prolonged, which is not observable in NAM.

In conclusion, we believe that the kurtosis parameter, and especially the ΔK-parameter, can provide new clinically relevant information about stroke lesions, since changes in cell morphology (in terms of cellular sizes) as well as in cell physiology (in terms of cellular membrane functionality) are represented by the ΔK parameter. For example, determining tissue at risk and diagnostic prediction of lesion development over time might be improved.

References

[1] JH. Jensen *et al*. MRM:2005;53;1432-40
[2] J. Lätt *et al*. MRI; 2008;26(1);77-87
[3] J. Pfeuffer *et al*. MRI; 1998;16:1023-32
[4] J. Lätt *et al*. in Proc. ISMRM; 2008, p. 1796