Differing Fractional Anisotropy Changes in Grey Matter and White Matter in Early Ischemic Stroke

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Introduction

Fractional anisotropy (FA) decreases in chronic ischemic stroke (>15 days after injury) [1-3]. However, within the acute period (< 24 h from stroke onset), changes in FA are less consistent. Studies have reported FA in grey matter (GM) to be unchanged [4,5], increased initially with a subsequent decrease [6], or decreased only [7]. In white matter (WM), FA has been observed to decrease [8], decrease after an initial delay [9], initially increase and then progressively decrease [10], or remain unchanged [11]. In order to further characterise the time course of FA changes after ischemic stroke, we analysed diffusion tensor imaging (DTI) data from patients who had serial DTI scans at < 26 h after onset, and during chronic stroke (> 22 days).

Methods

Thirteen patients (8F, 5M; age = 47–87 years) presenting with acute ischemic stroke were imaged on a 3 T MR scanner (Signa VH/i; General Electric Healthcare, Waukesha, USA) at < 26 h of onset of symptoms (acute), and > 21 days later (follow-up). Separate diffusion-weighted images (DWI) and DTI (TR/TE = 9000/75.3–101.2 ms; 11 directions, b = 1000 s/mm^2 (DTI only); and 144 × 144 (acute) or 192 × 192 (follow-up) acquisition matrices); FLAIR (TI, TR/TE/TI = 9002/140.7–146/2250 ms and acquisition matrix of 256 × 192); and inversion-recovery spin-echo planar imaging (IRSEPI) (TR/TE/TI = 15000/17.8–18.5/400 ms; and acquisition matrix of 144 × 144) were performed. All images were reconstructed to 256 × 256 with FOV of 24 cm^2. Contiguous oblique axial slices (5 mm) with whole brain coverage were acquired. We generated ADC, FA, and eigen value maps using FSL (FMRIB, http://www.fmrib.ox.ac.uk/fsl). The follow-up FLAIR was registered to the acute FLAIR and this transformation matrix was applied to the follow-up DTI as well as follow-up ADC, FA, and eigen value maps. Regions of interest (ROI) were manually drawn to outline the stroke on the follow-up FLAIR images. This whole stroke ROI along with a contralateral ROI in normal-appearing tissue were segmented into GM and WM using IRSEPI data. In three cases, pre-existing WM disease made necessary using the acute DWI as an additional guide for drawing the ROIs on the follow-up FLAIR scans. Mean ADC, mean FA, and mean eigen values were calculated for the lesion and contralateral control for both GM and WM. Relative changes (ipsilateral/contralateral) were calculated for ADC (rADC), FA (rFA), and the eigen values (rλ1, rλ2, rλ3) for each patient in GM and WM. Statistical differences for the change from acute to follow-up were tested using two-tailed paired t-tests ($p < 0.05$). Means ± standard deviations are reported.

Results

In both GM (n=12) and WM (n=13), mean rADC decreased acutely and then increased at follow-up. In acute stroke, mean rFA increased in GM, but did not significantly change in WM. At follow-up, the rFA for GM and WM decreased, though not significantly for WM. Acutely, the mean eigen values for GM decreased by differing amounts, but for WM, they decreased by comparable amounts. At follow-up, rλ1, rλ2, and rλ3 exhibited a more similar pattern in GM and WM.

Discussion

The rADC changes reconfirm that acute cytotoxic edema in ischemia restricts diffusion, but at follow-up, diffusion increases. These results are consistent with previous findings [12]. GM and WM exhibit similar absolute ADCs and rADCs at follow-up, possibly because microstructurally they become more similar (scarred). The eigen values show that, acutely, hindrance of water diffusion was asymmetric in GM, but symmetric in WM. By follow-up, though, there was a similar asymmetric hindrance of diffusion in both GM and WM.

Conclusion

Cellular swelling in acute stroke affects GM more than WM. However, in chronic stroke absolute ADC, rADC, and rFA in GM and WM converge with relative increases in diffusion perpendicular to the cellular axes. A possible mechanism could be that the myelination of WM cells offers additional resistance acutely to WM swelling compared to GM, but as the oligodendrocytes die, infarcted WM regions more closely resemble infarcted GM regions.

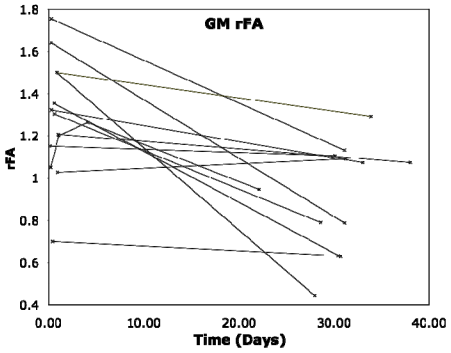

Figure 1. Evolution of rFA in GM (n=12) as a function of time from stroke onset.

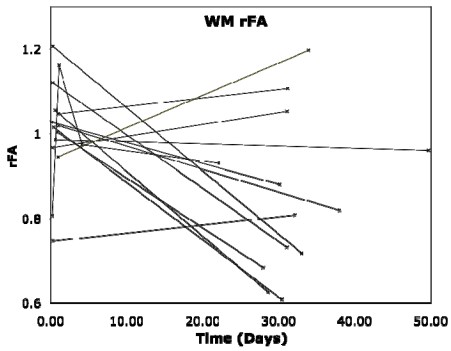

Figure 2. Evolution of rFA in WM (n=13) as a function of time from stroke onset.

Table 1: Changes at acute and follow-up times with p-value of change from acute to follow-up.

	Acute	Follow-up	p-value
GM rADC	0.68 ± 0.25	1.06 ± 0.37	< 0.001
WM rADC	0.86 ± 0.23	1.13 ± 0.32	< 0.001
GM ADC	(0.65 ± 0.17) ×10^{-3} mm^2/s	(1.03 ± 0.25) ×10^{-3} mm^2/s	< 0.001
WM ADC	(0.64 ± 0.13) ×10^{-3} mm^2/s	(0.96 ± 0.20) ×10^{-3} mm^2/s	< 0.001
GM rFA	1.29 ± 0.67	0.92 ± 0.37	0.004
WM rFA	1.00 ± 0.40	0.86 ± 0.32	0.06
GM rλ1	0.71 ± 0.26	0.99 ± 0.32	0.001
WM rλ1	0.86 ± 0.22	0.96 ± 0.26	< 0.001
GM rλ2	0.64 ± 0.26	1.04 ± 0.37	< 0.001
WM rλ2	0.85 ± 0.27	1.12 ± 0.37	0.004
GM rλ3	0.57 ± 0.27	1.16 ± 0.49	0.003
WM rλ3	0.89 ± 0.38	1.24 ± 0.56	0.007

References

1-Zelaya *et al*. *MRI*. 17(3):331– 348 (1999). 2-Yang *et al*. *Stroke*. 30:2382–2390 (2004). 3-Muñoz Maniega *et al*. *JNNP*. 75:1714–1718 (2004). 4-Sorensen *et al*. *Radiology*. 212:785–92 (1999). 5-Muñoz Maniega *et al*. *JNNP*. 75:1714–1718 (2004). 6-Baghat *et al*. *Proc ISMRM*. 16 (2008). 7-Zelaya *et al*. *MRI*. 17(3):331–348 (1999). 8-Muñoz Maniega *et al*. *JNNP*. 75:1714–1718 (2004). 9-Baghat *et al*. *Proc ISMRM*. 16 (2008). 10-Baghat *et al*. *JCBFM* 26:1442–1450 (2006). 11-Sorensen *et al*. *Radiology*. 212:785–92 (1999). 12-Lauzon *et al*. *Neuroimaging Clinics of North America*. 16:343–366 (2006).