

The power of the linear, planar, and tubular tensor in experimental stroke

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Introduction

Applications of the diffusion tensor imaging (DTI) are increasingly used in experimental and clinical studies to characterize the microstructural properties of tissue and cerebral connectivity and are found to be particularly useful in visualizing WM tracts in vivo (1, 2). Fractional anisotropy (FA) is one of the most widely used DTI indices. The contrast of the FA is based on differences between the three eigenvalues, which is similar to standard deviation calculations. As a result, FA can be sensitive to any types of differences of the eigenvalues. The degree to which the DT approximates the linear, planar, and tubular tensor are measured by the linear ($CL = (\lambda_1 - \lambda_2) / (\lambda_1 + \lambda_2 + \lambda_3)$), planar ($CP = 2(\lambda_2 - \lambda_3) / (\lambda_1 + \lambda_2 + \lambda_3)$), and spherical case ($CS = 3\lambda_3 / (\lambda_1 + \lambda_2 + \lambda_3)$) indices (3). Unlikely to more commonly used FA, these geometrics can be sensitive to the difference between eigenvalues.

We want to characterize the evolution of CL, CP, and CS following brain ischemia. We measure these indices intensively from the hyperacute to chronic phase. We follow the evolution of these indices in 3 different brain regions: cortex, subcortex, and corpus callosum. Following brain ischemia, no such systematic approach over an extended period of time, with CL, CP, and CS, has not yet been reported.

Material and methods

Wistar rats were subjected to focal cerebral ischemia by transient suture occlusion (n = 9). They were imaged in the hyperacute (2 and 3.5 hours), acute (1, 2, and 3 days), subacute (4 days, 1, and 2 week), and in the chronic phase (4, 6, and 8 weeks) after the MCAO. The MRI measurements were performed with a 4.7 T MR Scanner. The temporal evolution of the certain DT indices (CL, CP, and CS) was measured.

Result

In the hyperacute phase, CL and CS did not change but remained normal (Fig. 1B and D). In the acute phase CS became elevated and CL depressed, in all measured regions. In the subacute to chronic phase, CS remained elevated in all measured regions. In the subacute- and chronic phase, we found tissue-dependent behavior; CL of the cortex became depressed and remained significantly lower than the normal still at 8 weeks (Fig. 1B); while subcortex and corpus callosum with low CL, gradually increased until becoming normalized. CP remained relatively unchanged during the 8 weeks observation period (Fig. 1C). CS dominates through the 8 weeks observation period as it became significantly high compared to the normal in the acute phase, and remains high until the chronic phase, in all measured regions.

Conclusion

In the acute to subacute phase, significant reduction of CL was observed in all measured regions, while the time and the duration of the significant reduction varied between the 3 regions. This reduction of the CL perhaps aroused from the spatial distribution of barriers to diffusion. The constantly depressed CL in the cortex might refer to the severe damage in the neuronal cell bodies (4), while in the corpus callosum and subcortex, the normalization of the CL might indicate a partial recovery of the tissue (i.e., increased axonal number and/or density).

In the corpus callosum regions, where the tissue structure is originally more organized, the reduction of CL seemed to be more detectable than in the gray matter where the tissue is less organized. CP changed relatively little during the 8 weeks observation period.

References (1) Basser et al, J Magn Reson B. 1996;111:209-219, (2) Pierpaoli et al, J Magn Reson B. 1996;36:893-906, (3) Westin et al, Med Image Anal. 2002;6:93-108 (4) Carano et al, 2000;12:842-958

Fig 1. A: The volume of the lesion, expressed as the percentage of the right hemisphere (% hemispheric lesion volume, %HLV), $\%HLV = [(measured\ lesion\ volume - (left\ hemisphere\ volume - right\ hemisphere\ volume))/left\ hemisphere\ volume] \times 100$. **B, C, D:** The evolution of CL (B), CP (C), and CS (D) are presented over the 8 weeks observation period (2 hours – 8 weeks). Time points that are significantly different from the normal are marked in cortex (*), subcortex (#), and corpus callosum (**) ($p < 0.05$).

