

Combined DTI and Functional Connectivity Assessment of Cerebral Neoplasia

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Introduction

Diffusion tensor MRI (DTI) (1, 2) has been applied to a variety of CNS disorders including brain tumors (3-5). These studies demonstrated the high sensitivity of DTI in detecting damaged/compromised white matter tract integrity. An independent but related technique in development is functional connectivity mapping that exploits correlated low-frequency (< 0.1 Hz) spontaneous fluctuations of blood oxygenation level dependent (BOLD) MRI signals (6-8). Applications of functional connectivity in patients with brain lesions (9) showed reduced or altered inter-hemisphere (i.e., between lesion side and the contralateral normal side) functional connectivity. The purpose of this work reported herein was to combine DTI with functional connectivity to assess effects of cerebral neoplasms on structural (i.e. DTI) and functional connectivity, with one of the specific aims being to investigate the hypothesis that white matter fiber tracts contribute to cerebral structural connectivity that is underlying functional connectivity.

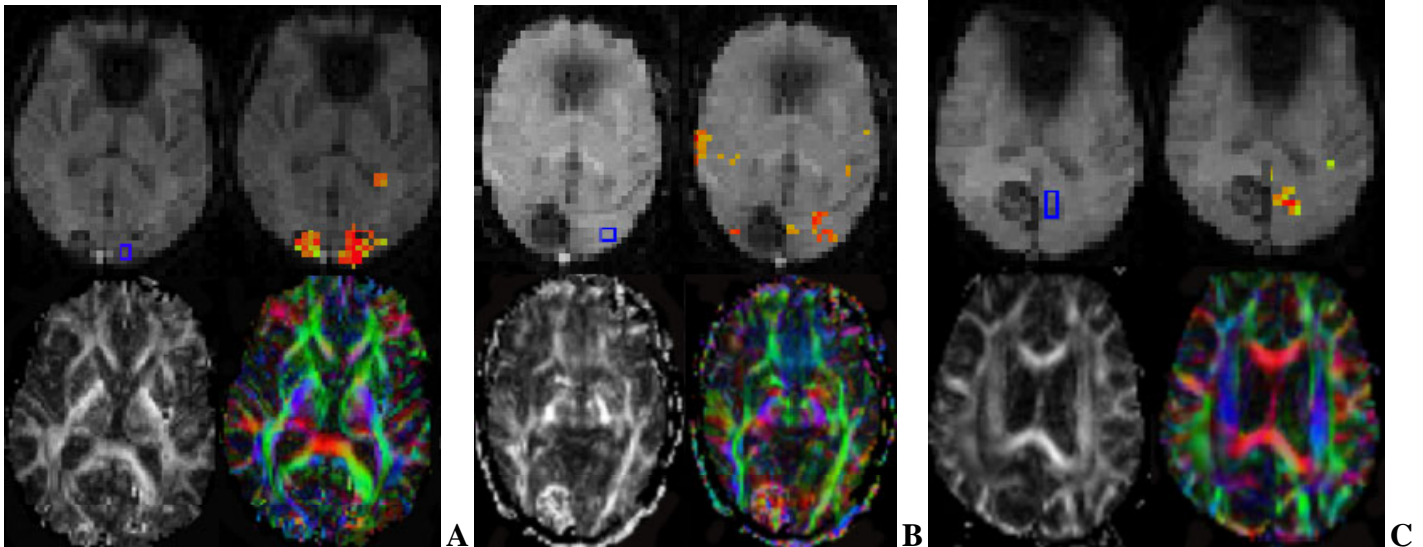
Materials and Methods

All MRI experiments were carried out on a standard 1.5T clinical MRI system using a circularly polarized head coil (Magnetom Vision, Siemens Medical Systems, Erlangen, Germany). Seven brain tumor patients (68.6 ± 8.5 years of age; 2 female, 5 male) were enrolled. DTI images were acquired with a single-shot pulsed gradient spin-echo EPI sequence (TR = 5s, TE = 100 ms, FOV = 256 mm, matrix = 128 x 128, slice thickness 4 mm, NEX = 12-16) that collected for each slice one T2WI image without diffusion gradient, and 6 diffusion-weighted images with a diffusion b-factor of 1025 s/mm². The DTI encoding used the icosahedron scheme optimal for 6 diffusion encoding directions (9), i.e., diffusion was measured along 6 non-collinear directions: (x,y,z) = [(u,1,0), (0,u,1), (1,0,u), (u,-1,0), (0,u,-1), (-1,0,u)], where u = (1 + √5)/2. DTI calculation involved the following steps: 1) image realignment using the Woods AIR algorithm; 2) correction of magnetic field inhomogeneity-induced geometric distortion of the EPI images; 3) calculation of the diffusion tensor using a weighted least square linear algorithm (10). (4) reconstruction of mean diffusivity maps (ADC), fractional anisotropy maps (FA), and FA-weighted diffusion tensor color map showing the main eigenvector direction. (5) White matter fiber tracking.

For functional connectivity, a BOLD sequence (single-shot gradient echo EPI, TR/TE/α = 0.38s/40ms/90°) was used to acquire a 3.5-minute (i.e., 550 time frames) resting state time series images, covering four 8-mm slices containing the brain tumors. The functional connectivity BOLD images and DTI images had the same slice orientation. A short TR = 0.38s was used in order to avoid cardiac signal aliasing. Using seed region-of-interest (ROI) in and around tumors as defined in DTI images, temporal correlation of the BOLD timecourse signals was conducted to find brain areas that had strong correlation (cc > 0.5) with the seed ROI.

Results and Conclusion

Three distinct patterns were observed: 1) when white matter fibers were not apparently affected by a lesion, functional connectivity was largely maintained. An example was given in Fig. A. 2) when a lesion caused displacement of white matter tracts without significantly affecting diffusion anisotropy, functional connectivity was found to be correspondingly distorted. An example was given in Fig. B. 3) when white matter tracts were disrupted, functional connectivity disappeared too. These results provided evidence that DTI can be used to examine structural connectivity underlying functional connectivity.



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References

- [1] Basser PJ, Jones DK. *NMR Biomed.* 2002;15:456-67. [2] Le Bihan D, et al., *J Magn Reson Imaging.* 2001;13:534-46. [3] Wieshmann UC, et al., *J Neurol Neurosurg Psychiatry.* 2000; 68:501-3. [4] Mori S, et al., *Ann Neurol.* 2002; 51(3):377-80. [5]. Field AS, et al., *ISMRM Workshop on Diffusion MRI,* March 2002, p. 137. [6] Biswal B et al., *MRM* 34: 537-41 (1995). [7]. Lowe MJ et al. *Neuroimage* 7: 119-132 (1998). [8]. Hampson M, et al., *Hum Brain Mapp.* 2002;15:247-62. [9]. Quigley M, et al., *AJNR Am J Neuroradiol.* 2001 22: 294-300. [10] Maldjian JA. *AJNR Am J Neuroradiol.* 2001; 22:239-40. [11] Hasan, KM et al., *JMRI* 13:769-80, 2001. [4] Mori S et al., *Annals of Neurology* 45:265-69, 1999.