

Diffusion Tensor MRI after Pediatric Brain Injury

K. M. Hasan¹, B. P. Kanabar², R. M. Santos³, M. Prasad⁴, L. A. Kramer⁵, L. Ewing-Cobbs⁴, P. A. Narayana⁵

¹Radiology, University of Texas, Houston, TX, United States, ²Biomedical Engineering, UH, Houston, TX, United States, ³UT, Houston, TX, United States, ⁴Pediatrics, UT, Houston, TX, United States, ⁵Radiology, UT, Houston, TX, United States

Introduction

Traumatic brain injury (TBI) is a major cause of disability and morbidity amongst school-age children. Diffuse axonal injury (DAI) caused by shear-strain deformation disrupting the cytoskeletal network and axonal membranes is the primary pathophysiological consequence of TBI [1]. DAI most prominently affects the subcortical white matter, corpus callosum, and dorsolateral aspect of the upper brainstem [2]. Following TBI, corpus callosum (CC) volumes are strongly related to indices of diffuse axonal injury as well as to cognitive measures. Atrophy is greatest in the posterior body and splenium in both children and adults [2]. Diffusion tensor imaging (DTI) has the potential to help in the localization and quantification of the extent of the damage and the correlation with outcome measures. In this preliminary report, we have used full brain, optimally designed DT-MRI protocol in combination with conventional anatomical T1w and dual fast spin echo (PD, T2w) MRI to assess the extent of injury in different white matter structures. This report focuses on the effect of TBI on the fractional anisotropy (FA) obtained from different subregions of CC.

Methods

Subjects: The four TBI cases that have been investigated in these studies had a mean age at injury of 8.1 years; the mean age at DT-MRI was 12.2 years. The seven normal controls, NDC1-NDC7, (all males, average age at scan \pm SD: 13 \pm 2, range 9-15 yrs), included one fraternal (NDC6) and identical twins (NDC7) to TBI3 and TBI4 respectively.

Conventional and DT MRI Acquisition: We have acquired entire brain data using a GE 1.5 T CNV4 system and a quadrature RF receive and transmit head coil. The MRI protocol included conventional T1w, PD, T2w dual spin echo images (TE1/TE2/TR=10/85/7000 ms) in addition to matching prescription of diffusion tensor MRI data. The DT-MRI data were acquired using a single shot dual spin echo diffusion sensitized EPI sequence with the balanced Icosa21 encoding scheme [3], b=1000 mm^2 , TR=7, TE= 84 ms. The slice thickness is 3mm with 42 slices, fov=24 cm, a matrix of 256x256 and NEX=4.

Data Processing and Analysis: Diffusion weighted data were distortion corrected and processed as described elsewhere [3]. To increase the accuracy and specificity of the ROI methodology, we have also implemented and adopted a DT-MRI guided subregional corpus callosum division of Witelson [4]. To minimize ROI contamination, all regions were selected by an experienced neurosurgeon. To assess tissue integrity, the FA value of a contiguous 9 pixel region was calculated within each of the 7 CC subdivisions (CC1-rostrum, CC2-genu, CC3-anterior midbody, CC4-middle body, CC5-posterior-body, CC6-isthmus and CC7 - splenium) (Figure 1).

Results

Figure 2 compares the DT-MRI and conventional MRI from one TBI child (TBI4) along with his identical twin brother (NDC7). Notice the loss of fibers in the splenium of the CC and the loss of contrast between WM and GM on the dual spin echo images. Figure 3 shows a summary of the pooled FA means of the TBI and control groups and the corresponding P obtained using the two-tailed t-test for unequal samples. In the controls, the FA values were smallest in the genu and largest in the splenium, which is consistent with previous studies [5]. FA values reflecting the integrity of white matter were related to callosal sub-regions. FA values were particularly sensitive to tissue integrity in posterior regions, including the isthmus and splenium, and in anterior regions, including the rostrum, genu, and rostral body. The FA values from CC subregions were more strongly related to the presence of TBI, this is consistent with a report on adult TBI [6].

Discussion and Conclusions

Assessment of FA in specific brain regions will allow stronger and more precise determination of brain injury to different regions. This information coupled with a neuropsychological behavioral, social and cognitive battery will be essential in the design of rehabilitation, surgical and therapeutic plans. FA values obtained from CC subregions were very sensitive to the effects of TBI. These findings are consistent with volumetric studies showing marked post-traumatic atrophy in the genu, the rostral and anterior body, and the splenium [7]. DT-MRI shows great promise for serving as an index of injury liability that impact specific brain regions. In this preliminary and on going study we have demonstrated the utility of full brain DT-MRI to assess the vulnerability of the CC (genu and splenium) to pediatric TBI.

Figure 1. Illustration of the DT-MRI guided implementation of the Witelson corpus callosum subdivisions. The map is an overlay of the FA modulated principal vector on the apparent mean diffusivity map. The directional DT-MRI color map is shown for clarity.

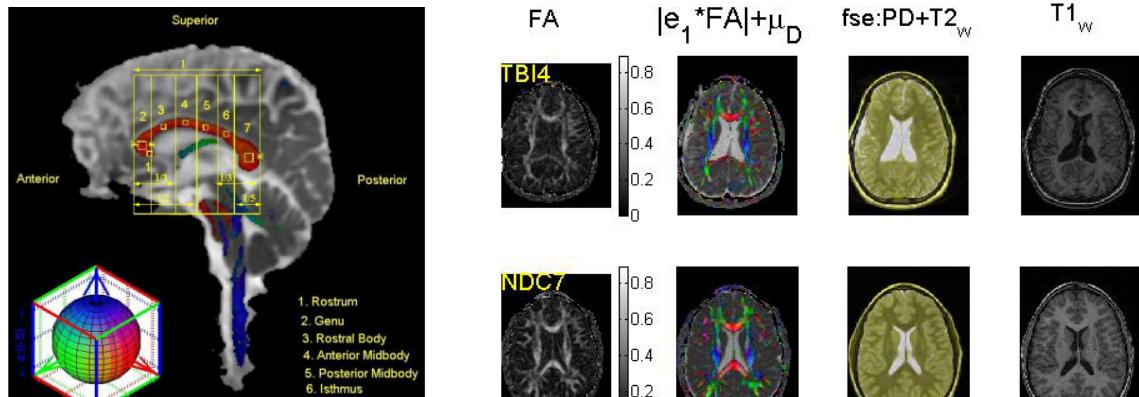


Figure 3. A bar plot of the pooled (mean \pm SD) FA between the 4 TBI and 7 controls; the P value is based on the t-test significance ≤ 0.05 . The structures included are the 7 subdivisions of the corpus callosum according to Witelson [4] (Figure 1): CC1-CC7 (CC1-rostrum, CC2-genu, CC3-anterior midbody, CC4-middle body, CC5-posterior-body, CC6-isthmus and CC7 - splenium).

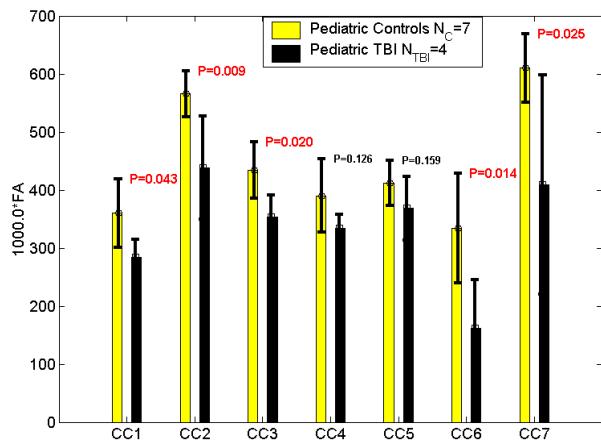


Figure 2. Comparison of the conventional and DT-MRI results on the identical twin brothers, TBI4 and NDC7. Notice the loss in splenium fibers and ventricular enlargement of TBI4 (upper) compared to his identical control brother, NDC7.

References

- [1] Hammoud DA, Wasserman BA. Neuroimag Clin N Am. 2002;12(2):205-16.
- [2] Poussaint TY, Moeller KK. Neuroimag Clin N Am. 2002;12(2):271-94, ix.
- [3] Hasan KM, Narayana PA. MRM 2003; 50:589-598.
- [4] Witelson SF. Brain 112:799-835, 1989.
- [5] Chepuri NB et al. AJNR 2002; 23:803-808.
- [6] Arfanakis et al. AJNR Am J Neuroradiol. 2002;23(5):794-802.
- [7] Levin et al. Neurology 54:647-653, 2000.