Correlation of Changes in Venous Cerebral Blood Flow after Trauma in Rats Measured by Susceptibility-weighted Imaging and Fractional Anisotropy in Diffusion Tensor Imaging

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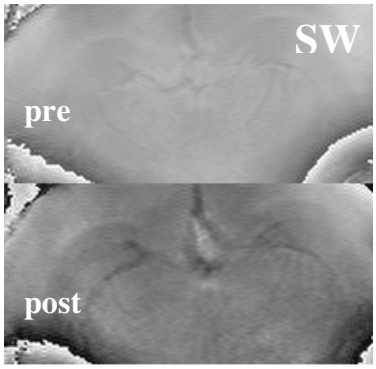
Introduction: Traumatic brain injury (TBI) is a leading cause of disability and death in the United States. Diffuse axonal injury (DAI) is a significant pathology following TBI and manifests a constellation of pathologies. However, DAI is very difficult to identify and quantify using conventional neuroimaging techniques, including MRI. As a relative new technique, susceptibility-weighted imaging (SWI) offers information about the intact structure of the venous system and the blood oxygen saturation, and it has been reported to measure venous cerebral blood flow (CBF) after trauma in animal models [1]. Furthermore, Diffusion Tensor Imaging (DTI), specifically fractional anisotropy (FA), has been reported to be sensitive to detect DAI [2-3]. The objective of this study was to utilize both SWI and DTI to investigate different aspects of DAI and their relationships in a comprehensive manner in animal models.

Materials and Methods: TBI was induced in six male Sprague Dawley rats utilizing the Marmarou impact acceleration model [4, 5]. All of the MRI measurements were performed on a 4.7-T Bruker AVANCE scanner. SWI is based on a fully flow compensated, high resolution, 3D gradient echo method. SWI sequence was used *in vivo* pre and 4 h post TBI with TR=83ms, TE=35ms, FA=30^o, FOV=40x40x18 mm^3, matrix size=1024x 512x18, interpolated to 1024x1024x24, Nacq=2, and total imaging time 34m. The venous CBF was measured indirectly using the SWI phase filtered images using our internally developed SPIN software. The 3D spin echo DTI sequence was used *ex vivo* for harvested brains 6-7 h post injury with 6 gradient directions, TR= 850 ms, TE=24 ms, b=0 and 1200 sec/mm^2, diffusion gradient duration/separation=6.6/12 ms, FOV= 26x26x20 mm^3, matrix size=128x80x32, interpolated to 128x128x32, Nacq=1, and total imaging time 4h 14m. DTI-studio (Johns Hopkins University, Baltimore, MD) and the SPIN software were utilized for the analysis of DTI data and to generate FA values. FA values for each rat were averaged over regions of the optic chiasm (och), anterior commissure (ac), corpus callosum (cc), left/right fimbria of the hippocampus (fi), internal capsule (ic), external capsule (ec) and forceps major of corpus callosum (fmj).

Results: An example of SWI pre/post TBI is shown in figure 1. Big changes were found in phase values in the internal cerebral veins. The venous CBF reduced by 50±8%, on average, for 6 TBI rats at 4h post TBI. An example FA map is shown in figure 2. Figure 3 shows the plots of FA for various brain white matter regions vs. changes in venous CBF averaged for each rat. FAs of ac, och, r-fi, l-fi, cc, l-ic and r-ic were positively correlated with changes in venous CBF for six rats. The correlation coefficients were ρ=0.83, 0.77, 0.72, 0.55, 0.54, 0.54 and 0.41. No correlation was found in the left/right ec and fmj regions with CBF. The correlation coefficients for these regions were ρ=0.02, 0.03, 0.21 and -0.19, respectively. The histology data also confirmed, by using beta amyloid antibody precursor protein (βAPP) immunohistochemistry, that the och region consistently has the largest number of axonal retraction balls (RB), and followed by the cc.



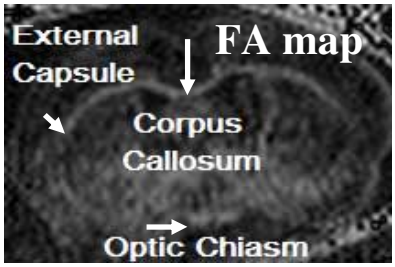
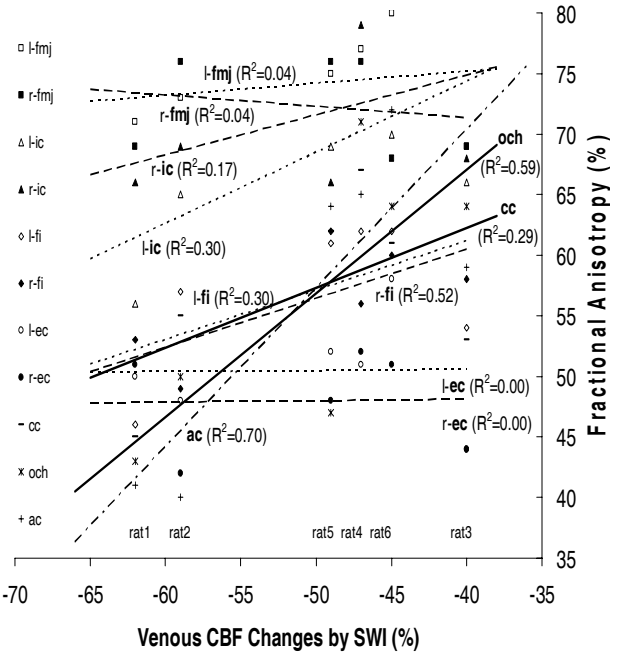


Fig. 1 SWI pre and post TBI (left).
Fig. 2 FA map of rat brain (middle).
Fig. 3 Correlation of FA and venous CBF in TBI rats (right).



Conclusions: Our MRI results demonstrate that there are decreases of global venous CBF and a drop of FA in major white matter tracts in this DAI rat model. Both the changes in CBF and FA are correlated with each other and could serve as an injury severity index. This is the first report on direct correlation between FA and CBF changes in TBI animal model. It also confirmed that a multimodal MR approach is a MUST to give a comprehensive view of traumatic brain injury.

References: 1.Y. Shen, Z. Kou, C.W. Kreipke, T. Petrov, J. Hu, E. M. Haacke, Magn. Reson. Imaging, 2006, to appear. 2. M. Inglese et al., J.Neurosurgery 103:298-303. 2005. 3. K. Arfanakis et al., American Journal of Neuroradiology, 23:794-802, 2002. 4. A. Marmarou et. al, J. Neurosurgery 80:291-300, 1994. 5. F. Abd-Elfattah et. al, J. Neurosurgery 80:301-313, 1994.