Diffusion Tensor Imaging of Post-traumatic Epilepsy

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Introduction: Posttraumatic epilepsy (PTE) is a consequence of head injury. The risk of developing PTE is related to the severity and type of injury. Factors that are known to contribute to the risk of PTE are duration of unconsciousness, dura penetration, degree of direct cortical damage and genetic predisposition to epilepsy. The investigation and management of patients after head injury must include the accurate and complete identification of cerebral damage. It has been shown that extent of the injury as defined by magnetization transfer MRI correlates with intractability in PTE[1]. DTI is a noninvasive modality that provides information about microstructural damage in brain parenchyma, which may not be seen on conventional MR imaging. Aim of this study was to look for any difference in the microstructural changes in normal appearing tissue surrounding conventional imaging visible abnormality using DTI in TBI patients with or without epilepsy.

Materials and Methods:

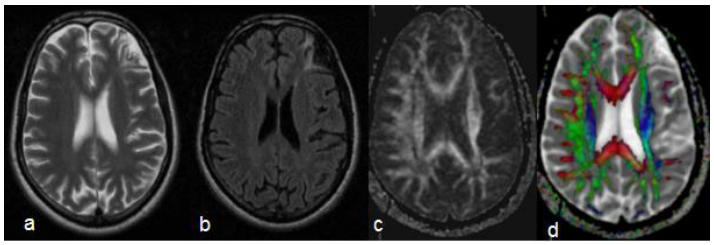
Subjects: Twenty patients with TBI (fourteen with epilepsy and six without epilepsy) and 11 healthy controls were studied. All patients with TBI had detailed clinical and electrophysiological workup before subjecting them to imaging.

MRI Protocol: Conventional MRI and DTI of brain were performed on a 1.5 Tesla GE MRI scanner using a standard quadrature head coil. DTI data was acquired using a single-shot echo planar dual spin echo sequence with ramp sampling. The acquisition parameters were: TR=8sec/TE=100ms/slice no=34/slice thickness=3mm/interslice gap=0/FOV=240mm image matrix=256x256 (following zero-filling)/NEX=8/ diffusion weighting b-factor=1000 s-mm^{-2}. A balanced rotationally invariant dodecahedral diffusion-encoding scheme was used for generating the DTI data[2]. Tensor field for each voxel was obtained by interpolating and decoding of distortion corrected data. The tensor field data were then diagonalized to obtain the eigenvalues (λ_1, λ_2 and λ_3) and the three-orthonormal eigenvectors (e1, e2 and e3). The tensor field data and eigenvalues were used to compute the mean diffusivity (MD) and fractional anisotropy (FA) for each voxel. Data processing and analysis were performed using an in-house developed DTI-Toolbox implemented under IDL. For quantitative analysis, the DTI-derived maps were displayed and overlaid on images with different contrasts to facilitate the region-of-interest (ROI) placement in the three orthogonal planes for visual inspection. Free hand ROIs were placed on region beyond T2 abnormality as confirmed by T2 and FLAIR imaging and normal appearing contralateral corresponding region for FA quantification in these patients. Then FA ratio in the region beyond T2 abnormality to contralateral corresponding region was calculated in the case of TBI patients. In controls, ROIs were placed in both frontal and occipital periventricular white matter to quantify FA ratio. The FA ratio in controls was compared to TBI patients with and without epilepsy using Student's t-test.

Results: The regional mean FA ratio and region mean MD ratios in all the groups are summarized in table. The FA ratio in the region beyond T2 abnormality was significantly lower in TBI compared to controls ($p<0.05$). The regional FA ratio was significantly lower in TBI patients with epilepsy as compared to TBI patients without epilepsy ($p<0.05$). The MD ratio was significantly lower in TBI compared to controls. Though the regional MD was lower in TBI with epilepsy compared to TBI without epilepsy, it did not reach the level of statistical significance ($p=0.06$). The Figure below shows a small area of gliosis in the left frontal region (place an arrow in power point) on T2 and FLAIR images (a, b). Note the large area of low FA in the left frontal region with extension on FA map (c) and color-coded FA fused with mean diffusivity image (d) in a patient with PTE.

Table: Summary of results

Subjects	Regional FA Ratio	Regional MD ratio	p for FA ratio	p for MD ratio
a. Controls	0.902 ± 0.067	0.952±0.027	a vs b <0.05 a vs c <0.05	a vs b <0.05 a vs c <0.05
b. TBI with out epilepsy	0.696± 0.039	1.085± 0.135	b vs c <0.05	b vs c =0.05
c. TBI with epilepsy	0.565± 0.059	1.154± 0.14		



Discussion: We observed lower FA ratio in the vicinity of the T2 abnormal region consistent with larger microstructural damage in patients with TBI. This observed low FA was found to be significantly lower in patients with seizure compared to no seizure group suggesting increased gliosis in PTE compared to non-epileptics. It has been observed in the earlier studies that the extent of axonopathy correlates with epileptogenesis in patients with PTE. The increased MD ratio in TBI patients compared to controls further confirms the larger area of gliosis in TBI patients than what was observed on conventional MRI. We conclude that DTI helps in demonstrating more extensive gliosis than what is seen on conventional imaging and may help in predicting the epileptogenesis in TBI on the basis of reduced regional FA ratio.

References:

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