Diffuse Axonal Injury in Corpus Callosum and Internal Capsule in Moderate to Severe Traumatic Brain Injury

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Introduction:

Traumatic brain injury (TBI) results in varying severity and regional distribution of primary and secondary lesions. Impairment in cognitive and multi-task execution sequelae have been attributed to diffuse brain/axonal injury or neural network disruption in the brain. Neuropathological and imaging studies have emphasized damage to white matter (1, 2). Diffusion tensor imaging (DTI) is a promising tool for noninvasive detection of the degree of white matter injury and predicting clinical outcome. Previous DTI studies have indicated reduced FA in white matter tracts in acute and chronic phases of injury in adults despite normal appearing white matter on MRI (3, 4). The purpose of this study is to diagnose and assess the distribution and severity of diffuse axonal injury (DAI) in corpus callosum (CC) and internal capsule (IC) of corticospinal tract (CST) following moderate to severe TBI.

Material and Methods:

Our study included 40 patients (13 females and 27 males, median age=30 years, range=18-61years) who sustained moderate to severe TBI with demonstrable computerized tomography (CT) findings at the time of injury. Patients underwent magnetic resonance imaging (MRI) within a mean interval of 12.6 days. (range, 2 to 30 days) after the traumatic incident. The control group comprised of 10, age and sex matched healthy volunteers (4 females and 6 males, median age=28 years) also underwent the same MRI procedure. Imaging was performed on a 1.5 Tesla MRI scanner (General Electric Medical System, Milwaukee, WI) using a standard quadrature birdcage head coil. In addition to conventional sequences (T2 weighted fast spin echo, T1 weighted spin echo, T2 weighted magnetization prepared gradient echo, T2 weighted fluid attenuated inversion recovery(FLAIR), T2 gradient recalled echo sequence) DTI was performed using single shot echo planar dual spin echo system with ramp sampling. The acquisition parameters were TR=8sec/TE=100ms/number of slices=36/slice thickness=3mm/inter-slice gap=0/ square FOV=240mm/image matrix =256 × 256 (following zero-filling) NEX=8/diffusion weighting b-factor=1000 s/mm². The DTI data were processed as described in detail elsewhere (5). The DTI derived maps were displayed and overlaid on images with different contrasts to facilitate the region of interest (ROI) placement. The ROIs were placed bilaterally in the white matter (WM) of posterior and anterior limbs of internal capsule (PLIC, ALIC) and genu, mid-body and splenium of CC for FA and MD quantification. (Fig. 1 & 2). The patients were further sub-grouped as frontal, parietal, fontotemporal, parieto temporal depending on the site of traumatic brain lesion on conventional MRI. Student's independent t-test was used to compare FA and MD values in patients group and controls. Statistical analysis was performed using SPSS (version 12.0, SPSS Inc.Chicago, USA). P value less than 0.05 was considered as statistical significant.

Result:

Hemorrhagic/ non-hemorrhagic contusions were present in frontal (n=16), parietal (n=5), frontotemporal (n=13), parietotemporal (n=6) regions of moderate to severe TBI with normal appearing white matter on MRI in early post-traumatic phase. The Glasgow coma scale (GCS) for all patients ranged from 3 to 11 at early post-traumatic injury time. The mean FA and MD values for the WM regions of IC and CC are summarized in Table 1.

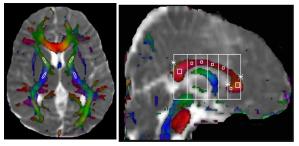
Table 1. A summary of group mean and standard deviation of FA and MD in WM regions of CC and IC in 40 TBI patients and 10 age sex matched controls.

	GENU		BODY		SPLINIUM		ALIC		PLIC	
	FA±SD	MD±SD (x10 ⁻³ mm ² /sec)								
Control (a)	0.50 ± 0.05	0.85 ± 0.04	0.48 ± 0.07	$090 \pm .08$	0.67 ± 0.05	0.77 ± 0.10	$0.41{\pm}0.04$	0.77 ± 0.04	0.55 ± 0.07	0.69 ± 0.04
Frontal (b)	0.39 ± 0.05	0.84 ± 0.07	0.42 ± 0.05	0.85 ± 0.07	0.55 ± 0.08	0.76 ± 0.08	0.35±0.04	0.74 ± 0.03	0.49 ± 0.03	0.66±.0.03
Parietal (c)	0.44 ± 0.02	0.83 ± 0.08	0.41 ± 0.05	0.82 ± 0.04	0.40 ± 0.05	0.75 ± 0.10	0.36 ± 0.06	0.74 ± 0.05	0.46 ± 0.05	0.67±.0.05
Frontotemporal(d)	0.42 ± 0.05	0.81 ± 0.06	$0.37{\pm}0.08$	0.83±0.09	0.42 ± 0.05	0.74 ± 0.06	0.37±0.04	0.74 ± 0.03	0.48 ± 0.03	0.65 ± 0.03
Parietotemporal(e)	0.42 ± 0.02	0.81 ± 0.07	0.42 ± 0.05	0.83 ± 0.08	0.58 ± 0.05	0.76 ± 0.05	0.37±0.05	0.76 ± 0.04	$0.49{\pm}0.05$	$0.66 \pm .0.03$
P values	pab= 0.00	pab= 050	pab= 0.01	pab= 0.10	pab= 0.00	pab= 0.84	pab=0.00	pab=0.02	pab=0.03	pab=0.03
	pac= 0.02	pac = 0.44	pac= 0.09	pac= 0.05	pac= 0.00	pac= 0.80	pac= 0.10	pac=0.24	pac=0.02	pac=0.33
	pad= 0.00	pad= 0.07	pad= 0.00	pad= 0.08		pad= 0.37	pad= 0.05	pad=0.02	pad=0.02	pad=0.02
	pae= 0.00	pae= 0.13	Pae= 0.13	pae= 0.11	pae= 0.00	pae= 0.93	pae= 0.10	pae=0.47	pae=0.09	pae=0.11

Compared with controls the average FA and MD values in the anterior and posterior limb of IC were significantly reduced in patients with frontal and frontotemporal lesions, while FA and MD values were not significantly reduced in patient group with parietal and parietotemporal lesions with an exemption in the PLIC of parietal group. Significantly decreased FA values with no change in MD values were observed in genu, mid-body, and splenium of CC in all patient groups relative to controls except in body of cc in parietal and pareitotemporal group. Highest reduction in FA was found in genu and splenium in patients with frontotemporal lobe lesions followed by frontal, parietal and parietotemporal lobe lesions.

Discussion:

DTI assesses microstructural integrity within the cerebral WM. We found a significant mean FA reduction in the CC of all patients irrespective of the region that is involved in trauma, emphasizing the unique vulnerability of CC to shear- strain forces in moderate to severe TBI. This is consistent with the published literature. It also suggests that genu and splenium are most affected in moderate to severe traumatic brain injury. The anterior and posterior limbs of IC are susceptible to DAI in patients with frontal and frontotemporal brain lesions. The results of this study show significant decrease in FA and MD that may suggest structural disorganization of axonal membrane leading to impairment of axoplasmic transport with subsequent swelling of axon. We conclude that DTI elucidates the extent and severity of early DAI of CC and IC in normal appearing white matter of moderate to severe TBI.



Figs. 1 & 2 ROIs placement on colour coded FA map fused with MD map.

References:

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