Diffusion Tensor Imaging of Cryptogenic and Acquired Partial Epilepsies

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Introduction

Diffusion tensor imaging (DTI) is a relatively novel imaging method of non-invasively evaluating the molecular movement of water in the brain (1). Diffusivity is a measurement of the magnitude of the diffusion. Pathological processes that change the microstructural environment, such as neuronal swelling or shrinkage, increased extracellular space and loss of tissue organisation, result in increased diffusivity. The aim of this study was to test the hypothesis that DTI would identify areas of increased diffusivity in patients with epilepsy and normal conventional MRI (MRI-negative patients).

Methods

We used DTI and Statistical Parametric Mapping (SPM) to objectively compare the diffusion properties, and hence the structural organisation of brains of patients with partial seizures and either chronic non-progressive acquired lesions or normal conventional MRI. Thirty healthy volunteers (20 women, median age 30 years, range 20-50 years) with no history of neurological disease and 40 patients with partial seizures (10 patients with acquired lesions (9 men, median age 36 years, range 20-53 years), and 30 MRI-negative patients (17 men, median age 36 years, range 18-55 years)) were scanned with conventional MRI (T1-weighted IRp-SPGR volumetric acquisition, contiguous 3mm oblique coronal T2-weighted, proton density and fast FLAIR) and DTI (1.5T Horizon Echospeed scanner (GE, Milwaukee, USA), single-shot CSF-suppressed diffusion weighted EPI (TR/TE/TI=5000/78/1788 ms), FOV 24cm, acquisition matrix 96x96, reconstruction matrix 128x128, 5mm slices) (2). Pulsed unipolar diffusion gradients were used for diffusion sensitisation (delta=28ms, DELTA=35ms). Two b-volumes were applied in seven non-collinear directions at 13 slice positions (b=703s/mm²). Two interleaved series with nine averages were acquired resulting in 1872 images. Images were transferred to an off-line workstation (Sun Microsystems, Palo Alto, CA). The images with no diffusion weighting were registered to a control subject in Talairach space using SPM (3) and spatially normalised maps of mean diffusivity (MD) were generated. The patients were individually compared to the 30 control subjects on a voxel-by-voxel basis and or MRI-negative patient groups. In all 10 patients with acquired lesions, SPM detected areas of increased MD corresponding to at least part of the abnormalities identified on visual inspection of the conventional MR images. In addition three patients had significant increases of MD in areas which appeared normal on conventional MRI. Eight of the 30 MRI-negative patients demonstrated significant increases in MD. In one patient the area of increased MD, in the right frontal lobe, concurred with both seizure semiology and ictal EEG recordings (see Figure 1). Group analysis was also performed on MRI-negative patients with EEG evidence of left temporal lobe seizures (9 patients). Compared to the 30 control subjects this group had a significant increase in MD within the white matter of the left temporal lobe.

Results

Two control subjects had significant increases in MD. At a statistical threshold of p<0.05 and 60 examinations (30 subjects with 2 contrasts each), up to 3 abnormal areas may have been anticipated by chance alone. No reductions in MD were seen within the control, acquired or MRI-negative patient groups. In all 10 patients with acquired lesions, SPM detected areas of increased MD corresponding to at least part of the abnormalities.

Discussion

DTI analysed using SPM was sensitive in patients with acquired cerebral damage, in some cases detecting abnormalities in cerebral tissue previously thought to be normal. This finding would be of importance should surgical management of similar patients be considered. Despite its sensitivity in patients with acquired lesions, the technique did not identify a clinically concordant abnormality in the majority of MRI-negative patients. The significant increase in MD in the left temporal lobe group however suggests minor structural disorganisation in the white matter associated with temporal lobe foci exists. This could be caused by either the aetiological factors, for example, occult dysgenesis, or as a result of chronic seizures, for example, atrophy, gliosis and expansion of the extracellular space.

References