Parahippocampal and thalamic diffusion abnormalities correlate with disease duration in temporal lobe epilepsy with unknown cause

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Introduction

Quantitative analyses of fractional anisotropy (FA) using magnetic resonance (MR) diffusion tensor imaging (DTI) are extremely sensitive to subtle pathological alterations in brain integrity. Patients with chronic temporal lobe epilepsy with unknown cause (TLEu), in whom no neuroanatomical abnormality can be identified using conventional MR methods, are an ideal sample to investigate subtle brain pathology that may reflect epileptogenic regions using quantitative FA methods and the effects of duration of chronic epilepsy on FA abnormalities.

Methods

Ten patients with TLEu (6 females, mean age 40.2 ± 14.2 SD; 4 men, mean age 42.3 years ± 9.7 years SD; 9 left TLEu, 1 right TLEu) and 81 healthy age-matched controls (42 women, mean age 35.5 ± 13.7 SD; 39 men, mean age 34.0 years ± 12.2 years SD) were recruited into this study. Interictal EEG, long-term video EEG monitoring, and conventional MRI (T1-weighted, T2-weighted and FLAIR) were performed for all patients. All conventional MRI techniques revealed no focal brain abnormality. For DTI we used echo planar imaging with 20 diffusion directions (two b-factors, 0 s/mm2 and 1000 s/mm2, TR=9.8 s / TE=95 ms, acquisition matrix: 128 x 128, voxel size: 1.8 x 1.8 x 3.6 mm3 (reconstructed to 0.89 x 0.89 x 3.6 mm3), 2 averages, scanning time 7:46 min). All DTI data were spatially processed using our recently developed toolbox incorporating optimal eddy current correction and multi-contrast image registration. Region-of-interest (ROI) masks were generated for the left and right hippocampus, parahippocampal gyrus, fimbria-fornix complex, thalamus, and entire white matter. Mean FA within the ROIs were statistically compared between patients and controls, and correlated with clinical data in patients.

Results

FA within all ROIs were significantly and substantially reduced in patients relative to controls, both ipsilateral and contralateral to the presumed epileptogenic zone (% FA change (min/max) -9.24% (right hippocampus, p=0.04) / 17.27% (left fimbria-fornix complex, p<0.00001). Duration of TLEu significantly negatively correlated with FA in the left and right parahippocampal gyri (r=-0.74, p=0.01 and r=-0.67, p=0.03, respectively) and with the left and right thalamus (r=-0.67, p=0.03 and r=-0.73, p=0.02, respectively), indicating that the longer the duration of chronic epilepsy, the greater the reduction of FA within these regions (Figure). No relationship existed between TLEu duration and the entire white matter. Age of onset of TLEu did not significantly correlate with FA values.

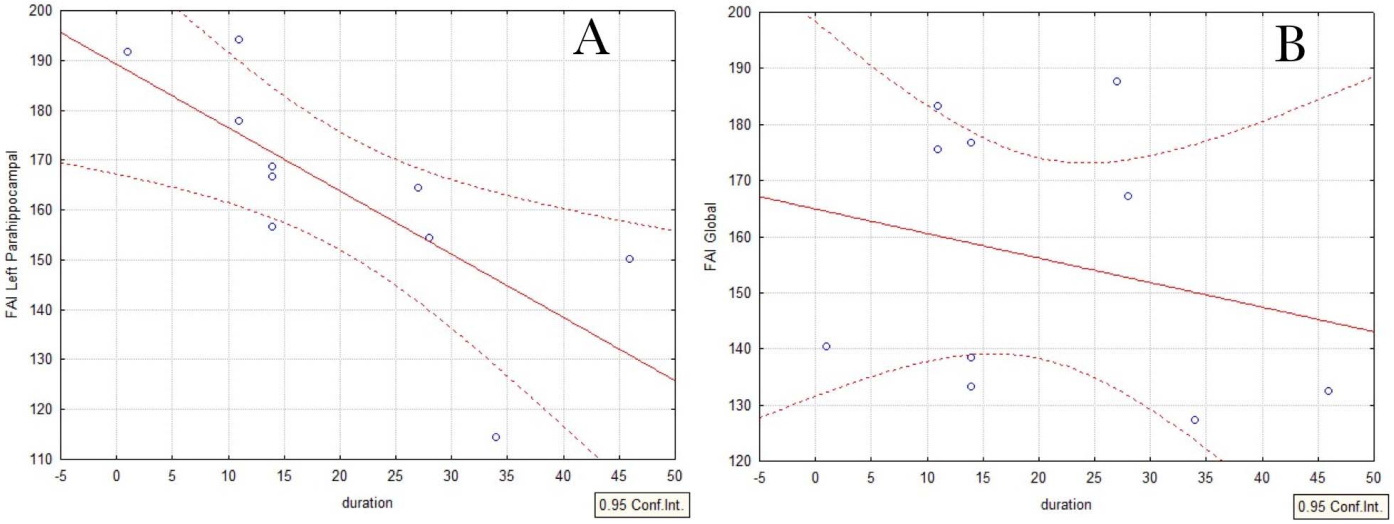

Figure. Relationship between duration of TLE and left parahippocampal gyrus FA (A) and entire white matter (B).

Discussion

Patients with TLEu show evidence of significant bilateral temporal lobe and thalamic diffusion abnormalities, with slightly greater involvement of structures ipsilateral to the side of seizure onset. The results presented here suggest the selective susceptibility of the parahippocampal gyri and thalami in response to the duration of chronic TLEu.