Diffusivity alterations in Temporal Lobe Epilepsy

P. B. Diniz[1,2], C. E. Garrido Salmon[2,3], T. Velasco[1,2], A. C. Sakamoto[1,2], J. P. Leite[1,2], and A. C. Santos[2,4]
[1]Neuroscience and Behavior, FMRP, University of São Paulo, Ribeirão Preto, SP, Brazil, [2]CInAPCe (Cooperação Interinstitucional de Apoio a Pesquisas sobre o Cérebro), Ribeirão Preto, SP, Brazil, [3]Physics and Mathematics, FFCLRP, University of São Paulo, Ribeirão Preto, SP, Brazil, [4]Internal Medicine, FMRP, University of São Paulo, Ribeirão Preto, SP, Brazil

Introduction: Temporal lobe epilepsy (TLE) is associated with hippocampal atrophy. However, the brain damage is not limited to limbic structures. The mechanisms underlying extra-hippocampal brain damage in TLE are unknown. Most MRI quantitative studies in TLE have used volumetric techniques to evaluate changes in the brain, but volumetric changes are not specific, because beyond axonal and cellular loss, the atrophy can be due to a decrease in the intracellular space or demyelination [1]. The increase of perpendicular diffusivity may indicate demyelination and its decrease would suggest axonal lesion [2]. In this study, we combined the use of fractional anisotropy (**FA**), mean diffusivity (**Dave**), parallel diffusivity (**Dpar**), and perpendicular diffusivity (**Dper**) to localize the regions where occur axonal lesion and demyelination in patients with TLE.

Method: High resolution T1-weighted anatomical images (3DT1) and Diffusion tensor imaging (DTI) were performed in 33 patients with unilateral TLE (15 left and 18 right) and 20 healthy controls. The DTI data were acquired in a 3T MR scanner (Achieva, Philips) using a single-shot echo planar imaging (TE = 82 ms), applying diffusion-sensitizing gradients in 32 directions (b = 1000 s/mm^2) and recording one reference image. The toolbox Tract-based spatial statistics (TBSS) [3] was used to a voxel-based analysis of the **FA** data comparing patients and controls. The regions with **FA** alteration were analyzed with diffusion maps.

Results: Patients exhibited widespread decrease of fractional anisotropy (Figure 1). From the diffusion map analysis we found evidence of demyelination in corpus callosum, corticospinal tract, fornix, internal capsule, corona radiate, sagittal stratum, cingulum, fronto-occipital fasciculus and uncinate fasciculus. Signs of axonal lesion were found in internal capsule, thalamic radiation, sagittal stratum, cingulum and uncinate fasciculus.

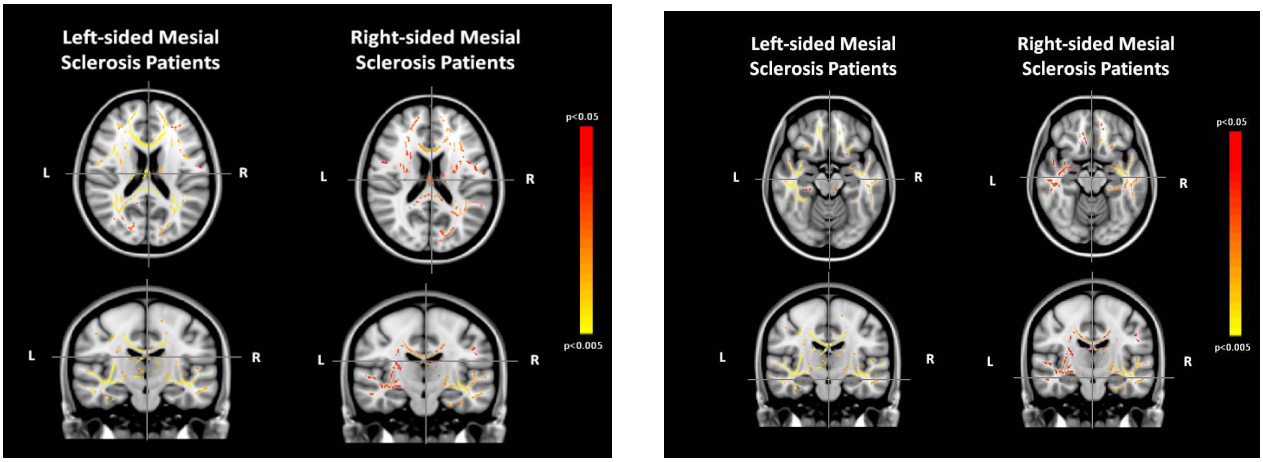

Figure 1: FA group differences superimposed on MNI305 template showing alterations in the TLE patients.

Discussion and conclusions: We found widespread clusters of abnormal DTI measures in prominent white matter tracts linking mesial temporal lobe structures to other brain areas. The **FA** abnormalities are very similar in both hemispheres for left-sided mesial sclerosis patients and more pronounced in the ipsi-lateral hemisphere in right-sided patients. Our results are consistent with the notion that demyelination and axonal damage may contribute to extra-hippocampal damage in TLE patients. We aim to prove the hypothesis that the analysis of perpendicular and longitudinal components of the diffusivity can add important insight about the pathophysiology of demyelination in refractory epilepsy.

References:

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