SPREAD OF DISCHARGES THROUGH TRACKS IDENTIFIED WITH DIFFUSION TRACTOGRAPHY MAY EXPLAIN TRANSIENT SPLENIAL "LESIONS" IN EPILEPSY

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Background: A characteristic transient lesion of the splenium of the corpus callosum is occasionally seen in patients with epilepsy. On MRI it appears as a symmetric ovoid focus which is hyperintense on T2-weighted images and hypointense on T1-weighted and ADC sequences.[1] This appearance is consistent with cytotoxic edema, however the lesion completely resolves within weeks to months. Various causes have been proposed, such as generalized seizures, focal seizures, abrupt withdrawal of anti-epileptic medications and medication toxicity, but the true aetiology of the lesion remains uncertain.[2]

Aim: To investigate the possible pathways of seizure propagation in such a patient, using diffusion tractography, and to describe how these pathways relate to the lesion of the splenium

Method: We present a 43 year old man with medically refractory temporal lobe epilepsy who developed this characteristic lesion of the splenium, on imaging performed 4 days after a period of video-EEG monitoring. Three seizures had occurred in the morning before the scan, and he was mildly confused but cooperative. Imaging was repeated 6 months later and showed that the lesion had resolved. Intracranial electrode monitoring was used to define the ictal onset zone. Seizures began at the left anterior temporal neocortex, then rapidly spread to broadly involve the right temporal neocortex and bilateral mesial temporal structures. Definitive treatment was the resection of the left anterior temporal lobe, resulting in a marked decrease in his usual seizures.

Diffusion imaging data were analyzed using constrained spherical deconvolution[3] and probabilistic fibre tracking using MRtrix.[4] A seed region was defined at the left temporal ictal onset zone, and tracked to a large region in the right temporal lobe, which encompassed the electrodes involved in the early contralateral seizure spread. The same procedure was followed for 5 non-epileptic controls, using homologous regions of interest derived from a common template space.

Results: In our patient, tracks from the ipsilateral temporal neocortex to the contralateral temporal lobe were identified crossing via the anterior commissure, hippocampal commissure and corpus callosum. The callosal tracks all passed through the lesion in the splenium. No tracks were seen in other parts of the corpus callosum. Tracks generated from imaging data acquired when the lesion was present, were the same as tracks generated from data obtained once the lesion had resolved. In the control group, the same tracks were identified, including the tracks which traversed the splenium.

Conclusions: The identified track via the splenium corresponds to a plausible normal anatomical tract, that has been previously identified in primate radio-tracer studies.[5] In a patient with neocortical temporal lobe seizures this may be a possible route for ictal spread to the contralateral temporal lobe. We propose that excess epileptic activity in this pathway may contribute to the development of this lesion in susceptible patients.

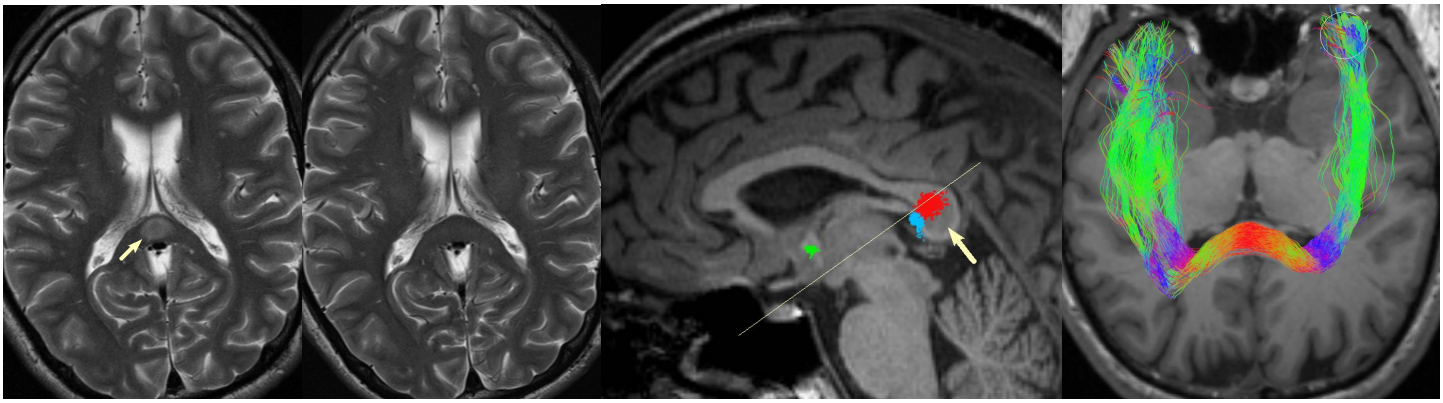

Figure 1: MRI of the described patient. a) Splenial lesion on T2-weighted imaging. b) Resolution of the lesion 6 months later. c) Midline sagittal T1-weighted image, with hypointense spenial lesion seen, showing tracks traversing the corpus callosum(red), hippocampal commissure(blue) and anterior commissure(green). Line indicates plane of figure 1d. d) Tracks from left temporal seed to right temporal target regions, which traverse the splenium, displayed on a co-registered T1-weighted image. Color indicates fibre direction.

References:

1. Hakyemez, B. et al., 2005. Transient splenial lesion of the corpus callosum associated with antiepileptic drug: Conventional and diffusion-weighted magnetic resonance images. *Acta Radiologica,* 46(7),734-736
2. Groppel, G. et al., 2009. Focal lesions in the splenium of the corpus callosum in patients with epilepsy. *Epilepsia,* 49(9), 1594-1601
3. MRtrix, Brain Research Institute, Melbourne, Australia, http://www.brain.org.au/software/
4. Tournier, JD. et al., 2007. Robust determination of the fibre orientation distribution in diffusion MRI: non-negativity constrained super-resolved spherical deconvolution. *Neuroimage,* 35(4), 1459-1472
5. Demeter, S., et al., 1990. Fields of origin and pathways of the interhemispheric commissures in the temporal lobe of macaques. *The Journal of Comparative Neurology*, 302(1), 29-53.