MRI study of the thalamus in Optic Neuritis and other presentations of Clinically Isolated Syndrome.

Su-Yin Lim, Ali Al-Radaideh, Christopher R. Tench, Penny Gowland, Cris S. Constantinescu

Division of Clinical Neurology, University of Nottingham, UK (SYL, CRT, CSC)
Sir Peter Mansfield Magnetic Resonance Centre, University of Nottingham, UK (AAR, PG)

Objectives: To detect and quantify thalamic pathology following a single attack of optic neuritis (ON) and other forms of clinically isolated syndrome (CIS) using magnetic resonance (MRI) volumetry and T1 relaxation times.

Background: The optic tracts synapse in the lateral geniculate nuclei (LGN) of the bilateral thalamus, the main thalamic relay nucleus in the visual pathway. Pathological changes have been observed in the thalamus of CIS and multiple sclerosis patients (Niepel et al 2006, Henry JNNP 2008), a proportion of whom may have had ON as an initial presentation or recurring attacks. The role of ON in contributing to thalamic damage however, remains uncertain. Volumetry and T1 relaxation measurements are useful MRI metrics that reflect demyelination and neurodegeneration.

Methods: 21 CIS patients, including 7 post-ON cases (all treated with intravenous steroids), and 11 matched controls were scanned using a 7T Philips MRI, obtaining a 3D MPRAGE sequence (o.6mm3 resolution) and T1 maps with 7 inversion times (range 150ms – 2500ms). Locally developed software, Neuroi (CRT) was used to semi-automatically segment the bilateral thalamus on a high-contrast T1 sequence, and to acquire the thalamic volume (later normalised to brain volume) and T1 relaxation times. Bedside visual function assessment was performed on the patients.

Results: There were no significant differences in the measured thalamic volume and T1 relaxation times of the ON group compared with the non-ON group. Overall, there were no significant differences in thalamic volume and T1 relaxation times between the patients (as a whole) and controls. Conclusion: No significant pathological changes affecting the thalamus were observed in our small cohort of patients who had treated ON.

[1]Niepel G. J Neurol 2006; 253(7): 896-902
[2]Henry RG. JNNP 2008; 79(1):1236-44
[3]http://www.nottingham.ac.uk/scs/divisions/clinicalneurology/index.aspx
[4]Korsholm K. Brain 2007; 130(5):1244-1253