OCT measures are associated with disease burden and inflammatory biomarkers in newly diagnosed MS and CIS

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Background:

Asymptomatic involvement of the optic nerve and the retina occurs in multiple sclerosis. Visual evoked potentials (VEP) and optical coherence tomography (OCT) could be used to detect this phenomenon.

In particular, the retinal ganglion cell-inner plexiform layer (GCIPL) present a progressive thinning, correlated with global neurodegenerative processes. While, the adjacent inner nuclear layer (INL) thickens in active MS.

Methods:

150 patients underwent a thorough diagnostic assessment after a first neurological episode and were diagnosed CIS (48%) or RRMS (52%). Patients performed within 15 days an OCT scan, lumbar puncture, multimodal evoked potentials (EP), brain MRI scan and clinical assessment of visual acuity and EDSS score.

Results:

Subclinical optic nerve involvement was detected using VEP or OCT in the 22% and 25% of patients respectively. Asymptomatic VEP involvement was associated with higher brain lesion load (p=0.01), conduction deficits along motor and somatosensory EP (p=0.002), frequency of oligoclonal bands (p=0.005) and longer time from the first neurological episode (p=0.02).

GCIPL correlated with low contrast visual acuity (p=0.025), EDSS (p=0.047), disease duration (p=0.049), number of T2 brain lesions (p=0.007) multimodal evoked potentials (p=0.017) and presence of oligoclonal bands (p=0.009) independently of clinical or subclinical (OCT or VEP) evidences of optic neuritis.

INL thickened 1 to 3 months after a relapse, this effect was reduced by concomitant steroid treatment. No linear correlation was found with markers of acute inflammation: gadolinium enhancing lesions, Link Index, serum neurofilaments. CSF levels of IL-7, MCP-1 and MIP-1b predict INL increase (R^2 0.4; p<0.001).

Conclusions:

Asymptomatic optic nerve involvement, revealed by OCT and VEP, is frequent in early MS and is associated with disease-burden. Moreover, GCIPL thinning, reveals an association with global neurodegenerative processes even after correcting for clinical and subclinical ON.

INL thickening occurs in a subacute phase and is associated with myeloid pro-inflammatory CSF cytokines. INL swelling might reveal a reactive response of Muller cells to neuronal injury.