Abstracts

Articles appearing in the January 2018 issue

Progressive inner nuclear layer dysfunction in non-optic neuritis eyes in multiple sclerosis

Objective To investigate primary retinal functional changes in eyes without optic neuritis (ON) of patients with multiple sclerosis (MS) by using full-field electroretinography (ERG).

Methods Seventy-seven patients with relapsing-remitting MS with no history of clinical ON in at least one eye and 30 healthy controls were recruited in the cohort study. Full-field ERGs were recorded and retinal optical coherence tomography scans were performed to assess the peripapillary retinal nerve fiber layer (RNFL) and retinal ganglion cell–inner plexiform layer (GCL-IPL) thicknesses. Annual MRI scans were also carried out to evaluate the disease activity in the brain. Patients were followed up for 3 years.

Results At baseline, a delayed b-wave peak time was observed in the cone response ($p < 0.001$), which was associated with the thicknesses of RNFL and GCL-IPL. The peak time of the delayed b-wave also correlated with the Expanded Disability Status Scale, T2 lesion volume, and disease duration. During the 3-year follow-up, progressive ERG amplitude reduction was observed (both a- and b-waves, $p < 0.05$). There was a correlation between the b-wave amplitude reduction and longitudinal RNFL loss ($p = 0.001$). However, no correlation was found between longitudinal ERG changes and disease activity in the brain.

Conclusions This study demonstrated progressive inner nuclear layer dysfunction in MS. The borderline a-wave changes suggested some outer retinal dysfunction as well. The correlation between full-field ERG changes and retinal ganglion cell loss suggested that there might be subclinical retinal pathology in MS affecting both outer and inner retinal layers.

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Covertly active and progressing neurochemical abnormalities in suppressed HIV infection

Objective To assess whether HIV-related brain injury is progressive in persons with suppressed HIV infection.

Methods Seventy-three HIV+ virally suppressed men and 35 HIV– men, screened for psychiatric and alcohol/drug use disorders, underwent neuropsychological evaluation and proton magnetic resonance spectroscopy (^1H -MRS) at baseline and after 23 ± 5 months. ^1H -MRS included brain regions known to be vulnerable to HIV and aging: frontal white matter (FWM), posterior cingulate cortex (PCC), and caudate area (CA). Major brain metabolites creatine (Cr: marker of cellular energy), NAA (marker of neuronal integrity), choline (marker of cellular membrane turnover), glutamate/glutamine (excitatory/inhibitory neurotransmitter), and myo-Inositol (mI: marker of neuroinflammation) were calculated with reference to water signal. Neurocognitive decline was corrected for practice effect and baseline HIV-associated neurocognitive disorder (HAND) status.

Results Across the study period, 44% had intact cognition, 42% stable HAND (including the single case that improved), 10% progressing HAND, and 4% incident HAND. When analyzing the neurochemical data per neurocognitive trajectories, we found decreasing PCC Cr in all subgroups compared to controls ($p < 0.002$). In addition, relative to the HIV– group, stable HAND showed decreasing FWM Cr, incident HAND showed steep FWM Cr reduction, while progressing HAND had a sharply decreasing PCC NAA and reduced but stable CA NAA. When analyzing the neurochemical data at the group level (HIV+ vs HIV– groups), we found stable abnormal metabolite concentrations over the study period: decreased FWM and PCC Cr (both $p < 0.001$), decreased PCC NAA and CA NAA (both $p < 0.05$), and PCC mI increase ($p < 0.05$). HIV duration and historical HAND had modest effects on metabolites' changes.

Conclusions Our study reveals covertly active or progressing HIV-related brain injury in the majority of this virally suppressed cohort, reflecting ongoing neuropathogenic processes that are only partially worsened by historical HAND and HIV duration. Longer-term studies will be important for determining the prognostic significance of these slowly evolving neurochemical abnormalities.

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