Visual and motor function in experimental autoimmune encephalomyelitis

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Experimental autoimmune encephalomyelitis (EAE) is associated with abnormalities in motor (MEP) and visual (VEP) evoked potentials and neuroretinal thinning at optical coherence tomography (OCT), consistently with clinical symptoms observed in multiple sclerosis. Understanding the time course of these abnormalities is pivotal for the translational testing of novel therapeutic strategies.
We performed VEP, MEP and OCT in 30 C57BL/6 mice immunized with MOG 35-55 (*vs* 10 controls), at 7, 14 and 31 days post-immunization-dpi (10 mice sacrificed at each timepoint-dpi). We report electrophysiological and OCT data.
Compared with controls, EAE mice had delayed VEPs at all consecutive time points (p=.00009, p=.014, p<.001, respectively; Student's t-test) and reduced neuroretinal thickness at 7 (p=.003) and 31 dpi (p=.013). EAE MEPs did not significantly differ from controls at 7 and 14 dpi, while at 31 dpi, MEPs were delayed in 5 hindlimbs from 4 mice (1 bilateral, 3 unilateral) and absent in 10 from 6 mice (4 bilateral, 2 unilateral). Optic nerve demyelination was significant in EAE mice vs controls at 7 (p<.0001) and 31 dpi (p<.0001), which correlated with increased VEP latency. Spinal cord demyelination was significant at 31 dpi (p=0,0002 vs controls), and smaller amplitudes correlated with increased demyelination, at 14 and 31 dpi. Increased MEP latency significantly correlated with stronger demyelination (p=.01) at 31 dpi. Abnormal VEPs were more frequent *vs* MEPs at 7 dpi (56.7% eyes *vs* 6.7% hindlimbs, p<.001), at 14 dpi (35% *vs* 10%; p =.031), and at clinical onset (13, 15 ±0.95 dpi, 42,5 % *vs* 10.5%, p=.004); with no significant group difference at 31 dpi (55.6% *vs* 83.3%; p=.227).
VEPs abnormalities appear before electrophysiological or clinical motor involvement, pointing to the relevance of electrophysiological measures to detect early, subclinical demyelination as a potential target of novel therapeutic approaches targeting inflammation, demyelination, and neuroaxonal loss.

Funding: Institute of Experimental Neurology (INSPE).

Disclosure of interests: None