Dynamic Patterns of Gray and White Matter Atrophy in Patients with Clinically Isolated Syndrome Suggestive of Multiple Sclerosis After 1 Year from the First Clinical Episode

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Target Audience. Neurologists, Radiologists and Neuroradiologists.

Purpose. To investigate the patterns of regional gray matter (GM) and white matter (WM) atrophy and their changes over one year in patients with clinically isolated syndrome (CIS) suggestive of multiple sclerosis (MS).

Methods. Clinical evaluation and brain dual-echo and post-gadolinium T1-weighted sequences were obtained from 41 CIS patients within 2 months from the clinical onset, and after 3 (M3) and 12 months (M12). Fourteen matched healthy controls were also studied. T2-hyperintense, T1-hypointense and gadolinium-enhancing lesions were measured. Longitudinal changes of GM and WM volumes were assessed using Tensor-Based Morphometry.

Results. At M12, according to 2010 diagnostic criteria, 36/41 (88%) patients had definite MS. At baseline, T2 lesions were mainly located in the periventricular WM. Normalized brain, GM and WM volumes did not change over time. At baseline, compared to controls, patients had increased GM volume in several regions of the temporal lobes and the right (R) paracentral lobule, which further increased at M3 and M12. The volume of a few GM cerebellar regions increased at M3 and normalized at M12 (*figure 1*).

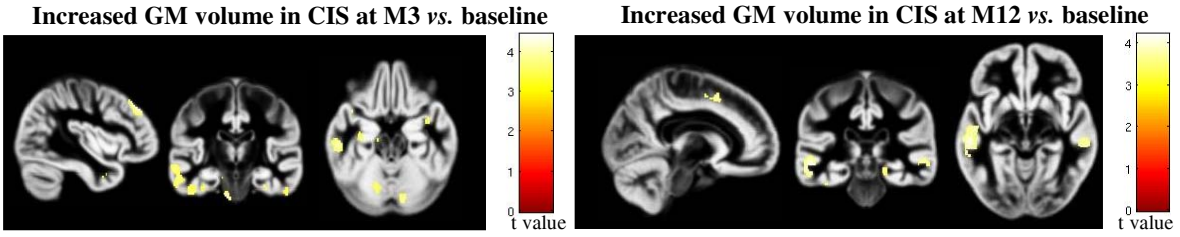

Figure 1. Areas with increased GM volume at M3 and at M12 *vs.* baseline.

WM volume of several regions of the temporal lobes, paracentral lobules, pons and frontal lobes decreased at M12. Compared to M3, at M12 patients had a decreased volume of the R thalamus, R caudate nucleus, bilateral cerebellum and temporal cortex, and decreased volume of the corpus callosum, posterior cingulum, R internal capsule and bilateral corona radiate (*figure 2*).

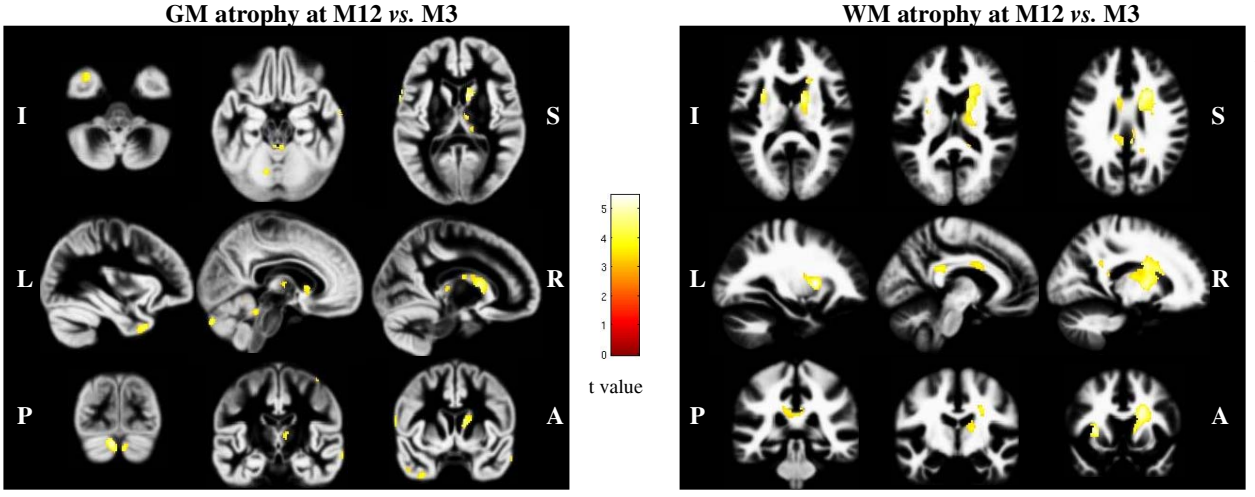

Figure 2. Patterns of GM and WM atrophy in CIS patients at M12

Discussion. After an acute inflammatory event, dynamic modifications of the regional distribution of GM and WM volume occur in CIS who will evolve to MS. While WM loss seems to follow a linear evolution from disease onset, a transient increase of GM volume in a few regions is detectable early in the disease course, which might reflect structural plasticity to damage.

Conclusion. The assessment of regional brain atrophy in CIS patients may improve our understanding of the pathophysiological mechanisms responsible for the accumulation of irreversible clinical disability and may help to predict the clinical evolution of CIS patients.

References.

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