

Lesion patterns topology is associated with regional cortical atrophy and predicts disease-related disability [Abstract]

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Angaben zur Veröffentlichung / Publication details:

Muthuraman, Muthuraman, Julia Kroth, D. Ciolac, N. Koirala, Gabriel Gonzalez-Escamilla, Vinzenz Fleischer, Angela Radetz, Sven G. Meuth, Frauke Zipp, and Sergiu Groppa. 2018. "Lesion patterns topology is associated with regional cortical atrophy and predicts disease-related disability [Abstract]." *Multiple Sclerosis Journal* 24 (S2): 205.
<https://doi.org/10.1177/1352458518798582>.



to discover distinct patterns from two inputs, first: WM lesion distribution derived from the T2-images and delineation with Lesion Segmentation Tool (LST under VBM8 and SPM12) and second: the rate of cortical atrophy (as derived from Freesurfer) from each voxel of the brain of the same patient. We then calculated the eigenvalue association of WM lesion patterns and the spatial extent of cortical atrophy. The predictive value of patient-specific lesion burden for each pattern has been used to predict emerging clinical disability (EDSS worsening) at the second time point by receiver operating characteristic analysis.

Results: We identified three significant associated patterns (bilateral, lateralized and cerebellar) of white matter lesions and corresponding cortical atrophy. The cerebellar lesion pattern was associated with the largest extent of cortical atrophy, mainly in the temporal and frontal regions. Each of the patterns discriminated progression of disability over one year as quantified by EDSS worsening. However, the lesion burden as determined by the cerebellar pattern was associated with the worst clinical outcome.

Conclusions: Our findings indicate that a distinct spatial distribution of focal WM lesions is associated with cortical atrophy, and is able to precisely predict the individual functional outcome over time together with disease progression. Thus, early identification of clinical phenotypes associated with a specific lesion distribution allows patient stratification for disease progression and clinical impairment.

Disclosure

Nothing to Disclose

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Lesion patterns topology is associated with regional cortical atrophy and predicts disease-related disability

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Background: Grey (GM) and whiter matter (WM) microstructural alterations are important hallmarks of neuroinflammation in multiple sclerosis (MS). Both processes contribute differently to disease progression and disability. It is however, not clear how the distribution of WM lesions influences GM atrophy and long-term clinical outcome. In this study, we investigated the interrelation of lesion topology and GM atrophy and its impact on emerging functional disability.

Methods: We included 119 patients (mean age \pm standard deviation: 34.6 ± 9.8 years, 38 males) with early (disease duration: 37.2 ± 1.5 months) relapsing-remitting MS (RRMS) and performed 3T magnetic resonance imaging (MRI) at baseline and one year later. We applied independent component analyses (ICA) on MRI data